Cardiopulmonary Physiology and Responses of Ultramarathon Athletes to Prolonged Exercise

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Abstract/Résumé

The purpose of this study was to determine the changes of pulmonary function and autonomic cardiovascular control after an ultramarathon and their relation to performance. Eight entrants to the Canadian National Championship 100-km running race participated in the study. Pulmonary function and 30-s maximum voluntary ventilation (MVV₃₀s) tests were conducted one day before the race and within 5 minutes of race completion. Heart rate and blood pressure data were collected 30 min before and 5 min after the race as well as during a 10-min stand test one day prior to the race. During the race, beat-by-beat R-R interval data were collected over the first and last 20 km. The results showed that MVV₃₀s and MVV₃₀s tidal volumes were reduced postrace (p < 0.001). Prerace supine total harmonic variation (p < 0.01) and prerace MVV values (10 s to 30 s) (p < 0.05) were correlated with race finish time. The changes in pulmonary function and MVV₃₀s values from pre- and postrace were not significantly correlated to race performance. We conclude that maximal sustainable ventilatory power and dynamic autonomic cardiovascular control are important factors in determining overall performance in an ultramarathon.
Le but de cette étude est d’analyser les variations des fonctions pulmonaires et du contrôle cardiovasculaire autonome à la suite d’un ultramarathon et d’en vérifier la corrélation avec la performance. Huit athlètes inscrits au Championnat Canadien du 100 km participent à cette étude. Des tests de fonction pulmonaire et de ventilation volontaire maximale en 30 s (MVV30s) sont administrés une journée avant la course et en moins de 5 min après la fin de la course. La fréquence cardiaque et la pression sanguine sont mesurées 30 min avant la course et 5 min après la course de même qu’au cours d’un test en position debout (stand test) d’une durée de 10 min une journée avant la course. Durant les 20 premiers et les 20 derniers kilomètres, la durée de l’intervalle R-R est enregistrée pour chaque battement. Les résultats démontrent qu’après la course, il y a une réduction de la MVV30s et du volume courant (p < 0,001). La variation harmonique totale en position couchée avant la course et les valeurs de MVV (10 s à 30 s) avant la course sont corrélées au temps de performance: p < 0,01 et p < 0,05, respectivement. Les variations des fonctions pulmonaires et de la MVV30s attribuables à la course ne sont pas significativement corrélées au temps de performance. La puissance ventilatoire maximale soutenable et le contrôle cardiovasculaire autonome dynamique constituent d’importants facteurs de la prédiction globale de la performance dans un ultramarathon.

Introduction

Endurance exercise may be defined as activity of at least 20 minutes duration in which heart rate is elevated to 60–80% of maximum. Ultra-endurance exercise is normally classified as activity of at least 3.5 hours in duration at a similar intensity of 60–80% of maximum heart rate. In the past decade ultra-endurance racing has become more popular and now encompasses single- and multi-day adventure races. Although the physiology of endurance exercise has been studied extensively, it is usually limited to less than 2 hours of observation in a laboratory. Of the research performed with ultramarathon athletes, the majority have focused on nutritional and hematological factors (Fallon et al., 1998; Glace et al., 2002; Neumayr et al., 2002). Past research into the cardiorespiratory aspects of exercise on performance has focused on VO₂max and running economy (Joyner, 1991; Maldonado, 2002) and energy utilization (Davies and Thompson, 1986). However, these studies indicate that other factors must contribute to the limitation of endurance performance.

Alterations in pulmonary function after endurance exercise were first noted in the 1923 Boston Marathon (Gordon et al., 1924) when vital capacity was found to decline. Subsequent studies have revealed significant reductions in vital capacity after running races ranging from 8 km to 80 km (Hill et al., 1991; Mahler and Loke, 1981; Maron et al., 1979), with greater declines following the longer events. However, the initial FVC or its race-induced deficit has not been found to be related to performance (Folinsbee et al., 1983; Maron et al., 1979). Maximal voluntary ventilation over 12 seconds (MVV12s) has not shown any reduction after a triathlon (Hill et al., 1991); however, MVV12s was found to account for almost 40% of the variance in running speed over a 24-hr ultramarathon (Warren et al., 1989).

The decrease in ventilatory muscle endurance may constrain running speed in prolonged running events. Utilization of a longer duration ventilatory “endurance” test was shown to have declined even 3 days after an ultramarathon (Ker and
Schultz, 1996). Respiratory muscle work in short-duration high intensity exercise (>90 % VO$_2$max) can also affect overall exercise performance (Babcock et al., 2002; Harms et al., 2000). As ultramarathon runners progressively fatigue, often reflected by a decline in running velocity (Fallon et al., 1998; Warren et al., 1989), their relative effort to continue becomes greater (Davies and Thompson, 1986; Utter et al., 2003).

Prolonged endurance training associated with ultramarathon affects autonomic cardiovascular control: endurance training increases parasympathetic activity and decreases sympathetic activity directed to the human heart at rest (Amano et al., 2001; Dixon et al., 1992; Goldsmith et al., 2000; Gregoire et al., 1996; Shi et al., 1995; Shin et al., 1995). These training-induced autonomic changes, coupled with a possible reduction in intrinsic heart rate, will decrease resting heart rate and increase resting heart rate variability (HRV) in an ultra-endurance athlete (Goldsmith et al., 2000; Shi et al., 1995; Shin et al., 1995). Heart rate variability is the most common and accepted method used to provide noninvasive indicators of the autonomic nervous system (Task Force, 1996). Two major spectral components have been identified: a low frequency (LF) and a high frequency (HF) component that can be used to determine markers of sympathetic and parasympathetic modulation of heart rate (Blaber et al., 1996; Task Force, 1996).

Following endurance training there is improved vascularization in trained muscles. With an increase in capillary pressure, the enlarged vascular bed will accommodate more fluid. This enhanced capacity for vasodilation, induced by training, may predispose an individual to an attenuated vasoconstrictor response (Fadel et al., 2001; Zhang et al., 1999). Very fit subjects also have an attenuated tachycardiac and vasoconstrictor response to orthostatic stress, suggesting that endurance training is associated with attenuated high-pressure baroreflex regulation of blood pressure during central hypovolemia (Raven et al., 1984). Orthostatic tests, such as stand tests, are commonly used to investigate autonomic control of cardiovascular function (Rowell, 1993). The shift in blood volume to the lower body decreases venous return to the heart and thus reduces cardiac output, producing an initial decrease in arterial blood pressure (ABP). In a healthy human, the decrease in blood pressure is quickly restored by the interaction of the parasympathetic and sympathetic nervous systems to increase heart rate and peripheral vascular resistance pressure (Rowell, 1993).

The Canadian National Championship 100-km running race provided an opportunity to study ultramarathon athletes in a competitive setting. We used this event to examine pulmonary function (FVC, extended MVV) before and after the race, and autonomic cardiovascular control before (Stand test, ABP and HRV), during (HRV), and after the race (ABP and HRV). In addition, we explored the relationship of the cardiorespiratory variables to 100-km running performance.

Methods

SUBJECTS

Fifteen athletes volunteered for this study; however, due to time and scheduling constraints, data were collected from only 7 males and 1 female out of a field of 19 registered entrants in the Canadian National Championship 100-km running race.
The Institutional Ethics Review Board of Simon Fraser University approved the study and written informed consent was obtained from all subjects after they read a full description of the testing procedures. The finishing positions of the 8 competitors ranged from 1st to 13th. The physical characteristics of the 8 ultramarathon athletes were as follows: age $35.8 \pm 7$ years; weight $63.8 \pm 5$ kg; height $1.77 \pm 0.06$ m. Each athlete was in regular training ($160 \pm 25$ km·wk$^{-1}$) 4 weeks prior to the race, at which time they entered their prerace taper. Their individual physical characteristics are presented in Table 1.

Table 1 Physical Characteristics, Training, and Race History of Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Training (yrs)</th>
<th>Marathon (number)</th>
<th>100 km (number)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>167</td>
<td>57.0</td>
<td>15</td>
<td>24</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>179</td>
<td>71.6</td>
<td>25</td>
<td>100</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
<td>175</td>
<td>70.6</td>
<td>22</td>
<td>37</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>61</td>
<td>178</td>
<td>75.4</td>
<td>30</td>
<td>43</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>56</td>
<td>181</td>
<td>63.8</td>
<td>28</td>
<td>77</td>
<td>7</td>
</tr>
<tr>
<td>6</td>
<td>33</td>
<td>181</td>
<td>69.4</td>
<td>6</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>43</td>
<td>179</td>
<td>86.0</td>
<td>17</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>44</td>
<td>172</td>
<td>69.0</td>
<td>20</td>
<td>11</td>
<td>8</td>
</tr>
</tbody>
</table>

None of the athletes had a history of cardiovascular or pulmonary disease, and preliminary respiratory measures conformed to American Thoracic Society tables of normal respiratory function (American Thoracic Society, 1987). The group of ultramarathon athletes studied was active in competition and may be regarded as representative of those who regularly compete in long-distance events in Canada. They were all capable of running the traditional marathon distance (42.2 km) in less than 3 hrs. All were in their final taper before the race and had not performed strenuous exercise 48 hours prior to the race. All subjects abstained from alcoholic or caffeinated beverages the morning before the physiological tests.

RACE COURSE

The course was certified and accurately measured with a Jones counter and bicycle and consisted of 10 laps of 10 km each. The race course was one of the flattest in Canada, with elevation changes of less than one metre. This provided the best possible conditions for continuous heart rate variability analysis since changes in heart rate would be associated with changes related to the competitor, not course topography. Lap and total times were collected for each athlete using the official race clock.
DATA COLLECTION AND ANALYSIS

Pulmonary measures were collected using the K4b² portable breath-by-breath gas exchange system (COSMED Ltd., Rome, Italy). This device meets the standards of the American Thoracic Society for pulmonary function testing. All measurements were performed with the subject seated. Prior to the competition, athletes practiced the pulmonary function test until maximal reproducible ventilatory efforts were within 5%. A standard forced expiratory pulmonary function test was performed. The best of 3 efforts was recorded. From these data the forced expiratory vital capacity (FEVC) and forced expiratory volume in 1 second (FEV₁.₀) were determined. A modified maximal voluntary ventilation protocol of 30 s (MVV₃₀s) was also performed whereby the subjects ventilated at maximal effort for 30 s. Raw data from these tests were stored on a computer at a sample rate of 25 Hz for postrace analysis. Pulmonary function and MVV₃₀s measures were performed one day prior to the competition between 10:00 a.m. and 4:00 p.m. and within 5 minutes of completing the competition. The MVV₃₀s data were averaged over 5-s intervals and recorded as the MVV at 2.5 s, 7.5 s, 12.5 s, 17.5 s, 22.5 s, and 27.5 s, respectively.

The MVV₃₀s test is quite similar to the Wingate 30-s test for leg cycle ergometry. The initial increase to maximal power occurs in 5–10 s and is quite reproducible. The decline in power thereafter is motivation-dependent. In all MVV₃₀s tests the subjects were verbally encouraged to maintain their maximal effort. The MVV₃₀s data (ventilation, tidal volume [Vₖ], and respiratory frequency) were divided into 5-s intervals with the mean reported at the middle time of each interval for statistical analysis.

Cardiovascular reactivity was determined one day prior to the race using a stand test. Subjects were placed in the supine position for electrocardiograph (ECG) and blood pressure instrumentation. A standard 3-lead ECG (LifePak-8 Cardiac Monitor, Physio-Control, Redmond, WA) was used. For safety reasons, during the stand test a continuous noninvasive finger photoplethysmograph blood pressure monitor (Finapres™, Ohmeda, Inglewood, CO) was used over that of periodic manual measurement. The monitored finger was fixed at heart level by supporting the arm with a shoulder sling to obtain heart-level blood pressure values. The Finapres display allowed the experimenter to visually assess the onset of syncope (presyncope), at a glance, through changes in heart rate, pulse pressure, and systolic and diastolic blood pressures. If the subjects became presyncope during the stand, they were returned immediately to the supine position. As mandated by the medical ethics review board of Simon Fraser University, subjects were deemed to be presyncope if they indicated symptoms such as dizziness or nausea, or if they were observed to have any of the following: a decrease in HR of more than 15 bpm; a decrease in systolic BP of more than 25 mmHg·min⁻¹; or a decrease in diastolic BP of more than 15 mmHg·min⁻¹.

Analog signals from the ECG and blood pressure monitors were recorded simultaneously at 1,000 Hz per channel using a computer strip chart recorder. Beat-by-beat analysis of these data was performed offline. Each peak of the R wave of the ECG tracings was marked by an automated computer system and was manually reviewed for anomalies and movement artefact that may have affected this process; missed beats were marked manually. These data were then used to gener-
ate an R-R interval (RRI) time series. Mean arterial blood pressure (MAP) was calculated as the arithmetic average of the data points in each beat. Systolic arterial blood pressure (SBP) and diastolic arterial blood pressure (DBP) were defined as the maximum and minimum pressures, respectively, in each beat. Any unusable blood pressure data were removed. The average of SBP, MAP, and DBP was then calculated for each 5-min segment used in HRV analysis.

Each subject was supine for 20 minutes before being assisted to the stand position by three investigators. To minimise blood pressure changes due to the effort of standing, subjects were lifted from behind both shoulders while their feet were swept off the bed. The subjects remained unassisted in the standing position for up to 8 minutes to allow for sufficient data to perform heart rate variability analysis.

On race day, heart rate and blood pressure were recorded with the subject in the seated position immediately prior to the start of the 100-km race (within 20 minutes) and within 5 minutes of finishing the race using a digital automated oscillometric blood pressure monitor (A & D Medical, Milpitas, CA). Data from this device were also collected, by the same operator, at the beginning and end of the stand tests for comparative purposes. These values were found not to be significantly different from the Finapres.

Beat-to-beat measures of R-R interval were collected in the first and last 20 km of the race via a Polar™ S810 watch (Polar Inc., Kempele, Finland). A recent literature review of heart rate monitoring (Achten and Jeukendrup, 2003) concluded that the Polar system was both reliable and valid for R-R interval collection during exercise as well as for heart rate variability analysis. The watch was removed from the subject at the end of 20 km, when the subject entered the replenishment station of the 10-km race loop, and the stored data were downloaded onto a laptop computer. The watch was then reset and returned to the subject for the final 20 km of data collection as the subject passed through the replenishment station at 80 km. At the finish of the race the watch was again collected and the data were downloaded. Since these watches do not store ECG data, only the R-R interval determined via R-wave detection, the R-R interval data were manually reviewed prior to spectral analysis.

Five of the cleanest 5-min steady-state segments from 10 to 20 km and 80 to 90 km were used. Interactive software was used to identify segments with missed beats, possibly due to movement of the Polar ECG band during the run. These data were removed and the time series was adjusted by interpolating new values from the two valid points surrounding the excluded segment. If more than 10% of any 5-min segment required for spectral analysis was interpolated, the results were deemed invalid. Outputs from the HRV analysis of each of these five segments were then averaged to produce a single value for each distance range.

HEART RATE VARIABILITY (HRV) ANALYSIS

Autonomic activity at the heart was determined by heart rate variability analysis using 5-min data segments. Measurements of HRV during short time periods (5 minutes) are stable and may be regarded as characteristic of an individual (Kleiger et al., 1991; Sinnreich et al., 1998).
Observations of HRV indicate that underlying the harmonic components analyzed by spectral analysis is a pattern of fractal variability (Goldberger, 1992; Peng et al., 1993; Saul et al., 1988). It has been speculated that this pattern is important for the maintenance of cardiovascular homeostasis (Butler et al., 1993; 1994; Peng et al., 1993). This fractal component has linear scaling across a wide range of frequencies when the data are plotted as log spectral power vs. log frequency. This is what is commonly called the $1/f^\beta$ relationship (Pomeranz et al., 1985; Yamamoto and Hughson, 1994), where $\beta$ is the positive value of the slope of the linear regression applied to these data.

When the value of $\beta$ is close to 1, there is a high level of complexity because the data frequently change direction toward or away from the mean. In contrast, a value of $\beta$ close to 2 represents a low level of complexity with less complex changes of the measured data (Yamamoto and Hughson, 1994). In a less complex system, feedback control is probably dominated by a reduced number of inputs (Mayer-Kress et al., 1988). Fractal analysis can be used as an indicator of homeostatic stress with a value of $\beta$ that is close to 1 and represents the most favourable position. In clinical studies of patients recovering from myocardial infarction, a trend of $\beta$ toward 2 has been found to be a powerful prognosticator of mortality (Huikuri et al., 2000).

We evaluated the beat-by-beat variability of R-R interval with coarse graining spectral analysis (CGSA) (Yamamoto and Hughson, 1993). This is the only HRV analysis method with the ability to extract the fractal ($P_{FRAC}$) and harmonic ($P_{HARM}$) components from the total HRV spectral power ($P_{TOT}$) (Butler et al., 1993; Yamamoto and Hughson, 1993). The harmonic component can then be further divided into two frequency regions: high frequency (HI: $>0.15$ Hz) and low frequency (LO: 0.0–0.15 Hz) (Yamamoto and Hughson, 1993). The high frequency region is respiratory related and mediated by the parasympathetic nervous system (PNS), whereas the low frequency region is a consequence of several factors and is a combination of both PNS and sympathetic nervous system (SNS) activity. From these the integrated low frequency (0.0–0.15 Hz, $P_{LO}$) and high frequency (0.15–0.50 Hz, $P_{HI}$) power can be determined. Previously we found that the harmonic values should be normalized by taking their square root (Blaber et al., 1996). In effect these are then the amplitudes of the total harmonic ($A_{HARM}$), low ($A_{LO}$), and high ($A_{HI}$) frequency regions (Blaber et al., 1996).

Although the recommended standard for the lower limit of the low frequency range of heart rate variability is 0.04 Hz (Task Force, 1996), this was based on methods that did not isolate harmonic from fractal and was designed to minimize the effect of the fractal component on low frequency power. However, the CGSA algorithm has been demonstrated to be able to efficiently extract the harmonic from the fractal component over the total frequency range (0.0–0.50 Hz) (Yamamoto and Hughson, 1993).

Given the distribution of sympathetic and parasympathetic nervous system activity with frequency at the sinus node, $P_{HI}/P_{TOT}$ is often used as an indicator of PNS activity and $P_{LO}/P_{HI}$ as an indicator of SNS activity in the control of heart rate (Yamamoto and Hughson, 1994; Yamamoto et al., 1991) or as a measure of sympathovagal balance (Pagani and Malliani, 2000). This estimate, $P_{LO}/P_{HI}$, can be problematic when there is not measurable high frequency variation, such as occurs
during heavy exercise. It has been suggested that modulation of SNS activity on the heart may be best estimated when normalized to total power ($P_{LO}/P_{TOT}$) rather than $P_{HI}$ (Pagani and Malliani, 2000). Computation of the powers by CGSA is different from the autoregressive technique used in the above research; however, $P_{LO}/P_{HARM}$ would be comparable. We have used this ratio as our indicator of SNS activity.

Since heart rate variability analysis relies on the respiratory frequency residing above 0.15 Hz, during instrumentation for the stand test each subject was asked to breathe at 0.25 Hz (15 breaths per minute), as cued by a watch metronome, and to continue this respiratory frequency during the entire test. Each subject was visually monitored and coached throughout the stand test procedure to maintain this breathing rate. Analysis of HRV revealed a frequency peak above 0.15 Hz in all subjects during the stand test. Although many subjects did not have a high frequency peak during the race, respiratory frequency was assumed to be over 0.15 Hz.

STATISTICAL ANALYSES

The JMP-IN statistical package (SAS Institute Inc.) was used for all statistical analysis. Repeated-measures analysis of variance was used to compare variables over the test and race conditions (supine, stand, 10–20 km, and 80–90 km). All data are quoted as a mean $\pm$ SEM (standard error of the mean). Significance was accepted at $p < 0.05$. Linear regression analysis was performed between all variables and 100-km finish times.

Results

The race had ideal overcast weather conditions with the temperature varying between 9 and 11 °C throughout the day. Of the 19 runners who registered for the race, 19 started and 15 finished. All of the test subjects completed the race with finish times that ranged from just over 7 hours to just under 11 hours (median: 9:20, h:min). Race pace decreased from $5.0 \pm 0.5$ min·km$^{-1}$ (mean $\pm$ SD) during the second 10-km loop to $6.4 \pm 1.0$ min·km$^{-1}$ over the ninth 10-km loop.

PULMONARY

There was a significant difference in MVV$_{30s}$ from pre- to postrace as well as over the 30-s duration of the test ($p < 0.001$) (Figure 1). There was also a significant pre/post interaction with duration ($p = 0.046$). Both pre- and postrace MVV$_{30s}$ were not different in the first 10 s of the test; however, after 10 s, postrace values were less than their prerace values. As well, total ventilated volumes over 5-s intervals decreased more rapidly postrace (Figure 1). The difference of postrace MVV$_{30s}$ measurements from prerace MVV$_{30s}$ measurements was: $-0.4 \pm 5.0$ (2.5 s), $-6.0 \pm 6.2$ (7.5 s), $-12.9 \pm 4.7$ (12.5 s), $-24.1 \pm 9.9$ (17.5 s), $-20.7 \pm 12.7$ (22.5 s), and $-26.4 \pm 9.9$ (27.5 s). Ventilation is the combination of both respiratory frequency and tidal volume. The average respiratory frequency, during the MVV$_{30s}$ test, did not change from pre- to postrace or with duration and had a mean value of $103 \pm 1.2$ breaths·min$^{-1}$. There was a significant difference in the 5-s average tidal volumes (MVV-$V_T$) pre- to postrace ($p < 0.001$) as well as with test duration ($p =$
0.002). The interaction term was not significant. At postrace, the average MVV-V_{T} was less than for prerace at all times, and MVV-V_{T} decreased significantly by 12.5 s both pre- and postrace (Figure 1). The ratio of expiratory volume in 1 second to total forced expiratory volume (FEV_{1.0}/ FEVC) was reduced postrace ($p = 0.046$, Table 2).

**CARDIOVASCULAR**

Heart rate and diastolic and mean arterial blood pressure increased from supine to stand while systolic blood pressure did not change (Table 3). An example of blood pressure and heart rate changes to stand in a single subject is shown in Figure 2. Heart rate measured in the seated position 20 minutes prior to the race was elevated from supine measures, but was not different from stand measures made the previous day. All measures of blood pressure made immediately prior to the race were elevated compared to both supine and stand values from the previous day (Table 3). Compared to prerace values, within 5 min after the race, heart rate was elevated and blood pressure was reduced. Postrace blood pressures were not different from stand measures on the previous day (Table 3).
Table 2  Mean (± SEM) Predicted (Cotes, 1993) Pulmonary Function Values for the 8 Racers

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>Baseline</th>
<th>Postrace</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1.0s (L)</td>
<td>3.94 ± 0.15</td>
<td>4.17 ± 0.16</td>
<td>3.87 ± 0.17</td>
<td>−0.29 ± 0.22</td>
</tr>
<tr>
<td>FEVC (L)</td>
<td>4.86 ± 0.18</td>
<td>5.05 ± 0.18</td>
<td>5.04 ± 0.19</td>
<td>0.00 ± 0.11</td>
</tr>
<tr>
<td>FEV1.0s / FEVC</td>
<td>0.80 ± 0.01</td>
<td>0.83 ± 0.02</td>
<td>0.77 ± 0.03†</td>
<td>−0.06 ± 0.03</td>
</tr>
</tbody>
</table>

Note: Mean (± SEM) measured pulmonary function values 1 day prior to the race (baseline) and within 5 min of completing the race (postrace), and the difference of postrace from baseline (postrace – baseline).
†Different from prerace, p < 0.05.

Table 3  Cardiovascular Variables During 6 Measurements

<table>
<thead>
<tr>
<th>Measure</th>
<th>Supine</th>
<th>Stand</th>
<th>Dif-stand</th>
<th>Prerace</th>
<th>Postrace</th>
<th>Dif-race</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>55 ± 2</td>
<td>68 ± 2*</td>
<td>−12 ± 1</td>
<td>64 ± 3*</td>
<td>0 ± 8*#†</td>
<td>24 ± 7</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>116 ± 3</td>
<td>122 ± 3</td>
<td>6 ± 4</td>
<td>155 ± 7*#</td>
<td>119 ± 4†</td>
<td>−36 ± 5</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>70 ± 2</td>
<td>79 ± 2*</td>
<td>9 ± 3</td>
<td>97 ± 3*#</td>
<td>78 ± 3*†</td>
<td>−19 ± 7</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>86 ± 2</td>
<td>94 ± 2*</td>
<td>8 ± 3</td>
<td>115 ± 4*#</td>
<td>93 ± 3*†</td>
<td>−21 ± 6</td>
</tr>
</tbody>
</table>

Note: Supine rest, the stand test, 20 min prerace, and within 5 min of completing the race (postrace), as well as the difference of stand from supine (Dif-stand: stand – supine) and the difference of postrace from prerace (Dif-race: postrace – prerace).
Significant, p < 0.05, from supine (*), stand (#), or prerace (†).

Coarse graining spectral analysis of R-R interval data revealed a decrease in the harmonic high frequency region and the parasympathetic indicator with stand, while the sympathetic indicator and the spectral exponent (β) were increased with stand (Table 4). During the race, the parasympathetic indicator and the harmonic high frequency region were less than both the supine and stand values from the previous day. Both the sympathetic nervous system indicator and the spectral exponent increased over the 100-km race (Table 4) and were not different from stand measurements made 1 day prior to the race.

CORRELATIONS WITH RACE FINISH TIME

Of the pulmonary function tests performed, only pre- and postrace MVV₃₀s measures had absolute correlation coefficients greater than 0.6. Linear regression analy-
sis of these data indicated that MVV\textsubscript{30s} values from 7.5 s to 27.5 s prerace and MVV\textsubscript{30s} values from 7.5 s to 17.5 s postrace (Figure 1) were significantly correlated (prerace: \(r = -0.80, -0.71, -0.75, -0.76, -0.82\); postrace: \(r = -0.84, -0.75, -0.61\)) with race finish time (example: Figure 3a). Total harmonic variation, \(A_{\text{HARM}}\), were also significantly correlated (supine: \(r = -0.83\); stand: \(-0.72\)) with race finish time (supine: Figure 3b), with higher variability associated with faster race times. Changes in the athletes’ cardiovascular (Table 3) and HRV (Table 4) variables between stand and supine, or in pulmonary function (Table 2) and MVV\textsubscript{30s} values between pre- and postrace, were not correlated with finish time. No significant correlation was found between the competitors’ height, weight, age, years of training, or race history (Table 1) with race finishing time (all \(r < 0.5, p > 0.2\)).

**Figure 2.** Cardiovascular stand test data (left) and race heart rate data (right) for Subject 3. *Stand test:* The subject was assisted to the stand position at 8 minutes. Blood pressure values are shown in top graph. Systolic, mean, and diastolic arterial blood pressure tracings are present in order of top to bottom. Heart rate response to stand is shown in bottom graph. Solid bars represent the region of data used for spectral analysis (256 beats). *Race heart rate:* 30 minutes of data from the second (top graph) and the eight 10-km lap (bottom graph).
In this study we have explored cardiopulmonary physiology in ultra-endurance athletes before, during, and after a 100-km race. First, an extended MVV, with maximal voluntary ventilation for 30 seconds compared to the usual 12 seconds, was used to examine the relationship between ventilatory power and race performance. Second, a stand test was performed on these athletes to describe the cardiovascular physiology and reflexes. Both tests revealed new information and insight into cardiovascular control and pulmonary function over an ultramarathon, and of the relationship between pulmonary function, ventilatory power, and cardiovascular control on performance in an ultramarathon.

**Discussion**

In this study we have explored cardiopulmonary physiology in ultra-endurance athletes before, during, and after a 100-km race. First, an extended MVV, with maximal voluntary ventilation for 30 seconds compared to the usual 12 seconds, was used to examine the relationship between ventilatory power and race performance. Second, a stand test was performed on these athletes to describe the cardiovascular physiology and reflexes. Both tests revealed new information and insight into cardiovascular control and pulmonary function over an ultramarathon, and of the relationship between pulmonary function, ventilatory power, and cardiovascular control on performance in an ultramarathon.

**PULMONARY**

The shape of the MVV\textsubscript{30s} curve, both pre- and postrace, was similar to the Wingate anaerobic test in which a subject is required to leg-pedal an ergometer as fast as he or she can at a high resistance for 30 s. The extension of the MVV test from 12 to 30 s provided more information on the effect of endurance exercise on respiratory power. If the test had been restricted to the standard 12 seconds, there would have

<table>
<thead>
<tr>
<th>Measure</th>
<th>Supine</th>
<th>Stand</th>
<th>Dif-stand</th>
<th>10-20 km</th>
<th>80-90 km</th>
<th>Dif-race</th>
</tr>
</thead>
<tbody>
<tr>
<td>RRI (ms)</td>
<td>1091</td>
<td>844</td>
<td>−197</td>
<td>425</td>
<td>505</td>
<td>81</td>
</tr>
<tr>
<td>± 28</td>
<td>± 22*</td>
<td>± 21</td>
<td>± 12*#</td>
<td>± 13*#†</td>
<td>± 16</td>
<td></td>
</tr>
<tr>
<td>(A_{\text{Harm}}) (ms)</td>
<td>12.5</td>
<td>8.7</td>
<td>3.8</td>
<td>5.5</td>
<td>6.7</td>
<td>1.2</td>
</tr>
<tr>
<td>± 2.7</td>
<td>± 2.7</td>
<td>± 2.7</td>
<td>± 1.1*</td>
<td>± 1.1*</td>
<td>± 0.9</td>
<td></td>
</tr>
<tr>
<td>(A_{\text{HI}}) (ms)</td>
<td>10.4</td>
<td>4.1</td>
<td>6.2</td>
<td>1.6</td>
<td>2.0</td>
<td>0.4</td>
</tr>
<tr>
<td>± 1.4</td>
<td>± 1.4*</td>
<td>± 3.9</td>
<td>± 0.6*</td>
<td>± 0.6*</td>
<td>± 0.4</td>
<td></td>
</tr>
<tr>
<td>(A_{\text{LO}}) (ms)</td>
<td>5.6</td>
<td>6.8</td>
<td>1.2</td>
<td>3.7</td>
<td>5.1</td>
<td>1.3</td>
</tr>
<tr>
<td>± 2.1</td>
<td>± 2.1</td>
<td>± 2.0</td>
<td>± 0.9</td>
<td>± 0.9</td>
<td>± 0.7</td>
<td></td>
</tr>
<tr>
<td>PNS indicator</td>
<td>0.13</td>
<td>0.06</td>
<td>−0.08</td>
<td>0.01</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>± 0.01</td>
<td>± 0.01*</td>
<td>± 0.04</td>
<td>± 0.01*#</td>
<td>± 0.01*#</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>SNS indicator</td>
<td>0.29</td>
<td>0.68</td>
<td>0.40</td>
<td>0.38</td>
<td>0.51</td>
<td>0.13</td>
</tr>
<tr>
<td>± 0.12</td>
<td>± 0.012*</td>
<td>± 0.11</td>
<td>± 0.04*#</td>
<td>± 0.05*†</td>
<td>± 0.5</td>
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</tr>
<tr>
<td>beta ((β))</td>
<td>1.01</td>
<td>1.50</td>
<td>0.48</td>
<td>1.06</td>
<td>1.33</td>
<td>0.27</td>
</tr>
<tr>
<td>± 0.15</td>
<td>± 0.15*</td>
<td>± 0.18</td>
<td>± 0.06#</td>
<td>± 0.06*†</td>
<td>± 0.5</td>
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</tbody>
</table>

**Note:** Difference of stand from supine (Dif-stand: stand − supine) and the difference of postrace from prerace (Dif-race: postrace − prerace) in CGSA variables. Significant, \(p < 0.05\), from supine (*), stand (#), or 10–20 km (†).
been no difference between pre and post results. The conclusion would have had to be that maximal ventilatory power was unaffected, similar to what was found after a triathlon (Hill et al., 1991). However, extending the test to 30 seconds elucidated a substantial reduction of approximately 30 L·min⁻¹ in ventilatory power in the last 15 s of the pretest and close to 50 L·min⁻¹ in the posttest. The reduction in ventilatory power from 10 to 30 s may be similar to the 5-min ventilatory endurance test reduction observed after an ultramarathon (Ker and Schultz, 1996). This decrease in MVV was due to a drop in tidal volume rather than respiratory frequency (Figure 1). The drop in force vs. frequency is common in fatigue studies and has been noted many times for ventilation in which fatigue induces a decrease in tidal volume and not respiratory frequency (Hue et al., 1999; Mador and Acevedo, 1991).

The determination of fatigue is a difficult task in the lab and even more so in the field. Power is the product of force and velocity. As fatigue progresses, how do each of these three (power, force, velocity) change? Sometimes only one is measured and sometimes more than one, but rarely all three. To further complicate interpretation, they are usually measured as one-time maximal efforts and not in an endurance manner.
After 5 to 15 min of fatiguing leg exercise, maximal inspiratory pressure decreases in normal subjects (Coast et al., 1993; McConnell et al., 1997) but not in trained subjects (Coast et al., 1993). By contrast, after exhaustive running in the same time range, Perret et al. (1999) did not find any reduction in inspiratory pressure but found a substantial decline in ventilatory endurance to task failure in the 3- to 6-min range. If the exercise duration is extended to a triathlon, then trained subjects do show a reduction in maximal inspiratory pressure (Hill et al., 1991). Three days after an ultramarathon, subjects showed a reduction in inspiratory endurance time but not in inspiratory pressure. Two and a half hours after a marathon, maximum inspiratory pressure is reduced but not 12-s MVV (Chevrolet et al., 1993). With the equipment limitations and field conditions in the current study, we chose the FVC test and prolonged the standard 12-s MVV to 30 s.

Respiratory chemosensitivity changes may affect the MVV_{30s} test. However, these changes would be very small for the within-subject variation between pre- and posttesting, especially when maximal voluntary efforts are being used. Certainly the trend of MVV_{30s} with time was very similar to data collected at rest under isocapnic conditions (Mulvey et al., 1991). The results showing an MVV_{30s} in the 5- to 10-s range as a strong predictor of performance would not be expected to be influenced by hypocapnia.

Although actual ventilatory rates during the last 10 km of the race were not measured, they were probably below the somewhat asymptotic final value (145 L·min^{-1}) of the MVV_{30s} test. This does not imply that respiratory muscle fatigue was not occurring. The significant decline in the MVV_{30s} postrace test indicates the respiratory muscles were fatigued. Whether this fatigue limits performance is another question. Cardiac output is shared in part by the leg muscles and the respiratory muscles. If the respiratory muscles received an extra amount of blood flow relative to the leg muscles as the run progressed, then one could say that respiratory muscles probably limited leg performance. It has been speculated that metaboreceptors in the respiratory muscles can increase leg muscle vascular resistance (Rodman et al., 2003). This would attenuate leg muscle performance.

It is also likely that the body has a built-in design to protect homeostatic function. Leg muscles can fatigue (i.e., a decrease in maximal effort) during submaximal exercise and can reach exhaustion when they can no longer maintain the desired output (Bigland-Ritchie et al., 1986). When the leg muscles exhaust, the subject can always lie down and recover. The respiratory muscles do fatigue, but they cannot exhaust. If the respiratory muscles can no longer maintain the ventilatory rates required for adequate homeostatic intake of O_2 and elimination of CO_2, whether during exercise or postexercise, there will be deleterious consequences. Thus there may be central mechanisms that will attenuate leg muscle activation in order to preserve homeostatic function (Walsh, 2000) or, via peripheral metaboreceptors, shift blood flow to those vascular beds required for homeostatic function.

There was a significant correlation between many of the MVV_{30s} values and race finishing time (Figure 3). This relationship was independent of whether the test was performed before or after the race. The numerous multiple correlations performed increased the risk of a type II error. However, there were many significant correlations and they were not randomly distributed. Rather they were in con-
secutive order, indicating the presence of an underlying trend. The change in MVV\textsubscript{30s} from pre- to postrace was not correlated with race performance. These data suggest that the greater the subject’s initial ventilation power, the better the race performance. At the same race ventilatory rate, the runner with the larger MVV will have the advantage of ventilating at a lower percentage of his or her maximum ventilation. It is more likely that the better runner, with the higher MVV\textsubscript{30s}, will operate at a greater percentage of his/her MVV (along with a greater cycling power output), as has been shown for elite cyclists compared to sedentary subjects during short-duration cycling (Folinsbee et al., 1983).

What also cannot be ruled out in interpreting pulmonary function data is the athlete’s motivation to perform postrace cardiorespiratory testing. It must be recognized that after such extreme prolonged exercise, at least some of the athletes were not lining up to be tested. From undocumented clinical observation, vigilance and other general mental functioning appeared temporarily diminished in some of the athletes immediately after the race.

CARDIOVASCULAR

We found a strong correlation between harmonic heart rate variability and race performance (Figure 3b), but not with the sympathetic or parasympathetic indicators. We can only report that there is a relationship between heart rate variability associated with autonomic nervous system control (A\textsubscript{HARM}) and race performance (finishing time) of an ultramarathon athlete. As has been reported previously from cross-sectional studies comparing subjects of varied athletic ability, endurance training is associated with increased HRV (Achten and Jeukendrup, 2003; Carter et al., 2003).

Although athletic training is related to increased HRV (Carter et al., 2003), all of the athletes had at least 6 years of training and performance at the marathon level. It is possible that the observed relationship of HRV and 100-km performance may be related to overtraining. Overtraining can lead to a decrease performance. In a study of half-marathon and marathon runners, Portier et al. (2001) observed an increase in high frequency power and a decrease in low frequency power and total variance of the HRV signal when comparing intensive training vs. rested training. The association of decreased variance with decreased performance is similar to what we observed with harmonic variance (A\textsubscript{HARM}) and performance. Our results are likely a combination of at least 6 years of athletic training, and the effects of recent overtraining, on HRV and performance.

To the best of our knowledge, these data are the first to provide beat-by-beat heart rate information during an ultramarathon. Heart rate variability analysis revealed several interesting aspects related to cardiac control from the beginning to the end of the race. Parasympathetic activity, sympathetic activity, and beta were significantly different at the beginning of the race when compared to the stand test the previous day (Table 4). Both exercise and stand are associated with a decrease in PNS activity. Withdrawal of cardiac PNS activity allows for an increase in heart rate. As expected, running heart rate was significantly higher than standing heart rate (Table 4). During the race, and with tilt we observed an increase in sympa-
thetic activity and beta (Table 4). Increases in sympathetic activity are normally associated with a response to increased cardiovascular stress; this is often coupled with an increase in beta. Beta is inversely related to the complexity of HRV and is thought to represent the number of inputs used to regulate heart rate, which decrease with increased stress (Blaber et al., 1996; Butler et al., 1993; Mayer-Kress et al., 1988).

Surprisingly, although the sympathetic indicator was increased near the end of the race, heart rate was reduced. It is possible that the effects of fatigue have altered the reactivity of the heart to sympathetic activity. In the last two stages of the race the participants are experiencing the effects of fatigue and have reduced their running pace (5.0 ± 0.5 min·km⁻¹, 10 to 20 km vs. 6.4 ± 1.0 min·km⁻¹, 80 to 90 km). Although the experimental protocol does not allow us to determine heart rate and sympathetic activation at a 6.4 min·km⁻¹ pace under fresh conditions, the lower heart rate with higher cardiac sympathetic tone could also suggest the possibility of cardiac fatigue (Dawson et al., 2003).

When interpreting HRV, care must be taken to ensure that the data segment was obtained during steady-state conditions (stationarity, Task Force, 1996). In this study we took care to ensure that data used for HRV analysis was from segments that were closest to being steady state. In the stand test the subject was supine for at least 10 minutes before HRV analysis, and during stand the HRV analysis was not performed until at least 1 minute had passed, allowing for heart rate and blood pressure to stabilize (Figure 2). During the race, data from the 10-to 20-km and the 80- to 90-km segments were used (Figure 2). The former was chosen to allow the runners to reach a comfortable pace. As the race continued, the running pace declined in all subjects, and in the final 10 km some were varying the pace considerably. However, in the 80- to 90-km portion of the race all racers maintained a steady pace.

We chose to use CGSA to investigate HRV due to its unique ability to allow for examination of fractal variability. However, results from the harmonic component of CGSA are compatible with those from a general spectral analysis fast Fourier transform (FFT) using the Task Force (1996) guidelines. A standard FFT algorithm with a suitable windowing function (Hanning window) applied to the stand data demonstrates the compatibility of CGSA with HRV guidelines (Task Force, 1996). The square root of the high frequency (FFT-A_HI: 0.15–0.4 Hz) and low frequency (FFT-A_LO: 0.04–0.15 Hz) showed similar trends (FFT-A_LO: supine 13.3 ± 0.6 ms > stand 14.8 ± 0.6 ms; FFT-A_HI: supine 14.9 ± 1.8 ms > stand 7.4 ± 1.8 ms, p = 0.019) to their CGSA counterparts (Table 4).

It is possible that underlying physical characteristics may explain some of the significant correlations found relating cardiorespiratory variables to performance. Certainly pulmonary function is related to height, age, and gender (Cotes, 1993). Respiratory functioning generally declines with age but 100-K running performance increases with age (up to a certain point) because it usually takes so much training volume to be successful. Furthermore, training can improve respiratory function (Pelkonen et al., 2003). These complicating factors make it difficult to relate common physical characteristics of cardiorespiratory functioning to performance, especially with the number of subjects evaluated in the present study.
SUMMARY

In conclusion, we have provided preliminary evidence that maximal sustainable ventilatory power and dynamic autonomic cardiovascular control are important factors in determining overall performance in an ultramarathon. The cardiovascular data support the theory that heart rate variability is directly related to endurance fitness level. Completion and performance in a 100-km running race is inarguably a reflection of overall endurance exercise fitness. Even at this extreme level of exercise, high resting HRV was directly associated with race performance. Although this evidence is correlative and may be a reflection of other unmeasured factors, we cannot exclude genetics or differences in training adaptation. These data provide new insights into the critical interaction of maximum sustainable ventilatory power and cardiovascular control over a continuous endurance event lasting several hours.

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References


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