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Effects of optimal pacing strategies for 400-, 800-, and 1500-m races on the $\dot{V}O_2$ response

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Abstract

The aim of this study was to compare the evolution of oxygen uptake ($\dot{V}O_2$) in specifically trained runners during running tests based on the 400-, 800-, and 1500-m pacing strategies adopted by elite runners to optimize performance. Final velocity decreased significantly for all three distances, with the slowest velocity in the last 100 m expressed relative to the peak velocity observed in the 400 m (77%), 800 m (88%), and 1500 m (96%). Relative to the previously determined $\dot{V}O_{2\max}$ values, the respective $\dot{V}O_{2\text{peak}}$ corresponded to 94% (400 m) and 100% (800 and 1500 m). In the last 100 m, a decrease in $\dot{V}O_2$ was observed in all participants for the 400-m (15.6 ± 6.5%) and 800-m races (9.9 ± 6.3%), whereas a non-systematic decrease (3.6 ± 7.6%) was noted for the 1500 m. The amplitude of this decrease was correlated with the reduction in tidal volume recorded during the last 100 m of each distance ($r = 0.85$, $P < 0.0001$) and with maximal blood lactate concentrations after the three races ($r = 0.55$, $P < 0.005$). The present data demonstrate that the 800 m is similar to the 400 m in terms of decreases in velocity and $\dot{V}O_2$.

Keywords: *Pacing strategy, oxygen uptake, running, performance*

Introduction

The strategy of pacing, defined as varying the speed throughout a race by regulating the rate of energy expenditure (Foster, Schrager, & Snyder, 1994), is proposed to be a marker of underlying physiological regulation (Tucker & Noakes, 2009). When compared directly, the performances of gold, silver, and bronze medallists are within 1% of each other and even small variations in pacing strategy may have marked effects on race outcome (de Koning, Bobbert, & Foster, 1999). Of the competitive athletics distances, only the 400-, 800-, and 1500-m races require substantial contributions from the both aerobic and anaerobic systems (Hill, 1999), due to high relative values of oxygen uptake ($\dot{V}O_2$) (Hanon, Lepretre, Bishop, & Thomas, 2010; Hanon, Leveque, Thomas, & Vivier, 2008; Thomas et al., 2005) and blood lactate concentrations above 15 mmol · L⁻¹ that have been reported previously (Hill, 1999; Lacour, Bouvat, & Barthélémy, 1990). Thus, an analysis of the optimal pacing strategies during these particular athletics events potentially could inform coaches and researchers, and provide insight into the underlying physiological processes involved in the performance.

Few studies have focused on the pacing strategies used in athletics competitions. In studying the pacing strategies adopted during world-record performances in 800-, 5000-, and 10,000-m races, Tucker and colleagues (Tucker, Lambert, & Noakes, 2006) noted key differences between the 800 m, for which the second lap was significantly slower than the first lap, and the 5000 m and 10,000 m, where an end spurt was observed due to the maintenance of a reserve during the middle part of the races. From an energetics point of view, the 800- and 5000-m races appeared to be regulated differently. Hanon and Gajer (2009) described the pacing strategies used by elite 400-m runners, and found that the last 100 m was also the slowest of the entire race, as was the last 100 m of elite 800-m races (Gajer, Hanon, Marajo, & Vollmer, 2001).

Differences in race strategy may have impact on physiological responses. In manipulating start pacing strategies during the first 4 min of a 20-km cycling time trial, Mattern and colleagues (Mattern, Kenefick, Kertzer, & Quinn, 2001) observed an impact both on the initial and subsequent $\dot{V}O_2$. A positive effect of an all-out pacing strategy compared with constant pacing on a 2-min paddling performance

was reported by Bishop and colleagues (Bishop, Bonetti, & Dawson, 2002), who thought it the result of a greater total $\dot{V}O_2$ with a similar accumulated oxygen deficit. Nevertheless, although demonstrated as optimal, these fast-start pacing strategies imply a slowing down in velocity (Gajer et al., 2001; Hanon & Gajer, 2009) and a decrease in $\dot{V}O_2$ (Billat, Hamard, Koralsztein, & Morton, 2009; Hanon et al., 2008, 2010; Thomas et al., 2005) towards the end of races. To date, no study has compared the $\dot{V}O_2$ responses of the 400-, 800-, and 1500-m race distances.

Using experimental designs based on the pacing strategies adopted during athletics events when runners realized performances equal to or very near their best, the aim of the present study was to describe and compare the effects on oxygen uptake. In light of past studies, since an aggressive pacing strategy is necessary for a best performance, and because of the supposed influence on metabolic responses, we hypothesized that the 400 m, 800 m, and 1500 m could be characterized by gradual $\dot{V}O_2$ responses: (1) a greater and earlier $\dot{V}O_{2peak}$ when the race distance is shorter, and the start velocity greater; and (2) a greater reduction in $\dot{V}O_2$ when the race distance is short, and a higher contribution of the anaerobic metabolism to the energetic needs. To address these hypotheses, we review and synthesize data previously published by our team in scientific or technical journals.

Methods

Data from six studies by our team, undertaken between 2000 and 2008, were analysed. These studies were published as original articles in scientific (Hanon & Gajer, 2009a; Hanon, Thomas, Leveque, & Vivier, 2007; Hanon et al., 2008, 2010; Thomas et al., 2005) or technical publications (Gajer et al., 2001; Hanon et al., 2007), with the aim of either describing optimal pacing strategies or the consequences of them on the time course of $\dot{V}O_2$. The analyses of pacing strategies were for elite runners (allowing comparisons to be made) with the aim of achieving a best performance (Part A), whereas the ventilatory studies (Part B) were realized with non-elite runners (inter-regional or national level). To allow comparison of the 400-, 800-, and 1500-m data, new techniques have been applied that allowed modification of previously published results. In particular, the same method of determination for the reduction in $\dot{V}O_2$ (final 100-m values expressed relative to peak values) was applied to the three distances that resulted in a less marked reduction in $\dot{V}O_2$ compared with the original 800-m study (24%). Furthermore, in the 1500-m study (Hanon et al., 2008), the reduction in $\dot{V}O_2$ was not reported. The three studies of Part B conformed to the recommen-

datations of the Declaration of Helsinki, and participants provided voluntary written consent to participate in this experiment, which was approved by the local ethics committee.

Part A: Chronometric velocity profile analysis

Velocity profiles of elite athletes competing in the 400-, 800-, and 1500-m events have been characterized using video analysis. Three groups of 20–50 elite athletes each were used for the 400, 800, and 1500 m. From all three groups, individuals were eliminated whose performances were 0.5 s (400 m), 1 s (800 m), and 1.5 s (1500 m) below their best time and all 800- and 1500-m runners whose successive distance marks were not discernible. Finally, three groups of 5, 25, and 10 athletes were analysed for the 400, 800, and 1500 m. The averaged performances of each group (44.4 s, 1 min 43 s, and 3 min 35 s for the 400, 800, and 1500 m respectively) corresponded to roughly the world best performances for the athletics season considered in each study, which corresponds to 1220, 1232, and 1175 points respectively (IAAF Scoring Tables 2008, Revised Edition).

Protocol

Because races are run in lanes and the subsequent gaps between the corridors, the 400 m was analysed differently (additional cameras) from the 800 m and 1500 m (for more details, see Hanon & Gajer, 2009). The 800-m and 1500-m analyses were based on images from personal video of national athletics coaches or from IAAF data. The times of passage were determined each 50 m (400 m) or 100 m (800 and 1500 m) by means of a time code on the TV monitor. Sequences were digitized to register the instant at which each athlete passed the markers that had been filmed previously. The average velocity for each segment was calculated from times recorded for the 50- or 100-m segments.

Part B: Ventilatory parameters

Participants

In three different studies by our team (2005, 2008, 2010), 10, 6, and 12 specifically trained 400-, 800-, and 1500-m runners (5–7 training sessions per week) were asked to reproduce these models of pacing strategies. These athletes were successful in regional and national running competitions [average performance of 50.9 ± 1.2 s for the men and 57.4 ± 3.7 s for the two women ($1 \text{ min } 55 \text{ s} \pm 0.03 \text{ s}$ and $3 \text{ min } 56 \text{ s} \pm 0.21 \text{ s}$) in the 400, 800, and 1500 m respectively].

Protocol

For all tests, oxygen uptake ($\dot{V} O_2$) was recorded continuously by means of a telemetric gas analyser (Cosmed K4, Roma, Italy) as previously described (Hanon et al., 2008, 2010; Thomas et al., 2005). Capillary blood samples were analysed with a Lactate Pro analyser (Arkray, Japan) (800 and 1500 m) and a Biosen Lactate analyser (Biosen C-line analyser, EKF Industrie, Elektronik GmbH, Barleben, Germany) (400 m).

Incremental protocols

Both $\dot{V} O_{2\max}$ and $v\text{-}\dot{V} O_{2\max}$ (the slowest speed at which an athlete was running when $\dot{V} O_{2\max}$ occurred) were determined using an incremental test conducted on an outdoor track as previously described (Hanon et al., 2008, 2010; Thomas et al., 2005).

400-, 800-, and 1500-m running tests

The warm-up was standardized based on a regular pre-event warm-up (15–20 min of jogging, stretching, and sprints at the respective start velocity). Since 400-, 800-, and 1500-m performances were different between athletes, cardiorespiratory data obtained as a function of time (5 s central moving average) were averaged over 25-m intervals (400 m, 800 m) or 50-m intervals (1500 m). The highest 5-s value for each distance was taken to be $\dot{V} O_{2\text{peak}}$. The end $\dot{V} O_2$ ($\dot{V} O_{2\text{end}}$) value was defined as the average of the last 100-m values, and the decrease in $\dot{V} O_2$ ($\dot{V} O_{2\text{decrease}}$) was defined as the difference between $\dot{V} O_{2\text{peak}}$ and $\dot{V} O_{2\text{end}}$ expressed relative to the peak values. Blood samples were taken from the ear lobe just before the tests and then at 1, 4 (400 m), 3 and 5 (800 and 1500 m), and 7 and 10 min (400, 800, 1500 m) during a passive recovery. The start and final velocities corresponded to the mean velocity during the first and the last 100-m segments. The velocity measured at 25% was the average velocity during the 25-m (400 m, 800 m) or 50-m (1500 m) segment preceding 100, 200, and 350 m after the onset of the test for the 400, 800, and 1500 m respectively.

Statistic analysis

Data are reported as means + standard deviations (s). Relationships between variables (running velocity at different times of the race, metabolic parameters, and $\dot{V} O_2$ kinetics) were analysed by Pearson's correlation coefficient. The relationship between blood lactate concentration (x) and the decrease/increase in $\dot{V} O_2$ (y) was assessed by regression

analysis. All statistical analyses were conducted using Statview software (version 5.0). Statistical significance was set at $P \leq 0.05$.

Results

Part A: Chronometric velocity profile analysis

As shown in Table I, and in spite of the start inertia, the start velocities recorded during the three races were always faster than the respective mean velocity. For the 400- and 800-m distances (Figure 1), peak velocities were reached early during the first (400 m) and second 100 m (800 m), although the fastest 100 m was covered between 1200 and 1300 m of the 1500-m distance. The velocity in the last 100 m decreased significantly in the 400-, 800-, and 1500-m races and was the slowest of the segments in the 400 and 800 m. Expressed relative to the peak value, the slowest final 100 m was observed for the shortest distance (Table I).

As observed in Table II and Figure 2, $\dot{V} O_{2\text{peak}}$ was higher and reached later when the distance run was long. A decrease in $\dot{V} O_2$ when the distance was short was observed in all participants in the 400- and 800-m races and in 9 of 12 participants in the 1500 m. Furthermore, a decrease in average tidal volume was systematically observed for all three distances. A decrease in minute ventilation was observed in the 400 m and 800 m and a decrease in respiratory frequency was noted in the 400 m only (Figure 3). There was a strong correlation between $\dot{V} O_2$ and tidal volume recorded during the last 100 m of each race ($r^2=0.85$, $P \leq 0.0001$).

Part B: Ventilatory parameters

The velocity measured at 25% of the distance (100, 200, and 350 m after the onset of the test for the 400,

Table I. Start and final velocities.

	400 m	800 m	1500 m
Mean velocity ($\text{m} \cdot \text{s}^{-1}$)	8.99	7.76	6.97
First 100 m ($\text{m} \cdot \text{s}^{-1}$)	9.06	7.86	7.35
Segment time (s)	11.04	12.73	13.61
Last 100 m ($\text{m} \cdot \text{s}^{-1}$)	8.12	7.32	7.13
Segment time (s)	12.31	13.65	14.02
Last 100 m (%)	76.60	88.40	95.94

Note: Velocity ($\text{m} \cdot \text{s}^{-1}$) is measured over the complete distance (mean velocity) and over the first and last 100 m of each distance. The first and the last 100-m segment times (s) correspond to the times to cover the first and the last 100 m of each distance. The last 100 m is expressed (%) relative to the fastest portion (50 m for the 400 m; 100 m for the 800 m and 1500 m) of each corresponding distance.

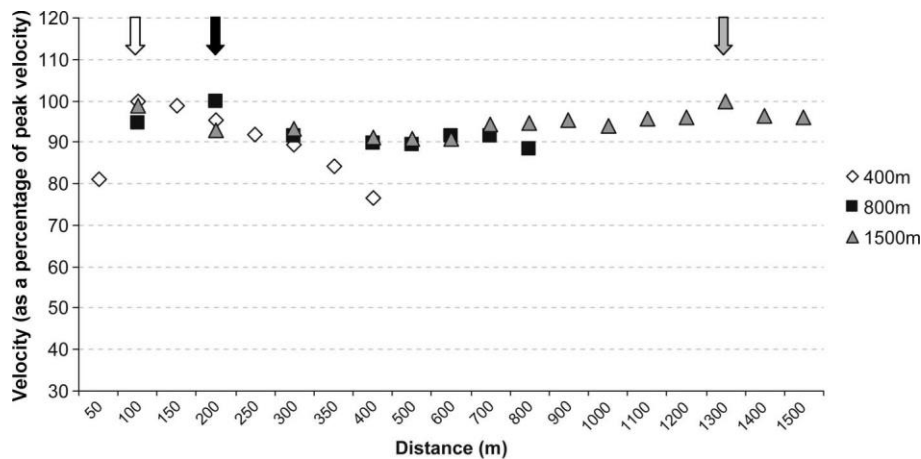


Figure 1. Time course of velocity during the 400-, 800-, 1500-m running elite competitions. Velocities expressed relatively to the peak of velocity of each distance (white, grey and black symbols correspond to 400, 800 and 1500-m velocities and respective arrow correspond to 400, 800 and 1500-m peak velocities). The 400, 800 and 1500-m velocities are presented every 25, 50 and 100 m, respectively.

Table II. Peak values measured during (cardiorespiratory variables) and after (blood lactate concentration) the 400-, 800-, and 1500-m tests (mean + s).

	$\dot{V} O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\dot{V} O_{2peak}$ (% $\dot{V} O_{2max}$)	Distance at $\dot{V} O_{2peak}$ (m)	$\dot{V} O_{2decrease}$ (% $\dot{V} O_{2peak}$)	Peak lactate ($mmol \cdot L^{-1}$)
400 m	54.2+5.7	93.9+3.9	192+22	15.6+6.5	22.0+1.9
800 m	66.3+2.3	103.3+3.8	316+75	9.9+6.3	17.8+1.4
1500 m	69.2+6.5	105.1+10.5	459+59	3.6+7.6	14.9+0.9

Note: $\dot{V} O_{2peak}$ was determined as the highest 5-s central moving average. $\dot{V} O_{2decrease}$ is expressed relative to the peak values defined as the difference between the $\dot{V} O_{2peak}$ and $\dot{V} O_{2end}$.

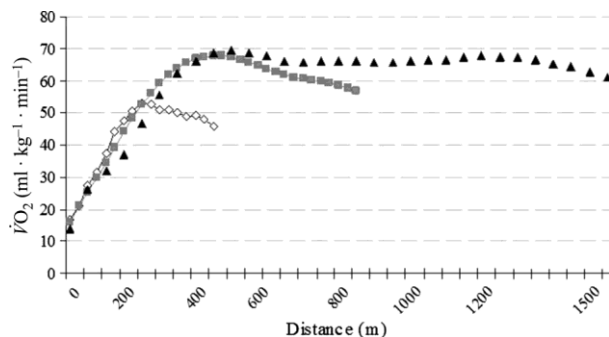


Figure 2. Time course of $\dot{V}O_2$ during the 400-m (○), 800-m (■), and 1500-m (▲) running tests.

800, and 1500 m respectively) was the parameter most strongly correlated with the time to reach $\dot{V} O_{2peak}$. The time to reach $\dot{V} O_{2peak}$ was shorter when the start velocity at 25% of the distance was high ($r^{1/4} 70.79$, $P \leq 0.0001$). Other correlations (12%, 5%, and 33% of the distance) with time to reach $\dot{V} O_{2peak}$ were calculated but found to be weaker ($P \leq 0.0005$ and $P \leq 0.005$ respectively). The amplitude of the decrease in $\dot{V} O_2$ was related to peak blood lactate concentration measured after the 400-, 800-, and 1500-m tests ($r^{1/4} 0.55$, $P \leq 0.005$, $n^{1/4} 28$) but no relation was observed

between velocity and decrease in $\dot{V} O_2$ ($r^{1/4} 0.29$). In the 1500-m runners, the decrease in $\dot{V} O_2$ was correlated with the velocity at 25% of the distance covered (measured between 300 and 350 m) expressed as a percentage of $v - \dot{V} O_{2max}$ ($r^{1/4} 70.59$, $P \leq 0.05$). The significant relationship between the magnitude of the decrease/increase in $\dot{V} O_2$ and peak lactate concentrations post-race ($r^{1/4} 0.55$, $P \leq 0.005$, $r^2^{1/4} 0.30$) indicates that 10 $mmol \cdot L^{-1}$ is the minimum value under which no decrease in $\dot{V} O_2$ is likely to occur in such exercise (Figure 4) and that blood lactate concentration can explain 30% of the magnitude of the decrease in $\dot{V} O_2$.

Discussion

Athletics distances of 400, 800, and 1500 m are characterized by common (fast-start) but also distinct (final velocity) characteristics, making them unique in their own way. From 400 to 1500 m, the links between pacing strategies and $\dot{V} O_2$ are clearly demonstrated: (1) a strong relationship between the velocities measured at 25% of each distance and the time to reach $\dot{V} O_{2peak}$, and (2) the occurrence of a decrease in $\dot{V} O_2$ that is related to the final peak lactate concentration.

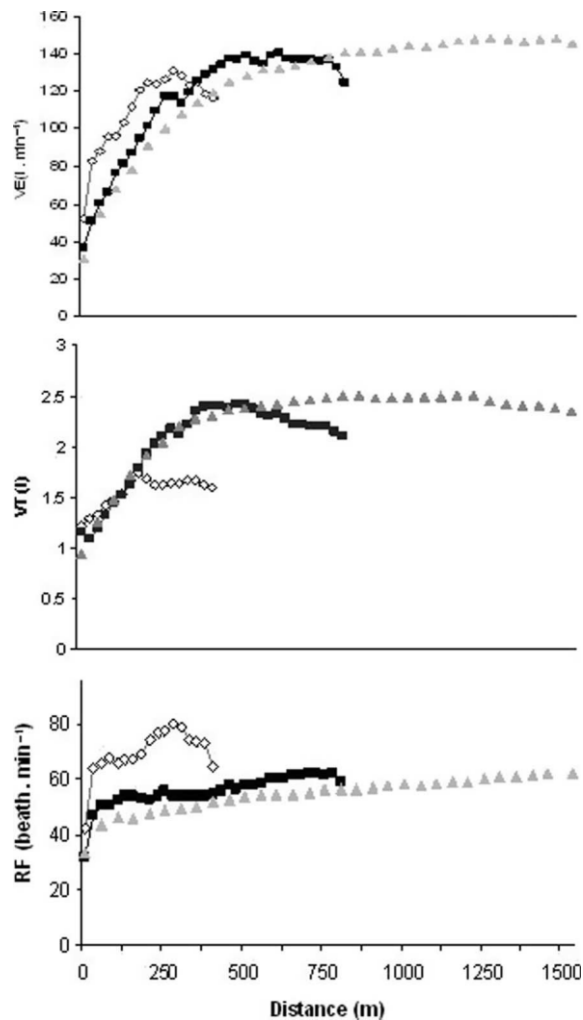


Figure 3. Ventilatory parameters time course during 400, 800 and 1500-m running tests. RF: respiratory frequency, VT: tidal volume, VE: minute ventilation measured during the 400-m (○), 800-m (◻) and 1500-m (◄) distances.

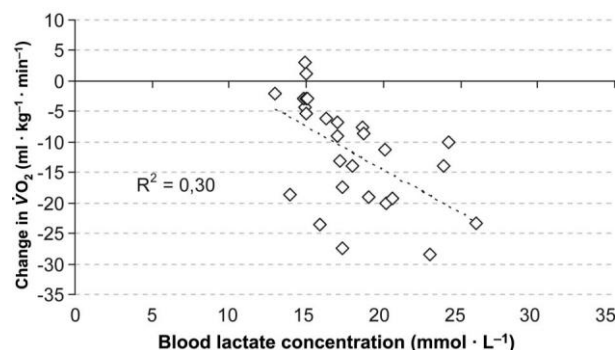


Figure 4. Blood lactate concentration ($\text{mmol} \cdot \text{L}^{-1}$) vs. change in $\dot{V}O_2$ ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$).

Consequence of the start velocity

The pacing strategies used by elite 400-, 800-, and 1500-m runners show some common characteristics, such as a fast start and a slowing of velocity in the last 100 m (Gajer et al., 2001; Hanon & Gajer, 2009;

Hanon et al., 2007). However, whereas no acceleration was observed in the second half of the 400- and 800-m races, peak velocity in the 1500 m was observed 300 m before the finish line.

In contrast, in studies performed on a treadmill at average constant velocity (Draper & Wood, 2005; Draper, Wood, & Fallowfield, 2003; Spencer & Gatin, 2001), $\dot{V}O_2$ has previously been shown to be at or close to maximum (Duffield, Dawson, & Goodman, 2005a, 2005b; Hanon et al., 2008, 2010; Thomas et al., 2005) in races of 400 to 1500 m using this positive (athlete's speed gradually declines) pacing strategy. Some authors have tested the positive effect of this pacing on the amplitude of $\dot{V}O_{2\text{peak}}$, which results in a better performance in 2-min paddling (Bishop et al., 2002), running (Gatin, Costill, Lawson, Krzeminski, & McConell, 1995; Sandals, Wood, Draper, & James, 2006), and 60-s cycling (de Koning et al., 1999) exercises. The correlation ($r = 0.7079$, $P = 0.0001$) observed in the present study between the velocity at 25% of each distance and the time to reach $\dot{V}O_{2\text{peak}}$ ($r = 0.7079$, $P = 0.0001$) is in agreement with the literature (Åstrand & Saltin, 1961; Hill, 1999). Therefore, this positive or "all-out" pacing strategy appears necessary to reach a high relative $\dot{V}O_2$ in such a short time, and to realize one's best performance.

Decrease in $\dot{V}O_2$ in the last part of the races

In our three previous studies, a decrease in $\dot{V}O_2$ was observed in the final part of the race whereas the velocity was always greater than $v \cdot \dot{V}O_{2\text{max}}$ for all participants, even in the last 100 m of the 400-, 800-, and 1500-m distances. Whereas this phenomenon was observed in every runner in the 400 and 800 m, this decrease was less marked and not systematic in the 1500 m (Billat et al., 2009; Hanon et al., 2007). This decrease in $\dot{V}O_2$ was shown to be related to the final decrease in velocity over the 400- and 800-m distances but not the 1500-m study, no more than observed in the total population (400, 800, and 1500 m). Nevertheless, for the 1500 m, this final $\dot{V}O_2$ response was correlated with the velocity at 25% (300–350 m) expressed as a percentage of $v \cdot \dot{V}O_{2\text{max}}$, indicating that the outcome of pacing depends on the duration of the race, with the 1500 m being regulated differently than the 400 and 800 m. Furthermore, as a decrease has previously been reported (Nummela & Rusko, 1995) and studied (Perrey, Candau, Millet, Borrani, & Rouillon, 2002) at a constant pace on exhaustive treadmill running tests, causality cannot be inferred in the relationship between the decrease in velocity and decrease in $\dot{V}O_2$. During treadmill running tests to exhaustion, biomechanical adaptations such as a decrease in stride length are possible (Hanon,

Thepaut-Mathieu, & Vandewalle, 2005). This decrease in stride length could be the result of a progressive failure to produce force (Taylor, Bronks, Smith, & Humphries, 1997), since Nummela and colleagues (Nummela, Vuorimaa, & Rusko, 1992) found that drop-jump performance was impaired by *39% following a maximal 400-m sprint, during which a decrease in stride length was observed. The significant correlation between the magnitude of decrease in $\dot{V}O_2$ and blood lactate concentration observed in the present study suggests that this phenomenon is related to the high glycolytic contribution and therefore to a high level of fatigue, as shown by the decrease in stride length and/or stride frequency. To reinforce this result, a correlation was observed between pH values and the final $\dot{V}O_2$ in the 400-m race (Hanon et al., 2010). Based on the present results and those in the literature (Billat et al., 2009; Nummela & Rusko, 1995), a large glycolytic contribution could partly explain why a decrease in $\dot{V}O_2$ is likely to occur during short and intense exercise. This relationship between the magnitude of the decrease in $\dot{V}O_2$ and blood lactate concentration could explain the absence of a decrease in $\dot{V}O_2$ with a less aggressive pacing strategy (Duffield, Dawson, & Goodman, 2004; Duffield et al., 2005b; James, Sandals, Draper, Maldonado-Martin, & Wood, 2007). One can then postulate that the reductions in $\dot{V}O_2$ and velocity (or only stride length) are both the consequences of extreme fatigue.

Furthermore, the strong positive relationship observed between tidal volume and $\dot{V}O_2$ recorded during the last 100 m of the three races could be a consequence of the hyperpnoea due to metabolic acidosis (Forster & Pan, 1995). These results could indicate that the runners hyperventilated so as to partially compensate for their metabolic acidosis and to maintain an effective alveolar O_2 pressure (Miyachi & Katayama, 1999). This decrease in tidal volume associated with an increase in breathing frequency, observed for the 800- and 1500-m distances, was reported previously by Cibella and colleagues (Cibella et al., 1996), during exhaustive exercise under chronic hypoxia, and by Perrey et al. (2002) in participants who demonstrated a decrease in $\dot{V}O_2$. Gallagher and colleagues (Gallagher, Im Hof, & Younes, 1985) have shown that this particular respiratory pattern observed during exhausting exercise could be considered an indirect sign of respiratory fatigue. Nevertheless, it is noteworthy that decreases in all ventilatory parameters (minute ventilation, breathing frequency, and tidal volume) were observed in the 400-m runners, demonstrating another respiratory pattern.

Why is this pacing strategy the most efficient?

This pacing strategy also possesses the potential to cause premature fatigue because of disturbances in muscle pH. This acidosis has been shown to inhibit aerobic capacity (Jubrias, Crowther, Shankland, Gronka, & Conley, 2003) and to cause low final $\dot{V}O_2$ values (Hanon et al., 2010; Thomas et al., 2005). As reported by Foster et al. (1994), athletes learn how to sense low muscle pH and adjust their pace accordingly, so that they ideally reach critical low values of pH near the end of a race. Interestingly, in 1997, Kipketer beat the 800-m world record when using this pacing strategy. The pacing strategies described in this study confirm the model based on the two equations derived from Newton's second law and that for power balance of metabolism described by Maronski (1996). In this model, the author concluded that the optimal tactic for performance over these intermediate distances, which are neither sprints nor long-distance events, is an initial acceleration phase and a final reduction in velocity.

Based on the intermediate pH values recorded for the 400 m (Hanon et al., 2010), well-trained athletes appear to be able to run about 15 s with a pH of 7.1, but must reduce the pace when pH falls below 7.0. Many authors (Abbiss & Laursen, 2008; Billat et al., 2009; de Koning et al., 1999; Foster et al., 1994; Hettinga, De Koning, Broersen, Van Geffen, & Foster, 2006) have concluded that anaerobic resources seem to be critical in regulating pacing strategies. Successful performance on the 400 m and 800 m requires full use of the buffering capacity (Ward-Smith, 1999), and therefore critical final values of pH (Foster et al., 1994). To accelerate in the last straight line is not compatible with these criteria, and does not allow a best performance.

The 1500-m distance appears to be regulated differently from the shorter distances as there was a significant and negative relationship between start velocity, expressed as a percentage of $v\text{-}\dot{V}O_{2\max}$ and final performance. This result is in good agreement with the conclusions of Billat et al (2009), who demonstrated that 1500-m runners who had the longest limits at their anaerobic power in the first part of the race (that is, close to the present notion of velocity expressed as a percentage of $v\text{-}\dot{V}O_{2\max}$) were those who achieved the best final performance. As in Billat and colleagues' study, this relation was obtained only in the 1500 m, which indicates a different pacing regulation compared with the shorter distances. Furthermore, the difference in competitive standard of participants (more than 20 s between the present study and that of Billat et al., 2009) could indicate that this result depends on the

duration of exercise rather than on the standard of competition.

Conclusions

When exercise is less than 3 min 30 s, the only way to ensure the total depletion of anaerobic reserves is to reach the exact sustainable pH value at the finish line. It appears that this is only possible with a fast start and a slowing of velocity in the last metres of the race, as calculated by Maronski (1996). The decrease in $\dot{V}O_2$ observed in the final stages of the three events examined here appears to be a consequence of aggressive pacing strategies and, therefore, extreme exhaustion. The high correlation between the decreases in $\dot{V}O_2$ and tidal volume indicates that ventilatory regulation likely induced by metabolic H⁺ accumulation is the major cause of the decrease in $\dot{V}O_2$. The 1500 m, which is regulated differently from the shorter distances, appears to be intermediate between predominantly anaerobic distances (400- and 800-m races) and predominantly aerobic middle- and long-distance running.

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