Optimal pacing strategy in competitive athletic performance

'Wie sterk is moet ook slim zijn'

Florentina J. Hettinga

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Optimal pacing strategy in competitive athletic performance

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Ο λαγός και η χελώνα Μύθος του Αισώπου

Ένα ανοιξιάτικο πρωινό ένας λαγός είχε βγει έξω από την φωλιά του και έτρωγε φρέσκο χορταράκι. Καθώς έτρωγε, είδε μια χελώνα να περνάει λίγο πιο μακριά και του φάνηκε τόσο αστείο το περπάτημα της, που άρχισε να την κοροϊδεύει ότι ήταν πιο αργή και από τα σαλιγκάρια. Η γελώνα σταμάτησε, γύρισε προς τον λαγό και του είπε:-Τι θα έλεγες να τρέξουμε σε ένα αγώνα δρόμου για να δούμε ποιος είναι πιο γρήγορος από τους δυο Αυτό ήταν! Ο λαγός έπεσε κάτω και άρχισε να χτυπιέται από τα γέλια. Βλέποντας όμως ότι η χελώνα παρέμενε σοβαρή, κατάλαβε ότι δεν του το είπε για αστείο και έτσι δέχτηκε την πρόκληση. Η αλεπού ως καταλληλότερη, όρισε το σημείο που θα ξεκινούσαν, την διαδρομή και το σημείο τερματισμού. Ο αγώνας ορίστηκε για το επόμενο πρωινό και πράγματι, οι δύο διαγωνιζόμενοι καθώς και πολλά ζώα του δάσους βρίσκονταν πρωί πρωί στην αφετηρία. Η αλεπού έδωσε το σύνθημα και ο αγώνας ξεκίνησε. Η χελώνα χωρίς να χάσει χρόνο άρχισε να περπατάει, αργά βέβαια, και ήδη είχε καλύψει τα πρώτα εκατοστά της διαδρομής. Ο λαγός βλέποντας τον ρυθμό της αντιπάλου του, και νυστάζοντας μιας και ήταν πολύ πρωί, σκέφτηκε να κοιμηθεί λιγάκι και όταν ξυπνήσει θα έτρεχε όπως μόνο αυτός μπορεί και θα τερμάτιζε σίγουρα πρώτος. Έτσι η χελώνα συνέχισε να περπατάει, στην ορισμένη από την αλεπού διαδρομή, ενώ ο λαγός το έριξε στον ύπνο. Πέρασε αρκετή ώρα και κάποια στιγμή ο λαγός ξύπνησε. Καιρός για τρέξιμο είπε και ξεκίνησε. Παραξενεύτηκε πολύ που δεν συναντούσε την χελώνα και για μια στιγμή σκέφτηκε ότι θα είχε εγκαταλείψει τον αγώνα αφού έτσι κι αλλιώς τον είχε χαμένο από χέρι. Περισσότερο όμως ξέρετε πότε παραξενεύτηκε; Όταν έφτασε στο σημείο τερματισμού και είδε την γελώνα να τον περιμένει μασώντας ένα φυλλαράκι και έγοντας μια έκφραση θριάμβου στο πρόσωπο της. Έτσι η χελώνα κέρδισε τον λαγό σε αγώνα δρόμου, όχι βέβαια γιατί τρέχει πιο γρήγορα από αυτόν, αλλά γιατί παρέμεινε πιστή στον σκοπό της και δεν έδειξε όπως ο λαγός αλαζονεία.

AESOP (620-560 BC)

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CHAPTER 1: PROLOGUE

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The tortoise and the hare, Aesop (620 - 560 BC)

The hare was once boasting of his speed before the other animals. "I have never yet been beaten," he said, "when I put forth my full speed. I challenge anyone here to race with me." The tortoise said quietly: "I accept your challenge." "That is a good joke," said the hare. "I could dance around you all the way!" "Keep your boasting until you've beaten," answered the tortoise. "Shall we race?" So a course was fixed and a start was made. The hare darted almost out of sight at once, but soon stopped and, to show his contempt for the tortoise, lay down to have a nap. The tortoise plodded on and plodded on, and when the hare awoke from his nap, he saw the tortoise nearing the finish line and he could not catch up in time to save the race.

Source: Joseph Jacobs, The Fables of Aesop (London, 1894), no. 68.

This fable shows that pacing strategy was already an issue about 2500 years ago, when the story was written by the Greek storyteller Aesop. Nowadays, it is still an issue in everyday life. In sports performance, it is not only about being stronger than the opponent, but also about being smarter, as the tortoise was, who made optimal use of his capacities. This is illustrated by the words of Dutch elite speed skater Simon Kuipers after his winning race against his direct opponent Erben Wennemars to earn a world cup ticket.

Simon Kuipers vs. Erben Wennemars: Daarom besloot Kuipers zijn tactiek aan te passen aan de opponent. "Ik heb op de persoon gereden," erkende hij, "ik moest snel openen, anders kon Erben op de kruising achter mij aan schaatsen. Ik concentreerde me vooral op die eerste 700 m. Dat was de enige manier om deze rit te winnen."

Source: www.knsb.nl.

[That was the reason Kuipers decided to adjust his tactics to the opponent. "I chose my pacing strategy based on the opponent. I had to open really fast, otherwise Erben could draft behind me at the crossing. So I focused on the first 700 m, that was the only way to win the race."]

Because the tortoise knew the capabilities of his own body (keeping a steady pace), he was able to win. Being aware or not aware of your own capabilities is also a familiar issue in sports performance, as illustrated by the following:

Bedrogen door het eigen gevoel. Dat overkwam de atleten Bram Som en Arnoud Okken op de 800 m al in de series met een kansloze uitschakeling. "Ik had erop gegokt dat ik zou kunnen meesprinten. Dat gaf mijn gevoel aan, en op dat gevoel heb ik altijd kunnen vertrouwen. Wat me het meeste dwarszit, is dat mijn gevoel me nu bedriegt." Ook Okken had zich laten misleiden door zijn gevoel. Kansloos kwam hij de laatste bocht uit. "Ik voelde me sterker dan ik was."

Source: Trouw (08-31-2007), Rob Velthuis

[Being deceived by one's own feeling. That is what happened to the track and field athletes Bram Som and Arnoud Okken, during the 800-m series, resulting in a failure to qualify. "I thought I could win the sprint. That is what my feeling told me. My feeling has always been right, but deceived me now, which

bothers me." Okken was also mislead by his feeling. He stood no chance coming out of the last curve.' I felt stronger than I was']

Choosing the right pacing strategy thus seems to be of considerable importance in sports performance and makes pacing strategy an interesting research topic. Different pacing strategies can be studied experimentally or by the use of modeling. The present thesis will experimentally explore several aspects that are relevant for pacing strategy, in order to provide the input to simulate different pacing strategies accurately by the use of an energy flow model (17, 18). Using this input, different pacing strategies will be modeled and compared to self-paced performance, resulting in possibilities for athletes to improve performance.

Modeling performance

Modeling performance is an appealing way to study pacing strategy. Various patterns of energy expenditure and their effect on performance can be simulated (6, 7, 18, 19, 20), while environmental circumstances can be held constant. Performance can be described with the use of an energy flow model (equation 1), that is based on power equations describing mechanical power production (power that is generated by the athlete) on the one side and mechanical power losses (power used to overcome frictional forces) and changes in kinetic energy (power involved in acceleration or deceleration of the center of mass) on the other side.

$$P_{tot} = P_{lost} + dE_{kin}/dt$$
 [1]

 P_{tot} is the mechanical power output generated by the athlete, P_{lost} is the mechanical power used to overcome frictional forces and dE_{kin}/dt is the change in kinetic energy $(\frac{1}{2} \cdot m \cdot v^2]$, m is body mass and v is velocity) of the center of mass of the athlete over time.

Performance can be optimized by maximizing the mechanical power that can be produced (P_{tot}), but also by distributing energy in such a way that the available resources are used in the most efficient way, and a high mean velocity over the race is achieved.

To model performance accurately, it is important to have a good knowledge of factors playing a role in performance, both on the power production side (aerobic and anaerobic) as well as on the opposite side of the equation. The present thesis will first study the P_{tot} (chapters 2, 3, 4, 5 and 6) side of the energy flow model related to pacing strategy. Next, performance and different pacing strategies will be modeled. The interplay between the different components of equation 1 will be studied to explain for example differences in day to day performance and the importance of choosing the right pacing strategy in order to obtain optimal performance (chapters 7 and 8). All experiments investigated supra-maximal middle distance race performance (1500 to 4000 m) in cycling (chapters 2 to 7) or speed skating (chapter 8). In exercises of this duration and intensity, both aerobic and anaerobic pathways

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are very important. In addition, these are air resisted sports where high velocities are attained, making minimization of energy losses to air frictional forces of great importance in choosing optimal pacing strategy.

As mentioned, the first step in the present thesis will be to investigate factors associated with the power production (P_{tot}) side of the power equation in sports performance. An athlete possesses the ability to generate a certain amount of metabolic energy over a certain amount of time, that can be transformed into mechanical energy ('movement') and/or heat. It will be explained how the mechanical equivalent of metabolic energy production can be determined and modeled and how pacing strategy might affect P_{tot} , leading to the questions that are addressed in chapter 2 to 6. In the last paragraphs, the interplay between all components of equation 1 will be described, as well as the modeling of different pacing strategies, leading to questions addressed in chapters 7 and 8.

Metabolic energy production

The main source of energy production in the human body is adenosine triphosphate (ATP). ATP plays a central role in energy exchanges in biological systems (17). ATP is an energy rich nucleotide, consisting of an adenine, a ribose and a triphosphate unit. By hydrolizing ATP to adenosine diphosphate (ADP) and orthophosphate (Pi), a large amount of free energy is liberated necessary for muscle contractions (equation 2). This reaction is catalyzed by actomyosine ATP-ase (8).

$$ATP + H_2O \leftrightarrows ADP + Pi + H^{\dagger} + 'energy'$$
 [2]

ATP serves as an immediate donor of free energy. The turnover of ATP is very high. During supra-maximal exercise, the rate of utilization of ATP may be as high as 0.5 kg per minute (1). There is merely enough ATP for contracting 3 twitches or sustain contractile activity for less than a second. So, to continue muscular activity, ATP has to be regenerated. This can be done anaerobically, via the creatine kinase reaction (equation 3) and the anaerobic glycolysis (equation 4), or aerobically (equation 5). Anaerobic metabolism, associated with sprinting capacity, exceeds aerobic metabolism in supra-maximal exercise lasting 60 s or less and remains a significant contributor (> 15%) in exercise lasting up to 5 min (4). Aerobic metabolism, associated with endurance performance, is important in shorter and supra-maximal events too. It has been shown that in the early stages of a single bout of a 20-s sprint exercise, the rate of ATP turnover from anaerobic sources is reduced during the second half resulting from a low phosphocreatine (PCr) and a decrease in glycolysis. The thereby expected decrease in power output is largely compensated by a 2-fold increase in aerobic energy contribution (3).

Anaerobic pathway

PCr stored in the muscle is the main source of anaerobic ATP production (equation 3) for the first 3 to 15 s of an all-out sprinting effort (17, 21). PCr can transfer its phosphoryl group to ATP (equation 3), catalyzed by creatine kinase.

$$ADP + PCr + H^{+} \subseteq ATP + Cr$$
 [3]

After several seconds, PCr stores are exhausted and anaerobic glycolysis, the breakdown of glucose, becomes of great importance in supra-maximal exercise. Equation 4 gives the net equation for anaerobic glycolysis. Glucose or a glycosyl unit of glycogen (during high intensity exercise most of the energy from carbohydrate comes from glucose stored as intramuscular glycogen) is transformed into glucose-6-phosphate, which is transformed into pyruvate (17) (equation 4a). In the absence of oxygen, pyruvate is reduced, catalised by lactatedehydrogenase (17), while forming lactate and NAD⁺ (equation 4b). The supply of NAD⁺ is limited and if it is not regenerated, glycolysis could only be continued for about 2 s. The regeneration of NAD⁺ sustains the continued operation of glycolysis under anaerobic conditions.

glucose + 2 ADP + 2
$$P_i \leftrightarrows 2$$
 lactate + 2 ATP + 2 H_2O [4]

glucose + 2 ADP + 2
$$P_i$$
 + 2 NAD⁺ \rightleftharpoons 2 pyruvate + 2 ATP + 2 NADH + 2 H⁺ + 2 H₂O [4a]

pyruvate + NADH +
$$H^{\dagger} = \text{lactate} + \text{NAD}^{\dagger}$$
 [4b]

Because of the limited stores of PCr and because the concentration of lactate in muscle almost never rises above 30 mmol·kg⁻¹ wet muscle mass, it is assumed that energy produced via anaerobic processes has a maximum: anaerobic capacity (14). Also, Gastin et al. (12) suggest that the maximal amount of anaerobic work is a constant value. Data from our own lab suggested otherwise and showed an increasing 'anaerobic capacity' with time trial duration up to 6 min. This will be discussed further in chapters 3 and 4.

Aerobic pathway

Only a small fraction of energy content of glucose is released in its anaerobic conversion into lactate. Much more energy can be extracted aerobically (net equation is presented in equation 5), by means of the citric acid (Krebs) cycle and the electron transport chain (17). So, besides providing energy, glycolysis fulfills another function: providing pyruvate as a substrate for oxidative metabolism (13).

The entry point of aerobic energy production is acetylcoenzyme A (acetyl CoA), which is formed by oxidative decarboxylation of pyruvate inside mitochondria. During intense exercise, acetyl CoA is completely oxidized into CO₂ by the citric acid cycle, resulting in 1 molecule ATP per acetyl-unit. This irreversible process links glycolysis

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to the citric acid cycle. The pyruvate formed in the glycolysis is used in this process for oxidative metabolism and cannot be re-transformed into pyruvate.

The energy rich molecules NADH and $FADH_2$, formed during the glycolysis and in the citric acid cycle, are phosphorilized oxidatively by transferring electrons from these molecules to O_2 by a series of electron carriers via the respiratory or electron transport chain. NADH enters the electron transport chain at the beginning of the chain, producing 3 ATP, while $FADH_2$ enters at a later stage and produces 2 ATP electrons and protons to form water at the end of the chain (13). NAD^+ is regenerated when NADH transfers its electrons to O_2 through the electron transport chain in the mitochondria.

$$3 \text{ ADP} + 3 P_i + \text{NADH} + \text{H}^+ + 0.5 O_2 \rightarrow 3 \text{ ATP} + \text{NAD}^+ + \text{H}_2\text{O}$$
 [5]

Power production in modeling performance

Only part of the total metabolic energy produced by the athlete is transformed into mechanical energy ('movement'). The energy that is not used for propelling the athlete is transformed into heat. For modeling performance, we are interested in movement, therefore it is necessary to describe performance in mechanical terms. In cycling, the mechanical equivalent of the metabolic energy produced over time can be measured accurately by multiplying torque on the pedals by pedal rotational velocity. For speed skating, total mechanical power can also be calculated as will be described in chapter 8. In modeling performance, mechanical equivalents of power production, aerobic power and anaerobic power are used and mechanical power production (Ptot) can be described as shown in equation 6.

$$P_{tot} = P_{aer} + P_{an}$$
 [6]

 P_{aer} is the aerobic contribution to the mechanical power output and P_{an} is the anaerobic contribution to the mechanical power output.

 P_{aer}

To obtain the mechanical equivalent of aerobic metabolic power (P_{aer}) during a race, the metabolically produced aerobic power (P_{met}) must be multiplied with gross efficiency (GE). Metabolic aerobic power production can be calculated by multiplying O_2 consumption with the energy equivalent of O_2 (11). GE is the ratio between the mechanical power that is used to propel the athlete and the produced metabolic power and can be calculated in sub-maximal exercise. Metabolic power production (P_{met}) is then generated solely via the aerobic pathways and is thus measurable as described above. By dividing total mechanical power output (P_{tot}) by P_{met} , GE can be obtained (equation 7), resulting in a percentage of total metabolic power that is used for the movement:

$$GE = P_{tot}/P_{met} \cdot 100\%$$
 [7]

Since the determination of GE is important for calculations of aerobic, and thereby anaerobic, mechanical power from aerobic metabolic power, it is an important factor in our models. Unfortunately, using the described method (equation 7), GE cannot be determined during a supra-maximal race because of the additional anaerobic contribution that cannot be measured accurately. GE can only be obtained in sub-maximal exercise preceding the race. GE might be influenced by ambient temperature or intensity of exercise and experiments were designed to test the assumption of a constant GE. The effect of ambient temperature on GE will be studied and discussed in chapter 5. Another experiment was designed to calculate GE at sub- and supra-maximal intensities via the heat balance The supportability of the assumption of a constant GE with intensity will be discussed in the epilogue.

By multiplying the obtained GE with the O_2 consumption and the energy equivalent of oxygen, P_{aer} can be calculated on a second by second basis. The response evoked by exercise can then be modeled mono-exponentially (equation 8) as is commonly done for $\dot{V}O_2$ response (2, 15).

$$P_{aer} = P_{aermax} (1 - e^{-\lambda(t-td)})$$
 [8]

 P_{aermax} is the maximal mechanical aerobic power, λ is the aerobic rate constant, t is time and td is the time delay. A mono-exponential fit resulted in good descriptions of the \dot{VO}_2 response and accurate predictions of supra-maximal performance were obtained (5, 6, 7, 15, 18, 19, 20). Chapter 6 will discuss this matter more extensively and will study possible effects of pacing strategy on the \dot{VO}_2 response to exercise.

 P_{an}

Assuming a constant GE, P_{an} can be calculated by subtracting P_{aer} from P_{tot} and can be described by a mono-exponential pattern (5, 6) (equation 9).

$$P_{an} = P_{anmax} \cdot e^{-\gamma t}$$
 [9]

 P_{anmax} is the maximal anaerobic power at t=0, t is time and γ is the anaerobic time constant. However, this equation is based on fixed duration experiments and anaerobic energy contribution at the end of the race is assumed to be 0, at least, if the race is as long as it takes to reach the asymptote. Recently however, spontaneously chosen pacing strategy in competition were studied and it was shown that anaerobic energy was used throughout the end of the race (10). To include this phenomenon in the model, a constant anaerobic power (P_{ancon}) is added to the equation, representing an asymptotic value for anaerobic energy expenditure (7). P_{an} is now characterized by the first order system in equation 10.

$$P_{an} = P_{ancon} + P_{anmax} \cdot e^{-\gamma t}$$
 [10]

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Now that mechanical descriptions of P_{an} and P_{aer} can be determined, different patterns of energy production can be modeled, forming the basis of simulating different pacing strategies with the use of the energy flow model.

Power production and pacing strategy

Several assumptions about P_{tot} relative to pacing strategy need to be addressed. P_{anaer} is the most important for the simulation of different pacing strategies in middle distance exercise (2 to 6 min). When modeling optimal performance with an energy flow model, as done by De Koning et al. (6), it is assumed that variations in pacing strategy are determined by variations in anaerobic energy distribution, keeping aerobic energy distribution constant. Also, the amount of anaerobic energy that is produced over the race is kept constant. The first challenge of the present thesis is to evaluate whether the distribution of anaerobic energy is indeed the determining factor in pacing strategy (chapter 2). Extreme pacing strategies will be imposed, as done in Foster et al. (9), and effects of energy distribution pattern (both anaerobic and aerobic) will be analyzed. Also, possible effects of central fatigue on pacing strategy and performance will be studied in chapter 2. The concept that anaerobic work over the trial is a constant value will be challenged, and the effect of different pacing strategies and different durations of exercise on anaerobic capacity will be studied in chapters 3 and 4, respectively. In chapter 5, the effect of ambient temperature on gross efficiency will be determined, as a first step in testing the supportability of a constant GE. In addition to the results regarding the global distribution of aerobic power over the race (chapter 2) the VO2 response will be studied more closely in chapter 6, to gain more insight into a possible intensity-dependent speeding of the VO₂ response, which could be relevant in modeling pacing strategy and optimizing performance.

Interplay between different components of the power equation on the basis of finding the optimal pacing strategy

So far, only P_{tot} has been explained and discussed and one way of improving performance is of course by increasing P_{tot} . But on the other side of the power equation are two other components that are also of importance in improving performance: P_{lost} and dE_{kin}/dt (equation 1). The importance of choosing the right pacing strategy lies in maximizing the amount of produced energy that is used to propel the athlete during the race. Available energy stores must be distributed in such a way that frictional losses are minimized and available energy sources are efficiently used over the race to maintain mean velocity as high as possible (5, 6, 7).

The interplay between P_{tot} and P_{lost} results in temporal changes in kinetic energy (dE_{kin}/dt) of the athlete, and thereby determines a certain velocity profile over the race, that determines final time. For example in a 1500-m cycling or skating race, a certain amount of energy (anaerobic + aerobic) is available that has to be distributed over the approximately 2 min it lasts. In air-resisted middle distance events, where

relatively high velocities are attained, the power produced by the athlete is predominantly used to overcome air frictional forces and rolling (or ice) resistance (P_{lost}). Furthermore, part of the available energy used over the duration of the race is used to accomplish a change in kinetic energy (dE_{kin}/dt). If velocity is reduced, dE_{kin}/dt is negative, if velocity is increased dE_{kin}/dt is positive. When insufficient power is generated to maintain a constant velocity, frictional forces are overcome at the expense of kinetic energy and the athlete will slow down.

In both P_{lost} and dE_{kin}/dt , velocity is involved non-linearly. Consequently, different profiles of velocity distribution affect both the amount of power that is used to overcome frictional forces and the pattern of changes in kinetic energy, affecting performance. In cycling, P_{lost} is determined by power that is lost to overcome air frictional forces (P_{air}) (equation 11) and by power lost to overcome rolling resistance (P_{rol}) (equation 12).

$$P_{air} = k_1 \cdot v^3 \tag{11}$$

$$P_{rol} = k_2 \cdot v \tag{12}$$

 k_1 is the air frictional constant and k_2 is the roll resistance constant (see chapter 7 for cycling and chapter 8 for speed skating, where power is lost to overcome ice resistance instead of rolling resistance). P_{rol} is linearly related to velocity. P_{air} on the other hand is non linearly related to velocity, and variations in velocity profile will influence the pattern of P_{air} over the race and also the amount of total energy produced that has to be used to overcome air frictional forces. For example when velocity increases to twice the initial value, P_{air} will be 8 (2^3) times higher than in the initial situation. In this respect, for finding optimal pacing strategy, it is important to find the velocity-profile where the amount of the total energy produced that is lost to frictional forces is lowest, so that more energy can be used to accelerate or maintain velocity of the center of mass of the body and final time will be faster. If only this mechanism is taken into account, distributing energy evenly over the race after the initial start results in optimal performance.

However, by distributing the available anaerobic energy equally over the course of a race of short duration, velocity at the end of the race will be rather high, representing a certain amount of kinetic energy $(\frac{1}{2} \cdot m \cdot v^2)$ which was produced but not used throughout the race. The amount of the total available mechanical energy that was used at the finish line and responsible for the accomplished final time is lower when velocity at the finish line is higher, since the remaining energy is transported over the finish line and no longer contributes to performance. In that respect, a pacing strategy with a high initial velocity and a low finish velocity is advisable, where a large part of the total amount of energy produced is used before crossing the finish line, and contributed to maintaining a high mean velocity.

An optimal balance needs to be found between benefits of starting fast, having all available energy used before crossing the finish line, and benefits of distributing

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energy as evenly as possible (6). The interplay between P_{lost} and dE_{kin}/dt determines the most efficient way to use the available energy resources and forms the basis of the pacing studies described in chapter 7 and 8 of the present thesis. Time trial length is of importance in answering the question which strategy is optimal. In sprinting events, performing an all-out strategy is advised, because having a lower E_{kin} is of more importance than minimizing air frictional forces. For example, in a simulation study of Van Ingen Schenau et al. (20) on the distribution of anaerobic energy in cycling bouts, a 1000 m cycling time trial was simulated. Results showed, that using an all-out strategy, cyclists spend 1090 J (2.3% of total) more energy to air friction than in an even paced strategy. However, in the all-out race, their kinetic energy was 1591 J lower than in cycling the even paced strategy. In this case, the lower kinetic energy in the all-out strategy is obviously of more importance than the higher frictional losses. In longer distances, it is more important to minimize power losses to overcome air friction than having a low E_{kin} at the finish line.

The next step is now to focus on the interplay between the different components of the power equation. This will be done in the final chapters of the present thesis. Optimal pacing strategy will be modeled and compared to day to day performance (chapter 7). Finally, it will be tried to impose optimal pacing strategy upon athletes in the technically complicated sport of speed skating (chapter 8).

Modeling different pacing strategies

To find optimal pacing strategy, ideally, an athlete has to perform a multiple pacing strategies under controlled circumstances, to see which one is optimal. This is usually done in the early career of an athlete by trial and error. The energy flow model can easily simulate a lot of different pacing strategies under the same environmental and internal conditions. If velocity is the only unknown variable in the overall equation (1), velocity can be calculated numerically. For each step in the simulation, this differential equation is integrated. The anaerobic power distribution and therewith the velocity profile can be systematically changed, keeping anaerobic capacity constant.

The first study that simulated different pacing strategies numerically using the energy flow model was Van Ingen Schenau et al. (19). A 'time to constant power' was introduced by De Koning et al. (6), to find the point in time from where on it is beneficial to start distributing energy equally over the remaining part of the race. The 'time to constant power' indicates at which time an athlete has to change from following the traditional anaerobic profile to performing an 'even-paced' strategy and distributing the remaining anaerobic energy equally over the race. It was shown that for shorter distances (e.g. 1000 m cycling, 1 min), all-out performance was optimal and for the longer distances (4000 m, 5 to 6 min), energy could best be distributed equally over the race after a fast start of 10-15 s. Though this clearly showed that distance to be covered was an important issue in pacing strategy, this rather global way of varying pacing patterns must be refined to study pacing strategy per athlete individually on middle distance exercise, as will be done in the present thesis.

In the range of competitive performance, it was shown that anaerobic energy distribution could best be described by the mono-exponential equation as given in equation 10 and chapters 7 and 8. Generally, this equation says that performance/pacing strategy is dependent of the athlete's anaerobic peak power output (P_{anmax}), the athlete's 'decay' during the race (γ), and the power the athlete has left at the end of the race (P_{ancon}). Anaerobic pattern of energy distribution will now be varied by manipulating these three parameters iteratively, keeping total anaerobic energy contribution the same in all simulations.

Concluding remarks

In summary, to simulate pacing strategy, a differential equation (equation 1) is integrated numerically to compute velocity profiles and performance times in order to find the optimal pacing strategy for completing a race. The input for this differential equation is of great importance, since a model only predicts reality well if its input factors are well-described. Many factors interact in performance which makes modeling performance a challenging task. The main variables of importance in pacing strategy are energy, technique and fatigue, all influencing one another (Fig. 1).

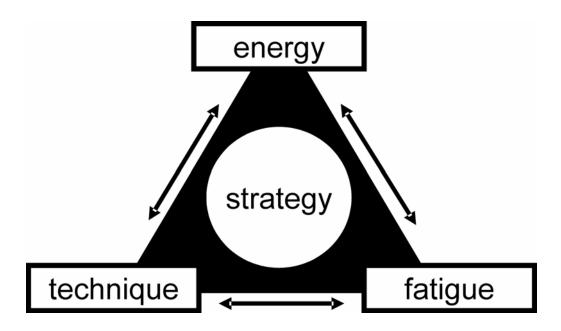


Fig. 1: Interacting complex of 'energy', 'fatigue' and 'technique', all influenced by strategy.

To understand this interacting complex, the first chapters of the present thesis will study the effect of pacing strategy on P_{tot} (the 'energy' part in Fig. 1), making it possible to model different pacing strategies and performance accurately, and study pacing strategy optimally. Determining factors of pacing strategy will be investigated (chapter 2), and anaerobic (chapter 3 and 4) as well as aerobic pathways (chapter 6) related to pacing strategy will be studied more closely. The assumption of a constant

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GE, that is important for calculating P_{aer} and P_{an} , will be studied and discussed (chapter 5 and epilogue). The last chapters will use the energy flow model to study the interplay between the different components P_{tot} , P_{lost} and dE_{kin}/dt and explain differences in day to day performance and the importance of choosing the right pacing strategy to obtain optimal performance (chapters 7 and 8).

Fatigue is implicitly included in the energy flow model, since it results in a drop in power production during the race and is thus taken into account in describing P_{tot} parameters. In chapter 2, a possible occurrence of central fatigue will be studied, to further investigate this topic relevant to modeling pacing strategy. In chapter 8, the 'technique' part of the figure will be studied by comparing different pacing strategies in the technically difficult sport of speed skating. In this way, the present thesis will focus on studying and modeling the complete athlete, to establish more insight in the complex totality of interacting factors involved in performance.

In the different chapters, the following will be discussed:

- Chapter 2: Does anaerobic energy expenditure determine pacing strategy and does central fatigue play a role in pacing?
- Chapter 3: Anaerobic capacity: Do different extreme pacing strategies lead to changes in anaerobic work?
- Chapter 4: Anaerobic capacity: Is anaerobic capacity a fixed amount independent of time trial length?
- Chapter 5: Does ambient temperature influence gross efficiency?
- Chapter 6: Is the VO₂ response influenced by starting intensity?
- Chapter 7: Interplay between variables: What determines variations in selfpaced exercise, and how do athletes pace their winning race compared to worse performance?
- Chapter 8: Interplay between variables: Is it possible to impose a more optimal pacing strategy in a technical difficult sport such as speed skating?
- Chapter 9: Epilogue.

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CHAPTER 2 PACING STRATEGY AND THE OCCURRENCE OF FATIGUE IN 4000-M CYCLING TIME TRIALS

Hettinga FJ, De Koning JJ, Broersen FT, Van Geffen P, Foster C. Pacing strategy and the occurrence of fatigue in 4000-m cycling time trials. Med. Sci. Sports Exerc. 2006; 38(8): 1484-1491.

Abstract

The present study was designed to examine the role of central and peripheral fatigue on 4000-m cycling time trial performance by comparing changes in mechanical power output (Ptot) and integrated electromyography (iEMG) in differently paced maximal efforts. Eight well-trained males performed three randomly ordered time trials with different pacing strategies, in which the first 2000 m were manipulated to evoke an increasing, even and decreasing power output profile (SUB, EVEN and SUPRA respectively). Subjects were instructed to finish the last 2000 m of all trials in the shortest time possible. iEMG of the rectus femoris (RF), vastus lateralis (VL) and biceps femoris (BF) muscle, Ptot and gas exchange variables were measured. Anaerobic and aerobic contributions to mechanical power output were calculated from gas exchange data. The increase in Ptot during the SUB-time trials was always associated with an increase in iEMG in all muscles. A decrease in Ptot near the end of the time trials was also marked by an increase in iEMG for all muscles, except for the RF. Comparing the last 2000-m interval with the first, aerobic power output increased for all strategies. Anaerobic power output increased in SUB and decreased in EVEN en SUPRA. The relationship between iEMG and Ptot pattern was consistent with peripheral fatigue rather than central downregulation of Ptot. Specifically anaerobic energy resources seem to be of importance in regulating pacing strategy.

Introduction

Pacing strategy refers to the variation of speed over the race by regulating the rate of energy expenditure (4). This involves controlling multiple variables, all interacting with each other. First, biomechanical factors are of importance to regulate energy expenditure efficiently. External power losses must be minimized. For a 4000-m time trial, it is clear that an even paced strategy is optimal to minimize external power losses (4, 28). Because athletes are very fatigued at the end of the time trials however, it can be hypothesized that attempts to maintain power output were achieved only by progressively increasing the subjects' effort. Thus, fatigue is another important factor in pacing strategy and may depend on the presence of a preestablished estimate of the power output that can be sustained over the duration of the event.

Fatigue has been described as a decrease in force production and power output, or as the inability to maintain force despite the presence of an increased perception of action (13). It has traditionally been believed that decreases in force production and power output are attributable to an impaired muscle contractile function caused by physiological changes within the muscle. This phenomenon is generally described as 'peripheral fatigue' and appears in the presence of an unchanged or increasing neural drive (26). If power output is regulated by a peripheral fatigue mechanism, an increased central neural recruitment of additional motor units would be expected to occur in an attempt to compensate for the reduced power output from the fatiguing motor units (12).

Bangsbø et al. (2) showed that metabolic changes in muscles were not tightly correlated with decreasing power output. They suggested that particularly central factors might be the cause of fatigue during prolonged exercise. An alternative hypothesis has been proposed (20) in which a central neural governor acts to reduce muscle recruitment during fatiguing exercise. In the light of this hypothesis, it has been suggested that in time trials, central fatigue occurs as a safety mechanism for preventing potentially dangerous metabolic disturbances and preserving the integrity of the muscle fibers (21). In this case, the reduction in power output is suggested to be the result of a reduction in central neural drive. In support of this hypothesis, studies comparing integrated electromyography (iEMG) and power output in time trial exercise with self-paced strategy demonstrated that iEMG decreased in parallel with power output during bouts of high intensity time trial exercise during a 100-km time trial (25).

Further support for the central governor hypothesis was provided by Ansley et al. (1), who found that iEMG and power output rose at the end of 4000-m cycling time trial exercise. They concluded that power output paralleled iEMG and that fatigue is not regulated by peripheral mechanisms but is centrally controlled. This conclusion was drawn based on iEMG measurements during self-paced exercise in the rectus femoris muscle only. To determine whether power output parallels iEMG, and whether central fatigue plays a role in time trial exercise, a range of pacing strategies must be performed, measuring iEMG in various muscles. If fatigue during time trial

exercise was centrally controlled as suggested in the governor hypothesis of Noakes (20), it should be visible in a pacing strategy where a relative high power output is delivered in the beginning of the trial, with a subsequent slowdown in the latter part. This study was conducted to create a better understanding of the role of central and peripheral fatigue on performance during middle-distance exercise. Therefore, influence of pacing strategy on the occurrence of fatigue was investigated by comparing changes in power output and iEMG activity during 4000-m cycling time trials. Furthermore, the contribution of the anaerobic and aerobic energy system to the total mechanical power output (P_{tot}) was calculated for the different pacing strategies.

Methods

Subjects

Eight male subjects, highly motivated to produce maximal time trial efforts, were recruited for this study. They were all well-trained and familiar with cycling exercise. Before the experiment, all subjects were informed of the nature of the investigation, after which they gave written informed consent. The protocol has been approved by the human ethics committee. The characteristics of the subjects are given in Table 1.

Table 1: Subject characteristics: age, height, body mass, maximum heart rate (HR_{max}), maximum oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained ($P\dot{VO}_{2max}$).

	Mean ± SD
age (yr)	25.4 ± 5.6
height (cm)	185.2 ± 5.6
body mass (kg)	76.3 ± 2.9
HR _{max} (bpm)	189 ± 5
$\dot{\mathbf{VO}}_{\mathbf{2max}}$ (I · min ⁻¹)	4.1 ± 0.2
PVO _{2max} (W)	345 ± 22.9

Values are means ± SD.

Experimental design

The experiment consisted of five tests with at least 48 h between trials. All subjects completed a maximal incremental exercise test and four cycling time trials of 4000 m, all conducted on a custom-made electronically braked laboratory cycle ergometer simulating real competition. The incremental test was cycled at a pedal frequency of 90 revolutions per minute (rpm). After a warm-up of 2 min at a mechanical power output (P_{tot}) of 100 W, the test started at a P_{tot} of 150 W. P_{tot} was increased by 30 W every 3 min until the subject could no longer maintain the required power output or pedal frequency dropped below 80 rpm. All trials were performed under standard conditions with a constant temperature of 15°C, 50% relative humidity (RH). Oxygen consumption ($\dot{V}O_2$), respiratory exchange ratio (RER), P_{tot} and heart rate (HR) were

recorded during all tests. Respiratory gas exchange was measured breath by breath using open circuit spirometry (Oxycon Alpha, Mijnhardt, The Netherlands). Before each test, the gas analyzer was calibrated using a Jaeger 3-l syringe, room air and a standard gas mixture (5.04% CO₂). HR was recorded every 15 s using radio telemetry (Polar Electro, Kempele, Finland).

First trial

The first time trial was designed to determine at which P_{tot} subjects had to cycle their different strategies. After warming up for 7 min and 30 s (100 W, 90 rpm), including 3 sprints of 15 s at min 5 (200 W), 6 (400 W) and 7 (300 W), subjects were told to complete the distance as fast as possible, as in competition. Subjects remained seated during the whole trial and oxygen consumption was measured. After the time trial, subjects cooled down for 5 min at 100 W (90 rpm). The profile of the entire protocol is shown in figure 1.

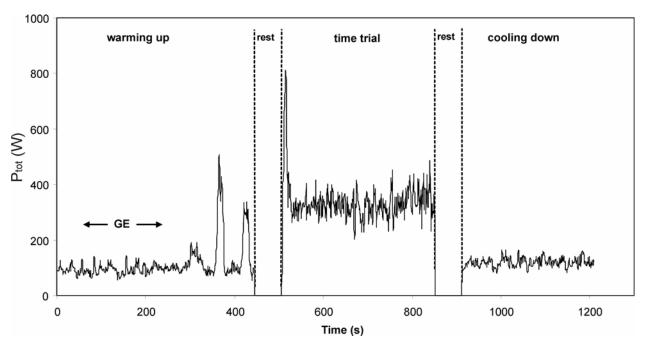


Figure 1: Typical example of a P_{tot} profile conform the protocol of the tests, including a warming up, the 4000 m time trial (EVEN) and a cooling down.

Time trials

The three remaining trials, performed in random order, were aimed at an even paced time trial (EVEN), a sub-maximal time trial (SUB) and a supra-maximal time trial (SUPRA). All trials started with a warm-up at 100 W (90 rpm) of 7 min and 30 s (see figure 1). During the 5 min of the warm-up, gross efficiency (GE) was estimated (11). After 5, 6 and 7 min, short sprints of 15 s at a higher P_{tot} (200, 400 and 300 W) were performed. After warming up, the subjects had 1 min of rest before the time trial. As in the first trial, subjects remained seated during the entire trial. P_{tot} was dictated during

the first 2000 m of all three time trials, based on the mean P_{tot} measured during the first trial. Elapsed distance and virtual velocity were the only feedback given to the subjects. During the first 2000 m of the SUB trial, the subjects were instructed to maintain a virtual velocity corresponding to a P_{tot} representing 95% of the mean P_{tot} measured during the first trial. During EVEN, the subjects rode the first 2000 m at a velocity corresponding to the mean P_{tot} of the first trial. During the first 2000 m of the SUPRA, 105% of the mean P_{tot} measured during the first trial was dictated. In all time trials, after the first 2000 m, the subjects were instructed to complete the time trial in as little time as possible. After performing the time trial, subjects cooled down for 5 min at 100 W (90 rpm).

According to the protocol, three different strategies performed by all subjects were the aim, which are broadly representative of spontaneous variations in pacing strategy adopted by athletes (5, 10). Because in all strategies, subjects were instructed to get the bike going as fast as possible and therefore, all strategies had a high peak power output during the first 200 m of the trial, which is not representative for the rest of the race. Therefore, the first 200 m were not incorporated in these analyses.

During all time trials, electromyographic muscle-activity (EMG), virtual velocity, P_{tot} , $\dot{V}O_2$, RER and HR were measured. EMG was measured over the Rectus Femoris (RF), Vastus Lateralis (VL) and Biceps Femoris (BF) muscle. These muscles are highly active during cycling (16). Anaerobic power output (P_{an}) and aerobic power output (P_{aer}) were calculated per 200 m as described in De Koning et al. (5), using $\dot{V}O_2$, RER and efficiency estimated during the warm-up (11). At the conclusion of 2000 and 4000 m, the rate of perceived exertion (RPE) was noted on a scale of 1 to 10, based on the Borg-scale (3). Blood lactate concentration (BLC) was measured before the time trial, after 2000 m and at the end of the time trial using dry chemistry (Lactate Pro, Arkray, Kyoto, Japan).

Electromyographic activity

To normalize EMG data, the maximal EMG level of each muscle was measured during maximal voluntary contractions (MVC). Before warming up for the time trials, each subject performed six isometric MVC's (three extensions and three times flexion of the knee joint of the left lower limb) of 5-s duration with 2 min of rest in between. During MVC, subjects sat in a specially designed chair in which the knee-angle was set at 90° and torque was measured by a built-in load cell. During extension, maximal EMG level of the RF and VL muscle was measured and during flexion, maximal EMG level of the BF muscle was measured. During the MVC tests, and all time trials, muscle recruitment was assessed by measuring EMG activity of the RF, VL and BF muscle of the left upper leg. EMG activity was recorded during the whole time trial with a sample rate of 2000 Hz (15). The EMG activity coinciding with peak torque of the best effort MVC was used to normalize the EMG values recorded during the time trials.

To remove external interference noise and movement artifacts, the raw EMG signals were filtered with a second-order Butterworth band-pass filter (10-400 Hz). The

already filtered EMG data were full-wave rectified and smoothed with a low-pass second-order Butterworth filter with a cut-off frequency of 10 Hz. Mean EMG (iEMG) was calculated over every successive 200 m.

Statistical analysis

Differences in mean values for efficiency (measured before the time trial) and final time between strategies were tested using ANOVA repeated measures.

For P_{tot} , P_{aer} , P_{an} , RPM, HR, BLC, RPE and iEMG, the mean values for the 200 to 2000-m interval were compared to mean values for the 2000 to 4000-m interval per strategy using 3 x 2 (strategy x interval) ANOVA repeated measures. If main effects were found, a pairwise comparison with Bonferroni adjustment was performed to find which variables differed significantly between strategies. In case of an interaction effect of strategy and interval, paired-sample t-tests were performed (p < 0.05) to compare mean values of variables over the first interval of the time trial to mean values of the corresponding variables over the last 2000-m interval.

Results

Power output

 P_{tot} , P_{aer} and P_{an} profiles are shown per 200 m segment for SUB, EVEN and SUPRA in figure 2. A 3 x 2 ANOVA with repeated measures revealed a main effect for strategy for P_{tot} . A pair wise comparison (with Bonferroni adjustment) showed a significant difference between SUB and SUPRA. Furthermore, an interaction effect was found. Paired samples t-tests showed that P_{tot} increased significantly in SUB. In EVEN, there was no significant difference and in SUPRA, P_{tot} decreased significantly (Table 2). For P_{aer} , a main effect of strategy was found, but a pair wise comparison did not show any significant differences between strategies. For interval, a main effect was found for P_{aer} . Also, an interaction effect was found. For all strategies, paired sampled t-tests showed that P_{aer} was significantly higher in the second interval of the trial compared with the first interval (Table 2). For anaerobic power output, no main effects were found for strategy and interval. An interaction effect was found. A significant increase in P_{an} comparing the second interval of the trial with the first interval was found in the SUB trial. In the SUPRA time trial, P_{an} decreased significantly (Table 2).

Table 2: Mean values for mechanical power output (P_{tot}), aerobic power output (P_{aer}), anaerobic power output (P_{an}) and iEMG for the RF, VL and BF over the first 2000 m of the time trial compared to the mean values over the second 2000 m of the time trial for each strategy.

	SUB		EVEN		SUPRA	
	200-2000 m	2000-4000 m	200-2000 m	2000-4000 m	200-2000 m	2000-4000 m
P _{tot} (W)	262.6 ± 32.2	$349.7 \pm 28.7^{\uparrow}$	311.8 ± 22.6	320.2 ± 35.1	338.6 ± 26.1	310.1 ± 25.8 _↓
$P_{aer}(W)$	203.6 ± 13.4	$248.1 \pm 12.3^{\uparrow}$	227.6 ± 10.2	$255.4 \pm 19.8^{\uparrow}$	228.3 ± 14.9	$254.0 \pm 18.7^{\dagger}$
$P_{an}(W)$	58.9 ± 24.0	$101.7 \pm 24.9^{\uparrow}$	84.3 ± 23.9	64.8 ± 23.8	110.4 ± 25.4	$56.0 \pm 28.1_{\downarrow}$
EMG _{RF} (%MV	(C) 9.4 ± 6.7	11.4 ± 6.7	10.5 ± 6.7	12.1 ± 7.2	12.0 ± 10.0	11.4 ± 7.8
EMG _{VL} (%MV	(c) 17.1 ± 7.6	$24.8 \pm 9.3^{\uparrow}$	21.9 ± 10.4	$26.3 \pm 11.3^{\uparrow}$	25.8 ± 12.6	$29.0 \pm 12.9^{\uparrow}$
EMG _{BF} (%MV	(C) 19.0 ± 6.8	$35.8 \pm 14.0^{\uparrow}$	26.7 ± 10.1	$38.9 \pm 15.0^{\uparrow}$	27.4 ± 11.6	$37.9 \pm 17.0^{\uparrow}$

Values are means \pm SD. The mean value over the 2000 to 4000-m interval is significantly higher than the mean value over the 200 to 2000-m interval when marked with a ' \uparrow ' and lower when marked with a ' \downarrow ' (p < 0.05).

Electromyography

iEMG and P_{tot} profiles are shown per 200-m segment for each strategy in figure 3. For iEMG, a 3 x 2 ANOVA repeated measures performed per muscle revealed main effects for strategy and interval for the VL and RF muscle, but not for the RF muscle. For all three muscles, an interaction effect was found. Paired sampled t-tests revealed a significant increase between iEMG in the second half compared to the first half of the race for the VL and BF (Table 2). For the RF, no significant differences were found between the time trial intervals. For the VL and BF muscle, the iEMG of the second 2000 m of the time trial was higher than the first part for all strategies. For the RF muscle however, a significant increase in iEMG level was not found. Thus, iEMG increased significantly (VL and BF) or remained constant (RF) in SUPRA, even when P_{tot} decreased significantly (Fig. 3). It can also be seen that iEMG never rises above 50% MVC in any of the pacing conditions.

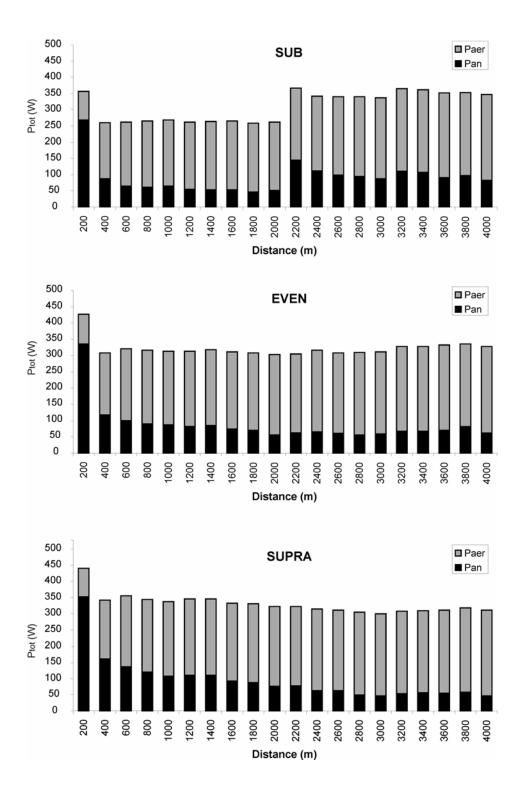


Fig. 2: Aerobic power output (P_{aer}) and anaerobic power output (P_{an}) per 200 m segment for SUB (upper panel), EVEN (center panel) and SUPRA (bottom panel). Total mechanical power output (P_{tot}) is the sum of P_{aer} and P_{an} .

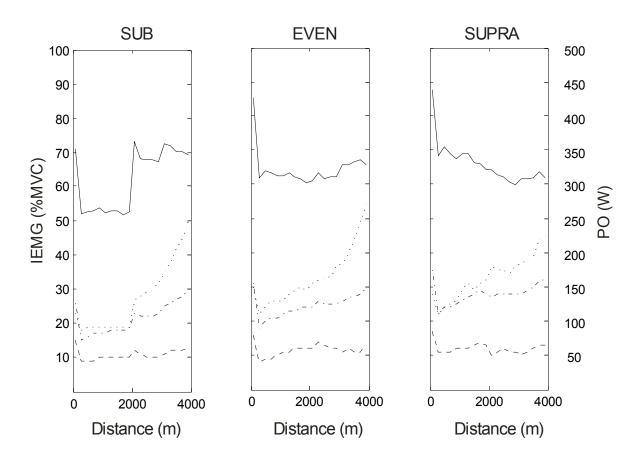


Fig. 3: Mechanical power output (P_{tot} , solid line) and iEMG for VL (dotted line), RF (dashed line) and BF (dash-dotted line) per 200-m segment for each strategy.

Heart rate, pedal frequency, blood lactate concentration and rate of perceived exertion

For RPM, a main effect of strategy was found as well as an interaction effect. Paired sampled t-tests comparing the second interval of the trial with the first revealed a significant increase in the SUB strategy and a significant decrease in the SUPRA strategy. Mean values for first and second interval per strategy are shown in table 3. For HR, strategies did not differ significantly. HR increased significantly in the second half compared to the first half for all strategies. For BLC, a main effect for strategy was found, but no significant differences between strategies were found in the pairwise comparison. Furthermore, BLC was significantly higher in the second interval compared to the first interval for all strategies (Table 3). For RPE, a main effect for strategy was found. A pairwise comparison with Bonferroni adjustment showed a significant difference between SUB and SUPRA. Also, a main effect for interval was found as well as an interaction effect. Paired samples t-tests revealed significant increases in RPE in SUB and in SUPRA (Table 3).

Table 3: Pedal frequency (RPM), heart rate (HR), blood lactate concentration (BLC) and rate of perceived exertion scores (RPE) over the first 2000 m of the time trial compared to the mean values over the second 2000 m of the time trial for each strategy.

	SUB		EV	EVEN		SUPRA	
	200-2000 m	2000-4000 m	200-2000 m	2000-4000 m	200-2000 m	2000-4000 m	
RPM (r·min ¹)	104.0 ± 1.5	113.5 ± 5.4 [†]	109.5 ± 1.5	109.7 ± 4.8	112.6 ± 1.2	108.9 ± 3.4 ↓	
HR (bpm)	164 ± 8	181 ± 8 [↑]	172 ± 9	184 ± 8 [†]	173 ± 8	182 ± 8 [†]	
BLC (mmol·l ¹)	7.6 ± 3.1	15.1 ± 2.4 [†]	12.2 ± 4.0	15.0 ± 3.4 $^{\uparrow}$	12.7 ± 2.9	16.6 ± 1.7 [↑]	
RPE	4.1 ± 0.8	$8.1 \pm 0.8^{\uparrow}$	5.3 ± 0.8	8.6 ± 0.7	6.1 ± 1.2	$9.0 \pm 0.8^{\uparrow}$	

Values are means \pm SD. \uparrow or \downarrow = The mean value over the 2000 to 4000-m interval is significantly higher respectively lower than the mean value over the 200 to 2000-m interval (p < 0.05).

Mean values for efficiency and final time

Mean values for efficiency measured during the warming up before the trial did not differ significantly between SUB (0.17 \pm 0.01%), EVEN (0.18 \pm 0.02%) and SUPRA (0.17 \pm 0.01%). Also, final times were not significantly different per strategy (351.1 \pm 6.2 s, 348.9 \pm 7.5 s and 345.8 \pm 5.8 s respectively).

Discussion

The main focus of the present study was to investigate the influence of different pacing strategies on fatigue by comparing changes in P_{tot} and iEMG activity during 4000-m cycling time trials, to create a better understanding of the role of central and peripheral fatigue on performance during middle-distance time trial exercise. Previous studies indicated that P_{tot} profile mirrored iEMG pattern in self-paced exercise. In these studies, iEMG was measured in the RF muscle solely (1, 15, 17, 24). We measured iEMG in three leg-muscles (RF, VL, BF) and found that iEMG patterns of the muscles differed. iEMG of the RF did not change or decreased slightly in all strategies, whereas iEMG of BF and VL increased significantly in all strategies, even when P_{tot} decreased. An increase in iEMG is contrary to the predictions of the central governor hypothesis but consistent with a peripheral locus of fatigue.

Ebenbichler et al. (6) has also found differences in EMG patterns of different muscles, in particular between mono and bi-articular muscles. Their functional difference might explain the occurrence of differences in fatigue patterns found between mono- and bi-articular muscles. Bi-articular muscles (RF, BF) seem to be responsible for controlling the distribution of net moments around the joints crossed (29), and thus are particularly involved in the regulation of movement direction and its external force (e.g. net torque on an environment). As a consequence, differences in seating position and technique will influence iEMG pattern. Conversely, mono-articular muscles (VL) seem to work mainly as work generators and thus differences in seating position and technique are expected to have little influence on the iEMG pattern. Therefore, the mono-articular VL muscle may potentially be the most appropriate muscle to monitor for evidence of central downregulation. The choice of which muscle

to monitor may be a significant issue, relative to future studies of the causes of fatigue.

In the previously mentioned studies about iEMG and P_{tot} during time trials, P_{tot} seemed to mirror iEMG. Additionally, these studies measured iEMG activity only in the RF muscle, and only employed self-selected pacing strategies. The P_{tot} profile was either even or slightly increasing towards the end of the trial, as was iEMG pattern of the RF. We studied a range of strategies, with a particular interest in a strategy during which P_{tot} could not be maintained despite increasing effort.

In SUB, where Ptot increased towards the end of the trial, we found both iEMG and Ptot to increase in all muscles. This is in accordance with earlier studies. However, iEMG of the VL and BF muscles showed a significant increase towards the end of the trial in all conditions, irrespective of Ptot profile. RF activity was independent of Ptot. This leads us to the main finding of our study: a decrease in Ptot was accompanied by constant or increasing iEMG activity. This is contrary to one of the central tenants of the central governor hypothesis which suggests that decreases in Ptot occur secondary to down-regulation of iEMG, potentially to prevent the development of unreasonable disturbances of homeostasis within the muscle. Although subjects experienced subjective fatigue in all trials, as shown by the high RPE and BLC concentration at the end of the trial, no evidence was seen of a central downregulation of muscle recruitment with decreasing Ptot. On the basis of these findings, centrally mediated downregulation of neural drive does not necessarily accompany fatigue in middle-distance time trial exercise. Peripheral fatigue, as shown by the decreasing Ptot in the presence of an unchanged or increasing central neural drive and the high occurring BLC, may be a better explanation for the cause of fatigue.

In their recently published papers, Noakes et al. and Lambert et al. (18, 22) addressed the importance of peripheral fatigue in pacing again in a different form. They suggested that changes in peripheral physiological systems act as afferent signalers and that peripheral metabolites provide information to the central controller by way of afferent neural pathways and are therefore an integral part of the regulatory process (22).

Another finding of our study was, that in all strategies, maximal iEMG level was not higher than approximately 50% MVC for BF, 30% MVC for VL and 15% MVC for RF. St Clair Gibson et al. (24) found that 20% of MVC was attained in RF muscle. The fact that fewer fibers are recruited during cycling time trial exercise than during MVC was also interpreted in favor of the governor hypothesis, as an indication that exercise performance had to be regulated by the central nervous system to ensure that catastrophic loss of homeostasis does not occur during normal exercise (22). It has to be pointed out that in calculating iEMG at MVC, the highest mean value from over 2 to 4 s of a 5-s isometric knee-extension (RF, VL) or knee-flexion (BF) contraction was used. This value was used to normalize iEMG over a 200-m segment. Because cycling is a cyclic movement, bursts of high EMG activity repeatedly occurred, alternating with zero activity intervals. In attaining iEMG during

cycling over 200-m segments, the zero activity is also included in the calculations, whereas the value for iEMG at MVC is calculated during the burst of activity only. Accordingly, iEMG during cycling will therefore not reach the maximal MVC values. Furthermore, iEMG at MVC is attained during isometric exercise. Maximal iEMG during cycling in a particular muscle might differ from maximal iEMG during isometric exercise. Hunter et al. (14) showed that iEMG for MVC was significantly greater than iEMG during a dynamic single maximal revolution of a cycle pedal, which reaches a value of about 75% of MVC. This might be explained by the fact that muscle coordination and timing are very important in dynamic tasks (29). For a dynamic movement like cycling, coordination is therefore important. It might just not be favorable to recruit all possible fibers maximally and for the same duration as during the MVC. Therefore, conclusions about the causes of fatigue based on comparing maximal iEMG during dynamic exercise with iEMG attained during isometric MVC, have to be interpreted with caution.

We were also interested in the contribution of aerobic and anaerobic energy systems to total P_{tot}, because pacing strategy refers to the variation of speed over the race by regulating the rate of energy expenditure (4) and thereby the pattern of Ptot. The different pacing strategies performed in this study were defined by Ptot pattern. In the SUB trial, Ptot increased significantly from the first half of the trial to the second half; in the EVEN trial, Ptot remained constant, and in the SUPRA trial, Ptot decreased significantly. Ptot was subdivided in aerobic and anaerobic power. For Paer, a main effect was found when comparing the first and second interval of the trial, which showed that aerobic contribution increased towards the end of the race, independent of strategy. This seems to indicate that the aerobic energy contribution in supramaximal exercise is not a defining variable of pacing strategy. No matter what strategy is chosen, even if Ptot decreases significantly, Paer increases significantly. Also, a main effect for HR was found, which showed that HR increased significantly in all strategies. Because athletes are operating close to $\dot{V}O_{2max}$ velocity in time trial exercise, they do not seem able to vary aerobic energy contribution much. Pan on the other hand seems to be the more important variable in pacing. Pan increases significantly from the first to last 2000 m in SUB, as does Ptot. In SUPRA, Pan decreases significantly, as does Ptot. In the EVEN trial, Pan and Ptot do not differ significantly between intervals. Although not significant, the somewhat higher value in the first interval for Pan in the EVEN trial can be expected, because to establish a constant power output profile, the increasing Paer has to be compensated. Additionally, in the first part of the race (first 500 m), anaerobic energy contribution is relatively high because anaerobic energy is immediately available, whereas aerobic energy production needs some time to increase. Noticing the comparable profiles of Pan and Ptot, it seems that the different pacing strategies, as already suggested by De Koning et al. (4), are mainly regulated by variation in anaerobic energy expenditure. In accordance with this finding, a terminal acceleration in self-paced time trials is often found, accompanied by an increase in anaerobic energy production (9, 10). This increase in P_{tot} at the end of exercise has also been found by Ansley et al. (1) in self-paced time trials, accompanied by an increase in iEMG. Because the amount of anaerobic energy that can be generated during a 4000-m time trial is limited (19), it is important to distribute this amount optimally over the race.

No significant differences were found for gross efficiency measured before each time trial per strategy. In calculating aerobic and anaerobic power, it is assumed that gross efficiency measured at submaximal intensities does not change towards higher intensities. For the profile of energy distribution, we do not expect this assumption to have big impact on the outcome. It should be kept in mind that gross efficiency might have a slightly differing value at higher intensities, which might result in an underestimation of aerobic power and an overestimation of anaerobic power.

Also, no significant differences were found between final times per strategy. Although not significant, it can be seen that the SUPRA strategy has the fastest final time and SUB the slowest. Additionally, for P_{tot} , a main effect of strategy was found. A pairwise comparison showed that P_{tot} in SUB was lower than in SUPRA. This could mean that in an aggressive strategy (SUPRA), subjects are able to win so much in the first interval of the race, that it is impossible to make up for that in the second interval of a conservative (SUB) strategy. For P_{tot} , strategy thus affects energy distribution, but can also have an influence on the total amount of energy that can be generated during the race. For BLC, a pairwise comparison clearly revealed that the greatest although not significant difference can be found between SUB and SUPRA. This, combined with an absence of a decrease in P_{tot} in SUB, indicates that in SUB, peripheral mechanisms interfere less with performance than in SUPRA, where they even evoke a decrease in P_{tot} . An effect of pacing strategy was also found on P_{aer} , EMG (VL, BF), RPM, and RPE (mean values over the whole trial) and pacing strategy influences these variables. It is therefore very important to gain more insight into fatigue.

Our findings suggest that reaching unsustainable metabolic disturbances during time trial performance might be prevented by peripheral regulation. This does not mean that central factors are not playing a role in pacing. Pacing is about regulating speed by varying energy expenditure and thereby Ptot pattern (4). Successful athletes are skilled in this monitoring process and prevent a significant slowdown late in the event by regulation of their early pace, e.g. Ptot (5). Pacing is about managing energy expenditure with its resulting peripheral fatigue so that no factor will be limiting before the end of the trial. Otherwise, performance will be suboptimal. Although homeostasis is not endangered during a time trial, as can also be seen in the variety of possible pacing strategies, energy must be distributed during the race to attain optimal performance. This is regulated centrally, by regulating muscle activation, as is also mentioned by Rauch et al. (23). Tucker et al. (27) showed, that during self-paced time trials in the heat, reduced Ptot and EMG activity compared with normal conditions occurred before any abnormal increase in rectal temperature was shown. The subjects, apparently anticipating these circumstances, avoided reaching limiting factors and distributed energy resources optimally. Rauch et al. (23) found, that

carbohydrate-loaded subjects had a higher mean power output during exercise than subjects who were not carbohydrate-loaded. It was suggested that the brain was able to anticipate the rate of muscle glycogen concentration according to an internal metabolic calculation based on each individual's own critical level end-point muscle glycogen ('glycostat'). Carbohydrate-loaded subjects had more energy to distribute and thus could afford an increase in skeletal muscle recruitment compared with those who were not.

As mentioned, we found that anaerobic energy resources seem to be of importance in regulating pacing strategy. This corresponds to Foster et al. (9), whose data showed that athletes monitor some aspect of anaerobic energy expenditure during high intensity exercise so that near zero values are not reached until the finish line is approached. When instructed to finish as fast as possible, athletes apparently 'negotiate' regarding an estimation of task remaining, momentary P_{tot} and remaining anaerobic reserves (10). This monitoring process to optimize distribution of energetic resources is consistent with the governor hypothesis, but not reaching near-zero values of anaerobic energy expenditure can also be seen as support for a metabolite accumulation or phosphagen-depletion model of fatigue (9). Possible mechanisms in regulating pacing strategy should be sought in the anaerobic energy production system.

Lastly, the fact that pacing is possible during a time trial makes riding a time trial different from exercise till exhaustion, in which pacing is not an option, because a fixed intensity has to be attained. In time trial exercise, limiting factors can be avoided or postponed by proper pacing. Central fatigue does not necessarily occur. However, an athlete might fail to select a proper pacing strategy, for example in unexpected circumstances. In such a case, peripheral fatigue is not well managed and down-regulation of EMG as a protection mechanism may reasonably be expected.

Conclusion

In time trials with different first half strategies imposed, no reduction in iEMG occurs with decreasing power output. Centrally mediated downregulation of mechanical power output does not necessarily accompany fatigue in middle-distance time trial exercise. However, to maximize performance by varying energy expenditure, central neural control is suggested. Specifically anaerobic energy resources seem to be important in regulating pacing strategy.

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CHAPTER 3 EFFECT OF PACING STRATEGY ON ENERGY EXPENDITURE DURING A 1500-M CYCLING TIME TRIAL

Hettinga FJ, De Koning JJ, Meijer E, Teunissen L, Foster C. The effect of pacing strategy on energy expenditure during a 1500-m cycling time trial. Med. Sci. Sports Exerc. 2007; 39(12): 2212-2218.

Abstract

A critical assumption in modeling optimal pacing strategy is that the amount of anaerobic energy that can be produced during a time trial is a constant value, independent of pacing strategy. To test this assumption, the effect of manipulations of pacing strategy on anaerobic work produced during a 1500-m cycling time trial was studied. Additionally, the effect of pacing strategy on aerobic and total work was studied. Nine well-trained cyclists performed three 1500-m cycle ergometer time trials with different strategies (conservative (SUB), even-paced (EVEN) and aggressive (SUPRA)). Anaerobic work, aerobic work and total work were calculated based on VO₂, RER, gross efficiency and external power output. ANOVA showed that total anaerobic work did not differ per strategy (EVEN: 27.6 ± 1.1 kJ, SUB: 26.5 ± 2.0 kJ and SUPRA: 26.9 ± 2.1 kJ). No differences in aerobic work (EVEN: 28.3 ± 1.6 kJ, SUB: 28.0 ± 1.4 kJ, SUPRA: 27.8 ± 2.0 kJ) were evident either. Subjects were able to accomplish significantly (p < 0.05) more total work during EVEN (55.9 ± 2.2 kJ) than during SUB and SUPRA (54.4 ± 2.3 kJ and 54.8 ± 2.4 kJ, respectively). Though relevant for sports performance, the differences in total work were relatively small (~2%) considering the broad range of imposed strategies. The assumption that anaerobic work is a constant value independent of pacing strategy seems valid in the range of different strategies that are currently simulated in the energy flow models.

Introduction

Pacing strategy is becoming a critical factor in sports competition, since athletes may be approaching the species limits of performance (21). So far, several studies have been conducted exploring the importance of the pattern of energy expenditure to performance (1, 5-9, 13, 15, 23, 24). The use of energy flow models to simulate various patterns of energy expenditure and their effect on performance (5, 6, 23) is a very appealing way to study different patterns of energy expenditure. By modeling performance using energy flow models, environmental circumstances can be held constant and the quantitative effect of a wide range of different pacing strategies can be tested under the same environmental circumstances.

A critical assumption in these energy flow models is that the amount of anaerobic energy that can be produced during a time trial is a constant value, independent of pacing strategy (23). In the literature, several studies have investigated possible effects of pacing strategy on anaerobic capacity by measuring maximal accumulated oxygen deficit (MAOD) (4, 7, 11). Foster et al. (7) did not find differences in MAOD in differently paced 2000-m time trials. Gastin et al. (11) and Craig et al. (4) did not find a difference between all-out and constant intensity exercise, but they compared trials of different duration. This could have influenced their conclusions involving pacing strategy. Overall, the assumption of a constant anaerobic energy contribution seems to be supported by the literature, though it has been shown that different exercise conditions influence MAOD (4).

Recently, we have shown that the pattern of anaerobic energy distribution varies with pacing strategy (13). This raised the issue again whether anaerobic capacity is indeed a fixed amount. So far, the effect of pacing strategy on anaerobic capacity has been tested experimentally only within a small rage of strategies, mostly within the normal competitive range (7). It might be that this range was too small to detect differences in anaerobic work. Since modeling performance with energy flow models makes it possible to test the influence of a very wide range of pacing strategies on performance under the same environmental circumstances, it is important to determine whether the assumptions regarding anaerobic capacity are robust. Therefore, the main purpose of the present study was to test whether anaerobic capacity was a constant value by imposing extreme pacing strategies. These pacing strategies were imposed in a 1500-m cycling time trial of approximately 2 min, since anaerobic capacity is widely assumed to be fully used within 90 s to 2 min of exercise (17, 25).

Anaerobic energy is thought to contribute for about 50% to performance in supramaximal exercise of about 2-min duration (12). The remaining 50% is produced aerobically. Thus it is also of interest whether there is an effect of pacing strategy on aerobic work over the time trial. No effect of pacing strategy on aerobic work was found in differently paced 4000 m time trials (13), but changes in \dot{VO}_2 kinetics in response to pacing strategy were shown to occur in 500-m kayaking (1). In agreement with what was assumed in modeling studies, we hypothesized that there was no effect of pacing strategy on the anaerobic and aerobic work performed during

a 1500-m time trial. Because the sum of aerobic and anaerobic work is total work, also no changes in total work over the race were expected. This would imply that the variables defining performance are restricted to the distribution of power output and the power losses attributable to varying air resistance at different riding velocities.

Methods

Subjects

Nine well-trained male cyclists participated in this study. The physical characteristics of the subjects are presented in Table 1. Before the experiment, subjects were informed about the nature of the experiment, after which they gave written informed consent. They were requested to follow their usual diets and reduce physical activities the day before each trial. They were also instructed to refrain from food for at least 2 h before each test. The experiment was approved by the ethics committee at the VU University Amsterdam.

Experimental Design

An incremental test, two habituation 1500-m cycling time trials and three 1500-m cycling time trials with different strategies were performed. All time trials were commenced after completing a 10-min warm-up protocol (Fig. 1). During the fifth minute of the warm-up, gross mechanical efficiency was determined, as described below. After the warm-up the subjects rested for 3 min, then the time trial was started.

Table 1: Subject characteristics: age, height, body mass, maximal oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained ($P\dot{VO}_{2max}$).

Mean value ± SD
26.4 ± 7.0
185.6 ± 7.9
76.5 ± 8.2
4.5 ± 0.2
383.2 ± 26.6

Values are means ± SD.

Subjects were instructed to finish as fast as possible and provided with distance feedback. The time trial was followed by a 3-min recovery period and a cooling-down of 6 min of cycling at 100 W. Subjects had two or more days off after each trial and performed all trials on a custom made electronically braked laboratory cycle ergometer (FBW-MTO, Amsterdam) designed to simulate real competition. Pedaling rate, power output, virtual velocity and virtual distance were calculated based on torque, crank rotational velocity and gear ratio. The ergometer was linked to a computer which stored power output, pedaling rate and torque at 20 Hz. Handlebars

and saddle height were adjusted to the preferences of the subject and were kept constant for each trial.

Incremental test and habituation trials

All subjects first completed a maximal incremental test that started at 180 W with a pedaling frequency of 80-100 rpm. Before commencing the incremental test, they warmed up for 2 min at 100 W and 3 min at 150 W. During the test, power output was increased by 20 W every minute, until the subject could no longer maintain the required power output or until the pedal frequency dropped below 80 rpm. Subsequently, subjects were instructed to cycle on different days two 1500-m habituation even-paced time trials at maximal intensity, to ensure that subjects were fully habituated to the task of an even-paced time trial.

Experimental trials

The first 1500-m experimental time trial was performed with an even-paced strategy (EVEN), based on the second habituation even-paced time trial. Subjects were instructed to finish in the shortest possible time and to keep their speed as even as possible. The EVEN paced strategy was chosen because Foster et al. (7) found that an evenly paced strategy resulted in optimal performance on a 2000-m (3 min) cycling time trial. Subsequently, subjects performed two randomly ordered 1500-m trials with experimentally imposed extreme pacing strategies, to cover a wide range of pacing strategies. Because pacing strategy refers to the variation in velocity over the race by regulating the rate of energy expenditure (5), power output was used to impose and evaluate the pacing strategies. The first 750 m of the imposed strategy trials was cycled at a velocity requiring either 95% (SUB) or 115% (SUPRA) of the mean power output of the even-paced trial. In all trials, subjects were instructed to start rapidly, to get to mean required velocity as soon as possible. Mean power output for these trials was calculated without the initial peak in power output (over approximately the first 15 to 20 s). For the second 750 m of both trials, subjects were instructed to finish in the shortest time possible, as in competition. Velocity and achieved distance were continuously displayed to help the subjects pace during the time trials. All tests were done on separate days and at about the same time of the day in a temperature and relative humidity controlled (18°C, 50% RH) environment. During the imposed strategy trials, respiratory gas exchange was measured breathby-breath using open circuit spirometry (Oxycon alpha, Mijnhardt, the Netherlands), a valid and reliable method to determine $\dot{V}O_2$, $\dot{V}CO_2$ and V_E (2, 20). The gas analyzer was calibrated before each test using a 3-I syringe (Jaeger, Germany), room air and a standard gas mixture. VO2, VCO2, VE and RER were continually monitored. Heart rate (HR) was recorded every 5 s using radio telemetry (Polar Electro, Finland). Blood lactate concentration (BLC) was measured three minutes after finishing each time trial from a fingertip blood sample and analyzed using dry chemistry (Lactate Pro, Arkray Inc, Kyoto, Japan). Power output (Ptot) was measured as explained with a sample frequency of 20 Hz.

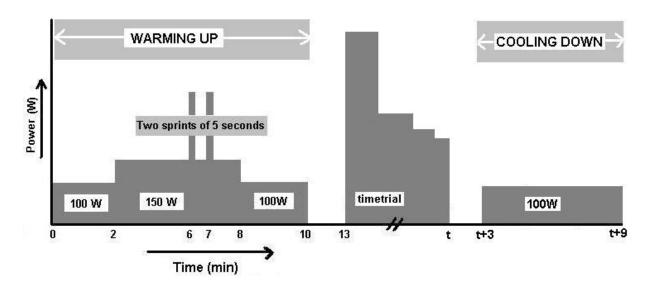


Fig. 1: Protocol of warm-up, 1500-m time trial and cooling-down.

Metabolic aerobic power (P_{met}) was calculated by multiplying oxygen consumption with the oxygen equivalent: P_{met} (W) = $\dot{V}O_2$ ($I\cdot min^{-1}$) · ((4940 · RER + 16040)/60) (10). We assumed that respiratory exchange ratios (RER) in excess of 1.00 were attributable to buffering of lactate by bicarbonate and they were thus treated as if they equaled 1.00. Gross mechanical efficiency was determined before each time trial at a power output of 150 W by dividing P_{tot} by P_{met} (3). For each subject, gross efficiency values were averaged. Aerobic mechanical power output (P_{aer}) during the time trial was calculated by multiplying P_{met} by gross efficiency. Anaerobic mechanical power output (P_{an}) during the time trial was calculated by subtracting the calculated mechanical aerobic power output from the total measured mechanical power output ($P_{an} = P_{tot} - P_{aer}$).

Statistics

The effect of time trial strategy was determined using a two-way repeated-measures analysis of variance. Pairwise comparisons were made with a Bonferroni adjustment. Statistical significance was accepted when p < 0.05.

Results

Pacing strategies

All subjects completed the 1500-m trials according to the prescribed strategy. Mean gross efficiency was $18.3 \pm 0.9\%$. In figure 2, the mean velocity profiles of the three different pacing strategies were shown. The patterns of P_{tot} , P_{an} and P_{aer} per 100 m during each trial were presented in figure 3. In figure 4, P_{tot} , P_{an} and P_{aer} of both SUPRA and SUB strategies were expressed per 100 m relative to the corresponding EVEN paced 100 m.

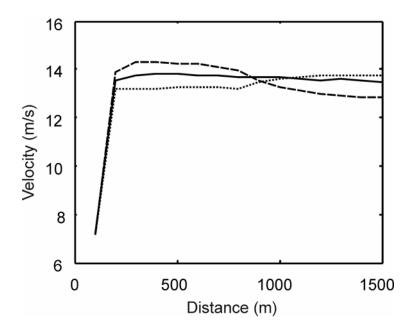


Fig. 2: Mean velocity per 100 m for SUB (dotted line), EVEN (solid line) and SUPRA (dashed line).

Mean P_{tot} , P_{an} and P_{aer} over the first (100-700 m) part of the time trial were compared per pacing strategy (Table 2). Main effects of pacing strategy on mean P_{an} and mean P_{tot} were shown. Mean P_{aer} did not differ per pacing strategy. During the second (800-1500 m) part of the time trial, main effects of pacing strategy on mean P_{an} and mean P_{tot} were shown. Mean P_{aer} during the second part did not differ per pacing strategy. In table 3, percentages of SUB and SUPRA compared to EVEN are shown per part. The start segment (0-100 m) and the middle segment (700-800 m) were not incorporated in these calculations to make sure that the start and transition phases between strategies did not influence the comparisons.

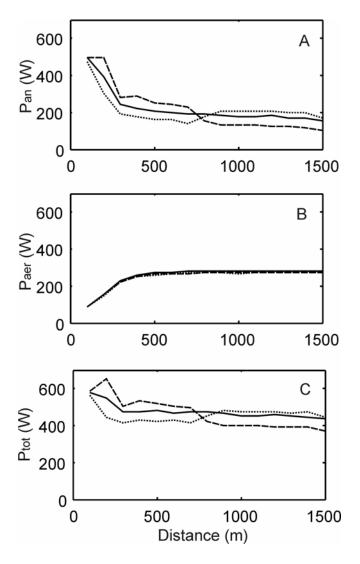


Fig. 3 (A, B, C): Mean anaerobic power (P_{an}), aerobic power (P_{aer}) and total power (P_{tot}) per 100 m for SUB (dotted line), EVEN (solid line) and SUPRA (dashed line).

Total, anaerobic and aerobic work during the race and final time

No significant differences between pacing strategies on anaerobic and aerobic work over the time trials were found (Table 4). For total work during the time trials, a main effect was found. Total work was highest for EVEN compared with SUB and SUPRA (Table 4). For final time, a main effect of pacing strategy was found. A pairwise comparison revealed that EVEN was faster compared with SUB.

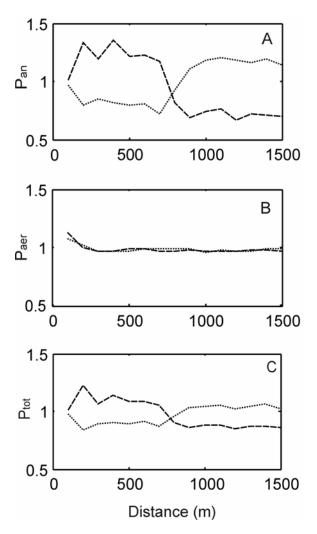


Fig. 4 (A, B, C): Mean anaerobic power (P_{an}), aerobic power (P_{aer}) and total power (P_{tot}) per 100 m for SUB (dotted line) and SUPRA (dashed line), expressed relative to EVEN.

Physiological variables: $\dot{V}O_2$, RER, V_E , HR and BLC

No significant differences in mean \dot{VO}_2 and mean \dot{VO}_2 for the first 30 s (\dot{VO}_{230}) were found between pacing strategies. For mean RER, V_E , mean HR and BLC no effect of pacing strategy was found. All mean values were shown in table 4.

Table 2: Mean anaerobic power (P_{an}), aerobic power (P_{aer}) and total power (P_{tot}) over the first (100-700 m) and second (800-1500 m) part of the time trial per pacing strategy (SUB, EVEN, SUPRA).

		SUB	EVEN	SUPRA
P _{an} (W)	First part (100-700 m)	183.2 ± 21.7 ^{ac}	232.9 ± 28.1 ^{bc}	287.5 ± 21.4 ^{ab}
	Second part (800-1500 m)	191.0 ± 22.5 ^{ac}	167.3 ± 12.3 ^{bc}	116.4 ± 22.9 ^{ab}
$\mathbf{P}_{aer}(W)$	First part (100-700 m)	229.1 ± 11.2	234.4 ± 15.5	230.2 ± 20.8
	Second part (800-1500 m)	263.8 ± 12.2	271.6 ± 12.6	263.9 ± 15.8
$P_{tot}(W)$	First part (100-700 m)	412.3 ± 20.6^{ac}	467.2 ± 33.5 ^{bc}	517.7 ± 22.4 ^{ab}
	Second part (800-1500 m)	454.9 ± 19.2^{ac}	438.9 ± 14.8 ^{bc}	380.3 ± 19.7^{ab}

Values are means \pm SD. Significant (p < 0.05) differences with EVEN are marked with an 'a'. Significant differences with SUPRA are marked with a 'c'.

Table 3: Mean anaerobic power (P_{an}), aerobic power (P_{aer}) and total power (P_{tot}) over the first (100-700 m) and second (800-1500 m) part of the time trial for SUPRA and SUB, expressed as a percentage relative to EVEN.

		First part (100-700 m)	Second part (800-1500 m)
P _{an} (%EVEN)	SUPRA	1.25 ± 0.08	0.71 ± 0.17
	SUB	0.79 ± 0.09	1.14 ± 0.13
P _{aer} (%EVEN)	SUPRA	0.98 ± 0.05	0.97 ± 0.05
	SUB	0.98 ± 0.06	0.97 ± 0.06
P _{tot} (%EVEN)	SUPRA	1.11 ± 0.05	0.87 ± 0.04
	SUB	0.88 ± 0.04	1.04 ± 0.03

Values are means ± SD.

Table 4: Time, anaerobic work (W_{an}), aerobic work (W_{aer}), total work (W_{tot}), revolutions per minute (RPM), mean oxygen consumption ($\dot{V}O_2$), mean oxygen consumption over the first 30 s ($\dot{V}O_{230}$), ventilation (V_E), blood lactate concentration (BLC) and heart rate (HR) per pacing strategy (SUB, EVEN and SUPRA).

	SUB	EVEN	SUPRA
Time (s)	117.9 ± 2.0 ^a	116.4 ± 1.7 ^b	117.1 ± 2.1
$\mathbf{W}_{an}\left(kJ\right)$	26.5 ± 2.0	27.6 ± 1.1	26.9 ± 2.1
$\mathbf{W}_{aer}\left(kJ\right)$	28.0 ± 1.4	28.3 ± 1.6	27.8 ± 2.0
\mathbf{W}_{tot} (kJ)	54.4 ± 2.3^{a}	55.9 ± 2.2^{bc}	54.8 ± 2.4^{a}
$\dot{\mathbf{VO}}_{2}$ (l·min ⁻¹)	3.73 ± 0.21	3.82 ± 0.21	3.73 ± 0.21
$\dot{\mathbf{VO}}_{230}$ (l·min ⁻¹)	2.24 ± 0.23	2.30 ± 0.24	2.29 ± 0.26
RER	1.30 ± 0.07	1.29 ± 0.13	1.32 ± 0.07
V _E (I·min ⁻¹)	141.9 ± 15.3	146.8 ± 15.7	154.2 ± 22.6
BLC (mmol·l ⁻¹)	14.1± 2.1	15.6 ± 2.3	15.7 ± 2.2
HR (bpm)	169 ± 16	172 ± 14	171± 16

Values are means ± SD. Significant (p < 0.05) differences with EVEN are marked with an 'a'. Significant differences with SUB are marked with a 'b'. Significant differences with SUPRA are marked with a 'c'.

Discussion

To study the effect of pacing strategy on energy expenditure, two extreme profiles of total energy distribution (SUB and SUPRA) were compared to a relatively even-paced pattern of total energy distribution (EVEN). In a self-paced 1500-m time trial, athletes regularly perform a rapid first 30-60 s, followed by a gradual decline in power output to a rather constant value (6, 8, 9). Though a shorter rapid start (of about 15 s) was performed in the present EVEN protocol, our EVEN strategy was comparable with pacing strategies performed in real competitive settings.

Based on the EVEN strategy, two extremely different profiles of (specifically anaerobic) power output were imposed by constraining the first half of the time trial, as was done by Foster et al. (7). They studied pacing strategies with a first half ranging from 48 to 55% of best time, which is within the range used in competition. The present study intentionally covered an even wider range of pacing strategies. Subjects were instructed to cycle the first half of the time trial at a velocity resulting in a mean power output of 95% relative to EVEN and of 115% relative to EVEN. Actual mean power outputs over the first half of the time trial were respectively 88% relative to EVEN and 111% relative to EVEN (Table 4, Fig. 4C). The different pacing strategies were clearly visible in velocity and total power distribution (Fig. 2, Fig. 3C). All pacing strategies were cycled at supra-maximal intensities, because mean values for RER were > 1.00.

Variations in total power distribution were shown to be mainly caused by changes in the pattern of anaerobic energy distribution (13). The present study also showed that anaerobic power output profiles clearly varied with pacing strategy. (Fig. 3A; Table 2). Clearly opposite patterns of P_{an} were found for SUPRA and SUB, with first halfs of 125% relative to EVEN and 80% relative to EVEN, respectively (Table 4, Fig. 4A). These large variations in pattern of anaerobic energy distribution did not lead to meaningful differences in the total amount of anaerobic work generated over the time trial. This corresponds with the findings of Foster et al. (7), Gastin et al. (11) and Craig et al. (4), who did not find any differences in MAOD for differently paced exercise trials. The assumption that anaerobic work is a constant value independent of pacing strategy seems to be a valid assumption in the range of different strategies that are currently simulated with the energy flow models (5).

In contrast with the findings for the patterns of anaerobic energy distribution, the patterns of aerobic energy distribution were remarkably similar (Fig. 3B). Neither the first nor the second part of the race differed per strategy, and mean P_{aer} for both parts was about 97-98% relative to EVEN (Table 4, Fig. 4B). The pattern of aerobic energy contribution was thus not influenced by pacing strategy, which is in agreement with the findings of Hettinga et al. (13). Additionally, no differences in the total amount of aerobic work generated over the race were found. Though it was found that VO_2 kinetics were shown to speed up by a fast 10-15 s start (1), in the present study starting procedure did not influence VO_2 kinetics. Subjects were instructed to accelerate the first 10-15 s maximally towards the intended velocity in all protocols and no differences in mean VO_2 for the first 30 s of exercise were found.

As hypothesized, it was possible for an athlete to cycle two completely different pacing strategies at supra-maximal intensity, resulting in no changes in anaerobic and aerobic work or in any of the mean values of the measured physiological variables (HR, V_E, VO₂ and BLC). Total work on the other hand, which is the summation of anaerobic and aerobic work, did differ per strategy in favor of EVEN compared to SUB and SUPRA. Apparently, slight non-significant differences in anaerobic and aerobic work add up to a significant difference in total work. It seemed that the difference in total work between SUB and EVEN was mainly caused by the impossibility of increasing Ptot enough during the second part of the trial to make up for the lower Ptot in the first part. During the second part of SUB, Ptot was only 4% higher than EVEN while it was 12% lower than EVEN in the first part. Since Paer hardly varied, Pan seemed to be the limiting variable here and the 'already performed exercise' in the first part seemed to set a limit for possible use of energy sources in the second part. As suggested in literature (8), athletes monitor some aspect of anaerobic energy expenditure so that near zero values are not reached during a time trial. This monitoring process was suggested to be based on task remaining, remaining anaerobic reserves and momentary power output (9). The 'already performed exercise' seems to be an additional variable that is used in this monitoring process involved in pacing strategy. Peripheral changes resulting from the 'already performed exercise' seem to be integrated in the monitoring process. In relation to this, St Clair Gibson et al. (22) suggested that, though initial pace is set at the beginning of exercise, continuous adjustments in power output are made, that are

associated with information processing between the brain and peripheral physiological systems.

A physiological explanation for the impossibility to increase P_{tot} above a certain level might be found in the fact that [H⁺] was suggested to counteract the depressing effects of elevated [K⁺] on force production (19). Halfway SUB, [H⁺] was expected to be relatively low compared to the other strategies, leading to a relatively lower counteractive effect and thereby a possible lower maximal force production over the second part of the race. On the other hand, maximal muscle lactate concentration was also found to correlate to perception of exhaustion (16) and a fast start as performed in SUPRA was presumably accompanied with larger disturbances in homeostasis. A too rapid acceleration in the first part of the race was suggested to cause premature fatigue due to too large disturbances in muscle pH (24). In the present study, no effect of pacing strategy on mean blood lactate concentration was found, but values were only measured after completing the time trial and not halfway during the trial. Further, no effect of pacing strategy over the entire time trial was found on mean HR and mean V_E, so cardiovascular stress and stress on ventilation did not seem to be influenced by pacing strategy either and did not seem to be causing differences in total work.

Another possibility to explain the differences in total work per strategy could be that these differences were caused by changes in gross efficiency over the race. Mean gross efficiency was $18.34 \pm 0.85\%$. This corresponded with values found in literature, varying between 17 and 22% in non-professional cyclists (3, 8, 14, 18). Gross efficiency was measured at sub-maximal intensities and was assumed to be constant during the time trial. A possible non-linear increase in gross efficiency could have occurred towards the higher intensities, since basal metabolism is relatively smaller compared to lower intensities (14, 18). It might be that this non-linear increase affected SUB and SUPRA differently than EVEN because SUB and SUPRA were cycled at different profiles compared to EVEN. Based on the literature (18) and the fact that the change in total work was relatively small, changes in gross efficiency were also assumed to be small.

Because total work differed per pacing strategy, the conclusion that anaerobic work was not influenced by pacing strategy might have to be interpreted with caution. The difference in P_{tot} was relatively small (~2%) considering the wide range of imposed pacing strategies and the fact that anaerobic and aerobic work separately were not affected by pacing strategy. In terms of sports performance on the other hand, 2% is a large difference. Our EVEN strategy was the most favorable pacing strategy regarding energy production and resulted in a 1.5 s faster final time in favour of EVEN compared to SUB. Energy flow models (5) have suggested that adopting an un-even fast start velocity profile would lead to optimal performance in supra-maximal exercise of relatively short duration, mainly because of the efficient use of the available energy resources. Theoretically, optimal pacing strategy must thus be found towards our SUPRA strategy. The present study showed that in addition to using the available energy resources efficiently, the total amount of energy that can be generated during

the race was influenced by pacing strategy, leading to a slight shift of optimal performance on a 1500-m time trial towards a more even-paced strategy. This is confirmed by final times in the present study, being fastest for EVEN though not significantly differing from SUPRA.

Conclusion

The main finding of the present study was that even when extreme patterns of distributing (anaerobic) power output were imposed, total anaerobic and aerobic work did not differ per strategy. Total work differed in favor of EVEN compared to both other strategies. Though relevant for sports performance, differences were relatively small (~2%). The assumption that anaerobic work is a constant value independent of pacing strategy seems to be a valid assumption in the range of different strategies that are currently simulated in the energy flow models.

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CHAPTER 4

THE EFFECT OF TIME TRIAL DISTANCE ON ANAEROBIC WORK DURING CYCLING

De Koning JJ, Hettinga FJ, Goede SL, Groen W, Foster C. The effect of time trial distance on anaerobic work during cycling. (under revision).

Abstract

Previous studies have suggested that the total amount of anaerobic work that can be performed during exhausting work bouts is constant for work tasks of 2-5 min duration. These studies were based on constant intensity exercise to fatigue, rather than on time trials in which the athletes are free to regulate their momentary power output. The aim of the present study was to quantify and compare the contribution of anaerobic work in 750-m, 1500-m, 2500-m and 4000-m cycling time-trials. Ten welltrained, task habituated, male athletes performed four randomly ordered cycle time trials. The contribution of aerobic and anaerobic energy systems was calculated from measurements of mechanical power, gross efficiency and oxygen uptake. The time trials of 750 m, 1500 m, 2500 m and 4000 m were completed in 57.2 \pm 1.7 s, 116.3 \pm 1.7 s, 198.5 ± 4.9 s and 325.3 ± 9.4 s, respectively. The total anaerobic work accomplished in the four time trial distances was 22.70 ± 2.70 kJ, 26.95 ± 2.38 kJ, 29.75 ± 4.68 kJ and 33.86 ± 7.01 kJ, respectively. Anaerobic work in the 750 m was significantly (p < 0.05) less than in all other trial distances. The mean values in the two middle distances (1500 m and 2500 m) did not differ significantly and the value in the 4000-m trials was significantly greater than all other trial distances. Based on the results of the present study it can be concluded that:

- 1) The absolute amount of anaerobic work is dependent on time trial distance, with a general trend towards larger amounts with increasing distance.
- 2) The hypothesis that the anaerobic capacity in supramaximal exercise is a well defined entity is not supported.

Introduction

Adenosine triphosphate (ATP) broken down during exercise is continuously resynthesized by both aerobic and anaerobic processes. Aerobic generation of ATP is dominant when exercise intensity is relatively low. At high relative exercise intensities anaerobic ATP generation becomes relatively more important. Anaerobically produced ATP can be attributed either to the breakdown of phosphocreatine or to muscle glycogenolysis. It has been hypothesized that the anaerobic capacity is a limited entity (13, 14, 15, 18). Medbø et al. (13, 14, 15) proposed a method to provide an estimate of the anaerobic capacity by measuring the accumulated oxygen deficit during exercise. In this method the amount of anaerobically attributable energy produced was calculated by subtracting the aerobically attributable energy from the total energy demand, determined by extrapolation of the relationship between exercise intensity and oxygen uptake (2, 13, 14, 15). This method of quantifying the anaerobic capacity is conceptually similar to the method recently used in our laboratory (3, 4, 5, 9, 10).

Although it is known that there are individual differences in anaerobic capacity between sprint trained athletes, endurance trained athletes and non-athletes (1, 2, 7, 8, 17) there is still debate about the time needed to fully use the anaerobic capacity during supra-maximal exercise. Estimates of the time to depletion range from 60 to 90 s during all-out tests (8, 23) to 300 s during constant intensity exercise (2). However, efforts ranging from 120-300 s are commonly thought to be sufficient to fully exhaust the anaerobic capacity (13, 14, 15, 24). In an experiment with trained cyclists, Foster et al. (5) found that the amount of anaerobically attributable energy produced increased slightly with increasing distance, but the differences were not significant. However there was a trend toward an increase in total anaerobic work for trials with durations of more than 120 s. More recently, De Koning et al. (3) found significant increases in anaerobic energy contribution during simulated competitions of longer than 120 s. The results of Foster et al. (5) and De Koning et al. (3) are contrary to the concept that anaerobic capacity is a well defined entity which can be fully utilized within 120 s. To gain more insight in this matter, the present study evaluated anaerobic energy expenditure during time trials with distances ranging from 750 to 4000 m (60-330 s). The longest distance is 1000 m (~90 s) longer than the longest distance in the study of Foster et al. (5). It was hypothesized that there would be an increase in the total anaerobic energy contribution with increasing time trial distance.

Methods

The protocol was approved by the ethics committee of the Faculty of Human Movement Sciences at the VU University Amsterdam and the subjects gave written informed consent. The subjects were regional level male cyclists and speed skaters (n=10). Descriptive data of the subjects are presented in Table 1. Subjects were instructed to follow their usual diets and to reduce their training the day before each trial, just as they normally would before competition.

Table 1: Subject characteristics: age, height, body mass, maximal oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained (\dot{PVO}_{2max}).

	Mean value ± SD
Age (yr)	26.4 ± 7.0
Height (cm)	185.6 ± 7.9
Body mass (kg)	76.5 ± 8.2
$\dot{\mathbf{VO}}_{2max}$ (I·min ⁻¹)	4.51 ± 0.22
PVO _{2max} (W)	383 ± 26

Values are means ± SD.

The subjects reported to the laboratory on six separate days. The first day was used to obtain anthropometric data, perform a VO_{2max} -test and habituate the subjects to the experimental set-up. The test protocol for the incremental VO_{2max} test consisted of a 5-min warm-up at 150 W. After the warm-up the exercise intensity was set to 180 W and increased by 20 W per minute. VO_2 and mechanical power were measured and VO_2 was averaged over the last 30 s of every stage. The VO_{2max} was defined as the highest 30 s VO_2 attained. PVO_{2max} was defined as the highest power output measured over a 60-s period during the test. On the second day, both 1500-m and 4000-m habituation time trials were performed. The subjects rested for ~30 min. between trials. Before the habituation time trials the only instruction given to the subjects was to complete the trial as quickly as possible, as in competition. On the remaining four days, one time trial was performed each day, with 48-96 h between trials. During all trials, VO_2 was measured using calibrated open circuit spirometry (Oxycon Alpha, Mijnhardt, The Netherlands).

On the four experimental days, the subjects performed randomly ordered time trials of 750 m, 1500 m, 2500 m and 4000 m. Before the time trials, the subjects followed a standard warm-up protocol. The mechanical power output (Ptot), pedaling rate and torque were measured at 20 Hz on a calibrated custom made cycle ergometer (MTO-VU, Amsterdam). The ergometer was designed to simulate riding on a conventional bicycle, including inertia and drag forces. Respiratory metabolism was measured as in the practice trials. Heart rate was measured using radiotelemetry (Polar, Finland) at 5-s intervals. Post exercise blood lactate concentration ([La]) was measured in capillary blood (Lactate Pro, Arkray Inc, Japan) 4 min after the time trial during a standard cool-down protocol of cycling at 100 W. A schematic representation of the test protocol is given in figure 1.

Gross mechanical efficiency (GE) was determined by dividing the external power output by the metabolic power output measured from minute 4 to 5 of the warm-up, at a power output of 150 W. Metabolic energy yield of O_2 (P_{met}) was calculated according to Garby and Astrup (6):

$$P_{\text{met}} = 4.94 \cdot \text{RER} + 16.04 \text{ (kJ)}$$
 [1]

In which RER is the respiratory exchange ratio. Throughout this study, respiratory exchange ratios in excess of 1.00 were assumed to be attributable to non-metabolic CO₂ production caused by buffering and were therefore set to 1.00 in computing metabolic work (5). GE was then calculated using equation 2:

$$GE = (P_{tot}/P_{met})$$
 [2]

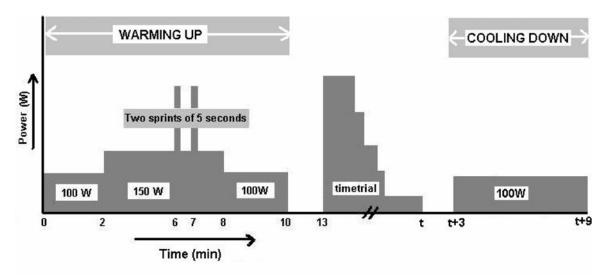


Fig. 1: Schematic representation of the test protocol used during the time trials including the warm-up and the cooling-down period. Note that the values on the Y axis are arbitrary and that 't' stands for the time of cessation of the time trial.

The aerobically produced energy during the time trials was calculated by multiplying the $\dot{V}O_2$ by the metabolic energy yield per liter O_2 (6) and by the GE. The amount of mechanical energy attributable to anaerobic processes was then calculated by subtracting the aerobically produced mechanical energy from the total mechanical energy produced. This technique, which has been used both in work from our laboratory (3, 4, 5, 9, 10) and elsewhere (18), requires that GE was assumed to remain constant during the time trials.

Statistics

The effect of time trial distance on total anaerobic work was determined using a two-way repeated-measures analysis of variance. In the case of a significant F value, pairwise comparisons were made using the Tukey test. The significance level was set at p < 0.05.

Results

Final time, mean velocity and post-exercise lactate concentration are presented in table 2. Post-exercise lactate concentration was only significantly different in the 1500-m trial compared to the 4000-m trial. The average GE of the subjects was 18.3 \pm 0.8%. The work attributable to anaerobic and aerobic processes as well as the total work accomplished during the time trials is presented in table 3. The amount of anaerobic work in the 750-m trial was significantly less than during all the other distances. In the 4000-m trial more anaerobic work was accomplished than in the 1500-m and 2500-m trials. The total, aerobically attributable and anaerobically attributable power output during each time trial is presented in figure 2. Subjects accomplished significantly more anaerobic work than on the other distances during the first part of the 750 m.

Table 2: Final time, mean velocity (v_{mean}) and blood lactate concentration ([LA]).

	Final time (s)	V _{mean} (m·s ⁻¹)	[LA] (mmol·l ⁻¹)
750 m	57.2 ± 1.7	13.1 ± 0.4	15.1 ± 2.4
1500 m	116.3 ± 1.7	12.9 ± 0.2	16.0 ± 2.4^{a}
2500 m	198.5 ± 4.9	12.6 ± 0.3	14.4 ± 1.8
4000 m	325.3 ± 9.4	12.3 ± 0.4	13.9 ± 1.9 ^b

Values are means \pm SD. Differences with 4000 m are marked with an 'a'. Differences with 1500 m are marked with a 'b' (p < 0.05).

This pattern of energy expenditure is comparable to what in the literature is called an 'all-out' pacing strategy. When the anaerobic work (normalized to the value observed during the 1500-m time trial) is plotted as a function of distance (Fig. 3A) or time (Fig. 3B) and compared to a previous study from our laboratory (5), it is evident that there was a progressive increase in the anaerobically attributable energy across the distances represented in the experimental design.

Table 3: Total, aerobic and anaerobic work performed during the time trials.

	Total work (kJ)	Aerobic work (kJ)	Anaerobic work (kJ)
750 m	34.32 ± 2.38^{bcd}	11.62 ± 0.93 ^{bcd}	22.70 ± 2.70^{bcd}
1500 m	55.25 ± 2.88^{acd}	28.31 ± 1.56 acd	26.95 ± 2.38 ad
2500 m	80.77 ± 5.51^{abd}	51.02 ± 2.33 abd	29.75 ± 4.68^{ad}
4000 m	121.60 ± 6.75^{abc}	87.74 ± 3.77^{abc}	33.86 ± 7.01^{abc}

Values are means \pm SD. Differences with 750 m are marked with an 'a'. Differences with 1500 m are marked with a 'b'. Differences with 2500 m are marked with a 'c'. Differences with 4000 m are marked with a 'd' (p < 0.05).

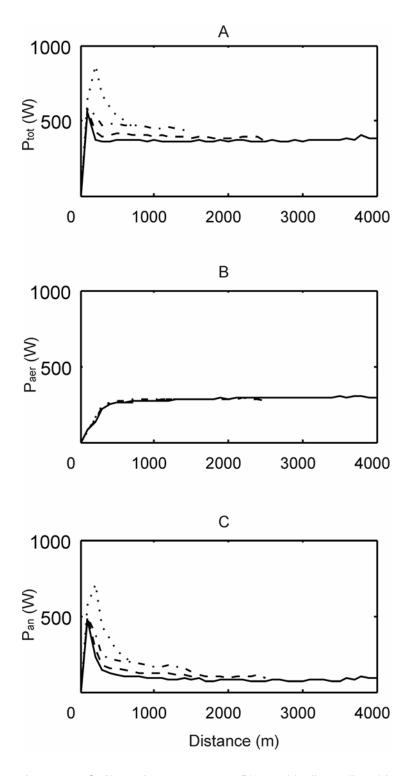


Fig. 2: Temporal pattern of: A) total power output, B) aerobically attributable power output, and C) anaerobically attributable power output in the 750-m (dotted), 1500-m (dashdot), 2500-m (dashed) and 4000-m (solid) time trials.

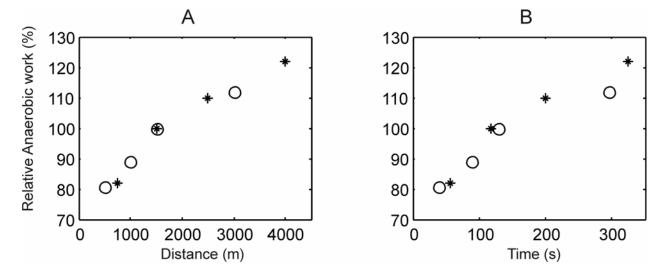


Fig. 3: Accumulated anaerobic work of the present study (*) and data from the related study of Foster et al. (5) (o) are shown in relation to A) distance, and B) time requirement of the time trials. For comparative purposes, the anaerobic work during a 1500-m time trial (~120 s) is set to 100%.

Discussion

The main purpose of this study was to determine the influence of time trial distance on total anaerobic work in trained athletes. It was found that the total anaerobic work accomplished during the trials differed significantly across distances, with the exception of the 1500 m compared with the 2500 m. There was a clear pattern of an increase in anaerobic work with an increase in time trial distance, even for the trials with duration of more than two minutes. This finding is not consistent with the concept of an individual maximal anaerobic capacity introduced by Medbø et al. (13, 14, 15). Other studies from our laboratory have anticipated this finding. De Koning et al. (3) have shown similar results in speed skaters, who were able to generate 63% more anaerobic work in a 5000-m trial (~7 min) versus a 1500-m trial (~2 min). Foster et al. (5) also found a trend towards an increase in anaerobic work with increasing cycle time trial distance, also observed by Craig et al. (2), although not statistically significant. For exercise shorter than 2 min of constant-load supra-maximal cycling, exercise time is simply not sufficient to fully attain anaerobic capacity (15). Conform the present results comparing 750 m with the longer distances, it was shown that athletes with a high anaerobic capacity were not able to recruit this capacity within 30 s or 45 s, despite all-out testing that produced exhaustion (1). Additionally, it looks like sprint cyclists can fully express their anaerobic capacity within an event specific 70-s all-out test (2), where track endurance cyclists seem to have the highest anaerobic capacity during an event specific test of 300 s. It seems that time trial length and type of athlete (sprint vs. endurance) are of influence on anaerobic work that can be generated during the race.

The longer time available to liberate energy explains the increase in MAOD with increasing intensity below 2-min (1500 m) duration. But in the longer time trials such

as the 4000 m compared to 1500 m, for example, where enough time is available to liberate the available energy, MAOD still increases with intensity. An explanation for this could lie in the fact, that during time trial exercise, an athlete is able to choose the distribution of power output over the race. Previous studies of MAOD have focused on either open loop exercise models where a fixed power output must be maintained for as long as possible or on fixed time exercise bouts, where the goal is to accomplish the maximum work within a specified time period. Neither of these two models is representative of the way in which athletes usually expend their energetic resources in competition, which is to minimize the time required to complete a certain task. Although not evident in the distance smoothed data (Fig. 2), it was very evident that there was substantial second to second variation in power output during the time trials (Fig. 5). In the situation where the subject was feeling momentarily fatigued, in a fixed power model the athlete would be compelled to terminate the effort. However, in a time trial, they have the option of brief reductions in the momentary power output, with a subsequent acceleration. The excellent reproducibility of time trial results (4) and the difficulty in changing pacing strategy even in the presence of external motivation (11) suggests that athletes similar to those used in this study have a well developed performance template (22). Viewed from the context where muscle metabolite accumulation may be thought to limit exercise performance (12), such brief reductions in power output may allow time to transport muscle metabolites to the body water pool and thus allow a greater total work to be accomplished before critical muscle metabolite concentrations are reached. Given that VO2 will remain elevated during brief reductions in power output, it may be that such 'breaks' in the continuity of power output may allow partial restoration of the muscle phosphagen pool. To gain more insight into this, research is needed where the option of brief reductions in the momentary power output during exercise is a controlled variable.

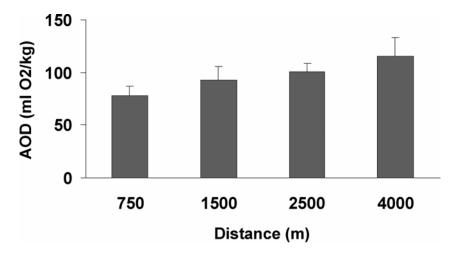


Fig. 4: Accumulated oxygen deficit during the different distance time trials, presented to allow comparison with other studies in the literature (1, 2, 8, 13, 14, 15, 23).

Besides the relatively small fluctuations of power output over the race, global pacing patterns also differed with time trial length, possibly affecting muscle metabolite concentrations. Although the only instruction given to the subjects was to finish as fast as possible, the subjects 'created' their own pacing strategy during the trials, which seems to depend on trial distance. Taking the end-point of exercise into account, they chose a lower starting pace for the longer distances. This is consistent with the concept of 'teleo-anticipation' described by Ulmer (22) and St Clair Gibson et al. (19, 20, 21). The teleo-anticipation hypothesis suggests that the distribution of energetic resources during trials is regulated in order to prevent the body from creating homeostatic disturbances that might cause poor performance or even injury. The use of a large proportion of anaerobic energy early in the race was evident in the shorter distances (i.e. 750 m and 1500 m). The longer distances on the other hand, especially the 4000 m, seem to naturally 'force' the subjects to be less generous with anaerobic energy expenditure during the early stages of the time trial, which could be the reason for the possibility to be able to reach a higher anaerobic capacity. Also the global distribution of energy over the race differs when comparing the two shortest distances with the two longest distances, as can be seen from figure 2.

These values are relatively high when compared to literature, particularly considering that our subjects were sub-elite athletes. One reason that explains part of this difference can be found in the reported rate of the O2 deficit by Medbø and Tabata (14, 15). O₂ deficit and the rate of this deficit over a range of 30 s to 3 min of exhausting cycling were calculated. If it is assumed that MAOD is reached in 3 min of exhausting cycling, the rate of the O₂ deficit at 3 min needs to be zero. The reported value for the rate of the O₂ deficit at 3-min duration is 13.5 μmol O₂·s⁻¹·kg⁻¹. This rate corresponds to an equivalent O₂ uptake of 18 ml O₂·min⁻¹·kg⁻¹ which, given a GE of 18.3% and a body mass of 76.5 kg as found in our subjects, corresponds to a mechanical power output of 88 W. This mechanical power output is from anaerobically attributable sources and corresponds to the magnitude of anaerobic power output reported at 2-3 min duration in time trials in speed skating and cycling (3, 4, 5) and to the anaerobic power output at the end of the longer time trials in this study. Based on the reported non-zero rate of the O2 deficit at 3-min duration of exhausting cycling (14, 15) it can be concluded that MAOD is not reached at 3 min and thus has been underestimated. Another factor that can explain some of the difference between the AOD in this study and in the literature is that in the literature the AOD is determined by linear extrapolation of the power output/VO2 relation, which effectively represents the delta efficiency (DE). In the present study, the gross mechanical efficiency (GE) was used. GE is always lower than DE, therefore an estimation of the anaerobic capacity using GE will be larger compared to an estimation using DE. However this difference is not enough to explain the discrepancy between the literature and the present results.

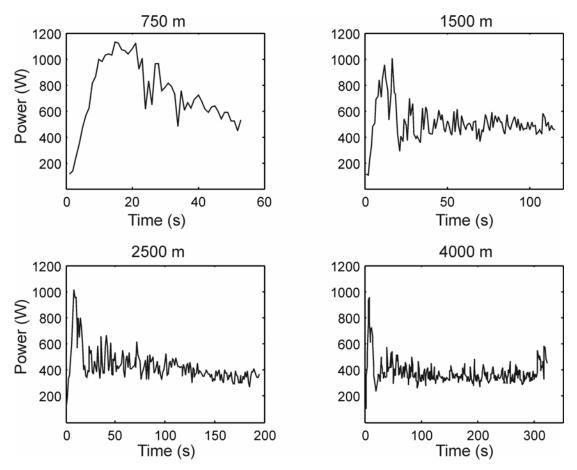


Fig. 5: Representative individual plot of power output during the different distance time trials. The individual data demonstrates the substantial second to second variation in power output in comparison to the average data in Figure 2A.

Summarizing, the results of the present study support the experimental hypothesis. There was an increase in the anaerobically attributable energy contribution with increasing time trial distance. Accordingly, the concept that the anaerobic capacity is a non varying entity at durations > 2 min is not supported. The open loop nature of previous studies of MAOD requires the maintenance of a fixed high power output for as long as possible and in this respect, when all-out exercise of a particular duration is concerned, anaerobic work over the race seems to be a fixed entity. In closed loop exercise, as employed during time trials, the possibility of varying power output over short periods of time may contribute to the larger total anaerobic work what may be accomplished when power output is forced to remain high. The next step is now to study a larger range of intensities/durations in closed and open loop exercise.

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CHAPTER 5 THE EFFECT OF AMBIENT TEMPERATURE ON GROSS EFFICIENCY IN CYCLING

Hettinga FJ, De Koning JJ, De Vrijer A, Wüst RCI, Daanen HAM, Foster C. The effect of ambient temperature on gross efficiency in cycling. Eur. J. Appl. Physiol. 2007; 101(4): 465-471. With kind permission of Springer Science & Business Media.

Abstract

Time-trial performance deteriorates in the heat. This might potentially be the result of a temperature-induced decrease in gross efficiency (GE). The effect of high ambient temperature on GE during cycling will be studied, with the intent of determining if a heat-induced change in GE could account for the performance decrements in time trial exercise found in literature. Ten well-trained male cyclists performed 20-min cycle ergometer exercise at 60% $P\dot{V}O_{2max}$ (power output at which $\dot{V}O_{2max}$ was attained) in a thermo-neutral climate (N) of 15.6 \pm 0.3°C, 20.0 \pm 10.3% RH and a hot climate (H) of 35.5 ± 0.5°C, 15.5 ± 3.2% RH. GE was calculated based on VO₂ and RER. Skin temperature (T_{sk}) , rectal temperature (T_{re}) and muscle temperature (T_m) (only in H) were measured. GE was 0.9% lower in H compared to N (19.6 ± 1.1% vs. 20.5 ± 1.4%) (p < 0.05). T_{sk} (33.4 ± 0.6°C vs. 27.7 ± 0.7°C) and T_{re} (37.4 ± 0.6°C vs. 37.0 ± 0.6°C) were significantly higher in H. T_m was 38.7 ± 1.1°C in H. T_m was not high enough to make mitochondrial leakage a likely explanation for the observed reduced GE. Neither was the increased Tre. Increased skin blood flow might have had a stealing effect on muscular blood flow, and thus impacted GE. Cycling model simulations showed, that GE decreased in heat to a degree that could explain at least part of the well-established performance decrements in the heat.

Introduction

Performance decrements are widely observed during exercise in the heat compared to thermo-neutral circumstances (6, 10, 17, 18, 21, 22). It has been shown that fatigue at exhaustion is related to factors associated with thermoregulation and hyperthermia (5, 16, 17). Time trial performance and fatigue (evidenced by decrements in power output over the race) and finish time have received much less attention in the literature. Accordingly, the present study will focus on the effect of a hot ambient temperature on thermal and cardio respiratory strain during the exercise that might contribute to the well-established decrease in power output.

Tatterson et al. (21) measured time trial performance on a 30-min self-paced cycling time trial in 32°C, 60% RH vs. 23°C, 60% RH. They observed that power output was reduced in the heat by 6.5% (345 \pm 9 W vs. 323 \pm 8 W). Tucker et al. (22) compared 20-km time trials in 35 and 15 °C and found a comparable reduction in average power output in the heat of about 6.3% (255 \pm 47 W vs. 272 \pm 45 W). This change in average power output led to a difference in final time on a 20-km time trial of about 48 s (29.6 \pm 1.9 min vs. 28.8 \pm 1.8 min), which equals about 2.8%. Possible causes for this deterioration in performance are associated with elevated body temperature (21) and anticipatory changes in pacing strategy to avoid hyperthermia (22). Although most research has revolved around neuromuscular function, central drive and fatigue at the point of exhaustion, a plausible explanation for the deterioration in time trial performance in heat may also be found in a temperature-induced change in gross efficiency (GE). GE is an important variable in cycling performance (12, 15) and a linear relationship between body temperature and GE has been observed (2).

A possible explanation for the decreased GE in the heat could be mitochondrial leakage. A temperature induced metabolic disruption caused by non-specific proton-leakage across the inner mitochondrial membrane has been shown to occur at high muscle temperatures (1, 23), resulting in a decrease in ADP:O ratio. Further, a heat induced skin vasodilatation could occur in heat. To prevent a resulting decrease in blood flow to exercising and respiratory muscles (16, 18, 19) a higher cardiac output must exist to continue supplying the muscles with the same blood flow, but still sending extra blood to the skin for cooling.

In the present study we sought to determine the effect of heat on GE. GE can be assessed accurately during sub-maximal exercise at intensities as high as 60-80% \dot{VO}_{2max} by calculating the ratio between mechanical and metabolic (mainly dependent on aerobic energy metabolism) power output, as has been done in literature (2, 8, 11, 12, 14, 15). The presence of a possible effect of ambient temperature on GE can serve as input for an energy flow model (3) to quantify the impact of this effect on time trial performance.

Methods

Subjects

Ten healthy, non-smoking, well-trained male subjects, familiar with cycling exercise at the club-level, participated in this study. They were informed of the nature of the experiment and provided written informed consent. Subject characteristics are presented in Table 1. The study was approved by the medical ethical committee of the University Medical Center Utrecht (The Netherlands).

Table 1: Subject Characteristics: age, height, body mass, maximal oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained ($P\dot{VO}_{2max}$).

	Mean value ± SD
Age (yr)	23.5 ± 4.4
Height (cm)	179.7 ± 9.1
Body mass (kg)	72.5 ± 7.2
$\dot{\mathbf{VO}}_{2max}$ (I·min ⁻¹)	4.78 ± 0.41
PVO _{2 max} (W)	354 ± 29

Values are means ± SD.

Incremental test

The subjects first performed an incremental bicycle test to determine at which power output \dot{VO}_{2max} was attained (\dot{PVO}_{2max}). The incremental test was performed in the heat, under similar conditions as the constant intensity bouts, to make sure that \dot{PVO}_{2max} would not be overestimated in the hot condition. This incremental test was used solely to determine the relative intensities for the constant intensity cycling bouts. The test began at a power output (\dot{P}_{tot}) of 150 W, after which \dot{P}_{tot} was increased with 15 W every minute. Exercise was performed on an electronically braked cycle ergometer (Lode Excalibur, Lode NV, Groningen, The Netherlands) until exhaustion or until pedal frequency dropped below 80 rpm. Oxygen consumption (\dot{VO}_2) was measured breath by breath, using open circuit spirometry (Oxycon ProDelta, Jaeger, Hoechberg, Germany). The gas analyzer was calibrated using a Jaeger 2-I syringe, room air and a calibration gas mixture. Heart rate (HR) was monitored using radiotelemetry (Polar Electro, Kempele, Finland).

Constant intensity exercise bouts

On separate days, subjects performed a constant-intensity exercise bout at 60% of $P\dot{V}O_{2max}$ in a thermo-neutral climate (N) and in a hot, dry climate (H). Temperature in the thermo-neutral climate was 15.6 \pm 0.3°C, relative humidity (RH) was 20.0 \pm 10.3%. In the hot, dry climate, the temperature was 35.5 \pm 0.5°C and RH was 15.5 \pm 3.2%. These temperatures were equivalent to the temperatures in the studies of Tatterson et al. (21) and Tucker et al. (22). All tests were performed in a climate-controlled room with a continuous airflow of 0.2 m·s⁻¹. RH was set low to increase the evaporative capacity of the environment. Further, all trials in the H-climate were

performed with a simulated wind velocity of 1.72 m·s⁻¹ (6.2 km·h⁻¹) to further increase evaporative heat loss in cycling in hot conditions (20). Prior to the experiments, the subjects were asked to refrain from vigorous exercise for at least 48 h. They were also asked not to consume coffee, alcohol or drugs after 10 p.m. the day before the exercise and not to eat for two hours prior to the experiments. Subjects drank water ad libitum before the experiment.

Before the constant intensity bout in the H-condition, the subject stabilized for 50 min in the 35°C, 20% RH room. For the comfort of the subjects and to prevent them from shivering, the stabilizing period was reduced to 35 min for the N-condition in the 15°C, 20% RH room. During the test, oxygen consumption $(\dot{V}O_2)$, respiratory exchange ratio (RER) and ventilation (V_E) were measured breath by breath. P_{tot} and heart rate were registered continuously.

After stabilization, a 5 min warm-up was performed at 100 W with a pedal frequency of ~100 rpm. After 1 min of rest, the constant intensity bout was started (~100 rpm). The subjects cycled for 20 min at a constant power output of 60% of the power output at which maximal $\dot{V}O_2$ was attained at the incremental test ($\dot{P}\dot{V}O_{2max}$) in the H condition. Directly after the end of exercise, blood lactate concentration (BLC) was measured (Lactate Pro, Arkray, Kyoto, Japan).

Rectal temperature (T_{re}) was monitored every 5 s during the entire test using a thermistor temperature probe (YSI 701, Yellow Springs Instrument, Dayton, USA) inserted about 10 cm in the rectum. Skin temperature (T_{sk}) was measured every 5 s at 14 different skin loci, conform ISO 9886, using thermocouples (YSI 709B, Yellow Springs Instrument, Dayton, USA). Data were recorded with a Data Translation acquisition board (DT2821, Viewdac, Keithley Instruments, Cleveland, U.S.A.). Muscle temperature (T_m) was measured at a minimum depth of two centimeters and recorded every 5 s.

Since this measurement was invasive, measurements were restricted to the constant intensity bout in H (n=6). T_m was measured with a sterile thermal thermocouple-probe (type MAC08170A275SM, Ellab A/S, Rodovre, Denmark) in the right vastus lateralis muscle, inserted by a physician. One hour before inserting the temperature probe, a lidocaine plaster was attached to the skin, as a local anaesthetic.

Calculating gross efficiency (GE)

Metabolic power (P_{met}) was calculated by multiplying oxygen consumption with the oxygen equivalent: P_{met} (W) = $\dot{V}O_2$ · [(4940 · RER + 16040)/60] according to Garby and Astrup (9), assuming that respiratory exchange ratio (RER) equaled respiratory quotient (RQ) at sub-maximal intensities. We assumed that respiratory exchange ratios in excess of 1.00 were attributable to buffering of lactate by bicarbonate. Ratios in excess of 1.00 were thus treated as if they equaled 1.00. The measured mechanical power output (P_{tot}) divided by the calculated P_{met} defined GE. GE was calculated from 90 s after the start of exercise until the end of exercise.

Energy flow model

The energy flow model as described by De Koning et al. (3) was used to calculate the effect of changes in GE on performance, by simulating a 20-km time trial as is studied in literature (21, 22). This model is based on power equations and has been reasonably successful in predicting performance in cyclic events as cycling (21) and speed skating (4). The energy flow model, also referred to as power balance model, relates to power production and power dissipation: $P_{tot} = P_{lost} + dE_{kin}/dt$, where P_{tot} is the total power that can be produced, P_{lost} is the power that is used to overcome frictional losses, and dE_{kin}/dt is the rate of change of kinetic energy of the mass center of the body. These terms can be calculated as described in de Koning et al. (3), and the influence of changing one single variable, in this case GE, can be predicted.

Statistics

Paired student t-tests were performed to test if data were significantly different between the H-condition and N-condition (p < 0.05).

Results

Power output at 60% PVO_{2max} was 211.5 \pm 18.6 W. All subjects were able to complete the trials in both H and N. VO_2 was significantly higher in H than in N, resulting in GE being significantly lower in H compared to N. Further, HR and V_E were higher in H compared to N. For RER and BLC, no main effect of temperature was found. Mean values are shown in table 2.

Mean GE over time is plotted for both conditions in figure 1. The difference in GE between conditions was 0.9% over the entire trial. From min 5-8, the difference between N and H was 0.6% \pm 0.7%, from min 15-18, the difference was 1.1 \pm 1.3%, both significant. No significant change in GE within trials was observed comparing GE over the first half (min 5-8) with GE over the second half (min 15-18).

 T_{sk} and T_{re} were significantly higher in H. Changes in T_{re} and T_{sk} combined, the difference in body heat content between N and H amounts to 138.5 \pm 35.1 kJ, assuming that the specific heat of the body tissue was 3.4 kJ·g^{-1.o}C⁻¹. Maximal muscle temperature (T_{mmax}) in H was 38.7 \pm 1.1°C. Mean temperature is shown in table 3.

Figure 2 shows T_{re} , T_{sk} and T_m plotted over time for both conditions. Figure 3 shows the correlation between T_{re} and GE for both conditions. R^2 in N was 0.04, R^2 in H was 0.36.

Table 2: Gross-Efficiency (GE), \dot{VO}_2 , respiratory exchange ratio (RER), respiratory minute volume (V_E), heart rate (HR) and blood lactate concentration (BLC) at 60% $P\dot{VO}_{2\,max}$ in the thermo-neutral (N) and in the hot dry (H) climate.

	60% PVO _{2max}		
	N	Н	
GE (%)	20.5 ± 1.4	19.6 ± 1.1*	
$\dot{\mathbf{VO}}_{2}$ (ml·min ⁻¹)	3002.8 ± 290.1	3126.5 ± 268.3*	
RER	0.89 ± 0.03	0.90 ± 0.01	
V_{E} (I · min ⁻¹)	76.9 ± 7.7	82.0 ± 9.4*	
HR (bpm)	145 ± 7	155 ± 12*	
BLC (mmol· l ⁻¹)	2.6 ± 1.0	3.9 ± 2.1	

Values are means \pm SD. Significant differences with N are marked with * (p < 0.05).

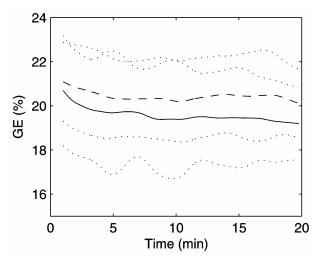


Fig. 1: Gross-efficiency (GE) plotted over time at 60% $P\dot{V}O_{2max}$ in the thermo-neutral (N) climate (dashed line) and in the hot dry (H) climate (solid line) \pm SD (dotted lines).

Table 3: Rectal temperature (T_{re}), skin temperature (T_{sk}) and maximal muscle temperature (T_{mmax}) at 60% $P\dot{V}O_{2max}$ in the thermo-neutral (N) and in the hot dry (H) climate.

	60% PVO _{2max}		
	N	Н	
T _{re} (°C)	37.03 ± 0.58	37.35 ± 0.63*	
$T_sk(^\circC)$	27.74 ± 0.71	33.39 ± 0.57*	
$T_{mmax}(^{\circ}C)$		38.7 ± 1.1 (n=6)	

Values are means \pm SD. Significant differences with N are marked with a * (p < 0.05).

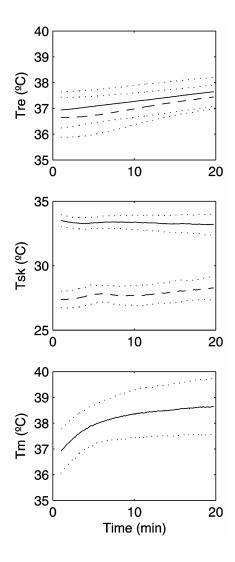


Fig. 2: Rectal temperature (T_{re}) plotted over time at 60% $P\dot{V}O_{2\,max}$ in the thermo-neutral (N) climate (dashed line) and in the hot dry (H) climate (solid line) \pm SD (dotted lines) (top panel). Skin temperature (T_{sk}) plotted over time at 60% $P\dot{V}O_{2\,max}$ in the thermo-neutral (N) climate (dashed line) and in the hot dry (H) climate (solid line) \pm SD (dotted lines) (center panel) Muscle temperature (T_m) plotted over time at 60% $P\dot{V}O_{2\,max}$ in the hot dry (H) climate (solid line) \pm SD (dotted lines) (bottom panel).

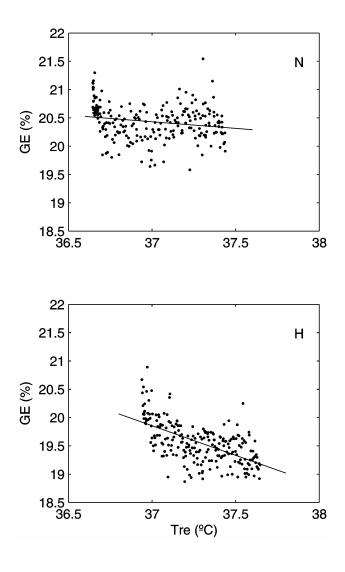


Fig. 3: Correlation between gross efficiency (GE) and rectal temperature (T_{re}) for the thermo-neutral (N) climate (top panel) and for the hot, dry (H) climate (bottom panel).

To quantify the effect of the decrease in GE of 0.9%, a simulation with an energy flow model (3) was performed. This simulation showed that a change in GE of 0.9% would result in a difference in final time of 25.6 s over 20 km. About half of the decrement in performance reported in the literature (a difference in final time of 48 s over 20 km (22)), could be accounted for by the measured decrease in GE. To explain the entire deterioration in time trial performance in the heat, the mean decrease in GE had to be 2%.

Discussion

GE during sub-maximal cycling exercise in the heat was significantly reduced compared to exercise in N by 0.9%. Values for GE corresponded to values found in literature, between 17 and 22% (8, 14). It has to be kept in mind that in calculating GE at 60% PVO_{2max}, anaerobic contribution was assumed to be negligible. But it might have been influenced by the different climates. Gonzalez-Alonso et al. (10) reported that exercise in a hot environment results in a higher anaerobic contribution and found an increase in carbohydrate utilization and lactate accumulation during exercise. Though not significant, BLC in the present study was higher in H. According to Di Prampero et al. (5), an increase of BLC of 1 mmol·l⁻¹ of blood is equivalent to an O₂ consumption of 3 ml ·kg⁻¹ body weight. Differences in BLC in the present study between climates (2.6 mmol·l⁻¹ in N vs. 3.9 mmol·l⁻¹ in H) thus correspond with a difference in O₂ consumption equivalent of ~14.1 ml·min⁻¹. Accordingly, differences in GE between climates might be even larger than reported in the present study. If one adds this 'anaerobic O2 equivalent' to the VO2, GE decreases about 0.19% in N compared to about 0.27% in H, which means that the difference in GE between N and H increases with 0.07%.

Mean T_{re} was about 0.3°C higher in H compared to N. Mean T_{sk} was about 5.7°C higher in H than in N. Comparable differences of 0.2-0.4°C in T_{re} (21, 22) and about 6.0°C in T_{sk} (21) were reported during cycling bouts of 30 min in climates comparable to the present study. Although the difference in rectal temperature between H and N was significant, it was not a large difference compared to literature. The measured absolute values for T_{re} were somewhat low, presumably due to the relatively short period of exercise in our protocol and the use of a fan to optimize losing heat to the environment, as was done in the studies of Tatterson et al. (21) and Tucker et al. (22). The fan was used to simulate competitive circumstances, and although wind velocity in this study was not as high as the cycling speed, combined with the relatively low RH, the effect would have been considerable (20).

The differences in ambient temperature between H and N represent a considerable difference in thermal stress. Thermal strain is mainly visible in the increased skin temperature, but although the difference of 0.3° C in core temperature is small, it is significant. Rectal and skin temperature changes combined, the difference in body heat content between N and H amounts to 138.5 ± 35.1 kJ, which is considerable. Further, the differences between climates have been shown to be large enough to evoke a difference in GE of 0.9%, the main purpose of the present study.

A possible explanation for the lower GE in the heat may be the higher core temperature. Daanen et al. (2) found a strong linear relationship between body temperature, mainly determined by T_{re} , and GE at 60% $P\dot{V}O_{2max}$ in 30°C. Their study showed that our difference in T_{re} of 0.3°C could account for a reduction in GE of ~0.2%, and thus was not large enough to explain the entire reduction in GE of 0.9%.

In the study of Daanen et al. (2), the skin temperature however was almost unchanged, while in the present study skin temperature in the H condition was about 5.6° C higher, indicating an increased blood flow to the skin. The present study did not find a strong correlation in heat (r^2 = of 0.36) between T_{re} and GE. Further, remarkably, in the N-condition, no correlation between T_{re} and GE was found at all, even though T_{re} reached values which are also observed in the H-condition, only later in time (see fig. 3). Apparently, T_{re} does not seem to be the main cause of the reduced GE. This is supported by the observation that even though T_{re} rose significantly during the time trial with about 1°C, GE did not increase significantly comparing the first half with the second half within the trials.

Two other explanations for the reduced GE in heat can be given. Firstly, an explanation for the temperature-induced reduction in GE might be a metabolic disruption that is known to occur at elevated muscle temperatures (1, 23). Though Ferguson et al. (7) found no effect of increasing muscle temperature on energy turnover in dynamic exercise in a range of 34.2-38.3°C, ADP:O ratio has been shown to decrease at higher muscle temperatures (1, 23). During heavy exercise in the heat, nonspecific proton leakage across the inner mitochondrial membrane is increased, resulting in a decrease in the efficiency of oxidative phosphorylation and increasing the resting metabolic rate (23). This will result in a reduction in GE. Willis and Jackman (23) found a decrease of 10%-20% in ADP:O ratio at muscle temperatures of 40°C and higher compared to 37°C. They found that this decrease resulted in a 400-800 ml·min⁻¹ increase in VO₂. In the present study, a smaller but significant increase in $\dot{V}O_2$ of 124 ml·min⁻¹ was found. Mean T_{mmax} in heat was 38.7°C, lower than the 40°C as found in Willis and Jackman (23), but higher than 34.2-38.3°C, the T_m range in which Ferguson et al. (7) found no effect of increasing T_m on energy turnover in dynamic exercise. Since T_m was not measured in both climates, it cannot be confirmed if muscle temperature was significantly different. T_{sk} and T_{re} were significantly different but did not approach 40°C in either condition. It seems that, as suggested by Brooks (1), the core of the body functions as a heat sink for the skeletal musculature helping to maintain T_m below the point where significant reductions in the ADP:O ratio occur.

Another potential cause for the decrease of GE in a hot ambient temperature was the larger vasodilatation of the skin to lose heat, as was shown by the significantly higher T_{sk} in H compared to N. The resulting decreased blood flow to exercising and respiratory muscles may be compensated by increasing cardiac output (15, 19), since a significant increase in heart rate was found in heat. The extra \dot{VO}_2 in the H condition is at least partially attributable to the extra myocardial \dot{VO}_2 , since a higher cardiac output has to exist to continue supplying the muscles with the same blood flow, but have to send extra blood to the skin for cooling. Additionally, ventilation was increased, which may also lead to a reduction in GE. Assuming that the mechanical work per breath is 80-125 J (13), it can be estimated that the higher V_E in this study can account for maximally 10% of the increase in \dot{VO}_2 .

Lastly, it has to be noticed, that GE decreases if the proportion of energy expenditure that is used to maintain homeostasis is increased. Thus, the lower GE in H could have been solely due to an increase in resting metabolic rate, while net efficiency remained unchanged. This would be consistent with the hypothesis that muscle temperature in the hot conditions was not high enough to make mitochondrial leakage a likely explanation for the observed reduced GE. Unfortunately, resting metabolic rate has not been measured.

To determine the potential importance of the measured decrease in GE on time trial performance, a 20-km time trial, which was also studied by Tucker et al. (22), was modeled by the use of the energy flow model (3). A time trial of this distance can be seen as a mainly aerobic exercise bout. Tucker et al. (22) found a difference in final time of 48 s between exercise in 35°C compared to 15°C. Using the energy flow model of De Koning et al. (3), it was calculated that the measured difference in GE of 0.9% would lead to a difference in final time between H and N of 25.6 s. This explains about half of the 48 s found by Tucker et al. (22). For the entire reduction in final time of 48 s, a decrease in GE of 2% would be necessary, which was not found. It can be concluded that about half of the decrease in time trial performance can be accounted for by the reduction in GE. It has to be noted that in this simulation, it is assumed that the difference in GE between H and N at higher intensities will not change. Although it has been shown that GE increases with exercise intensity, since the relative share of resting metabolism diminishes at higher sub-maximal intensities (14), this effect seems to be equal for both conditions and will at most have only a minor effect on the difference between conditions.

Conclusion

GE was lower in the heat. T_m was not high enough to make mitochondrial leakage a likely explanation for the observed reduced GE. Neither was the increased T_{re} . The extra \dot{VO}_2 in the H condition seems to be at least partially attributable to the extra myocardial \dot{VO}_2 , since a higher cardiac output has to exist to continue supplying the muscles with the same blood flow, but have to send extra blood to the skin for cooling and thus impacted GE. Based on our findings under the current circumstances, it can be concluded that the temperature-induced change in GE could account for about half of the well-established performance decrements found during time trial exercise in the heat.

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CHAPTER 6 VO₂ RESPONSE IN SUPRA-MAXIMAL CYCLING TIME TRIAL EXERCISE OF 750 M TO 4000 M

Hettinga FJ, De Koning JJ, Foster C. $\dot{V}O_2$ response in supra-maximal cycling time trial exercise. Med. Sci. Sports Exerc. (in press).

Abstract

Limited research has been done on the VO₂ response to time trial exercise in the supra-maximal domain or during free range exercise typical of competition. The present study was designed to measure and model the VO₂ response during supramaximal time trial exercise. Well-trained cyclists (n=9) performed a 1-min incremental exercise test to obtain maximal power output (PVO_{2max}) and 4 cycle ergometer time trials of different distances (750 m, 1500 m, 2500 m, 4000 m). Athletes were instructed to finish in as little time as possible. VO2 was measured breath-by-breath and modeled mono-exponentially over the first 54 s (750 m) or 114 s (1500 m, 2500 m and 4000 m) of the time trials. Mean $P\dot{V}O_{2max}$ in the incremental test was 383 ± 28 W. Mean $\dot{V}O_{2max}$ was 4.5 ± 0.2 l·min⁻¹. All time trials were characterized by an initial burst in power output during the first 15 s, (175 \pm 23%, 149 \pm 14%, 145 \pm 14%, 139 \pm 10% PVO_{2max}) being largest for 750 m. Simultaneously, the mean response time was significantly smaller in 750 m compared to all other trials (18.8 \pm 2.2 s; 20.9 \pm 1.9 s; $20.8 \pm 1.5 \text{ s}$; $21.2 \pm 2.2 \text{ s}$). Near maximal values of VO_2 can be reached within 2 min of strenuous exercise. The larger initial burst in power output in 750 m was accompanied by a faster VO2 response and seems to be of importance to trigger the aerobic system maximally.

Introduction

Explaining sports performance is complicated, even in comparatively simple 'pursuit' sports where power output and power losses are the primary performance related variables. Because of the complexity of sports performance, and the difficulty in experimentally controlling the behavior of athletes, mathematical models are attractive devices for exploring effects attributable to training, ergogenic aids, equipment and tactics. We have performed several studies using an energy balance model developed in our laboratory (10, 11, 32), where both expressions for biomechanical factors and energy supply have been manipulated to predict performance (25). In order to model sports performance, it is necessary to make several assumptions involving the parameters of energy supply and energy losses. We have demonstrated a time/distance dependent pattern of anaerobic energy use (10, 13, 14, 20) and a recent report from our laboratory has demonstrated the relative constancy of the anaerobic capacity in response to different patterns of total power output (e.g. pacing strategy) (18). An untested assumption in our model is the pattern of VO₂ response during supra-maximal exercise intensity, typical of speed skating, athletics, swimming and track cycling competitions. A fast start strategy appears to accelerate the VO₂ response (4, 21). However, to our knowledge, there are few data regarding VO₂ response during supra-maximal exercise intensities (9, 19, 34, 37), particularly when the power output is free to vary, rather than the square wave manipulations of power output that are traditionally used in studies on VO2 kinetics (1-3, 6-9, 12, 15, 19, 22, 23, 26-31, 33-37).

Numerous studies have been performed investigating VO₂ response. It has been shown that VO, kinetics are affected by different warm-up protocols potentially leading to subsequent performance benefits (5, 7, 8, 12, 15, 16) and are influenced by training status (6, 23, 24). Also, several studies have investigated the influence of exercise intensity on VO₂ response, but have shown different outcomes (1, 2, 9, 19, 21, 23, 26, 27, 31). Relatively little research has been performed at work rates exceeding peak VO₂. Since the aerobic system is maximally stressed in the supramaximal domain, changes in the VO₂ response to exercise have great implications for the use of the anaerobic capacity and are thus relevant for sports performance. Additionally, though the VO2 response to exercise has mostly been modeled in square-wave exercise bouts (1-3, 6-9, 12, 15, 19, 22, 23, 26-31, 33-37), sports performance is often characterized by a large, short duration burst of power output at the commencement of exercise (10, 13, 14). To address this gap in our understanding about how to construct models of high intensity exercise performance, the present study was designed to measure and model the VO₂ response to exercise during supra-maximal time trial exercise representative of that undertaken during competition. Time trials of maximal effort were performed and the VO₂ response was hypothesized to be invariant with starting intensity in the supra-maximal domain.

Methods

Subjects

Nine well-trained male cyclists participated in this study (Table 1). All subjects gave written informed consent before entering the study. The experiment was approved by the ethics committee of the Faculty of Human Movement Sciences at the VU University Amsterdam (The Netherlands). The subjects were requested to follow their usual diets and to lessen physical activities the day before each trial, just as they would before a competition.

Table 1: Subject characteristics: age, height, body mass, maximal oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained (\dot{PVO}_{2max}).

	Mean value ± SD
Age (years)	26.4 ± 7.0
Height (cm)	185.4 ± 8.3
Body mass (kg)	77.5 ± 8.1
VO₂max (I·min⁻¹)	4.5 ± 0.2
PVO _{2max} (W)	383 ± 28

Values are means ± SD.

Experimental Design

After performaing an incremental test, four maximal effort time trials (mean power output > 100% PVO_{2max}) were completed by each subject: (750 m, 1500 m, 2500 m and 4000 m). All tests were done on separate days and at the same time of the day. All time trials were commenced after completing a 10-min sub-maximal warm-up protocol including two 5-s all-out sprints and followed by a cooling down period as described by Hettinga et al. (18). The warm-up protocol was standardized so that results could not be attributed to differences in prior exercise (5, 7, 8, 12, 15, 16). Between warm-up and the beginning of the time trial, 3 min of rest were allowed. For the time trial, the subjects were instructed to finish as fast as possible. Athletes were provided with distance and velocity feedback, as they would have in competition. Subjects performed all trials on a custom made electronically braked laboratory cycle ergometer (FBW-MTO, Amsterdam) designed to simulate competition. Torque and crank rotational velocity were measured directly. Pedaling cadence, power output, virtual velocity and virtual distance were calculated based on these measurements and gear ratio. The ergometer was linked to a computer which continually measured and stored mechanical power output (Ptot), pedaling cadence and torque at 20 Hz. Handlebars and saddle height were adjusted to the preferences of the subject and kept constant for each trial. All subjects first completed a maximal incremental test to measure maximal rate of oxygen consumption (VO_{2max}) and maximal power output (PVO_{2max}). On a second day, the subjects cycled 1500-m and 4000-m habituation time trials at maximal intensity with 30 min of rest between trials. Subsequently, the experimental time trials were performed in random order, with 48-96 h between trials.

Respiratory gas exchange was measured breath-by-breath using open circuit spirometry (Oxycon alpha, Mijnhardt, The Netherlands) and afterwards interpolated to 1-s values. The flow meter and gas analyzer were calibrated before each test using a 3-l syringe (Jaeger, Germany), room air and a standard gas mixture. Blood lactate concentration [BLC] was measured 3 min after finishing each time trial from a fingertip and analyzed using dry chemistry (Lactate Pro, Arkray Inc, Kyoto, Japan). Conforming to our procedures in other modeling studies (10, 11), VO₂ response was fit by a least squares mono-exponential model (1, 3), minimizing the sum of squared error and are modeled mono-exponentially according to equation 1, with an error defined as the sum of squared errors per number of samples. For 750 m, VO₂ response was fit over 54 s (= duration of fastest 750 m). For the 1500 m, 2500 m and 4000 m, VO₂ response was fit over 114 s (duration of the fastest 1500 m).

$$\dot{V}O_2$$
 (t) = $\dot{V}O_{2rest}$ + A [1-e^{-(t-td)/T}] [1]

 \dot{VO}_{2rest} is the average over 1 min of \dot{VO}_2 immediately prior to exercise, t is time, A is the asymptotic amplitude, τ is the time constant and td is the time delay of the \dot{VO}_2 response. Because the cardio-dynamic component does not directly represent active muscle O_2 utilization (26), the \dot{VO}_2 response was calculated from the first point after the cardio-dynamic phase, based on visual inspection for each trial. Depending on this point, the first 10 to 20 s were omitted from the fitting field. For the 1500 m, 2500 m and 4000 m, the value of A at 114 s (= duration of the fastest 1500-m time trial) was calculated (A_{end}) to allow comparison of amplitudes at the same moment in time. Since the 750-m time trial was of shorter duration, A_{end} was calculated at 54 s (based on the shortest 750-m time trial). To characterize and compare the \dot{VO}_2 responses, mean \dot{VO}_2 over the first part of the race was calculated, and mean response time (MRT) was calculated as MRT = td + τ .

Statistics

 ${
m VO}_2$ responses were compared using a one-way repeated-measures analysis of variance. To reveal significant differences between trials in case of a significant main effect, paired sample t-tests were performed. The significance level was set at p < 0.05.

Results

Time trials

All subjects completed the time trials without problems. Mean P_{tot} profiles are shown together with standard deviations in figure 1. Power output was normalized to $P\dot{V}O_{2max}$ as determined in the incremental test. In table 2, final time and mean values for P_{tot} and $\dot{V}O_2$ were shown. Mean $\dot{V}O_2$ and P_{tot} were calculated over the first 114 s. For 750 m, mean $\dot{V}O_2$ and P_{tot} were averaged over the first 54 s (= duration of the

fastest 750 m time trial). Mean \dot{VO}_2 was also calculated over the first 15 s (\dot{VO}_{215}), the first 30 s (\dot{VO}_{230}) and from 15 to 30 s (\dot{VO}_{21530}).

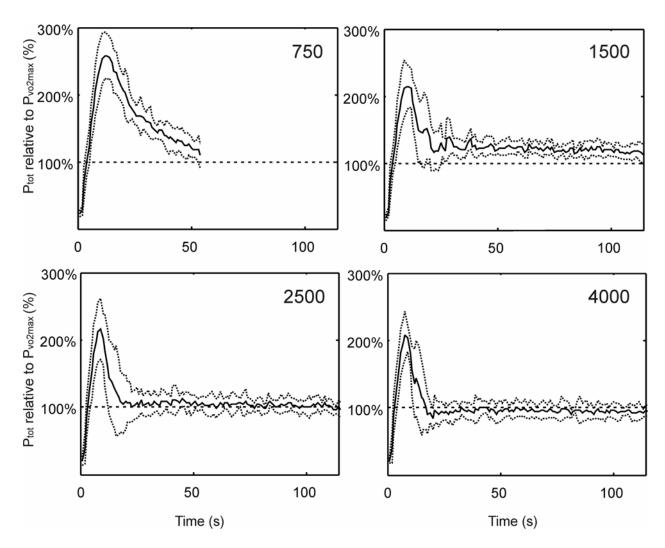


Fig. 1: Mean P_{tot} profiles (solid line) \pm SD (dotted lines) relative to maximal power output reached at the incremental test ($P\dot{V}O_{2\,max}$) for the 750-m time trial (left upper panel), 1500-m time trial (right upper panel), 2500-m time trial (left lower panel) and 4000-m time trial (right lower panel).

VO₂ response

Mean $\dot{V}O_2$ during the time trials is shown in figure 2, together with standard deviations. $\dot{V}O_2$ response was fit through these mean data. Results are shown in equations 2 to 5 and plotted in figure 3.

750 m:
$$\dot{VO}_2 = 0.82 + 3.35 \left[1 - e^{-(t-12.88)/5.78}\right]$$
 [2]

1500 m:
$$\dot{V}O_2 = 0.76 + 3.64 \left[1 - e^{-(t-9.83)/10.74}\right]$$
 [3]

2500 m:
$$\dot{VO}_2 = 0.69 + 3.58 \left[1 - e^{-(t-12.37)/8.29}\right]$$
 [4]

4000 m:
$$\dot{V}O_2 = 0.64 + 3.62 \left[1 - e^{-(t-11.60)/9.54}\right]$$
 [5]

Table 2: Mean values for final time, blood lactate concentration (BLC), mean \dot{VO}_2 (\dot{VO}_{2114}) over the first 114 s (or 54 s for 750 m), mean power output (P_{tot114}) over the first 114 s (or 54 s for 750 m) and mean power output over the first 15 s relative to $P\dot{VO}_{2max}$ (P_{tot15}) for the 750-m, 1500-m, 2500-m and 4000-m time trials.

	750 m	1500 m	2500 m	4000 m
Final time (s)	57.0 ± 1.8 ^{bcd}	116.4 ± 1.7 ^{acd}	198.6 ± 5.2 ^{abd}	325.9 ± 9.7 abc
BLC (mmol·l ⁻¹)	15.3 ± 2.4	15.6 ± 2.3	14.5 ± 1.8	13.9 ± 2.0
$\dot{\mathbf{VO}}_{215}$ (l·min ⁻¹)	1.26 ± 0.34	1.41 ± 0.36	1.35 ± 0.25	1.39 ± 0.27
VO₂₁₅₃₀ (I·min ⁻¹)	3.46 ± 0.37^{d}	3.28 ± 0.23^{d}	3.17 ± 0.22	3.11 ± 0.20^{ab}
$\dot{\mathbf{VO}}_{230}$ (I·min ⁻¹)	2.36 ± 0.26	2.35 ± 0.24	2.28 ± 0.22	2.26 ± 0.20
$\dot{\mathbf{VO}}_{2114} \ (\mathrm{l \cdot min}^{-1})$	3.10 ± 0.24^{bcd}	3.80 ± 0.22^{a}	3.70 ± 0.18^{a}	3.67 ± 0.18^{a}
P_{tot114} (W)	615 ± 51 ^{bcd}	481 ± 24 ^a	420.5 ± 46^{a}	386 ± 38 ^a
P_{tot15} (% $P\dot{V}O_{2max}$)	175 ± 23 ^{bcd}	149 ± 14 ^a	145 ± 14 ^a	139 ± 10 ^a

Values are means \pm SD. Differences with 750 m are marked with an 'a'. Differences with 1500 m are marked with a 'b'. Differences with 2500 m are marked with a 'c'. Differences with 4000 m are marked with a 'd'. (p < 0.05).

Also, individual \dot{VO}_2 data were fit per subject to make statistical analysis and comparisons possible. Parameters of the \dot{VO}_2 response are shown in table 3, as well as a typical example of the \dot{VO}_2 response and the residuals in a 1500-m time trial in figure 4. Comparing $A_{end} + \dot{VO}_{2rest}$ (I·min⁻¹) values with \dot{VO}_{2max} (4.5 ± 0.2 I·min⁻¹) attained at the incremental test, differences were significant with all but the 1500-m time trial.

Table 3: Mean values for mean response time (MRT), resting \dot{VO}_2 (\dot{VO}_{2rest}), time delay of the \dot{VO}_2 response (td), time constant of the \dot{VO}_2 response (τ), amplitude of the \dot{VO}_2 response (Δ_{end}) and the sum of Δ_{end} and Δ_{end} and Δ_{end} are trials.

Time trial	750 m	1500 m	2500 m	4000 m
MRT (s)	18.8 ± 2.2 ^{bcd}	20.9 ± 1.9 ^a	20.8 ± 1.5 ^a	21.2 ± 2.2 ^a
VO _{2rest} (I·min⁻¹)	0.82 ± 0.17^{d}	0.76 ± 0.19^{d}	0.69 ± 0.14	0.64 ± 0.10^{ab}
td (s)	13.8 ± 1.7	12.1 ± 2.3	12.4 ± 1.9	11.6 ± 1.6
T (S)	4.9 ± 2.1 bcd	8.8 ± 3.4^{a}	8.4 ± 2.5^{ad}	9.6 ± 2.9^{ac}
A _{end} (I·min ⁻¹)	3.35 ± 0.30^{bcd}	3.64 ± 0.34^{a}	3.60 ± 0.18^{a}	3.64 ± 0.28^{a}
A _{end} + VO _{2rest} (I·min ⁻¹)	4.17 ± 0.33^{b}	4.40 ± 0.25^{acd}	4.27 ± 0.20^{b}	4.26 ± 0.24^{b}

Values are means \pm SD. Differences with 750 m are marked with an 'a'. Differences with 1500 m are marked with a 'b'. Differences with 2500 m are marked with a 'c'. Differences with 4000 m are marked with a 'd'. (p < 0.05).

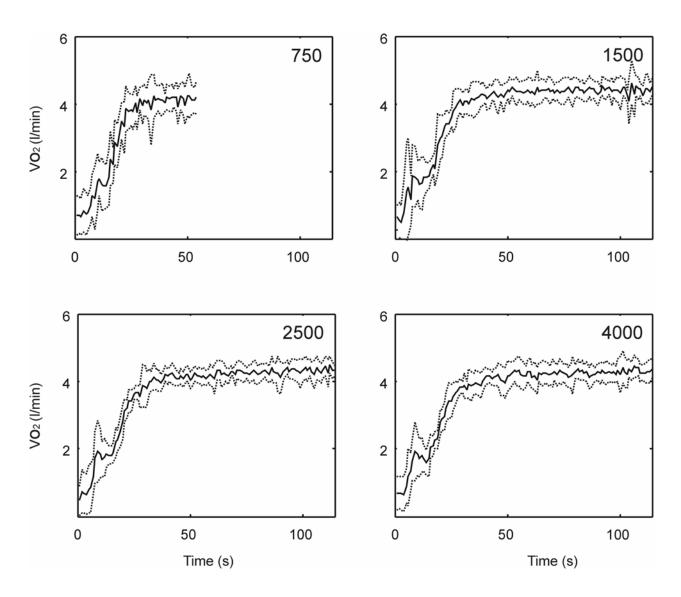


Fig. 2: Mean $\dot{V}O_2$ (solid line) \pm SD (dotted lines) for the 750-m time trial (left upper panel), 1500-m time trial (right upper panel), 2500-m time trial (left lower panel) and 4000-m time trial (right lower panel).

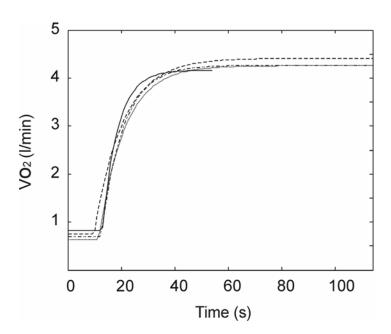


Fig. 3: \dot{VO}_2 response fit through mean \dot{VO}_2 -data (n=9) for the 750-m time trial (solid line), the 1500-m time trial (dashed line), the 2500-m time trial (dash-dotted line) and the 4000-m time trial (dotted line).

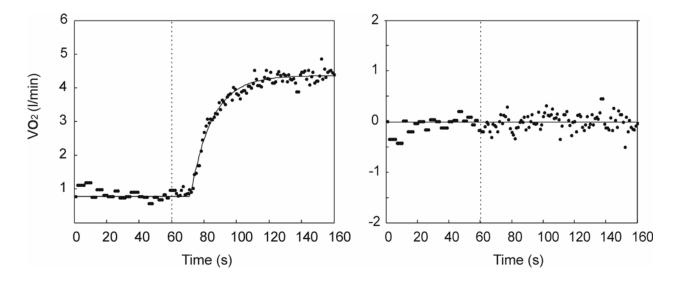


Fig. 4: Typical example of the \dot{VO}_2 response of a representative subject for the 1500-m time trial (left panel). Residuals were calculated by subtracting the measured \dot{VO}_2 from the mono-exponential fit (right panel). The 60-s period of rest preliminary to the time trial is included and the start of the time trial is marked at 60 s.

Discussion

The present study was designed to observe the $\dot{V}O_2$ response in maximal effort competitive simulations. All time trials were characterized by an initial burst of power output during the first 15 s, which is an important difference between competitive

exercise and the square wave exercise that is ordinarily used in studies of VO₂ kinetics. In the 750 m, the initial burst in power output was larger than in all other trials. Simultaneously, the VO₂ response was accelerated in the 750-m time trial, as shown by a significant reduction in MRT compared to all other trials (see also figure It seems that the initial burst of power output that is so characteristic for sports performance and was apparent in all exercise conditions, is of fundamental importance in terms of defining VO₂ response. Studying mean VO₂ over the first 30 s of exercise, no differences were found, mainly because $\dot{V}O_2$ over the first 15 s, a time period corresponding with the time delay (11.6-13.8 s), was not differing. From seconds 15 to 30, a significant higher mean $\dot{V}O_2$ was found for 750 m and also 1500 m compared to 4000 m. Accordingly, our experimental hypothesis was not supported. Jones et al. (21) also found that a fast start strategy resulted in a more rapid increase in VO₂ during high intensity exercise (exhaustion in 120 s) compared to a slower start strategy. Also, an initial burst in power output over the first 15 s of 500-m kayaking has been shown to result in a faster VO₂ response over the first 30-45 s of exercise, resulting in a significant increase in mean power output (4). A possible explanation for the faster VO₂ response in the time trial with the higher initial burst might be linked to the phosphocreatine (PCr) response to exercise. It has been shown that the time constants of decreases in [PCr] are identical to simultaneously measured VO2 onkinetics of the primary component (29). This equality was also present at high intensity exercise (30), supporting the concept that VO₂ kinetics are determined by intramuscular mechanisms. The higher initial burst in peak power could be a signal to accelerate oxidative phosphorylation and apparently leads to a faster increase in VO₂ on response, as is shown by the present data.

Though the high intensity domain has received attention in literature, exercise intensities that substantially exceed $P\dot{V}O_{2max}$ have not been studied extensively. It has been found that nitric oxide dependent metabolic inertia represents an important limitation to VO₂ kinetics following the onset of high intensity cycle exercise (37), where O_2 transport limitations were shown not to constrain VO_2 response (36). Further, the gain of VO₂ response was reduced at intensities above critical power (26, 31, 34). This was suggested to be caused by a direct effect of acid pH on mitochondrial respiration. Also, at higher intensities, anaerobic metabolism is becoming of increasing importance and VO2 will be constrained by the attainment of VO₂ peak. A lower response amplitude is reached than the one predicted by the VO₂-workrate relation and the VO₂ response in supra-maximal exercise is thus truncated, resulting in relatively higher T values (19, 33) compared to sub-maximal intensities. A semi-log model takes (19) this into account and assumes a predicted $\dot{V}O_2$ beyond the upper limit of $\dot{V}O_2$, resulting in a slower τ (9, 33). Since this predicted VO₂ cannot be attained in real life, as also noted by Carter et al. (9), it is not suitable for modeling the VO₂ response to describe athletic performance in competitive exercise, where we are mainly interested in describing the actual VO₂ response during time trial exercise. MRT shows that the response is clearly fastest at 750 m (Fig. 4).

For the present results, the main intention was to describe VO2 response in competitive exercise to test assumptions necessary for modeling sports performance as has been previously done in studies from our lab (10, 11, 32) and elsewhere (25). For 1000 m (~1 min) and 4000 m (~6 min) cycling (10, 25), 1500 m (~2 min) speed skating (11), modeling studies have led to accurate predictions of performance and excellent fits of VO₂ response with a mono-exponential model, with correlation coefficients ranging from 0.94-0.98 (25). As discussed, the initial burst of 10-15 s seems to be of fundamental importance in speeding $\dot{V}O_2$ kinetics. Additionally, at 114 s, $\dot{V}O_2$ values were only about 0.1 I-min⁻¹ lower than $\dot{V}O_{2max}$ attained at the incremental test and in ~20 s from start, 63% of the final amplitude was already reached. Apparently, values close to maximum VO2 can be reached well within 2 min of strenuous exercise, at least in well-trained (23) and warmed up (12, 15) athletes. A_{end} of the 1500-m, 2500-m and 4000-m time trials did not differ largely. Only during the 750-m time trials, athletes reached a lower maximum during exercise from baseline, probably caused by the shorter endurance of the event. Looking at the absolute amplitude, differences were found between 1500-m and all other trials, but were within a range of ~0.2 l·min⁻¹. It thus seems that for middle distance exercise, about the same pattern of VO2 response was found, as was already observed in a previous study (17), where different pacing strategies were mainly determined by differences in distribution of anaerobic energy, while aerobic energy contribution globally followed the same profile for differently paced supra-maximal 4000-m cycling time trials.

A remaining question is how much the VO2 response is influenced by the nature of the athlete and the nature of the sport. In the present study we used very well-trained sub-elite athletes, with a MRT of 18.8 s on a 1500-m cycling. In an earlier report from our laboratory (11) we noted an even faster VO2 response with a MRT of 15.2 s during simulated speed skating competition of a 1500-m time trial in elite junior speed skaters (including the junior World Champions and holders of 7 of 11 recognized junior World Records). MRT for a fast start cycling time trial of ~120 s (but not with the characteristic burst in peak power) in recreationally, but not highly trained cyclists was 37-40 s (21). In sub-maximal exercise, where truncation of the VO₂ response is not of influence on time constants, fastest T values were 8-10 s, observed in well-trained athletes (23). Training status has been shown to increase speed of muscle VO, kinetics (24) and trained individuals exhibit a faster VO2 response compared to untrained individuals (23). Though the influence of training on VO2 response has been well documented, it is still uncertain what the best actual type of training is to optimize VO₂ response (22). Based on the present results, it might be favorable to train the VO2 response not only by aerobic training, but also by intensive interval training. In terms of modeling, there is understandably an interest in modeling the behavior of elite athletes. It may be that even if the relative intensity of the start has little effect on the VO2 response to exercise, that these individuals still have remarkably fast VO2 kinetics. Likewise, at the other end of the performance continuum, sedentary individuals or patients with cardio-respiratory pathologies have slow $\dot{V}O_2$ kinetics (28) that need to be taken into account when modeling human locomotor activities.

Conclusion

The higher initial burst in peak power output in 750 m was accompanied by a faster $\dot{V}O_2$ response. To make optimal use of the aerobic system, the initial burst of power output that is characteristic for time trial exercise of maximal effort seems to be of high importance.

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CHAPTER 7 RELATIVE IMPORTANCE OF PACING STRATEGY AND MEAN POWER OUTPUT IN 1500-M SELF-PACED CYCLING

Hettinga FJ, De Koning JJ, Hulleman M, Foster C. Relative importance of strategy and mean power output in 1500-m self-paced cycling. (submitted).

Abstract

Both mean power output and the distribution of the available energy over the race, i.e. pacing strategy, are critical factors in performance. The purpose of this study was to determine the relative importance of both pacing strategy and changes in mean power output to performance. Six cyclists performed four 1500-m ergometer time trials (~2 min). For each subject, fastest (F) and slowest (S) time trials were compared. The relative importance of the measured differences in mean mechanical power output and pacing strategy was determined with the energy flow model. First, the effect of the difference in mean power output was calculated. Second, by systematically varying anaerobic energy distribution based on mean data over all trials, keeping total energy constant, performance outcomes of different pacing strategies were determined. The difference in final time between F and S was 4.0 ± 2.5 s. F was performed with a higher mean power output (F: 437.8 ± 32.3 W; S: 411.3 ± 39.0 W), a higher aerobic peak power (F: 295.3 ± 36.8 W; S: 287.5 ± 34.7 W) and a higher anaerobic peak power (F: 828.8 ± 145.4 W; S: 649.5 ± 112.2 W) combined with a relatively higher, but statistically unchanged anaerobic rate constant (F: 0.051 \pm 0.016 W; S: 0.041 \pm 0.009 W). The changes in mean power output (63% anaerobic, 37% aerobic) largely explained the differences in final times. Athletes chose a different pacing strategy close to optimal for their physiological condition in both F and S. Athletes seemed to be able to effectively adjust their pacing profile based on their 'status of the day'.

Introduction

Besides the absolute amount of energy that can be produced over a race, the distribution of this available energy, i.e. pacing strategy, is a critical factor in sports performance (4, 9, 28, 29). Optimal performance in endurance sports is determined by the mean power output that can be generated by an athlete in relation to the power losses to the environment, which in turn are determined by the chosen pacing strategy (28, 29). Several studies have already explored the importance of pacing strategy experimentally and a variety of pacing patterns have been found, depending on exercise duration, exercise conditions and sport (1-3, 6, 8, 10, 11, 14, 18, 26, 30). For the shorter distances (< 2 min), where mean velocities are higher and less time is needed to complete the race, the balance between power production and power losses is different from the longer distances and a pacing strategy with a relatively fast start is optimal (6). Within one competitive distance, results from competitions show that various pacing strategies (and thus velocity profiles) are used by different athletes. Since the power that is necessary to overcome air frictional forces is non linearly related to velocity, variations in the velocity profile over the race will influence the fraction of total produced energy that has to be used to overcome these forces. In principal, each deviation from the average velocity will result in higher costs associated with air resistance. The fraction of total produced energy that is inevitably carried over the finish line in the form of kinetic energy associated with the velocity of the athlete has its influence as well. With an equal amount of energy to distribute, a lower kinetic energy at the finish (and a lower velocity) needs to be compensated by a higher velocity earlier in the race, leading to an uneven velocity profile. Optimal performance would be reached when lower kinetic energy at the finish is not surpassed by higher frictional costs resulting from the uneven velocity profile (29). In particular for the middle distances of about 2-min duration, investigating optimal pacing strategy is of interest. Here, a fast start, leading to an uneven pacing profile. but lower finishing velocity, has important advantages for performance and the distribution of anaerobic energy over the race is therefore expected to be particularly relevant. It is unknown to what extent variations in power production and variations in power losses contribute to day to day variation in intra-individual self-paced performance (i.e. final times) and in which range variations in pacing strategy occur. It is not practical to experimentally validate the relative importance of minimizing energy losses and differences in power production on day to day variation in performance. However, performance can be modeled based on measurements of self-paced performance.

Modeling performance with an energy flow model provides a tool to study the effect of various performance determining factors in sports without the necessity for extensive experimentation (28) and has been shown to result in reasonably accurate prediction of performance in both cycling (6, 7, 20, 21, 28, 29) and speed skating (4,5,29). Simulation techniques can be used to optimize pacing strategy with the great advantage that numerous pacing strategies can be tested under constant environmental and internal conditions. An energy flow model that simulates different

profiles of energy distribution accurately has been introduced (4, 6, 29) and applied to optimize pacing strategies for Olympic athletes in middle distance exercise such as 1500-m speed skating or 1000 m and 4000 m cycling. By systematically varying anaerobic energy distribution, keeping total energy constant, performance outcomes of different pacing strategies were determined. The model predicted that a pacing strategy with a faster start resulted in better performance in middle distance exercise and also showed that the fastest performances were accomplished by the athletes generating highest mean power output over the race. The present study will now use the energy flow model and study multiple self-paced races. Within subject variations in pacing strategy will be investigated to: 1) determine the relative importance of changes in pacing strategy and mean power output for performance and 2) to determine how close well-trained athletes are to their optimal pacing strategy. Determinants of both inter- and intra-individual differences in performance will be analyzed.

Methods

Subjects

Six well-trained male cyclists participated in this study. The cyclists were competitive at a regional level and were habituated to cycling time trials in a laboratory setting. All subjects provided written informed consent prior to participation, and the protocol had been approved by the human subjects committee at the University of Wisconsin-LaCrosse. The testing took place approximately one month after the end of the competitive season, during a period of light training (90-120 min at intensities generally below the ventilatory threshold performed 3-4 days per week).

Table 1: Characteristics of the subjects: Age, height, body mass, ventilatory threshold (VT), maximal oxygen consumption (\dot{VO}_{2max}) and maximal power output at which \dot{VO}_{2max} was attained ($P\dot{VO}_{2max}$).

	Mean value ± SD
Age (years)	32.1 ± 10.8
Height (cm)	177 ± 6
Body mass (kg)	72.1 ± 7.2
$VT (\%\dot{VO}_{2max})$	68 ± 8
\dot{VO}_{2max} (I·min ⁻¹)	4.1 ± 0.1
PVO _{2max} (W)	354 ± 17

Values are means ± SD.

Experimental set up

Each subject completed an incremental test followed by four self-paced 1500-m time trials (~ 2 min), a type of exercise in which both aerobic as well as anaerobic energy expenditure are important (13). The time trials were performed with a 48 to 96 h interval between trials, on a racing cycle interfaced with a wind load simulator with a

heavy flywheel (Findlay Road Machine, Toronto, Canada). Previous studies from our laboratory have shown that this apparatus provides realistic perceptual and power output responses during time trials (8, 10, 11). Power output, accumulated distance and elapsed time were recorded every second using a SRM dynamometer (Koningskamp, Germany). Fastest (F) and slowest (S) time trials for every subject were compared and the relative importance of pacing strategy and mean power output to optimal performance in the day to day variation in self-paced performance was quantified by applying an energy flow model as described in de Koning et al. (4) to the data of these trials. To compare aerobic (Paer) and anaerobic power (Pan) distribution profiles between F and S, data were calculated and fit mono-exponentially per subject, leading to individual fits and parameters for Paer and Pan. For modeling and simulating performance, group mean data of F and S were used, which were first averaged and then fit, and thus give a better representation of the group average power output profiles than mean values for the individual fits, that are necessary to perform statistics. To simulate performances, data were averaged to decrease the fitting error and group mean data of F and S are used for further calculations and discussion.

Incremental test

The subjects completed an incremental exercise test on an electrically braked cycle ergometer (Lode, Groningen, The Netherlands) to allow definition of \dot{VO}_{2max} and the ventilatory threshold (VT). After a 10-min warm-up at a self-selected intensity, subjects cycled 3 min at 25 W. Thereafter, the workload increased by 25 W every min, until the subject could no longer continue. The highest power output maintained for a full minute plus the interpolated power output for fractions of the terminal min was defined as $P\dot{VO}_{2max}$. \dot{VO}_2 was measured by open circuit spirometry (Quinton Q-plex, Seattle, WA). \dot{VO}_{2max} was defined as the highest \dot{VO}_2 observed during 30 s.

Experimental trials

Each time trial session started with a standard warm-up protocol, as described by Foster et al. (10). Gross efficiency (GE) was calculated based on the \dot{VO}_2 and power output during the last 2 min of a 5 min sub-maximal ride at a velocity requiring a \dot{VO}_2 equal to 90% of ventilatory threshold observed during the incremental exercise test. Next, the 1500-m cycling time trial was performed. Before each subsequent time trial, the subjects were provided with feedback about their velocity profile and average velocity in consecutive segments of preceding time trials, just as an athlete would be aware of previous performances and split times in previous competitions. The only instruction given before each time trial was to finish in the shortest possible time. To provide general motivation, there was a 'lab record' sheet posted in the laboratory listing the best performances from previous studies in our laboratory.

Modeling performance

In level ground cycling, power produced by the cyclist is predominantly used to overcome air frictional forces and rolling resistance. The interplay between power production and these power losses result in temporal changes in kinetic energy (dE_{kin}/dt) of the rider (see equation 1), leading to a certain velocity profile over the race. Taking the universal law of conservation of energy as a basis, performance can be described by the use of a power equation (4, 5, 6, 20, 21, 28, 29) as presented in equation 1. By using this model including expressions for the power production (Ptot) and power dissipation (P_{friction}) from experimental data, performance can be simulated and the velocity profiles can be obtained.

$$P_{tot} = P_{friction} + dE_{kin}/dt$$
 [1]

The power used to overcome rolling resistance and air friction drag (P_{friction}) can be described by equation 2. To model the performed time trials the parameter values k₁ and k₂ were obtained from the relation between power and velocity (v) of the used ergometer ($k_1 = 0.249$, $k_2 = 4.25$).

$$P_{\text{friction}} = k_1 \cdot v^3 + k_2 \cdot v$$
 [2]

The mechanical power production (Ptot) was measured by the calibrated SRM system and equaled the sum of mechanical power production by the aerobic (Paer) and anaerobic (Pan) energy system (equation 3).

$$P_{tot} = P_{aer} + P_{an}$$
 [3]

Aerobic power

Conform our procedures in previous modeling studies (4, 6, 20, 21), the VO₂ response during the 1500 m was fit by a mono-exponential model (1) and multiplied with the energy equivalent of oxygen (20.98 kJ per liter O_2) and gross efficiency, resulting in a rate constant (λ) and a maximal aerobic power output (P_{aermax}) that can be reached during the time trial (equation 4). Because the cardio-dynamic phase in the early VO₂ response does not directly represent active muscle O₂ utilization, this phase was omitted from the fitting fields based on visual inspection.

$$P_{aer} = P_{aermax} \left[1 - e^{-\lambda(t)} \right]$$
 [4]

Anaerobic power:

Pan was calculated by subtracting Paer from Ptot on a second by second basis and was fit to the equation as described by equation 5 (4):

$$P_{an} = P_{ancon} + P_{anmax} \cdot e^{-\gamma(t)}$$
 [5]

 P_{ancon} is the asymptotic lowest value of anaerobic contribution that is reached, representing a constant anaerobic power during the time trial. Above the constant anaerobic contribution, an additional amount of P_{an} is generated that can be described by a mono-exponential equation including an anaerobic maximum P_{anmax} and the anaerobic rate constant γ . For comparing individual data, the value of P_{an} at 122 seconds was used ($P_{ancon122}$), since the fastest time trial lasted 122 s. Peak anaerobic power (P_{anpeak}) is the sum of P_{ancon} and P_{anmax} , which is a variable more relevant to performance than either model parameter P_{ancon} and P_{anneak} separately.

Calculating relative importance of changes in mean power output (MPO) and pacing strategy

In reality, anaerobic work is not a constant value, and aerobic power profiles might differ slightly between strategies. Using the energy flow model, effects of the measured changes in mean anaerobic power output and in mean aerobic power contribution to day to day variation in performance will therefore be calculated. Firstly, the model was applied to the measured data for the fastest (F) and slowest (S) trials. Secondly, in a substitution process values for variables found for the fastest trials were used to remodel the performance of the slowest trials in the following way: first Paer profiles of S were substituted by Paer profiles of F, and then the difference in mean power output between F and S was added to the anaerobic power output profile of S, keeping the relative distribution of anaerobic energy the same. The difference between the modeled performance and the remodeled performance accounts for changes in mean (anaerobic or aerobic) power output. The remaining part of the day to day variations in final time is attributable to variations in pacing strategy.

Contributions of variations in pacing strategy due to changes in the relative contribution of energy losses can also be calculated. Since pacing strategy in the supra-maximal domain is mainly determined by the anaerobic energy distribution (14), the effect of manipulations of the anaerobic parameters P_{ancon} , P_{anmax} and γ was studied, based on the group mean data of all F and S trials. The parameters of the anaerobic power expression were varied systematically with the restriction that total anaerobic work that could be generated over the race was kept constant. Different combinations of P_{anmax} , γ and P_{ancon} lead to different modeled performance times. The combination of P_{anmax} , γ and P_{ancon} leading to the fastest performance time is defined as the optimal performance and describes the optimal pacing strategy.

Inter-individual differences in performance: 'winner' vs. 'loser'

In addition to studying intra-individual differences in performance, the energy flow model was applied to data for the best subject ('winner') and slowest subject ('loser'), averaged over their 4 trials. The relative importance of changes in mean power output

and pacing strategy in inter-individual differences in performance was calculated using the method described above.

Statistics

Paired sample t-tests were used to test differences between each individual's fastest trial and the slowest one. Statistical significance was accepted when p < 0.05.

Results

Gross efficiency (GE)

Mean GE for all subjects was $19.5 \pm 0.7\%$ and did not differ between F and S trials.

Final time

Mean final time averaged over all trials was 130.8 ± 4.7 s. Mean final times for F and S were 129.1 \pm 4.1 s and 133.1 \pm 4.8 s respectively, differing significantly with a mean difference of 4.0 ± 2.5 s. The time at 750 m was 61.8 ± 1.9 s for F and 64.6 ± 2.8 s for S, with a mean difference of 2.8 ± 1.7 s.

Power output

Mean power output profiles for both F and S are shown in figure 1.

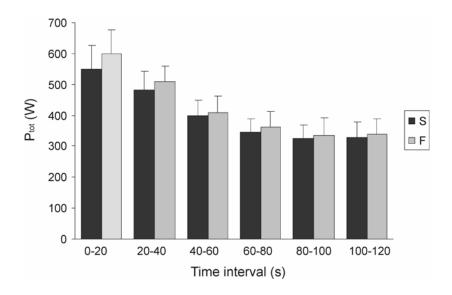


Fig. 1: Mean power output averaged over time intervals of 20 s for the faster (F) and slower (S) trials.

Group mean data, based on the individual fits

Group mean data based on the individual fits for aerobic and anaerobic power profiles were used for statistically comparing F with S and presented in table 2.

Table 2: Group mean data based on individual fits for aerobic and anaerobic power profiles of the fastest (F) and slowest (S) trials, simulated by the energy flow model are shown. Aerobic power profiles are defined by a mono-exponential equation described by the maximal aerobic power (P_{aermax}) and an aerobic rate constant (λ). Anaerobic power profiles are defined by a mono-exponential equation described by the anaerobic peak power (P_{anpeak}), an anaerobic rate constant (γ) and a constant value for anaerobic power, continuously generated over the time trial (P_{ancon}). Values for P_{ancon} are given at t = 122 s. Maximal anaerobic power, the sum of P_{anpeak} and P_{ancon} , is a variable more relevant for performance and thus also displayed. Lastly, average mean anaerobic (P_{an122}), aerobic (P_{aer122}) and total (P_{tot122}) power output over the race are presented, calculated by taking the surface under the first 122 s of the power curves are shown.

	Fastest	Slowest
P _{aermax} (W)	295.3 ± 36.8*	287.5 ± 34.7
λ (s ⁻¹)	0.087 ± 0.014	0.085 ± 0.017
P _{anmax} (W)	828.8 ± 145.4*	649.5 ± 112.2
P _{anpeak} (W)	794.2 ± 176.3*	623.5 ± 125.2
γ (s ⁻¹)	0.051 ± 0.016	0.041 ± 0.009
P _{ancon} (W)	38.9 ± 44.2	32.1 ± 17.6
P _{an122} (W)	170.0 ± 24.0	151.9 ± 16.2
P _{aer122} (W)	267.8 ± 29.8	259.5 ± 27.7
P _{tot122} (W)	437.8 ± 32.3*	411.3 ± 39.0

Values are means \pm SD. Significantly higher values are marked with * (p < 0.05).

Group mean data based on fitting group average data

Aerobic power curves based on fitting group average data are shown in figure 2. The mono-exponential equations for P_{aer} for both S and F are given in equation 1 and 2 respectively:

S:
$$P_{aer}(W) = 287.5 [1-e^{-0.078(t)}]$$
 [1]
F: $P_{aer}(W) = 295.4 [1-e^{-0.082(t)}]$ [2]

Anaerobic power curves based on fitting group average data are shown in figure 2. The mono-exponential equations for P_{an} for both S and F are given in equation 3 and 4 respectively:

S:
$$P_{an}(W) = 26.1 + 605.0 \cdot e^{-0.039(t)}$$
. $P_{ancon122} = 31.6 \text{ W}$, $P_{anmax} = 631.1 \text{ W}$. [3]

F:
$$P_{an}(W) = 30.8 + 712.8 \cdot e^{-0.042 (t)}$$
. $P_{ancon120} = 35.0 \text{ W}$, $P_{anmax} = 743.6 \text{ W}$. [4]

Mean P_{an} over the first 122 s (P_{an122}) was 166.2 W (F) and 151.3 W (S), calculated by taking the surface under the modeled curves of P_{an} . Mean P_{aer} over the first 122 s (P_{aer122}), calculated by taking the surface under the modeled curves of P_{aer} was 267.1 W for F and 258.3 W for S. Mean total power output over the first 122 s (P_{tot122}) thus was 433.3 W (F) and 409.6 W (S), resulting in a difference in P_{tot122} of 23.7 W. 63% of

this difference was caused by the difference in anaerobic contribution of 14.9 W, and 37% was caused by the difference in aerobic power contribution of 8.8 W.

Relative importance of changes in mean power output and pacing strategy, calculated by simulating performance

Simulated velocity profiles for F and S are shown in figure 2 (bottom left). Final time for the simulated F-trials based on group mean data was 129.6 s (0.5 s slower than measured final time of F) and final time for the simulated S-trials was 132.5 s (0.6 s faster than measured final time of S). Difference in simulated final time between F and S was 2.9 s. To obtain the same mean anaerobic power output for F and S, the difference in mean anaerobic power output of 14.9 W between the group mean data curves of F and S was added to the anaerobic power curve. Simulating S with the aerobic power profile of F, and the mean anaerobic power output of F, showed that differences in final time could almost fully be attributed to the difference in Ptot122. Pacing strategy seemed to be rather stable and could only account for a difference of 0.03 s in final time.

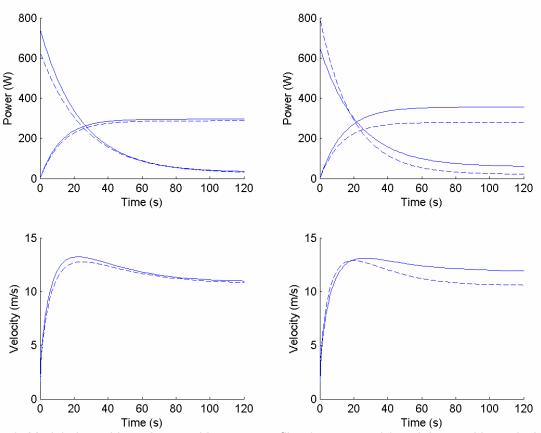


Fig. 2: Modeled aerobic and anaerobic power profiles (upper panels) and the resulting velocity profiles (bottom panels) of fastest (F) and slowest (S) (left panels) and 'winner' vs. 'loser' (right panels) are presented. Solid lines represent the F trials and the 'winner', dashed lines represent the S trials and the 'loser'.

Table 3: Aerobic and anaerobic power profiles of the 'winner' and 'loser', simulated by the energy flow model are shown. Aerobic power profiles are defined by a mono-exponential equation described by the maximal aerobic power (P_{aermax}) and an aerobic rate constant (λ). Anaerobic power profiles are defined by a mono-exponential equation described by the anaerobic peak power (P_{anpeak}), an anaerobic rate constant (γ) and a constant value for anaerobic power, continuously generated over the time trial (P_{ancon}). Values for P_{ancon} are given at t=122 s. Maximal anaerobic power, the sum of P_{anpeak} and P_{ancon} , is a variable more relevant for performance and thus also displayed. Lastly, average mean anaerobic (P_{an122}), aerobic (P_{aer122}) and total (P_{tot122}) power output over the race, calculated by taking the surface under the first 122 s of the power curves are shown.

	'Winner'	'Loser'
P _{aermax} (W)	355.7	279.2
λ (s ⁻¹)	0.072	0.081
P _{anmax} (W)	650.9	798.5
P _{anpeak} (W)	592.3	777.9
γ (s ⁻¹)	0.045	0.053
P _{ancon} (W)	60.94	21.81
P _{an122} (W)	164.1	138.2
P _{aer122} (W)	309.3	247.2
P _{tot122} (W)	473.3	385.4

Values are means ± SD.

Simulating optimal pacing strategy

Performance outcomes of different simulated combinations of anaerobic parameters are shown for both F and S in figure 3. The top panels display the effect of the combination of P_{anmax} , γ and P_{ancon} on simulated performance time. The lower the position on the surface, the faster the time will be. For clarity, the surface of the top panels is transformed to a contour in the bottom panels of the figures. The space between contour lines in the bottom panels indicates 0.1 s time difference. Because of the restriction that the amount of anaerobic work in the performed simulations is equal for all simulations, P_{anmax} , γ and P_{ancon} are not independent. For that reason only two of them need to be displayed. We chose for γ and P_{ancon} , but a different combination leads to comparable figures. The combination of P_{anmax} , γ and P_{ancon} as employed during the time trials is indicated in the bottom panels by an asterisk. It can be seen that subjects are close to an optimal pacing strategy within their range of possibilities in both their best and their worst trial.

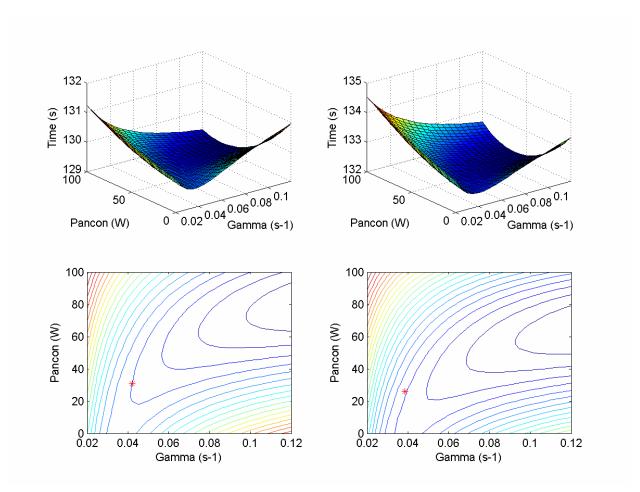


Fig. 3: Different combinations of the anaerobic parameters gamma (y) and P_{ancon} are shown, with corresponding final times, for fastest performance (F, left panels) and slowest performance (S, right panels). The lowest part of the surface represents the fastest final time. Contour plots of these graphs are also shown (bottom panels). The space between contour lines indicates 0.1 s. Only Pancon and the anaerobic rate constant (y) were displayed, since the amount of anaerobic work is the same in all simulations, meaning that P_{anpeak} , P_{ancon} and γ are interdependent. Displaying different combinations will lead to comparable plots.

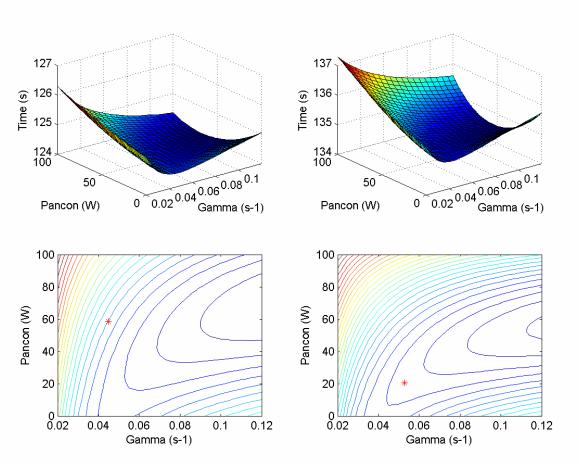


Fig. 4: Different combinations of the anaerobic parameters γ and P_{ancon} are shown, with corresponding final times, for the 'winner' (left panels) and 'loser' (right panels). The lowest part of the surface represents the fastest final time. Contour plots of these graphs are also shown (bottom panels). The space between contour lines indicates 0.1 s. Only P_{ancon} and γ were displayed, since the amount of anaerobic work is the same in all simulations, meaning that P_{anpeak} , P_{ancon} and γ are interdependent. Displaying different combinations will lead to comparable plots.

Inter-individual differences in performance: 'winner' vs. 'loser'

Studying inter-individual differences, it was found that the best performer cycled a final time of 122.8 ± 1.4 s, with a halfway passage at 61.3 ± 1.5 s (averaged over 4 trials). The slowest subject cycled the trial in a final time of 135.4 ± 1.3 s, with a halfway passage at 65.2 ± 2.3 s.

The modeled aerobic and anaerobic power profiles of the 'winner' and the 'loser' are presented in table 3 and figure 2 (upper right panel). The resulting simulated velocity profiles are shown in figure 2 (bottom right panel). Simulated final time of the 'winner' was 124.7 s, simulated final time of the 'loser' was 134.9 s, resulting in a difference in simulated final times of 10.2 s. This difference was largely caused by the difference in P_{tot122} between the 'winner' and 'loser' of 87.9 W. 62.1 W of this difference (71%) was

caused by a difference in aerobic contribution, 25.8 W of this difference (29%) was caused by a difference in anaerobic contribution.

When the energy flow model was remodeled in the way that the slowest performer had the mean power output of the fastest, final time would be 124.4 s, thus 0.3 s faster than the 'winner'. Performance outcomes of different simulated combinations of anaerobic parameters are shown for the 'winner' and the 'loser' are shown in figure 4. The 'winner' chose a pacing strategy that was relatively further away from the optimal pacing strategy.

Discussion

It has been shown that differences in pacing strategy and thus velocity profiles were largely determined by differences in the distribution of the available anaerobic energy (14). The present work studied pacing strategy by analyzing differences in simulated anaerobic power profiles. Maximal laboratory data seem to provide accurate means of modeling cycling performance (12) and an energy flow model has previously led to accurate predictions of sports performance (4, 5, 6, 20, 21, 28, 29). The energy flow model has successfully been used to model the effect of applying different pacing strategies on performance outcome (5, 6). To minimize errors, performance of F and S was in the present study modeled based on averaged data, resulting in final times within a range of accuracy of approximately 0.6 s. The measured difference of about 4 s between better and worse performance within individual athletes was reduced to about 3 s. Thus, the energy flow model underestimate the difference in final times, and differences might be even larger in real life.

Comparing simulated anaerobic power profiles, as well as 750-m passage times, it was clearly shown that faster performance was accomplished with a relatively faster pace early in the race. Peak anaerobic power was higher for F and the athletes performed the first 750 m of the race relatively fast compared to the second half. But though peak anaerobic power differed between F and S and different power and velocity profiles were shown for F compared to S (Fig. 1 and Fig. 2), athletes were close to their optimal pacing strategy in both F and S (Fig. 3), which did not leave much room for a pacing strategy related improvement in performance. This finding suggests that experienced athletes possess a 'performance template' that is relatively fast obtained (18), but not necessarily a 'fixed' pattern that is the same under all conditions. Athletes seem to be able to effectively adjust their pacing profile based on their 'status of the day' and a higher mean power output is accompanied by a higher peak anaerobic power output (see Fig. 2). Based on task remaining, momentary power output and estimated energetic reserves (11, 25) a performance template is formed, taking the finishing point of exercise into account (26), but apparently also taking the individual's sense of physical condition into account. According to Craig (3), humans possess a cortical image of homeostatic afferent activity reflecting physiological condition of the body tissues, located in the dorsal posterior insula. In the right anterior insula, a meta-representation of the primary interoceptive activity is

represented, causing a 'feeling' on the basis of the homeostatic condition of the individual. Possibly, this feeling enables the athlete to determine initial pace based on physiological state. It also might enable the athlete to choose the right pace during the exercise, where continuous adjustments in power output associated with information processing between the brain and peripheral physiological systems are made, also associated with homeostatic control (23, 24). Pacing strategy seems to be an internal negotiation from the start onwards, comparing actual sensed fatigue with the expected state of fatigue (11), where the rate of perceived exertion (RPE) might be the conscious interpretation of the actual fatigue (19, 23).

Though a different pacing strategy was chosen in F compared to S, the variation in pacing strategy was not responsible for explaining intra-individual differences in selfpaced performance. Better performance was largely explained by day to day variations in Ptot122 within a range of 24 W. The larger part of this difference (63%) was caused by differences in contribution of the anaerobic energy system. No differences in GE were found for F and S, so differences could not be attributed to a difference in GE. Interestingly, the aerobic system was of large importance explaining the difference in performance between 'winner' and 'loser' (the inter-individual performance). A difference in peak aerobic power of about 75 W accounted for 71% of the difference in mean power output (87.9 W) between 'winner' and 'loser'. 1500-m cycling is a supra-maximal middle-distance effort lasting about 2 min. In efforts at comparable intensities and comparable duration such as 800 m running, both the aerobic and anaerobic system are of great importance (17) and confirm the present result. It was also shown that superior performance in 800 m running depended more on the aerobic than on the anaerobic energy system (22). This finding suggests that not only sprint-type athletes, but also endurance athletes can perform well on middle distance exercise, which seems to be the case looking at candidates competing at world level in a comparable effort such a 1500-m speed skating.

Furthermore, the range in which pacing strategy can alter final times in self-paced exercise was not very large (Fig. 3) compared to differences that are made by increasing mean power output. A variation in peak power output of about 200 W, accompanied by the resulting small changes in the rate constant and Pancon, was found, affecting power balance, and thus performance. Applying different pacing strategies results in different performance outcomes depending on two mechanisms involved in efficiently distributing a given amount of energy to achieve the highest possible mean velocity over the race (6, 28, 29). The most important mechanism determining optimal pacing strategy for longer events is, that part of the produced energy has to be used to overcome frictional forces due to air friction. Pair is non linearly related to velocity, and variations in velocity profile will influence the fraction of total produced energy that has to be used to overcome air frictional forces. If the fraction of energy that is lost to overcome frictional forces is lower, more energy can be used to accelerate the center of mass of the body and final time will be faster. Taking only this mechanism into account, distributing energy evenly over the race results in optimal performance. An even paced strategy is described by a high y in

combination with a high peak power, to get up to speed as soon as possible. After the initial acceleration phase, anaerobic energy is distributed evenly, with as a result an even velocity profile (6). The other mechanism involved in achieving the highest possible mean velocity over the race with a given amount of energy is associated with the velocity at the finish and the related kinetic energy of the body at that point. It is clear that this kinetic energy is produced by the energy systems during the race, because the race was started with a standing start. Theoretically speaking, the smaller the kinetic energy at the finish, the larger portion of the produced energy is available to overcome resistive forces. As pointed out in the introduction, with equal performance a lower velocity at the end of the race must be accompanied by a higher velocity earlier in the race, leading to non linear increases in energy lost to air friction. Choosing the optimal pacing strategy has thus to do with finding a balance between a high velocity earlier in the race and a low velocity later on. Based on this mechanism, anaerobic energy at the finish (P_{ancon}) and the combination of P_{anmax} and γ leading to high power output in the first part of the race must be combined optimally. This is particularly relevant in short exercise events where high velocities are attained (6, 28, 29). A pacing strategy resulting in a fast 1500-m final time thus should get up to speed as fast as possible (high γ, high P_{anpeak}), with a corresponding P_{ancon} that is not too high. In the present 1500 m, benefits of a more even-paced pattern seem to be canceled out or close to canceled out by the relatively higher Pancon at the finish in F compared to S. This results in relatively small differences in performance caused by pacing strategy in the self-paced range as shown in figure 3. To see meaningful differences in final times, pacing strategies must be changed dramatically, outside the range of self-paced performance and it would be interesting to study what would happen if a theoretical optimal pacing strategy would be imposed and athletes are deliberately not performing their 'own' pacing strategy. Further, it seems that distribution of the available energy according to a fast start strategy is a more important factor in short lasting events, such as a 500 m speed skating, where a high peak power output was correlated with personal best times, and better sprinters seem to be able to liberate more energy at the onset of exercise than lower level performers (3), and thus the benefit of a lower amount of kinetic energy left at the finish outweigh the disadvantage of the higher resistive losses due to the non even-paced pattern of energy distribution.

Though pacing related differences in final times in the range of self-paced intraindividual performance are relatively small, a high initial peak power output seems to be associated with a faster final time and is associated with the better performance, supporting earlier findings (6, 28, 29). A beneficial result of a high initial peak power is an acceleration of the VO₂ response (16) and a higher aerobic peak power as shown in the present study. A fast start might even enable the athlete to realize a higher anaerobic mean power output, though a recent study from our lab did not find a significant difference in anaerobic work with different imposed pacing strategies (15). Studying the range of differences between the winning and losing athlete it was evident that the winner had a relatively low peak power output and was relatively far

away from the optimal pacing strategy, which cost about 0.3 s. Because of the higher generated mean power output, which is clearly of larger importance, the winner still won. Pacing strategy becomes more relevant when athletes have the same amount of power to distribute, and small differences in total power output can lead to the difference between winning or loosing. Alternatively, in athletes who are trying to improve their own performance, and have approximately the same total energy available to use, pacing strategy (i.e. efficient use of available energetic resources) can be critical. A mistake in choosing pacing strategy can be significant for individual performance.

Conclusion

The main finding of the present study was, that the day to day variation in intraindividual performance was mainly determined by differences in mean anaerobic
power output that was generated. To a lesser extent differences in mean aerobic
power output were of importance. In inter-individual performance, differences in
aerobic contribution had a large effect on performance and mainly determined the
difference between winner and loser. Pacing strategies were close to optimal in both
F and S and athletes seem to be able to effectively adjust their pacing profile based
on their 'status of the day'. The fastest trials were commenced with a clearly higher
peak anaerobic power output than the slowest trials, but the range in which pacing
strategy varies in self-paced performance does not lead to large differences in
performance outcome.

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CHAPTER 8 EFFECT OF IMPOSING A THEORETICAL OPTIMAL PACING STRATEGY COMPARED TO SELF-PACED COMPETITION IN 1500-M SPEED SKATING

Hettinga FJ, De Koning JJ, Schmidt L, Wind N, MacIntosh B, Foster C. Effect of imposing a theoretical optimal pacing strategy compared to self-paced competition in 1500-m speed skating. (submitted).

Abstract

Humans are well equipped to choose the right pace to perform well on a specific effort, such as the 1500-m speed skating. The purpose of the present study was to 'override' self-paced performance by instructing athletes to execute a theoretically optimal pacing profile. Seven national level speed-skaters performed a self-paced 1500 m and a 1500 m with an imposed pacing strategy. The races were analyzed by obtaining velocity (every 100 m) and body position (every 200 m) with video to calculate total mechanical power output. Together with gross efficiency and aerobic power output, obtained in separate trials, data were used to calculate anaerobic power output profiles. An energy flow model was applied to the 1500-m self-paced performance and a range of pacing strategies was simulated for each individual. Based on the outcome, a theoretically optimal pacing profile was imposed. The resulting performance was compared to self-paced performance. Final times of the imposed strategy trials were about 2 s slower. Total power distribution per lap differed, with a higher value over the first 300 m for the imposed strategy compared to self-paced performance (637.0 ± 49.4 W vs. 612.5 ± 50.0 W). Anaerobic parameters did not differ. The effort of a faster first lap resulted in a higher aerodynamical drag coefficient and probably a less effective push-off. Experienced athletes seem to possess a well-developed performance template and pacing strategy is not easily changed towards a faster start protocol. A faster start protocol seems to have relatively large consequences on speed skating technique, that seems to be incorporated in the pacing template.

Introduction

Modeling studies have suggested that in athletic events of <120 s duration, better results were obtained with a pacing strategy where initial pace was relatively fast (7, 16, 28). A recent study in our lab (16) demonstrated that when a higher mean power output could be generated over the race, athletes commenced their race with a higher peak power. However, in both better and worse performance within the intraindividual range of self-paced performance, the chosen pacing strategy was close to optimal. Pacing strategy seemed to be adjusted to changes in mean power output, that accounted for differences in final times in a range of 4 s and athletes seemed to choose a pacing strategy suitable for their physiological condition from the starting point of exercise. This suggests that the trade-off between extra power early in the race and earlier fatigue was solved by the athlete in a virtual calculation balancing homeostatic disturbances against the goal of competition. Continuous adjustments in power output within an event are associated with information processing between the brain and peripheral physiological systems (22). The conscious product of this processing is reflected quantitatively in the Rating of Perceived Exertion (RPE). Even when incorrect distance feedback is provided, RPE patterns do not change over the course of a race (1) and subjects seem to follow a predetermined exercise template with a growth in RPE that is proportional (scalar) to the estimated event distance (9, 18). Under the same circumstances, this exercise template appears to be rather fixed once learned (17) and it is of importance both in preventing catastrophic disturbances in homeostasis (19, 20) and in maximizing performance. The process of developing and using this template has been referred to as teleoanticipation (24), which may be viewed as internal negotiation regarding estimation of task remaining, momentary power output, the estimated energetic reserves (11, 23), and possibly also the already performed exercise (14).

Experienced athletes seem to be well equipped to determine pacing strategy remarkably accurately relative to momentary conditions. However, modeling studies have consistently suggested that in <120 s events like the 1500-m speed skating, a more all out starting pattern would be even more optimal (7, 16, 28). The present study was designed to investigate what would happen if the chosen self-paced strategy was 'overridden', outside the normal range of self-paced exercise, by imposing a specific pacing strategy based on a theoretically more optimal profile (7, 16, 28). It was hypothesized that a faster initial pace would result in better performance. In speed skating, pacing strategy is of special interest, since this is a sport where a high mean velocity is attained and a large within-race deceleration is routinely observed. To achieve optimal performance, it is important to minimize air frictional losses, which are related to velocity in a curvilinear manner. This means that they are influenced by the pattern of velocity distribution, and thus by pacing strategy. Available energy stores must be distributed in such a way that the interplay between frictional losses and kinetic energy carried over the finish line results in a mean velocity as high as possible (4, 7, 8, 16, 28). Additionally, variations in technique are of large importance in technically difficult sports such as speed skating, where the timing and direction of the push off and maintaining body position is of large importance.

Methods

Subjects

Seven well-trained sub-elite speed skaters participated in this study. All subjects provided written informed consent prior to participation, and the protocol had been approved by the University of Calgary Conjoint Health Research Ethics Board. Subject characteristics are shown in table 1.

Table 1: Subject characteristics: height, body mass and age.

	Mean value ± SD
Height (cm)	181 ± 6
Body mass (kg)	77 ± 5
Age (yr)	20 ± 3

Values are means ± SD.

Protocol

Athletes performed two competitive 1500-m races, one with their self-paced strategy and one with an imposed theoretically optimal pacing strategy based on calculations from the energy flow model. All skating measurements were performed on separate days at an indoor 400-m skating oval located at an altitude of 1035 m above sea level (Calgary, Canada). Before every trial, barometric pressure was measured. Mean ice temperature was measured at eight locations on the ice. From the established relationship between mean ice temperature and ice friction coefficient (5), ice frictional coefficient could be determined. To obtain the individual characteristics that are necessary as input for the energy flow model, gross efficiency (GE) was determined for each athlete in a trial of sub-maximal skating and VO2 response was obtained in a prelimanary trial. Both trials were preceding the experimental self-paced and imposed pacing strategy trials, so in total, 4 speed skating trials were performed. To obtain knee- and trunk-angles, video recordings were made on the straights by digital cameras. Timing of the skater was done by 4 digital cameras at the entry and exit of each corner. From the timing, the average velocity of each segment was calculated and power output was calculated (8). Because of the large acceleration in the initial 100 m of the race, calculating mean velocity over the first 100 m of the race was not accurate enough and velocity at 100 m was assumed to be 1.3 times the mean velocity over the first 100 m, based on second by second velocity profiles of 1500-m Salt Lake City-Olympic data.

Gross efficiency

To obtain gross efficiency (GE) for each subject, subjects were asked to skate 9 laps at sub-maximal effort at an intensity intended to be sustainable for 30-60 min. $\dot{V}O_2$

was measured breath-by-breath using an open-circuit spirometry with a portable metabolic system (Cosmed K4b², Rome, Italy). Before each test the gas analyzer was calibrated with room air and reference gas (15.19% O_2 , 6.06% CO_2). Volume measurement was calibrated using a Cosmed 3 I calibration syringe. Knee- and trunkangles were obtained, as well as skating velocity, to determine total power output. Based on calculations of total power output and $\dot{V}O_2$ and RER (12), GE could be determined, as described in De Koning et al. (8).

VO, response

In a 1500-m trial preceding the self-paced and imposed pacing strategy experimental trials, $\dot{V}O_2$ response was registered on a second by second basis. Subjects were instructed to finish as fast as possible and to skate as if it was a competition. The $\dot{V}O_2$ response was fit by a mono-exponential model (2), that has also been applied to time trial exercise (7, 8, 15, 21). Multiplied with gross efficiency and the energy equivalent of oxygen (20.98 kJ per liter O_2), this resulted in a description of performance involving a time constant (τ) and a maximal aerobic power output that can be reached during the time trial (P_{aermax}) (see equation 1). Because the cardiodynamic phase in the early $\dot{V}O_2$ response does not directly represent active muscle O_2 utilization, this phase was omitted from the fitting fields based on visual inspection.

$$P_{aer} = P_{aermax} \left[1 - e^{-(t)/\tau} \right]$$
 [1]

Experimental trials

The first experimental race was a self-paced race and was part of a national competition. Together with the GE obtained during the sub-maximal trial and the VO₂ response obtained in the pre-trial, this race was used to obtain model parameters for the energy flow model as described by De Koning et al. (7, 8). In the imposed strategy trial, a pacing strategy closer towards the theoretically optimal profile was imposed, which was outside their normal range of pacing strategies. Based on the predictions of the energy flow model, that will be described below, athletes were instructed to skate the first part of the race at a relatively faster pace, also conform to results of de Koning et al. (7) and Hettinga et al. (15). The skaters practiced the initial part of their imposed pacing strategy during two training sessions. During the first training session the skaters completed 3 x 300 m as fast as they could. On the second training, the skaters performed two or three times a 700 m as fast as possible. To compare pacing strategies between the two experimental trials, mean velocities were corrected for different circumstances by normalizing to mean velocity. To identify the effect of a changing skating technique during the race, air friction coefficient (kair) (25) was measured and compared between trials.

Modeling self-paced vs. imposed performance

To identify the optimal combination of anaerobic energy parameters, resulting in the fastest final time, an energy flow model was applied to the collected data. Mechanical

power produced by the speed skater (P_{tot}) is predominantly used to overcome air frictional forces and ice resistance. The interplay between P_{tot} and these power losses results in temporal changes in kinetic energy (dE_{kin}/dt) of the skater (see equation 2), leading to a certain velocity profile over the race. All components in this equation represent mechanical power. By using the energy flow model including expressions for the mechanical power production (P_{tot}) and mechanical power dissipation ($P_{friction}$) that were based on experimental data, performance of both the self-paced as well as the imposed trial was simulated and the velocity profiles over the race were obtained (7, 8, 21, 27, 28).

$$P_{tot} = P_{friction} + dE_{kin}/dt$$
 [2]

The power used to overcome ice resistance and air friction drag ($P_{friction}$) can be described by the summation of P_{air} (equation 3 and 4) and P_{ice} (equation 5).

$$P_{air} = k_{air} \cdot v^3$$
 [3]

The air friction coefficient (k_{air}) is described as in equation 4 and v equals velocity in a situation with no wind velocity, as in the present study.

$$k_{air} = (0.0205 \cdot I \cdot m^{(1/3)} \cdot (0.798 + 0.0132 \cdot F) \cdot (0.167 + 0.00757 \cdot G) \cdot H \cdot bp/1013$$
 [4]

This equation includes body height (I), body mass (m), trunk angle (F), knee angle (G), drag coefficient (H) that is dependent on v (25), and bp/1013 is barometric pressure expressed relative to normal pressure of 1013 hP.

 P_{ice} was calculated from the skater's body weight (m · g), the coefficient of ice friction (μ) and v (6) (equation 5). μ was determined based on the relation between the temperature of the ice and the ice friction (5).

$$P_{ice} = \mu \cdot m \cdot g \cdot v$$
 [5]

 P_{tot} equals the sum of mechanical power production by the aerobic (P_{aer}) and anaerobic (P_{an}) energy system (equation 6).

$$P_{tot} = P_{aer} + P_{an}$$
 [6]

 P_{an} was calculated by subtracting P_{aer} from P_{tot} and was fit to an equation as described by equation 7.

$$P_{an} = P_{ancon} + P_{anmax} e^{-\gamma(t)}$$
 [7]

A constant anaerobic power is generated during a time trial, described by P_{ancon} , the asymptotic lowest value of anaerobic contribution. Above the constant anaerobic contribution, an additional amount of P_{an} is generated that can be described by a mono-exponential equation including an anaerobic maximum P_{anmax} and the anaerobic rate constant γ (8, 16). For comparing individual data, the value of P_{ancon} at the end of exercise is taken. Peak anaerobic power (P_{anpeak}) is the sum of P_{ancon} and P_{anmax} , which is a parameter more relevant to performance than either model parameter P_{ancon} and P_{anpeak} separately.

Optimal pacing strategy

Since pacing strategy in the supra-maximal domain is mainly determined by the distribution of anaerobic energy (13), the effect of manipulations of the anaerobic parameters P_{ancon} , P_{anmax} and γ was studied. The parameters of the anaerobic power expression were varied systematically with the restriction that total work generated over the race was kept constant. Different combinations of P_{anmax} , γ and P_{ancon} lead to different modeled performance times. The combination of P_{anmax} , γ and P_{ancon} leading to the fastest performance time is defined as the optimal performance and describes the optimal pacing strategy. Based on the prediction of the energy flow model, the coaches' advice and 300-m sprint times, the skaters were instructed to start their imposed strategy trial at a pace that was relatively fast and outside their normal pacing range.

Statistics

Self-paced and imposed pacing strategies were compared per lap with ANOVA repeated measures (strategy x lap). The parameters of the anaerobic power profiles were compared with a paired sample t-test (p < 0.05).

Results

GE measurement

All subjects completed all trials. Mean GE of the skaters was $18.7 \pm 1.0\%$, skated at a mean power output of 185.7 ± 16.0 W, with a mean velocity of 9.59 ± 0.40 m·s⁻¹ and a mean RER of 0.98 ± 0.04 . Mean knee-angle was $110.7 \pm 9.8^{\circ}$, mean trunk-angle was $18.5 \pm 6.0^{\circ}$.

VO₂ response

The mean amplitude of the \dot{VO}_2 response was 3.3 ± 0.3 l·min⁻¹, which corresponds with a mean aerobic peak power output, its mechanical equivalent, of 218.7 ± 20.6 W. Mean time constant was 8.0 ± 2.7 s. Final times were 121.99 ± 2.94 s and relatively slow due to extra wind and breathing resistance from the metabolic system. The fact that it was a test trial and no real competition could have affected motivation. In figure 1, a typical example of \dot{VO}_2 response is shown of a representative subject.

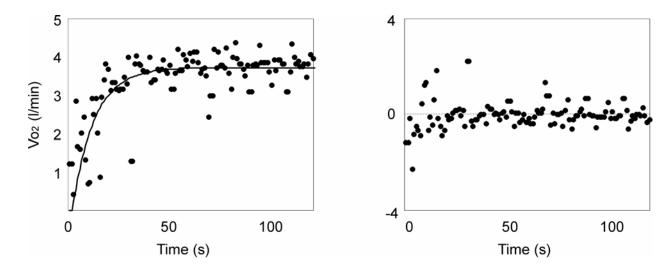


Fig. 1: Typical example of the \dot{VO}_2 response of a representative subject for the 1500-m time trial (left panel). Residuals were calculated by subtracting the measured \dot{VO}_2 from the mono-exponential fit (right panel).

Self-paced trial vs. imposed strategy trial

Ice-friction coefficient was slightly higher in the imposed strategy trial (0.0047 \pm 0.0000), where ice temperature was -4.5 \pm 0.0°C compared to the self-paced trial (0.0044 \pm 0.0002), where ice temperature was -6.2 \pm 1.1°C. Barometric pressure was also slightly higher in the imposed trial (897.9 \pm 1.2 hP) compared to the self-paced trial (896.6 \pm 0.4 hP).

Final times of the self-paced trial (115.39 \pm 4.45 s) were significantly faster than the final times of the imposed strategy trial (117.29 \pm 3.53 s). Lap times and velocity profiles per lap are shown in table 2. Main effects for absolute and relative velocity (normalized to mean velocity) were found. Values for the aerodynamical drag coefficient (k_{air}) are shown in table 3 and were higher throughout the entire race in the imposed trial compared to the self-paced trial. In table 4, mean P_{tot} per lap is presented. P_{tot} decreased significantly per lap. An inter-action (strategy x lap) effect was found for the distribution of P_{tot} per lap, demonstrating that indeed, different pacing patterns were performed, that resulted in different (relative) velocity profiles over the race.

Table 2: Mean lap times, absolute velocity (v_{abs}) per lap and relative velocity normalized to mean velocity (v_{rel}) per lap.

	Self-paced	Imposed	Self-paced	Imposed	Self-paced	Imposed
	Lap tim	ies (s)	v _{abs} (r	n·s ⁻¹)	v _{rel} (m	ı·s⁻¹)
200	25.80	25.71	12.04	12.08	0.93	0.94*
300 m	± 0.68	± 0.57	± 0.31	± 0.27	± 0.04	± 0.04
700	28.32	28.47	14.14	14.05	1.09	1.10
700 m	± 1.06	± 0.85	± 0.52	± 0.41	± 0.01	± 0.01
1100 m	30.16	30.82*	13.28*	12.99	1.02	1.02
1100 III	± 1.43	± 1.18	± 0.59	± 0.47	± 0.01	± 0.01
4500 m	31.11	32.30*	12.56*	12.09	0.96*	0.94
1500 m	± 1.91	± 1.78	± 0.72	± 0.64	± 0.03	± 0.03

Values are means \pm SD. Significantly higher values (p < 0.05) than the corresponding lap are marked with a *.

Model prediction of the theoretically optimal pacing strategy

Based on GE, \dot{VO}_2 and velocity data obtained in self-paced performance, optimal pacing strategy was predicted. For all subjects, pacing strategy must be changed towards a faster initial pace, conforming with previous data (7, 16, 28). Less energy must be left at the finish, as shown by a lower P_{ancon} and more energy must be used in the early part of the race. The predicted final times resulting from the different simulated pacing strategies for a representative subject are presented in figure 2.

Table 3: Mean air friction coefficient (k_{air}) per lap, mean knee-angles per lap and mean trunk-angles per lap.

	Self-paced	Imposed	Self-paced	Imposed	Self-paced	Imposed
	k _a	ir	Knee-ar	ngle (°)	Trunk-aı	ngle (°)
200	0.149	0.155*	105.1	106.0	18.3	20.4
300 m	± 0.012	± 0.014	± 6.0	± 9.7	± 5.6	± 3.9
700 m	0.136	0.142*	106.9	110.7	19.9	20.4
700 m	± 0.006	± 0.010	± 5.05	± 5.9	± 5.0	± 3.0
1100 m	0.139	0.149*	110.6	117.0	17.9	18.8
1100 111	± 0.010	± 0.012	± 3.0	± 7.9	± 6.0	± 3.5
1500 m	0.146	0.158*	110.6	117.0	19.9	20.8
1500 m	± 0.012	± 0.009	± 7.1	± 4.9	± 7.5	± 3.6

Values are means \pm SD. Significantly higher values (p < 0.05) than the corresponding lap are marked with a *.

Table 4: Total power output (Ptot) per lap.

	Self-paced	Imposed		
	P _{tot} (W)			
300 m	612.5 ± 50.0	637.0 ± 49.4*		
700 m	421.8 ± 62.2	418.7 ± 54.9		
1100 m	341.9 ± 54.2	352.6 ± 71.9		
1500 m	318.3 ± 64.7	291.8 ± 47.6		

Values are means \pm SD. Significantly higher values (p < 0.05) than the corresponding lap are marked with a * .

Anaerobic power distribution

In the imposed pacing strategy trial, anaerobic power profile changed towards the predicted direction, but no significant differences with self-paced performance were found in anaerobic power parameters. In table 5, anaerobic parameters are presented for self-paced and imposed strategy performance.

Table 5: Individual data for the anaerobic parameters P_{anmax} , P_{anpeak} , γ , P_{ancon} and E_{an} . The value for P_{ancon} is the constant value that is attained at the end of exercise.

	Self-paced	Imposed
P _{anmax} (W)	673.0 ± 80.5	710.9 ± 133.5
P _{anpeak} (W)	772.9 ± 36.1	799.0 ± 117.7
γ (s ⁻¹)	0.056 ± 0.013	0.054 ± 0.020
P _{ancon} (W)	102.2 ± 57.0	91.6 ± 49.1
E _{an} (kJ)	23.8 ± 6.1	24.2 ± 5.4

Values are means \pm SD. Significant differences are marked with * (p < 0.05).

Modeled performance

Modeling performance with mono-exponential equations for aerobic and anaerobic energy expenditure, based on data points once per 100 m, leads to an underestimation compared to 'real' performance. Modeled performance was in all cases slower than real performance, with final times for modeled self-paced performance of $116.27 \pm 4.42 \, \text{s}$ (which is $0.88 \, \text{s}$ slower than 'real' performance) and for modeled imposed performance $118.11 \pm 3.54 \, \text{s}$ (which is $0.81 \pm 0.38 \, \text{s}$ slower than 'real' performance). In table 6, differences between real and modeled performance were shown.

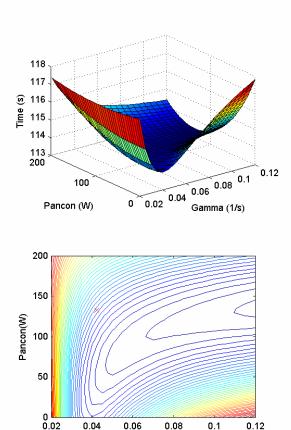


Fig. 2: Different combinations of the anaerobic parameters γ and P_{ancon} are shown, with corresponding final times (upper panel). The lowest part of the surface represents the fastest final time. Contour plots of these graphs are also shown (bottom panel). The space between contour lines indicates 0.1 s. Only P_{ancon} and gamma (γ) were displayed, since the amount of anaerobic work is the same in all simulations, meaning that P_{anpeak} , P_{ancon} and γ are interdependent. Displaying different combinations will lead to comparable plots.

Gamma (1/s)

Table 6: Individual differences between modeled performance and real performance are presented per lap. In the first lap, modeled lap times were faster than real performed lap times. In the rest of the race, modeled lap times were slower than real performed lap times.

	Self-paced	Imposed
0-300 m (s)	0.26 ± 0.17	0.26 ± 0.29
300-700 m (s)	-0.07 ± 0.28	-0.15 ± 0.32
700-1100 m (s)	-0.40 ± 0.34	-0.38 ± 0.39
1100-1500 m (s)	-0.68 ± 0.22	-0.54 ± 0.36

Values are means \pm SD. Significant differences are marked with * (p < 0.05).

Discussion

In the present study, we imposed a theoretically optimal pacing strategy on athletes competing at a national level. Conforming with previous predictions (7, 16, 28), starting with a higher initial peak power output was predicted to lead to better performance. Accordingly, athletes were instructed to start as fast as possible over the first 300 m. Velocity per lap was normalized to mean velocity, to correct for external changes in the environment, and it was shown that a relative increase in initial pace over the first 300 m of exercise occurred, caused by a significant difference in the distribution of Ptot. An interaction effect was found for distribution of P_{tot} per lap, with a higher power output over the first 300 m in the imposed strategy trial, resulting in a higher relative velocity over the first lap and a lower relative velocity over the last lap. Though the different anaerobic fitting parameters separately did not differ, differences in Ptot are suggested to be caused by combined effects of changes in P_{anpeak} and γ (where γ determines the distribution of energy after start). However, the profile of aerobic power distribution was assumed to be equal in both self-paced and imposed strategy performance, but has been shown to be influenced by starting procedure (15). A higher burst in peak power over the first ~15 s of the race seemed to accelerate VO2 response and thus might also account for a faster pace in the initial phase of the race. Unfortunately, VO₂ reponse was not obtained during the races.

Though pacing strategies shifted towards the predicted profiles, it was concluded that it was not easy to draw athletes far away from their self-selected pacing strategy, in agreement with data from Hulleman et al. (17), who showed that an incentive did not change pacing strategy compared to non incentive trials. Athletes seem to have a very accurate system of determining pacing strategy, forming a performance template based on endpoint knowledge, previous experience (11, 22, 24), physiological condition (3, 16) and peripheral factors (3, 22). Based on the awareness of metabolic reserves and homeostatic disturbance in the current exercise bout, this predetermined template is compared to previous experiences, a process that is not easily overridden (17) and more than two training sessions might be necessary for athletes to 'learn' a different pacing pattern. During an entire speed skating season, no differences in performance on a 30 s Wingate test were found (29), which indeed shows that it is not easy to achieve changes in anaerobic contribution in the initial part of the race. Over a longer period of time and training (4 seasons), it was shown that successful speed skaters distinguish themselves by the ability to produce higher power outputs (6). Though natural growth was involved (skaters were 17-21 years old), it also suggests that it might be possible to 'train' optimal pacing strategy and further studies are required.

Though the pattern of anaerobic energy distribution shifted towards a theoretically optimal pacing profile, performance in the imposed trial was about 2 s slower than in the self-paced trial. Mean absolute velocity was higher for self-paced compared to imposed performance, mainly caused by a higher velocity in the latter half of the self-paced race. The worse results of the imposed pacing strategy could not be explained by differences in physiological condition of the athletes, since mean power output was

the same in both trials. Possible differences in motivation were minor since they did not result in differences in total work. External conditions of the environment (barometric pressure and ice friction) varied only slightly in favor of the self-paced condition and could not account for the difference between performances. The explanation for the worse performance in the imposed pacing trial thus has to be found in the technical nature of speed skating, a predefined movement where maintaining body position is of large importance and body weight has to be carried continuously. Not getting preliminary fatigued is of extra importance, since a loss of technical ability results in a change in body position and thus in air frictional coefficient (kair) of the body. Speed skaters have to hold their trunk as horizontal as possible and maintain a small angle in the knee joint during gliding (26). This crouched position is most favorable for reducing wind resistance but also for a proper timing and direction of the push off, determining the work that can be generated per stroke (26). As athletes fatigue, body position changes throughout the race towards a less crouched position (8), which is also predicted based on evidence suggesting restricted blood flow over the race (10). The present study showed that in the imposed strategy trial, air frictional constant was clearly higher throughout exercise, mainly caused by the summation of increased knee- and trunk-angles possibly affecting the effectiveness of movement and decreasing gross-efficiency. It seems that the effort of increasing initial pace results in unfavorable technical changes in speed skating, that are larger than beneficial effects of a faster initial pace. As discussed, athletes seem to have a well developed sense of physical condition (3) and metabolic rate can be accurately adjusted by a feedback control system in which the finishing point of exercise is taken into account, but also biomechanical pattern of motion (24). By comparing their awareness of metabolic reserves and homeostatic disturbance in the current exercise bout to previous experience, a well-evolved system seems to exist making it possible for the athlete to skate a 1500 m without loosing the technical ability.

Unfortunately, it was not possible to register power output directly on a second by second basis as can be done in cycling (16). Anaerobic parameters were thus fit based on registrations of mean velocity once per 100 m. Particularly in the first 100 m, where acceleration is relatively large, this influences determination of total mechanical power output, and consequently the estimation of the anaerobic parameters that are varied to simulate different pacing strategies. Therefore, it was assumed that velocity at 100 m is 1.3 times mean velocity over the first 100 m, based on second by second Salt Lake City-Olympic speed skating data. As shown in table 6, deviations of real performance were equally large over the first 300 m and are not expected to influence existing differences between trials. Anaerobic parameters were in about the same range as shown in cycling and speed skating (8, 16) and differences were as expected based on level of the athletes and differences between sports.

Conclusion

Experienced athletes seem to possess a well-developed performance template and changing pacing strategy towards a theoretically optimal fast start protocol did not result in better performance. Slight deviations from the template apparently have relatively large consequences regarding technical aspects of the movement over the race.

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CHAPTER 9 EPILOGUE: GENERAL DISCUSSION

Epilogue 143

General discussion

The present thesis investigates pacing strategy in supra-maximal athletic performance. Performance can be described mechanically by the use of an energy flow model (13). With this energy flow model, a variety of pacing patterns can be simulated and the interplay of P_{tot} (anaerobic and aerobic), P_{lost} and dE_{kin}/dt , all mechanical terms, can be quantified. The effects of differences in the distribution of energy over the race on performance, i.e. final time, can be determined. In this method, it was assumed that variations in pacing strategy were caused by variations in anaerobic energy pattern. Successively, assumptions involving the anaerobic energy system, issues related to GE, issues related to the aerobic energy system and simulating different pacing strategies will be discussed.

Anaerobic energy system

As shown in chapter 2, variations in power output over supra-maximal races were determined by variations in the anaerobic contribution, while the pattern of aerobic energy distribution did not seem to vary with pacing strategy. This means that modeling different pacing strategies by varying anaerobic energy distribution is an accurate method to model different pacing strategies. In chapter 3, the assumption that anaerobic work is constant with pacing strategy was tested and seemed to be valid in the range of simulated performances. In chapter 8, imposing a different pacing strategy also did not result in differences in anaerobic work over the race, and simulating different pacing strategies keeping the total amount of energy constant appears to be a realistic assumption. It does have to be kept in mind that variations in self-paced performance were for the larger part caused by variations in anaerobic contribution, as shown in chapter 7. Further, a higher mean power output was associated with a higher initial peak power output. It seems that pacing strategy is adjusted to physical condition, and not the other way around. Though different pacing strategies do not influence the total anaerobic work over the race, total anaerobic work is affected by distance as shown in chapter 4. Therefore, it is advisable to measure the total amount of energy that can be generated over the distance of interest, as done in all experiments of the present thesis.

Gross efficiency (GE)

Anaerobic power is calculated by subtracting aerobically produced mechanical power of total mechanical power. The determination of GE is essential for converting the aerobic metabolic energy to mechanical energy. Chapter 5 investigated the effect of ambient temperature on GE showing that GE was lower at higher ambient temperatures (35°C). Consequently, it is important to measure GE under the same circumstances as the performance that is modeled. In speed skating, ambient temperature can be relatively low, while during cycling competitions, ambient temperature can be very high.

In speed skating, GE seemed to be affected by technique to a relatively large extent, as shown in chapter 8. If the ability of the athlete to transport energy to the ice

decreases, the athlete becomes less efficient. If fatigue sets in and knee-angles increase, the push-of becomes less effective and frontal area and thereby air resistance will increase, possibly resulting in a decrease in GE. In cycling, maintaining body position is less of a problem since the athlete's body is supported by the saddle and pacing related changes in technique are less relevant.

Originally, it was planned to investigate the supportability of the assumption of a constant GE over a range of exercise intensities from 20% to 110% $\dot{V}O_{2max}$. By determining heat balance (aerobic metabolic energy + anaerobic metabolic energy mechanical energy = wet heat loss per second + dry heat loss + heat storage), anaerobic metabolic energy could be calculated. Up till now, anaerobic metabolism could only be estimated, making it impossible to reliably estimate contribution via the anaerobic pathways. Unfortunately, wet heat loss could not be monitored due to movement artifacts for the weighing scale under the ergometer, and GE could only be determined in the range of sub-maximal intensities. In the sub-maximal domain, GE was found to increase with intensity (4, 10). GE was shown to reach a constant value (10) towards the higher sub-maximal intensities at ventilatory threshold or 50% of peak power output, mainly because the proportion of energy expenditure that is used for the baseline metabolic rate becomes smaller as total energy expenditure increases. It was also shown that GE should be measured over a period of at least 3 min (1), as is done in all experiments of the present thesis. It is advised to measure GE at an intensity of 50% of peak power output or at ventilatory threshold, averaged over a period of at least 3 minutes of steady state exercise. In the first studies of this thesis, GE was measured at relatively lower exercise intensities, possibly resulting in underestimating GE. An underestimation of GE results in an underestimation of aerobic mechanical power and in an overestimation of anaerobic produced mechanical power.

Aerobic energy system

Chapter 6 studied the effect of differences in pacing strategy on \dot{VO}_2 response. Though chapter 2 showed that the global pattern of aerobic mechanical work over the race did not vary, an effect of different time trial durations/intensities on \dot{VO}_2 response was found. The large initial burst of peak power, that is characteristic for time trial exercise, seemed to accelerate the kinetics of the aerobic system. For modeling performance, it is of importance to model \dot{VO}_2 response based on 'real (simulated) competition' data. In the simulated range of performances, variations in initial peak power are not expected to largely influence \dot{VO}_2 response, since initial peak powers are already relatively high and the initial burst characteristic for sports performance is already performed. For optimal performance, it is advised to start the first seconds of the race fast.

It has to be mentioned that both anaerobic and aerobic power production show a large day to day variation (chapter 7) in self-paced performance and measurements and predictions of optimal pacing strategy for a particular day of performance must be

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made close to the performance and under the same conditions as the performance, as performed in chapter 8, where optimal pacing strategy per subject was determined.

Simulating different pacing strategies

Now the assumptions involving anaerobic and aerobic energy expenditure on the power production side are tested, pacing strategies can be simulated with the energy flow model. Studying the interplay between the different components of the differential equation as discussed in the introduction (equation 1), it is shown that pacing strategy is not a determining factor in day to day variation in self-paced performance (chapter 7). Relative to the physical condition of the athlete, available energy stores were distributed in such a way that energy was efficiently used over the race resulting in a close to optimal mean velocity over the race. A high initial peak power output was associated with better performance and stressed the importance of choosing the right pacing strategy. However, because athletes seemed to follow a predetermined performance template resulting in a close to optimal pacing strategy in both better and worse performance, pacing strategy seems unlikely to account for differences in performance.

So far, all studies were performed in cycling. The last chapter was conducted in speed skating, a technically difficult sport, to investigate the contribution of technical aspects to pacing as well. We tried to impose a theoretically optimal pacing strategy (faster start) outside the range of self-paced exercise. Imposing a faster start strategy resulted in technical changes leading to an increase of the aerodynamic drag coefficient, caused by the combination of changes in knee-and trunk-angles. Possibly, this resulted in a change in GE because of a less effective push off and worse aerodynamics. It seems that adjusting pacing strategy towards a theoretically more optimal profile is not easily done and requires training in increasing P_{anpeak} and maintaining technical skills throughout the race. Technical aspects need to be taken into account in choosing optimal pacing strategy and athletes seem to possess an accurate system to incorporate technical aspects in choosing optimal pacing strategy.

As for the tortoise and the hare story, it can be concluded that when both hare and tortoise would follow their optimal pacing pattern, it is impossible for the tortoise to win. Mean power output is the determining variable in performance. But the hare misjudges his pacing strategy severely and though it seemed impossible: the tortoise wins. Being strong is not enough, you also have to be smart to win. In the athletic world, differences are very small, sometimes only 0.01 of a second separating winning and losing. With such small margins, what counts is how smartly the available energy is distributed. Of two equally strong athletes, the smarter will win by choosing a more optimal pacing strategy. Experienced athletes possess a pretty accurate system to determine their pacing strategies, as shown in chapter 7 and 8. Further research is required to investigate how this system works and can be

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influenced. For example, in a technical difficult sport such as speed skating, it might take a lot of training to be able to perform a theoretically better pacing strategy, since the athlete has to be able to maintain technical ability throughout the race.

As shown in the present thesis, the interplay between P_{tot}, P_{lost} and DE_{kin}/dt seems to be incorporated in a predetermined exercise template that is formed based on earlier experience and rather fixed once learned (5). Adjusting this template towards a theoretically more optimal profile must be accompanied with appropriate training, especially in sports with a large technical component such as speed skating.

Regulating pacing strategy

As described by Ulmer (12) and also discussed by Newsholme (8), in sports competition, the rate of energy consumption has to be arranged with respect to a finishing point, a process called teleo-anticipation. Metabolic rate has to be optimally adjusted. Extra-cellular regulation of metabolism can be brought about by hormones as well as the autonomous nervous system. Extracellular concentrations of serotonin (8), but also the interaction between dopamine and serotonin were suggested to have a regulative role in fatigue (7).

In the process of regulating metabolic rate, efferent signals containing biomechanical patterns of motion (force and displacement) are fed back through afferent somatosensory channels and optimized by motor learning (12), which explains that athletes are able to incorporate their technical abilities in a sport as speed skating into their pacing template. Afferent feedback may also arise from muscle (somatosensory) and peripheral organs, that could possibly alter the interpretation of the afferent feedback, resulting in an internal negotiation taking momentary power output, task remaining and prior experience into account (3). This model has also been applied to fatigue, suggesting that changes in peripheral physiological systems such as substrate depletion and metabolite accumulation act as afferent signallers to the brain (6) to be able to prevent catastrophic disturbances in homeostasis (6, 9) and to maximize performance.

Continuous adjustments in power output associated with information processing between the brain and peripheral physiological systems are made, associated with homeostatic control (11). A down-regulation of muscular activity has been suggested to be the possible cause of decreasing power output with fatigue, but central fatigue in this form does not seem to play a role in middle-distance exercise, as described in chapter 2. Though no down-regulation was shown with fatigue, it is still of interest which central and peripheral factors are taken into account in determining and adjusting pacing strategy. Chapter 2 also indicates that regulatory mechanisms of pacing strategy in supra-maximal exercise probably must be sought in factors associated with anaerobic energy expenditure, since that is the determining factor in pacing strategy.

An interoceptive system associated with autonomic motor control has been suggested, providing a basis for 'emotional awareness' of the state of the body (2). All

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aspects of the physiological condition of all body tissues are suggested to be reflected by a cortical image of homeostatic activity in the brain. This results in a 'feeling', that in high intensity exercise probably reflects fatigue. Based on this feeling, that seems to be measured as a rate of perceived exertion, athletes can choose and adjust their pacing strategies accurately, and form a predetermined exercise template. As suggested by chapter 7, physical condition indeed seems to be taken into account and when athletes are able to generate a higher mean power output, they also start with a higher initial peak power output. Peripheral factors seem to form the basis of central actions. It would therefore be of interest to investigate the relative importance of central and peripheral factors by manipulating both brain and muscles. Neurochemically manipulating central drive in the brain (7) and/or force generating capacity in the muscle during time trials would make it possible to study the effect of peripheral fatigue on central drive, and the effect of central drive on peripheral fatigue.

Optimal pacing strategy

The question particularly relevant for the athlete is of course: what is the optimal pacing strategy? Based on the present thesis, it can be concluded that it seems favorable to start the first 5-15 s as fast as possible. This speeds up the aerobic pathways (chapter 6), and a high initial peak power output also has beneficial effects on the efficient distribution of the available energy, as was shown by the predictions of optimal strategy in chapter 7 and 8. To be able to perform well with a faster start strategy, training is required. Especially in a sport like speed skating, where maintaining body position is of high relevance and fatigue has a relatively large effect on effectiveness of movement, this is essential.

It was shown that athletes seem to have an accurate 'sense' of optimal pacing strategy, since they are relatively close to their optimal pacing strategy under the given circumstances, as shown in chapter 7. Based on previous experience, athletes learn to judge the signals that their body gives them relative to the task they know they have to perform. As shown by the example given of the European Champions Arnoud Okken and Bram Som in the series of the world championships in Japan, even experienced athletes can be 'wrong' sometimes. To be able to judge possibilities accurately, it is advisable for athletes to experiment and keep experimenting with different pacing strategies together with their coaches and in this way obtain a well-documented up to date exercise template, based on prior experience. Accurate models predicting optimal performance could help directing the athlete towards favorable pacing patterns. The present thesis contributed to improve the energy flow model, that can be used to predict optimal performance and favorable pacing strategies.

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SUMMARY

The present thesis investigates several assumptions that are of critical importance in modeling performance and simulating various pacing strategies (chapters 2 to 6) in order to model the effect of different pacing strategies on performance (chapters 7 and 8). Modeling the effect of different pacing strategies can help athletes in their search for their optimal race strategy.

In **chapter 2**, different pacing strategies and their effect on patterns of anaerobic and aerobic energy distribution in cycling were studied. Distribution of anaerobic energy turned out to be the determining factor in pacing strategy. Pattern of aerobic energy distribution did not seem to vary with pacing strategy and different pacing strategies can be modeled by varying the distribution of anaerobic energy over the race, as done with the energy flow model in **chapters 7 and 8**. Also, electromyographic activity was measured and analyzed in combination with power output. This was done to study the central fatigue hypothesis that suggested that a drop in power output at the end of time trial exercise was caused by a central down-regulation. This was not the case and central fatigue in the form of a central down-regulation at the end of exercise did not seem to occur in a 4000-m supra-maximal time trial.

The total amount of anaerobic work that can be produced during a time trial is assumed to be a fixed amount. Chapters 3 and 4 studied this assumption by imposing different pacing strategies and different time trial lengths. Chapter 3 showed that indeed, even when extreme patterns of distributing (anaerobic) power output were imposed, anaerobic and aerobic work did not differ per pacing strategy. Total work differed in favor of an even-paced strategy. Though relevant for sports performance, differences were relatively small (~2%). The assumption that anaerobic work is a constant value independent of pacing strategy seems to be a valid assumption in the range of different strategies that are currently simulated in the energy flow models. Looking at time trials of different distances (750 m, 1500 m, 2500 m and 4000 m) on the other hand, total anaerobic work over the race increased with time trial distance, as shown in chapter 4. The closed loop nature of time trial exercise provides the possibility of varying power output over short periods of time. This may contribute to the larger total anaerobic work than may be accomplished when power output is forced to remain high in open loop exercise. This possibility might be particularly favorable for time trials > 2-3 minutes, the maximal duration assumed to be necessary to exhaust anaerobic resources.

One of the important ingredients in modeling sports performance is gross efficiency (GE). **Chapter 5** investigated the effect of ambient temperature on GE, and showed that GE was about 0.9% lower in heat compared with thermo-neutral circumstances. A high muscle temperature and increases in rectal temperature were not large enough to account for the complete difference and it is suggested that the extra \dot{VO}_2 in the heat is at least partially attributable to the extra myocardial \dot{VO}_2 . A higher

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cardiac output has to exist to continue supplying the muscles with the same blood flow, while extra blood has to be sent to the skin for cooling. The lower gross efficiency in heat could explain about half of the decrements in time trial performance that have been shown to occur in heat.

Limited research has been done on the \dot{VO}_2 response of time trial exercise in the supra-maximal domain or during free range exercise typical of competition. In **chapter 6**, \dot{VO}_2 response during supra-maximal time trial was measured and modeled for time trials of four different distances (750 m, 1500 m, 2500 m and 4000 m). The burst in power output over the first 15 s of exercise, that is characteristic for a start in time trial exercise, was largest in the 750-m time trial. The higher initial burst in power output in 750 m was accompanied by a faster \dot{VO}_2 response, possibly linked to the [PCr] response to exercise. To make optimal use of the aerobic system, the initial burst of power output that is characteristic for time trial exercise of maximal effort seems to be of high importance.

The obtained information from chapters 2 to 6 contributed to making the energy flow model more accurate. In chapters 7 and 8, the energy flow model was used to predict optimal performance and favorable pacing strategies. In chapter 7, the relative importance of changes in pacing strategy and changes in mean power output to variations in self-paced performance were investigated. Four self-paced time trials were performed. Comparing the fastest intra-individual time trial with the slowest, a difference of about 4 s was found, that was caused mainly by variations in mean power output. Pacing strategy was in both fastest and slowest performance close to optimal, and athletes seemed to be able to effectively adjust their pacing profile based on their 'status of the day'. In chapter 8, it was tried to 'override' the athlete's selfpaced performance, by imposing a theoretically optimal pacing profile based on the energy flow model. This study was performed in speed skating, a sport where maintaining body position and technique is of large importance. Athletes were instructed to start with a faster initial pace than their self-paced exercise. Distribution of power output over the race indeed slightly changed towards a faster initial pace, but in contrast to predictions, this did not lead to better performance. Technical aspects also changed with the changing of pacing strategy, resulting in a higher aerodynamical drag coefficient. Technical aspects seem to be integrated in the welldeveloped performance template of the athlete and deviating from that seems to have relatively large consequences for speed skating. Proper practicing before applying such a pacing strategy is necessary.

Optimal pacing strategy

The question particularly relevant for the athlete is of course: what is the optimal race strategy? Based on the present thesis, it can be concluded that it seems favorable to start the first 5-15 s as fast as possible. This speeds up the aerobic pathways (chapter 6), and a high initial power output also has beneficial effects on the efficient

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distribution of the available energy, as was shown by the predictions of optimal strategy in **chapter 7 and 8**. To be able to perform well with a faster start strategy, training is required. Especially in a sport such as speed skating, where maintaining body position is of high relevance and fatigue has a relatively large effect on effectiveness of movement, this important.

Further, it was shown that athletes seem to have an accurate 'sense' of optimal pacing strategy, since they are relatively close to their optimum depending on mean power output they can generate over the race and also technical aspects seem to be incorporated, as discussed in **chapters 7 and 8** respectively. Based on previous experience, athletes learn to judge the signals that their body gives them relative to the task they know they have to perform. To be able to accurately judge possibilities, it is advisable for athletes to experiment and keep experimenting with different pacing strategies together with their coaches and in this way obtain a well-documented up to date exercise template, based on prior experience. Accurate models predicting optimal performance could help directing the athlete towards favorable pacing patterns. The present thesis contributed to making the energy flow model, used to predict optimal performance and favorable pacing strategies, more accurate.

SAMENVATTING

Dit proefschrift onderzoekt verscheidene aannames die van groot belang zijn bij het modelleren van sportprestaties en het simuleren van verschillende wedstrijdstrategieën (hoofdstukken 2 tot en met 6) met als uiteindelijke doel om het effect van verschillende wedstrijdstrategieën op de prestatie te modelleren (hoofdstukken 7 en 8). Het modelleren van verschillende wedstrijdstrategieën kan atleten helpen in hun zoektocht naar de voor hen optimale race strategie.

In hoofdstuk 2 zijn verschillende strategieën en hun effect op de verdeling van de anaërobe en aërobe energie bestudeerd bij het fietsen van een tijdrit. De verdeling van anaërobe energie over de race was bepalend voor wedstrijdstrategie. De verdeling van de aërobe energie over de race leek onveranderd. Het modelleren van verschillende wedstrijdstrategieën kan dus gedaan worden door de verdeling van anaërobe energie over de race te variëren, zoals gedaan is in de hoofdstukken 7 en 8. Electromyografische activiteit was eveneens geregistreerd en geanalyseerd in combinatie met het geleverde vermogen. Het doel hiervan was om de centrale vermoeidheid hypothese nader te bekijken, die suggereert dat een daling in geleverd vermogen aan het einde van een tijdrit veroorzaakt wordt door een centrale afname in aansturing. Dit was niet het geval en centrale vermoeidheid in de vorm van een centrale afname in aansturing aan het einde van inspanning trad niet op in een 4000 m supra- maximale fietstijdrit.

Er wordt aangenomen dat de totale hoeveelheid anaërobe energie die geproduceerd kan worden gedurende een tijdrit een vaste hoeveelheid is. In de hoofdstukken 3 en 4 werd deze aanname getoetst door fietsers verschillende wedstrijdstrategieën en verschillende tijdrit afstanden op te leggen. Hoofdstuk 3 liet zien dat zowel de aërobe als de anaërobe arbeid die tijdens de tijdrit geleverd kon worden niet verschilde tussen verschillende wedstrijdstrategieën, zelfs niet wanneer extreme verdelingen van (anaërobe) energie werden opgelegd. De totale hoeveelheid geleverde arbeid verschilde wel en was het hoogst voor een strategie waarbij energie gelijkmatig over de race verdeeld was. Verschillen waren slechts 2%, maar wel relevant voor sportprestatie. De aanname dat de geleverde anaërobe arbeid over een tijdrit een vaste waarde is, onafhankelijk van de gekozen wedstrijdstrategie, kan gedaan worden voor het scala aan wedstrijdstrategieën, dat momenteel gesimuleerd wordt met het vermogensbalans model. Bij tijdritten van verschillende lengte en duur daarentegen (750 m, 1500 m, 2500 m, 4000 m) nam de totale hoeveelheid geleverde arbeid over de rit toe met de lengte van de tijdrit, zoals beschreven in hoofdstuk 4. Het rijden van een tijdrit (een 'gesloten circuit' inspanning) biedt de atleet de mogelijkheid om het geleverde vermogen over korte tijdsperioden te variëren. Dit draagt mogelijk bij aan de grotere hoeveelheid anaërobe arbeid die geleverd kan worden dan als het geleverde vermogen gedwongen hoog blijft in 'open circuit' inspanning.

Een van de belangrijke ingrediënten bij het modelleren van de sportprestatie is de 'bruto efficiëntie', ofwel 'gross efficiency (GE)'. **Hoofdstuk 5** heeft het effect van omgevingstemperatuur op de GE onderzocht en liet zien dat GE in de hitte 0.9% lager was dan in thermo-neutrale omstandigheden. Een hoge spiertemperatuur en een stijging van de kerntemperatuur konden de afname in GE niet geheel verklaren. Gesuggereerd wordt, dat de verhoogde zuurstofopname in de hitte deels veroorzaakt wordt door een hogere zuurstofbehoefte van de hartspier. Met een gelijk blijvende doorbloeding zal het hartminuutvolume verhoogd moeten worden om ervoor te zorgen dat spieren toch van genoeg bloed worden voorzien, terwijl er extra bloed naar de huid moet worden gestuurd om het lichaam te koelen. De lagere GE in de hitte is verantwoordelijk voor ongeveer de helft van de daling in tijdritprestatie die in de hitte optreedt.

Er is maar weinig onderzoek gedaan naar de VO₂ respons tijdens tijdritinspanningen op supra-maximale intensiteiten. In **hoofdstuk 6** werd de VO₂ respons gemeten en gemodelleerd tijdens vier tijdritten van verschillende lengte (750 m, 1500 m, 2500 m, 4000 m). De initiële piek in het geleverde vermogen tijdens de eerste 15 s van de inspanning, die karakteristiek is voor een tijdritinspanning, was het grootst tijdens de 750 m tijdrit. De hogere initiële piek in geleverd vermogen werd vergezeld door een snellere VO₂ respons, mogelijk verbonden met de [PCr] respons tijdens inspanning. Om tijdens een tijdrit optimaal gebruik te maken van het aërobe systeem lijkt deze geleverd initiële piek in vermogen, karakteristiek voor supra-maximale tijdritinspanningen, van groot belang.

De informatie verkregen in de **hoofdstukken 2 tot en met 6** heeft eraan bijgedragen het vermogensbalans model, dat gebruikt wordt voor het modelleren van sportprestatie accurater te maken. In de hoofdstukken 7 en 8 werd het vermogensbalans model gebruikt om de optimale prestatie en wedstrijdstrategieën te voorspellen. In hoofdstuk 7 werd het relatieve belang van de bijdrage van veranderingen in wedstrijdstrategie en veranderingen in het gemiddeld geleverde vermogen onderzocht. Er werden vier tijdritten met een zelfgekozen strategie gefietst. Bij het vergelijken de beste prestatie met de slechtste prestatie van dezelfde atleet werd een verschil van 4 s gevonden. Dit verschil werd voornamelijk veroorzaakt door variaties in het gemiddeld geleverde vermogen. De gekozen wedstrijdstrategie was voor zowel de snelle als de langzamere tijdritten bijna optimaal en het lijkt erop dat de atleten goed in staat waren hun strategie aan te passen op hun 'status van de dag'. In hoofdstuk 8 is geprobeerd de zelf gekozen strategie van de atleet te 'overschrijven' door de atleet een theoretisch optimaal patroon van energieverdeling op te leggen. Dit optimale patroon werd berekend met behulp van het vermogensbalans model. Deze studie is gedaan bij schaatsen, een sport waarbij het handhaven van lichaamshouding en techniek van groot belang is. Atleten werden geïnstrueerd om sneller te starten dan gewoonlijk. De verdeling van geleverd vermogen over de rit veranderde inderdaad richting een snellere start strategie, maar Samenvatting 155

in tegenstelling tot de voorspellingen leidde deze verschuiving niet tot een betere prestatie. Ook technische aspecten veranderden met het veranderen van wedstrijdstrategie, resulterend in een hogere aërodynamische weerstandscoëfficiënt. De technische aspecten die van belang zijn bij schaatsen lijken geïntegreerd te zijn in een goed ontwikkeld 'sjabloon' dat atleten bezitten. Het hiervan afwijken lijkt relatief grote gevolgen te hebben voor schaatsen. Goede training op het rijden van verschillende strategieën wordt dan ook geadviseerd.

Optimale race strategie

De vraag die relevant is voor atleten is natuurlijk: "Wat is nu de optimale wedstrijdstrategie?" Gebaseerd op dit proefschrift kan geconcludeerd worden dat het gunstig is de eerste 5 tot 15 s zo snel mogelijk af te leggen. Het aërobe systeem komt dan snel op gang (hoofdstuk 6) en een hoog piekvermogen heeft ook gunstige effecten met betrekking tot het efficiënt verdelen van de beschikbare energie, zoals de voorspellingen in de hoofdstukken 7 en 8 laten zien. Om goed te presteren met een snellere start strategie is training noodzakelijk. In het bijzonder voor een sport als schaatsen, waarbij het handhaven van lichaamshouding van groot belang is en vermoeidheid een groot effect heeft op de effectiviteit van de beweging, is dit belangrijk.

Verder bleken atleten een accuraat 'gevoel' voor het kiezen van een goede wedstrijdstrategie te bezitten. Ze kozen een strategie dicht bij de voor hen optimale strategie afhankelijk van het vermogen dat ze in staat waren te leveren tijdens een rit (hoofdstuk 7) en namen hierbij ook technische aspecten in ogenschouw. Gebaseerd op eerdere ervaring leren atleten in te schatten hoe ze de signalen die hun lichaam geeft kunnen interpreteren ten opzichte van de taak die ze moeten uitvoeren. Om in staat te zijn de mogelijkheden accuraat in te schatten wordt atleten geadviseerd samen met hun coaches te blijven trainen met verschillende wedstrijdstrategieën, om zo een goed gedocumenteerd en up-to-date 'sjabloon' te ontwikkelen, gebaseerd op eerdere ervaringen. Accurate modellen, waarmee gunstige wedstrijdstrategieën berekend worden, kunnen helpen de atleet in de richting van mogelijk gunstigere patronen van energieverdeling te sturen. Dit proefschrift heeft eraan bijgedragen het vermogensbalansmodel, dat gebruikt wordt om optimale prestatie en gunstige wedstrijdstrategieën te voorspellen, nauwkeuriger te maken.

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