J. Math. Biol. (1993) 31: 853–878

Journal of
Mathematical
Biology

A mathematical model for the force and energetics in competitive running

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Received 3 September, 1991; received in revised form 15 September, 1992

Abstract. A simple mathematical model for competitive running is developed. This model contains the force and energy reserves as key variables and it describes their relationship and dynamics. It is made up of three submodels for the biomechanics of running, the energetics and the optimization. The model for the energetics is an extension of the hydraulic model of Margaria and Morton. The key geometric parameters of this piecewise linear, three compartment model are determined on the basis of well known physiological facts and data.

Key words: Athletics – Running – Force – Energy – Hydraulic model

Introduction

The running performance of an athlete is primarily determined by his force, energy reserves and breathing capacity. In addition the build of the athlete, the composition of his muscles and other biomedical factors are important. Finally psychological factors influence his performance.

The main objective of this study is the construction of a simple optimization model for the force, velocity profile and energetics in running. Thus we shall neglect psychological factors and individual variations in build, biomechanics etc. This model is an extension of [3]. It is made up of three submodels, which describe the biomechanics, the energetics and the optimization. The model for the energetics is an extension of the hydraulic model of Margaria [16] and Morton [18]. Its geometric parameters will be determined on the basis of well known physiological facts and data. In a subsequent paper this model will be applied to world records and to estimate small effects in running.

It is obvious that such a model can also be applied to other disciplines like swimming, skating, rowing or bicycle riding.

The paper is divided into four sections:

I Biomechanics
II Energetics
III Optimization
IV Discussion

Throughout we use the m kg sec-system, though sometimes the energy will be given in cal, 1 cal = 4,184 J.

For reasons of normalization, which will be explained in Sect. I, most quantities will be defined per kilogram of bodyweight. This normalization procedure effectively makes the mass dimensionless. The derivative of a function g of a single variable will be denoted by g'.

I Biomechanics

For simplicity we describe the motion of the athlete as one dimensional motion, where the coordinate axis coincides with the track. Thus $x(t)$, $v(t)$ will denote the position respectively the velocity of the center of mass at time t.

By Newton's law the equation of motion is

$$Mv(t)' = F_l - R_{\text{int}}(v, x, t) - R_{\text{ext}}(v, x, t) \tag{1}$$

where M is the mass of the athlete and F_l the propelling force generated by the legs. R_{int}, R_{ext} describe the resistive internal respectively external forces.

From this formulation it is clear that we do not take into account the internal degrees of freedom of the athlete. The main reason is that these features would complicate the model considerably and that so far we do not know an adequate description of the biomechanics of sprinting. Indirectly the internal degrees are taken into account via R_{int}, which is used to describe the dissipation of energy within each step cycle. Each step consists of a push off phase, a free flight phase and a braking phase. Thus this periodic acceleration and deceleration or cycles of "positive" and "negative" work [16] is largely responsible for the internal dissipation of energy. The internal friction will contribute to a much lesser extent to R_{int}. We shall also assume that the track is smooth and homogeneous. Then R_{int} and R_{ext} are independent of x.

If an athlete runs with an almost constant velocity v and if $\pm\Delta$ is the variation of the velocity during each step cycle, the kinetic energy lost during each step is approximately given by $2M\,\Delta v$. Thus the dissipated power is $2M\,\Delta v \cdot \nu$, where ν is the step frequency. The corresponding contribution to R_{int} is thus $2M\,\Delta v$. Since runners increase their running speed by lengthening their strides and then by increasing ν, we expect R_{int} to grow at most linearly at higher velocities. This effect is partly counteracted by an increased saving of elastic energy in tendons and muscles at higher velocities. All this explains the following observed facts:

(i) At moderate velocities the oxygen consumption is proportional to v, i.e. R_{int} is approximately constant [22].

(ii) At higher velocities the oxygen consumption grows quadratically in v [8].

For this reason we define

$$R_{\text{int}} = r_{\text{int}} M = \begin{cases} Mr_0 & v \leqslant 6 \\ Mr_0 + Mr_1(v-6) + Mr_2(v-6)^2 & v \geqslant 6. \end{cases} \tag{2}$$

$v = 6$ m/sec delimits the moderate velocity domain, because speeds of $v \geqslant 6$ can be kept up almost indefinitely. For $v \geqslant 6$ (2) is of course just the Taylor expansion of R_{int}, and we shall use actual data to determine the coefficients r_0, r_1 and r_2. In addition we note that the internal mechanical energy is given in [24]

as a quadratic polynomial. R_{ext} describes the air resistance. It is given by

$$R_{ext} = \tfrac{1}{2} AC \cdot \varrho v^2 = Mcv^2. \tag{3}$$

Here A is the frontal area of the runner, ϱ the density of air and C a shape factor. The data of Davies [5] give approximately

$$c = 0.00375.$$

Simple scaling arguments show that the average force per step cycle is approximately proportional to M. Thus $F_l = M \cdot f$ and the equation of motion simplifies to

$$v' = f - r_{int} - cv^2 \qquad v(0) = 0 \tag{4}$$

where f has to be interpreted as force per kilogram of body weight.

(4) is now size independent and can thus be applied to all athletes alike. Since the energy reserves of an athlete are stored in the muscles and since the mass of the muscles is about 40% of the total mass, we expect the reserves to be roughly proportional to M also. For the same reasons as above we define all energy reserve quantities as quantities per kilogram of bodyweight. This will also be applied to the breathing rate.

Muscles and tendons have limited strength. Thus

$$0 \leqslant f \leqslant F.$$

Since man has to run in the field of gravity, the total force generated by the muscles in the legs should be given by

$$F_{tot} = M \cdot \text{Safetyfactor} \cdot g, \qquad g \approx 9.81 \text{ m/sec}^2.$$

The safety factors for animals are usually in the range between 1.5 and 4.5. Assuming a safetyfactor of 3 and an average angle of 20° of the push off leg with the vertical during the acceleration phase, we expect

$$F \approx (3 \cdot g - g) \cdot \sin 20 = 6.7 \text{ m/sec}^2.$$

For the acceleration at the start we get similarly $F \approx (3 \cdot g - g) \sin 45° = 13.7$. This estimate agrees well with values reported in the literature [1, 2, 8].

In the muscles chemical energy is transformed into mechanical energy of the limbs, which in turn is partially dissipated or transformed into energy of the center of mass.

$$\begin{array}{c} \text{Chemical Energy} \rightarrow \text{Total Mech. Energy} \rightarrow \text{Energy of the Center of Mass} \\ \qquad\qquad\qquad\qquad\qquad\qquad \downarrow \\ \qquad\qquad\qquad\qquad\qquad\qquad \text{Dissipated Energy } (R_{int} \cdot v). \end{array}$$

Even though we have described the second transition by the additive term R_{int}, we shall use the factor η, the efficiency, to describe the first transition from chemical energy to total mechanical energy. The use of an efficiency factor η is quite common in engineering and physics wherever energy losses occur for thermodynamic reasons. One knows that η is an increasing function of v [4] and from the result of [4] one deduces by linear regression

$$\eta(v) = 0.6 + 0.04 \cdot (v - 6) \quad \text{for } v \leqslant 6. \tag{5}$$

For simplicity and reasons explained above we put

$$\eta(v) = 0.6 \quad \text{for } v \leqslant 6.$$

Since the cost of transport at $v \leqslant 6$ is approximately 1 cal per meter and kilogram of bodyweight [16], we obtain for the power demand in a steady run at a velocity of 6 m/sec

$$6 \text{ cal/kg} = 6 \cdot 4.184 = 6[r_0 + c \cdot 36]/\eta(6).$$

This gives

$$r_0 = 2.3754. \tag{6}$$

The equation of motion (1) has to be modified when it is applied to the start. For this reason we introduce an extra starting phase, which is the interval $[0, t_0]$ in which the runner attains a velocity of 6 m/sec, which we have used to separate the moderate and high velocity domain. The start is characterized by a reaction time τ, a high initial acceleration for the first few steps and a rather small negative energy [1, 2]. Interpolating the term $r_{\text{int}} + r_{\text{ext}}$ linearly, we can write instead of (1) for the starting phase

$$v' = \begin{cases} 0 & 0 \leqslant t < \tau \\ 1.6F & \tau \leqslant t < \tau + 0.3 \\ F - 2.5104 \dfrac{(t - \tau - 0.3)}{t_0 - \tau - 0.3} & \tau + 0.3 \leqslant t < t_0 \end{cases} \tag{7}$$

with $v(0) = 0$. In addition we set $x(0) = -0.2$, because at the start the center of mass is slightly behind the starting line.

The reaction time τ for sprinters is known to lie between 0.1 and 0.2 seconds [10]. We therefore approximate it by

$$\tau = 0.15$$

for world class athletes.

As we will see later in Sect. III, see also [12, 3], sprints are run mostly with maximal force, i.e. $f = F$. With this, the equations of motion (7) for $t \leqslant t_0$ and (1) for $t \geqslant t_0$ can readily be integrated in closed form. One finds

$$\begin{aligned} t_0 &= (6 - 0.48 \cdot F) \cdot (F - 1.2552)^{-1} + \tau + 0.3 \\ x(t_0) &= 0.072 \cdot F - 0.2 + 0.48 \cdot F \cdot (t_0 - \tau - 0.3) \\ &\quad + 0.5 \cdot (F - 0.8368)(t_0 - \tau - 0.3)^2 \\ v(t_0) &= 6. \end{aligned} \tag{8}$$

Writing (1) in the form

$$\begin{aligned} v' &= F - r_0 - r_1(v - 6) - r_2(v - 6)^2 - cv^2 = a - b(v - 6) - (c + r_2)(v - 6)^2 \\ &= (c + r_2)(w_1 - (v - 6))(w_2 + (v - 6)) \end{aligned}$$

one gets with $c' = c + r_2$

$$\begin{aligned} v(t) &= 6 + w_1(1 - g(t)) \left(1 + \frac{w_1}{w_2} g(t)\right)^{-1} \quad \text{and} \\ x(t) &= x(t_0) + (6 + w_1)(t - t_0) - \frac{1}{c} \ln\left[\left(1 + \frac{w_1}{w_2}\right) \Big/ \left(1 + \frac{w_1}{w_2} g(t)\right)\right] \end{aligned} \tag{9}$$

where $g(t) = \exp(-c'(w_1 + w_2)(t - t_0))$.

By choosing the remaining parameter F, r_1 and r_2 appropriately (1) and (7) respectively (8) and (9) can be used to fit the data and results of Ballreich [1, p. 129]. With a fit of x in the acceleration phase of better than 0.2 m for all six groups one finds

$$r_2 = 0$$

and $5 \leqslant F \leqslant 6.5$, as expected. Moreover $0.685 \leqslant b \leqslant 0.855$. Since $c(w_1 + w_2) \approx 0.85$ the limit velocity v_∞ is attained rather quickly and $x(t)$ is, for $t \geqslant 4$ sec, almost a linear function of t.

In order to compute the power demand in the first seconds, we approximate the energy demand in the starting phase by

$$E_s = 1/2 \cdot 6^2 \cdot \frac{1}{0.6} + 10 = 40.$$

Here the second summand describes the lifting of the center of mass from a crouching position to its normal running position. For $t \geqslant t_0$ the power demand is given by

$$E'(t) = F \cdot \frac{v}{\eta(v)} \tag{10}$$

with v determined by (9). Though (10) can be integrated in closed form, the exact expression is of limited value here.

Sprints are almost entirely run with maximal force. A simple model for sprints is thus determined by (2), (4), (5) and (7) with F, $b = r_1 + 12c$ and τ as parameters. This allows us to describe the sprint world records by setting $\tau = 0.15$ and by choosing F and b appropriately. With

$$F = 6.95 \text{ (men)}, \qquad F = 6.30 \text{ (women) and } b = r_1 + 12c = 0.75 \tag{11}$$

we obtain

Table 1. World records sprint

	Men		Women	
D	T	D_{comp}	T	D_{comp}
45.72	5.20	45.76	–	–
50	5.55	49.81	6.06	51.64
60	6.41	59.83	6.96	61.42
100	9.86	100.29	10.49	99.95
$F = 6.95$	$v_\infty = 11.75$		$F = 6.3$	$v_\infty = 10.93$

Contrary to [3] we have chosen a common r_1 for men and women. The determination of F and r_1 from the sprint data is complicated by the fact that both quantities have an opposite effect on the results. Though the 100 m records will have to be corrected due to the power constraint in the last phase, the approximation is nevertheless quite good, in particular if one remembers that individual variations are of the order of .1 sec or 1.2 m for the 100 m sprint. Thus the error for F and b will be about .1 and 0.05 respectively. While F is of rather

limited importance for longer distance runs, b extends its influence to these distances via the energy consumption. This model extends earlier models for sprints by Keller [12] and Ward-Smith [23], because it takes into account a separate starting phase and because it uses a more general resistance term r_{int}. Numerical comparisons with these models show that (2), (3), (4) and (6) give a better fit of the sprint data and lead to more reasonable model parameters.

One finds that the model of Ward-Smith leads to an infinite acceleration for $t = 0$ and tends to overestimate the performance for short distances and short sprints. The model of Keller leads to unrealistic values for F, $F \geqslant 15$. In addition the values for r_1 for men and women differ too much, $r_1 = 1.8$ men and $r_1 = 2.3$ women. Consequently this leads to an energy consumption which is far too large.

II Energetics

The energy for running and other activities is mainly derived from the following sources, listed in their usual order of recruitment and power

(i) *ATP* and creatine phosphate, the alactic anaerobic store

(ii) Glycolysis, the anaerobic transformation of glucose or glycogen to pyruvic or lactic acid

(iii) the oxydation of carbohydrates, mostly glucose and glycogen

(iv) the oxydation of lipids.

The release of energy from these sources is regulated hormonally and enzymatically by feedback mechanisms in a rather complex way. In particular these processes are rate- and capacity-limited. The following values are given in the literature [16].

Table 2.

	ATP – PCr	Glycolysis	Oxydation
Power (cal/sec.)	13.5(18.1)	6.5	4(5.5)
Capacity (cal)	200	250–350	250000

The results on the capacities have been confirmed approximately also by determining the *ATP*, *PCr* and lactic acid concentrations in the muscle. These values hold for well trained people. For top athletes they will be higher, because such athletes have relatively more muscles, a higher lactic acid tolerance and a more efficient breathing system. In fact, the highest VO_2 max recorded is about 80 mlO_2/min. This corresponds to 6.7 cal/sec or 28.2 W. Values determined by di Prampero et al. measured with participants at the Olympic Games in Mexico City [6] are therefore given in brackets in the above table. In general the anaerobic alactic power seems to be about 3.3 times higher than the aerobic power. Ambiguities arise however, because η, the efficiency is incompletely known and depends on the specific energy providing process. In addition there is a small amount – 100–200 J – of oxygen bound to the myoglobin in the muscle

[13]. *ATP* is the only immediate source of energy for the muscle, and all other processes operate via *ATP* production. There is about three to four times as much phosphocreatine as *ATP* in the muscle [12, 15]. In intensive muscular exercise the *ATP* would be depleted in about 3 seconds. Its stores however are replenished immediately – within milliseconds – by the phosphocreatine. For this reason we shall treat the *ATP* and phosphocreatine system as a single compartment, the phosphagen compartment. This system is regulated enzymatically such that the *ATP* concentration is kept at a constant and high level, because a decrease in *ATP* concentration results in a decrease of power [15, 16, 17]. Thus one can expect the maximal power to remain constant until the phosphagen stores are reduced to about half its values. This would also explain the indication for a compartmentalization of this system [16].

The second most important source of energy is glycolysis. It is triggered by *ADP* and inhibited by *ATP* and breathing. Thus it sets in when the *ATP* concentration begins to decline, after 6 to 7 seconds with supramaximal work. Its main function is thus to offset the decrease in power due to a decrease in *ATP* concentration and to provide power at a high level for a longer time (about 30 seconds) until the breathing rate is sufficiently high. Since the rate of glycolysis depends critically on the ratio of the concentrations of *ATP* and *ADP*, it assumes its maximal power rather quickly, in 2–3 seconds, in maximal work. If this were not so the maximal power would decrease too strongly after 8 seconds. This maximal power output in strenuous exercise is kept up until the inhibition of this process through high lactate concentration sets in.

Since lactate formation is inhibited by breathing (Pasteur effect), the lactate concentration tends to a steady state and the glycolytic power is low after about 90 to 120 seconds of maximal work. Thus the highest lactate concentrations are observed with 400 and 800 m events [11]. Responsible for this inhibition is an increasing acidosis and probably the fact that glycolysis utilizes only about 5.5% of the energy of glucose.

Oxydation of carbohydrates and fats is the most important source of energy for longer lasting activities. The reserves of carbohydrates in general suffice for 1 to 2 hours of activity, e.g. a marathon run, only.

Increased breathing is triggered by *ADP*. Thus it is a delayed process with a time constant of 20–30 seconds and the anaerobic processes listed above are utilized to offset this delay. In addition to an increased burning of fats, fatigue is an important factor for very long distance runs.

So far only individual aspects of the thermodynamics and chemistry of the muscle system have been described and modelled, because a reasonable complete description would be much too complicated. Such a complete model would moreover require a large number of additional parameters and the description of processes, which are as yet only partially understood. For this reason we shall only use a simplified analog model for the energetic processes which take place in the muscle. This model is an extension of the hydraulic model proposed by Margaria [16] and extended by Morton [18]. Basically it is a three-compartment model in which the dynamics is determined by the hydraulic pressure of a liquid representing the energy. Our model consists of three vessels, one for each source of energy in the muscle. These sources are phosphagen, glycolysis, oxydation of glucose and lipids. The corresponding vessels are denoted by (P), (L) and (O) respectively. The volumes (capacities) of these compartments will be denoted by V_P, V_L, and V_O. In contrast to Margaria and Morton we assume V_O to be finite, because glycogen depletion and fatigue are serious problems for long distance

events e.g. 10,000 m or marathon runs. Even though it seems reasonable to consider separate compartments for glycogen and the oxydation of lipids, this cannot be done, because these processes are not additive as far as their power is regarded and because they run largely in parallel using the same substrate, namely oxygen. Thus we consider a single vessel O, which is the union of two vessels with cross-section A_O and A_F respectively. This allows us to model the different rates of depletion of these sources as well as their order of recruitment. Thus the lower part of this vessel has the cross-section A_F, because the oxydation of lipids sets in only when the glycogen is sufficiently depleted. In order to avoid a further unnecessary parameter we have allowed a discontinuous change of parameters. Since A_O and A_F are large this has only a minor effect on the dynamics. In contrast to this the vessels P and L have a constant cross-section A_P and A_L respectively. The basic outline of this model is given in Fig. 1.

Vessel (L) is connected to (P) by a pipe, allowing the liquid – energy – to flow from (L) to (P). This represents the formation of ATP from ADP via glycolysis. The maximal rate of flow in this pipe will be denoted by M_L. This is obviously the maximal glycolytic power. There is also a pipe from (O) to (P), which carries a maximal flow of M_O. M_O is also known as VO_2 max. There are two more pipes leading out of L, which describe the intracellular respectively extracellular oxydation of lactic acid. These processes however are important only in recovery and will not interest us here. Since glycolysis uses glucose as its fuel, one should also consider a connection between (O) and (L).

The primary source of muscular power W is ATP, since it is directly involved in the contraction process of the muscle. In this model the muscular power is represented by the liquid flowing through the tap T with a rate W. This flow causes a drop h in the fluid level in (P). This in turn induces drops in the level of fluid in (L) by l and in (O) by k. The rate of flow between these vessels is regulated in each case by the hydraulic pressure of the fluid. The connection between (L) and (O) however should not be governed by hydraulic forces. The dynamics of this analog model is thus determined by the volumes of the vessels, the maximal rates and the geometry of the system. The geometry of this model is defined as follows. Vessel (P) has a height normalized to 1. The pipe from (L) to (P) is at height λ and the ceiling of (L) is at θ. The pipe from (O) to (P) is at height ϕ, while the ceiling of the bottom part of (O) is at μ. For details and explanations of a closely related model the reader is referred to [18]. For easier comparison we have chosen the same parameters as in that paper. The geometric parameters θ, λ, ϕ and μ model in an indirect fashion the chemistry in the muscle

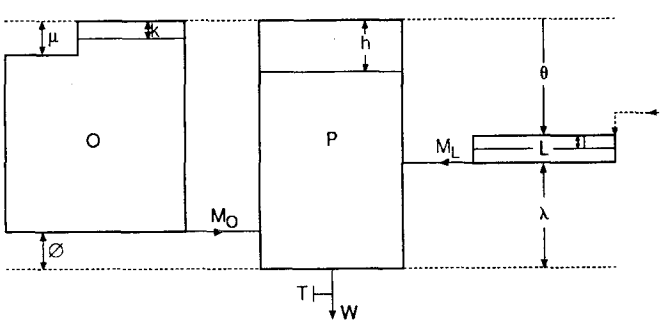

Fig. 1. The hydraulic model

cell. One would therefore expect these parameters to be almost the same for all athletes. We therefore call these parameters almost universal. To a lesser extent this will also hold for the ratios V_P/V_L and M_P/M_L since the muscles have a similar composition for all athletes. With this assumption we neglect of course individual differences in lactic acid tolerance and different amounts of fast twitch and slow twitch fibers. On the basis of our assumptions above we are thus led to

$$V_L \approx 1.4V_P, \qquad M_L \approx 0.45M_P. \tag{12}$$

We are keeping M_O as an independent parameter of course, because the breathing system is largely independent of the muscles.

It remains to determine the geometric parameters of the model. This will be done such that the dynamics of the hydraulic model corresponds most closely to experimental data or physiological principles. Thus we will have to solve the differential equations governing the behavior of this model and compare the solutions with observations. In doing this we shall use as reference parameters $V_P \approx 900-1200$ J, $V_L \approx 1400-1700$ J, $M_P \approx 90-100W$; $M_O \approx 24-29W$ as well as (12), because these values are representative for good athletes [3].

The state variables of the hydraulic model are h, l and k, the levels of the liquid in (P), (L) and (O). Of these h is the most important, since it models the concentration of phosphagen in the muscle.

Since the model is essentially a three compartment system with piecewise constant coefficients, the dynamics of this system is determined in each phase by three coupled linear constant coefficient differential equations. The corresponding characteristic values λ_1, λ_2, and λ_3 are real in all cases and thus the solutions can be written in closed form as sums or integrals of exponentials. Thus we shall only state the equations and the solutions. In some cases it is advantageous to approximate the solutions in order to estimate parameters.

The eigenvalues can usually be grouped into two classes with $\lambda_1, \lambda_2 \approx -0.5$ and $|\lambda_3| \ll 1$. We interpret this fact as a rapid transition to the quasistationary state governed by λ_3.

We shall now discuss the various phases of this model and consider further restrictions later on. Our guiding principle is to determine the values of λ, ϕ, θ and μ such that the dynamics of this model corresponds most closely to observed physiological facts or principles.

Phase 1 No glycolysis; $0 \leqslant h \leqslant \theta$.

The mechanical power W, produced by the muscle will be denoted by W. It is made up from the contributions of all vessels

$$W = W_P + W_L + W_O.$$

ATP is the primary fuel for the contraction of the muscle and the ability of the muscle to contract decreases with the concentration of *ATP* [15, p. 40, 45, 60, 61]. For this reason the enzymes in the muscle operate in such a way that the concentration of the *ATP* is kept constant at the cost of phosphocreatine splitting [17]. Once the phosphagen stores have been emptied to approximately 60% the rising *ADP* concentration triggers glycolysis via the activation of fructokinase. This effect counteracts the decrease in power due to a decrease in *ATP* concentration and provides a high power for about 30 seconds. Thus super-maximal workloads, $W \approx M_P$ can be maintained for about 6–7 seconds without the formation of lactic acid [17]. In terms of the hydraulic model this

means $\theta = 0.55$–0.65. We choose

$$\theta = 0.6. \tag{13}$$

This also explains the compartmentalisation of (P) mentioned in [16]. In the first phase we thus have

$$W = W_P + W_O = A_O k' + A_P \cdot h', \qquad A_O k' = M_O \frac{h-k}{1-\phi}. \tag{14}$$

This is a degenerate system of linear differential equations with constant coefficients. For constant W the solution is given by

$$h(t) = \frac{W}{A_P(\alpha+\beta)}\left[\beta t + \frac{\alpha}{\alpha+\beta}(1-e^{-(\alpha+\beta)t})\right] \tag{15}$$

$$k(t) = \frac{W}{A_P}\frac{\beta}{\alpha+\beta}\left[t - \frac{1}{\alpha+\beta}(1-e^{-(\alpha+\beta)t})\right] \tag{16}$$

where $\alpha = M_O/((1-\phi)\cdot A_P)$ and $\beta = M_O/((1-\phi)A_O)$.

Since the time constant for breathing is approximately 25–20 sec, we get $\alpha+\beta \approx \alpha \approx 1/27$ or $\phi \approx 0.25$–0.3.

If $W/(A_P\cdot(\alpha+\beta)) \approx W(1-\phi)/M_O \leqslant \theta$ then no lactic acid is formed for a long time. Thus the anaerobic threshold is given by

$$W = M_O \frac{\theta}{1-\phi}.$$

It is known that this threshold is approximately $0.6M_O - 0.85$ [15, p. 84, 86] and higher values are observed for better athletes. Thus $\theta \approx 0.8\cdot(1-\phi)$ or

$$\phi \approx 0.25. \tag{17}$$

Phase 1 ends at t_1 when $h(t_1) = \theta$.

Phase 2 The onset of glycolysis, $\theta \leqslant h \leqslant 1-\lambda+l$, $W_L \leqslant M_L$.
As soon as $h > \theta$ the flow from vessel (L) to (P), i.e., glycolysis starts in. Glycolysis is triggered by a higher ADP concentration, which corresponds in this model to larger h values. The differential equations for this process are

$$\begin{aligned} W &= W_O + W_P + W_L = M_O\frac{h-k}{1-\phi} + A_P h' + M_L\cdot\frac{h-l-\theta}{1-\theta-\lambda} \\ A_L l' &= M_L\frac{h-l-\theta}{1-\theta-\lambda} \quad \text{and} \quad A_O k' = M_O\frac{h-k}{1-\phi} + 19M_L\frac{h-l-\theta}{1-\theta-\lambda}. \end{aligned} \tag{18}$$

The second term in the equation for k' describes the glycogen depletion due to glycolysis. This term has not been considered by Morton, though it is of considerable importance. Because A_O is rather large, $A_O \approx 10^5$, the characteristic values of (18) are $\lambda_1 = 0$, $\lambda_2 \approx -0.0016$ and $\lambda_3 \approx -0.7$. Thus the general solution of (18) with constant W is a superposition of three exponentials. Of these the one associated with λ_3 describes the rapid growth of h and a corresponding rapid onset of glycolysis. (18) is only valid as long as $A_L\cdot l' \leqslant M_L$ or

$$h - l - \theta \leqslant 1 - \theta - \lambda. \tag{19}$$

It is known that glycolysis reaches its maximal value M_L in 2 to 3 seconds in supramaximal work. An explicit calculation with $W = 0.8M_P$, $M_P = 100$,

$M_O = 28$, $A_P = 1200$, $V_P = 1700$ gives $h(t_1 + 3) = 0.6638$ and $l(t_1 + 3) = 0.033$. This shows $1 - \theta - \lambda \approx 0.06$–$0.07$ and we set

$$\lambda = 0.33. \tag{20}$$

Analysing (18) further, one can show that the maximal glycolytic power is attained only if W is sufficiently large $W \gtrsim M_L + M_O$.

Phase 3 Maximal glycolytic power, $1 - \lambda + l \leqslant h \leqslant 1 - \phi$.
In this case $W_L = M_L$ and the determining differential equation becomes

$$\begin{aligned} W &= M_O \frac{h - k}{1 - \phi} + A_P h' + M_L \\ A_O k' &= M_O \frac{(h - k)}{(1 - \phi)} + 19 \cdot M_L. \end{aligned} \tag{21}$$

This is a degenerate system of two equations, similar to (14). It can be solved along the same lines as above, by considering first the equation for $(h - k)'$.

It should be noted that such a phase is not considered by Morton. Experimental evidence however seems to indicate that a maximal glycolytic phase exists and is attained rather rapidly in supramaximal work [16, p. 20 Fig. 1.12]. If the power W is not supermaximal one has $1 - \lambda + l > h$ for all t. Then this phase is absent and phase two is directly followed by the next phase.

Phase 4 Maximal breathing rate; $h - k \geqslant 1 - \phi$.
As soon as $h(t) - k(t) \geqslant 1 - \phi$, W_O is maximal and the equations for the system become

$$W = M_O + A_P h' + A_L l' \quad \text{and} \quad A_L l' = M_L \min\left[\frac{h - l - \theta}{1 - \theta - \lambda}, 1\right].$$

For both cases the solution can be determined along the lines stated above.

Maximal power

The equations derived above determine the dynamics of our system uniquely. The solutions would be unrealistic however, because this system does not describe the decline in power i.e., endurance and fatigue correctly. This model has to be augmented by further conditions. This problem has been discussed by Morton in [19], where he relates the maximal power to the remaining glycolytic store. For the following reason we do not consider this assumption to be reasonable.

ATP is the primary fuel for the contraction of the muscle and its ability to contract decreases with the *ATP* concentration, because *ATP* is directly involved in the contraction of the muscle filaments. The maximal power W_m should therefore be a function of the *ATP* respectively phosphagen concentrations alone. In addition the glycolytic power in the muscle is reduced in longer lasting events, due to the Pasteur effect, so that it is not a reasonable indicator for the maximal power.

We have seen above that the *ATP* concentration in the muscle is kept at a constant and maximal level initially at the cost of phosphocreatine splitting [17]. In terms of our model this holds as long as $h \leqslant \theta$. From the assumptions in this paper it is also obvious that $1 - h$, for $h \geqslant \theta$, is a measure for the *ATP* concentration, because then the phosphocreatine concentration is low.

Since this relation is linear, a first order approximation, as suggested by the principle of mass action, leads to

$$W_m = \begin{cases} M_P & h \leqslant \theta \\ M_P \dfrac{(1-h)}{1-\theta} & h \geqslant \theta. \end{cases} \tag{22}$$

It should be clear that W_m is the total maximal power of the muscle.

Since glycolysis is inhibited by high lactate concentrations and local glycogen depletion, the maximal rate of glycolysis decreases with increasing l. The results of Danforth [15, p. 117] suggest a sigmoidal relationship. Taking into account also the buffering in the muscle cell and the slightly elevated pH due to the split phosphocreatine, a piecewise linear approximation suggests

$$W_{L_m} = \begin{cases} M_L & 0 \leqslant l \leqslant \alpha \\ M_L \dfrac{(2\alpha - l)}{\alpha} & \alpha \leqslant l \leqslant 2\alpha \end{cases} \tag{23}$$

where $\alpha \approx (1 - \theta - \lambda)/2$.

This condition has to be modified further because of the Pasteur effect.

In this model (22) and (23) describe fatigue in a rather simple way as a power constraint. In reality fatigue is caused by accumulation of waste products, lack of substrates, increasing lack of neural control and other factors which are as yet not completely understood [14]. In a more extensive model this could be described by state dependent "constants" $F = F(h, l, k)$, $M_O = M_O(h, l, k), \ldots$. In particular F, the maximal force, depends on the present state. However since the force constraint is in general limiting only in the beginning of a run, see Sect. III for this, fatigue will operate mainly via power constraints. We will see later that this model also limits W_{Om}, because V_O is finite.

Maximal work

A work rate W will be defined to be maximal, if the maximal glycolytic rate M_L is attained. It is obvious that (22) and (23) do not influence the governing equations for phase 1. Phase 2, i.e., (18) is not modified either, as long as

$$W \leqslant M_P \frac{(1-h)}{(1-\theta)}. \tag{24}$$

This holds for most of phase 2 if

$$W \leqslant M_P \frac{(\lambda - l)}{(1-\theta)} \approx 0.84 M_P.$$

If (24) is violated during phase 2 then (18) has to be replaced by

$$M_P \frac{(1-h)}{(1-\theta)} = \frac{M_O h}{1-\phi} + A_P h' + \frac{(h - l - \theta)}{(1 - \theta - \lambda)} M_L$$

$$A_L l' = \frac{(h - l - \theta)}{(1 - \theta - \lambda)} M_L$$

once $W = W_m$ (22).

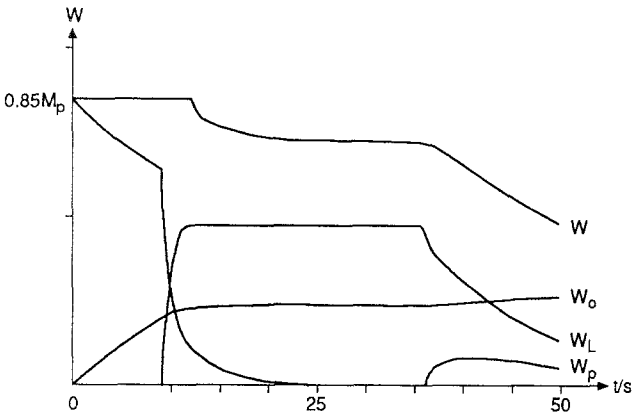

Fig. 2.

Here t_W is defined by equality in (24). This is again a system with constant coefficients. The solutions can therefore be written as a superposition of two exponentials and a constant term.

Thus the case of maximal work covers two subcases. In the first (24) has to be involved during phase 2. This happens for $W \geqslant 0.84M_P$, with our choice of parameters. In the other case (21) is not active during phase 2. An explicit calculation shows that in both cases the maximal glycolytic rate is reached rather quickly, in about 2 to 6 seconds. In general one can continue into phase 3 with W for some further seconds, depending on W, but then the maximal work $W_m = M_P(1-h)/(1-\theta)$ will decrease due to (22) and the decrease of h, which is not compensated by an increase in W_O. This decrease is slower and smaller if W is smaller. In fact it vanishes completely if $W \leqslant 0.737M_P$ – with our choice of parameters. Following this first decrease there is a phase of about 15 seconds in which W_m is nearly constant. Thus h and W_O are nearly constant, too. In this time the glycolytic power is maximal. If W_m is continued further, the maximal glycolytic power decays with a time constant of about 15 seconds. At first this can be compensated by phosphagen, but then h and W_m decay likewise. Thus this second decrease of W_m is caused by (23). In Fig. 2 the development of W_m for $W = 0.85M_P$ is shown.

Heavy work

Heavy work means a power demand W, which is nonmaximal in the above sense, but which is stronger than $0.4M_P$. Thus W is heavy, if $W_1 \leqslant W \leqslant W_2$ where $W_2 \approx 0.73M_P$ and $W_1 \approx 0.4M_P$. With heavy work it is the second part of (23), which becomes limiting at t_2, the end of phase 2, where $l(t_2) > \alpha$, $2\alpha = 1 - \theta - \lambda$ and $4\alpha - 2l(t_2) = h(t_2) - l(t_2) - \phi$. Then (23) effectively decouples the equation for l' from the others and we obtain for the glycolytic power

$$W_L(t) = W_L(t_2)\, e^{-d(t-t_2)} \quad \text{where } d = \frac{2M_L}{V_L}.$$

d^{-1} is thus the characteristic time with which the glycolytic reserves are depleted.

Inserting this expression into the remaining equations gives for $t \geqslant t_2$

$$\begin{aligned} W &= M_O \frac{h-k}{1-\phi} + A_P h' + W_L(t_2)\, e^{-d(t-t_2)}, \\ A_O k' &= M_O \frac{h-k}{1-\phi} + 19 W_L(t_2)\, e^{-d(t-t_2)}. \end{aligned} \tag{25}$$

This system is degenerate and the solution can be written as a superposition of two exponential terms with a linear factor if W is constant or maximal. The leading term describes a rise of h. It has a characteristic time of d^{-1}. This term arises because the phosphagen is used to offset the exponential decrease of glycolytic power. (25) has a quasistationary state at $h \approx W(1-\phi)/M_O$. This state however is not reached because after about d^{-1} seconds (22) becomes limiting and (25) has to be replaced by

$$\begin{aligned} \frac{1-h}{1-\theta} M_P &= M_O \frac{h-k}{1-\phi} + A_P h' + W_L(t_2)\, e^{-d(t-t_2)}; \\ A_O k' &= M_O \frac{h-k}{1-\phi} + 19 W_L(t_2)\, e^{-d(t-t_2)}. \end{aligned} \tag{26}$$

These equations govern the system until the oxydation of lipids starts in. However, in general the oxydation of lipids will be important only for levels of work of less than $0.4 M_P$.

Endurance work

Long lasting athletic events are governed by a power demand $W \leqslant 0.4 M_P$ so that the largest part of the power is provided by breathing. Thus glycolysis as a means to offset the decrease in phosphagen is of lesser importance. In addition, it exerts an adverse effect in endurance work, because it uses glycogen rather inefficiently and because the breathing rate stays almost constant at $M_O \cdot (1-\lambda)/(1-\phi)$ while glycolysis is active.

Numerical computations based on (18), (21) and the other equations indicate that glycolysis is advantageous only for running distances of less than 1500m. A numerical simulation of a 2000m run with V_L as a parameter, for example, yields an optimal result with $V_L = 0$. In addition one finds that the transition region of glycolysis to no glycolysis is rather narrow. The adverse effect of glycolysis in endurance work can also be seen from the following example. In an exercise of constant power $W \approx 0.5 M_P$ glycolysis sets in after about 10 seconds and the glycolytic reserves are used up by more than 95% after about 100 seconds. Thus after 100 seconds the glycogen reserves are reduced to $V_O - 19 \cdot V_L$. With $A_O \approx 10^5$ this corresponds to $k \approx 0.32$, which in turn implies that the maximal breathing rate in this model is at most $0.60 \cdot M_O$, an unrealistic low value. In nature it is the Pasteur effect which inhibits glycolysis at low power demands. Thus for endurance work or running distances $D \geqslant 2000$m, glycolysis should be neglected completely. For such workloads the three compartment hydraulic model reduces to a two compartment system. Thus we have to consider three phases only, phase 1, phase 4 and a power constraint phase, which is controlled by (22) respectively (26) with $W_L = 0$. For constant W, h and k in phase 1 are

given by (15) and (16). Phase 1 ends at t_1 when $(h-k)(t_1) = 1-\phi$. Phase 4 is governed by $h' = (W - M_O)/A_P$ and $k' = M_O/A_O$. It is attained only if $W \geqslant M_O(1 + (A_P/A_O))$. It ends at t_4 when

$$h(t_4) = 1 - \frac{W}{M_P}(1-\theta).$$

The equations in the power constraint case are

$$M_P \frac{1-h}{1-\phi} = M_O \frac{h-k}{1-\phi} + A_P h' \quad \text{and} \quad A_O k' = M_O \frac{h-k}{1-\phi}. \tag{27}$$

This system has $(h, k) = (1, 1)$ as a stable equilibrium. This state cannot be attained however since $k \geqslant \phi$. So (27) will be valid for working times of less than 6 hours only. Its characteristic values are

$$\lambda_1 \approx \frac{1}{A_P}\left(\frac{M_P}{1-\theta} + \frac{M_O}{1-\phi}\right) \quad \text{and} \quad \lambda_2 \approx -\frac{M_O}{A_O \cdot 0.87}.$$

The first eigenvalue describes a rapid transition, with a time constant of about 4 seconds, to a quasistationary state governed by λ_2. Thus for $t \geqslant t_4 + 4(-\lambda_1^{-1})$ we have $1 - h \propto \exp \lambda_2 t$, and correspondingly

$$W_O = \text{const.} \cdot e^{\lambda_2 t}.$$

This is valid as long as $k \leqslant \mu$. For $k > \mu$ we have to replace A_O by A_F. This results in a slight increase of λ_2 to λ_F and a corresponding slight change in the eigenvector. Since these changes are extremely small, they can be approximated by: $A_O \to A_F$, $\lambda_2 \to \lambda_F$, $k(t_4+) = \mu$. $h(t_4+)$ can be determined from the condition that $(h(t_4+) - 1, \mu - 1)$ is the eigenvector for the eigenvalue λ_F.

Thus endurance work is connected with two time constants λ_2^{-1} and λ_F^{-1}. The first describes the local depletion of the glycogen stores in the muscles, while the second is related to the longtime fatigue after the onset of the lipid metabolism. We should therefore expect $\lambda_2^{-1} \approx 5000$ and $\lambda_F^{-1} \approx 30{,}000$.

It should be noted, however, that it is not clear at all whether hydraulic forces can adequately describe the carbohydrate and lipid metabolism, because these substrates are released from their depots during activity. For fatigue the situation is even less clear. These considerations also show that for long times $M_{Om} \approx \text{const.} \cdot \exp(\lambda_2 t)$.

For long distance runs, $D \geqslant 5000$m, this leads in first order to the following simple model. We assume that the velocity profile for the first T_0 seconds, which correspond to $D = 5000$m, is the same for all these runs and that $k(T_0) \geqslant \mu$. Then

$$W(t) = W(T_0)\, e^{(t-T_0)/\tau}, \quad \tau = \lambda_F^{-1}.$$

In first approximation this gives

$$v(t) = v(T_0)\, e^{-(t-T_0)/\tau}$$

and

$$s(t) = 5000 + v(T_0) \cdot \tau(1 - e^{-(t-T_0)/\tau}).$$

These expressions underestimate v and s slightly, because the air resistance is

incompletely taken into account. The error however is less than 5%. For the long distance world records this gives

Table 3.

			Men ($T_0 = 778.39$)				
T (sec)	1628,23	3444,2	3600	4435.8	5358.8	7610	22220
D (m)	10000	20000	20944	25000	30000	42195	100000
D_{comp} (m)	9994	20256	21110	25631	30497	41825	99628
			$v(T_0) = 5.95$ $\tau = 34000$;				
			Women ($T_0 = 877.33$)				
T (sec)	1813,74	2837	4000	8466	26337		
D (m)	10000	15000	21097,5	42195	100000		
D_{comp} (m)	10082	15464	21368	42088	100676		
			$v(T_0) = 5.51$ $\tau = 31000$				

With $M_O = 28.4$ (men) and 23.5 (women) [3] this implies $A_F = 1.13 \cdot 10^6$ and $0.84 \cdot 10^6$ respectively.

In these computations one finds that the 25000, 30000 and 100000m distances are consistently overestimated. Apart from systematic deviations caused by the recruitment of lipids, this may also be due to the fact that these records are old and that these distances are run rarely.

Because of the glycogen depletion in the muscles due to glycolysis and because of the Pasteur effect the dynamics of the hydraulic model have to be modified further. This modification has to reflect the following observed facts: (i) For small W glycolysis is of less importance and the muscle cell prefers oxydative processes to glycolysis. (ii) Glycolysis is low when the breathing is large. Contrary to (ii) I believe that the final spurt in running is performed partly with the aid of glycolysis. Responsible for this effect is probably the recruitment of fast twitch fibres during the final sprint.

Since no lactate is produced below the anaerobic threshold, which is $0.8M_O$ in our model, the simplest way to satisfy (i) is to demand

$$M_L(W) = \begin{cases} M_L(\infty) & W \geqslant 0.8M_O + M_L(\infty) \\ W - 0.8M_O & 0 \leqslant W \leqslant 0.8M_O + M_L(\infty) \end{cases} \tag{28}$$

where M_L is the largest possible rate of glycolysis. In order to satisfy (ii) we also scale V_L like M_L. (28) could be generalized by replacing the factor of .8 above by another value. The motivation for (28) lies in the fact that for submaximal work lower lactate values are measured [9].

III Optimization

In competitive athletics the optimization of force, energy and other factors is of primary importance. It is this aspect, which we want to investigate here on the basis of the models developed above. In order to obtain a tractable model, we

will introduce a few simplifications. Thus we neglect the starting phase and assume that r is a sufficiently smooth version of

$$r(v) = \begin{cases} r(6)\dfrac{\eta(v)}{\eta(6)} & v \leqslant 6 \\ r_0 + r_1 v + cv^2 & v \geqslant 6. \end{cases}$$

Moreover we assume that the constants in the model have approximately the values employed above. Actually we shall consider four models, two for short and medium distances, where the effects of glycogen depletion are neglected. The others describe the situation for longer distances and one model serves to illustrate the Pasteur effect.

The main difficulty with the hydraulic energy model stems from the fact that the dynamics change from phase to phase. We can unify the description however if we use the appropriately modified equations of phase 2. Thus we restrict the optimization model to short and middle distances and we neglect k and write

$$\begin{aligned} v' &= f - r(v) \\ h' &= \frac{1}{A_P} f \cdot \frac{v}{\eta} - \frac{1}{A_P} M_O \frac{h}{1-\phi} - \frac{m}{A_P} \\ l' &= (1-\theta-\lambda)/V_L \cdot m \end{aligned} \tag{29}$$

where we have set $m = (h - l - \theta)/(1 - \theta - \lambda)M_L$ in order to describe phase 2. These equations cover the first phase if $m = m(l, h)$ is 0 for $h \leqslant \theta$ and they cover phase 3 if $m = M_L$ for $h > \theta$ and $h - l \geqslant 1 - \lambda$. They can also be used in the glycolytic constraint phase if m is replaced by

$$M_L \min\left(\frac{h-l-\theta}{1-\theta-\lambda}, \frac{2\alpha - l}{\alpha}\right)$$

for $\alpha \leqslant l \leqslant 2\alpha = 1 - \theta - \lambda$. It is now advantageous to define the various phases by their defining equations, i.e. the various forms of m.

Defined this way, m becomes a continuous piecewise linear function of h and l. Similarly one can handle M_O, but we shall neglect this phase. Thus we treat essentially four phases only, the phases 1, 2 and 3 and the glycolytic constraint phase. For simplicity we shall denote the latter by phase 4 henceforth. (29) has to be augmented by several inequalities. The first

$$0 \leqslant f \leqslant F \tag{30}$$

describes the limitations of the force due to the finite strength of tendons and muscles. It follows from (29) and (30) that v is limited by v_∞, where

$$F = r(v_\infty)$$

if $v(0) \leqslant v_\infty$. If one considers evolution as an optimization process, one would expect

$$\frac{F \cdot v_\infty}{\eta(v_\infty)} \approx M_P$$

so that the power constraint

$$\frac{f \cdot v}{\eta} \leqslant M_P$$

is superfluous. But we still need

$$\frac{f \cdot v}{\eta} \leqslant M_P \frac{(1-h)}{(1-\theta)} \tag{31}$$

which is limiting in the final part of a run. The aim of the athletes is to cover the distance D in the shortest time possible. This is equivalent to maximize

$$D = \int_0^T v(t)\, dt$$

for fixed time T subject to the constraints (29), (30) and (31). This is a problem of optimal control with phase inequality constraints and f as the control variable. The existence of a minimum with a measurable f follows easily from Filipov's theorem [7]. Though Filipov proves his result only for smooth coefficients, his method extends to this slightly more general situation. With the aid of the optimal solution (x, v, h, l, f) we can now define t_1, t_2 and t_3, the end of phase 1, 2 and 3 respectively. If they occur more frequently, we distinguish them by additional indices. The phase boundaries t_1, t_2 and t_3 are characterized by additional constraints. These are

$$\begin{aligned} h(t_1) &= \theta, \qquad l(t_1) = 0 \\ (h-l)(t_2) &= 1-\lambda \qquad \text{boundary between phase 2 and 3} \\ l(t_3) &= \alpha \qquad \text{boundary between phase 3 and 4} \end{aligned} \tag{32}$$

or

$$\begin{aligned} h(t_1) &= \theta, \qquad l(t_1) = 0 \\ (h+l)(t_2) &= 2-2\theta-2\lambda \qquad \text{boundary between phase 2 and 4.} \end{aligned} \tag{33}$$

Thus (32) describes the constraints for a run in which the maximal glycolytic rate M_L is attained, while (33) is valid for a longer distance run.

Necessary conditions for this problem are given by Neustadt [21]. Although his results require smooth coefficients, the proofs carry over to our situation as well [21]. To derive these necessary conditions we have to introduce a quadruple of adjoint variables ψ_0, ψ_1, ψ_2 and ψ_3 for x, v, h and l. They are absolutely continuous in the intervals $(0, t_1)$, $(t_1, t_2), \ldots, (t_3, T)$ and satisfy

$$\begin{aligned} \psi_0' &= 0 \qquad \psi_1' = -\psi_0 + r'\psi_1 - \frac{f}{A_P}\left(\frac{v}{\eta}\right)\psi_2 - \mu f\left(\frac{v}{\eta}\right)' \\ \psi_2' &= \frac{M_O}{(1-\phi)A_P}\psi_2 + \frac{1}{A_P}(\partial_h m)\psi_2 - \frac{(1-\theta-\lambda)}{V_L}(\partial_h m)\psi_3 - \mu\frac{M_P}{1-\theta} \\ \psi_3' &= \frac{1}{A_P}\cdot(\partial_l m)\,\psi_2 - \frac{(1-\theta-\lambda)}{V_L}(\partial_l m)\psi_3 \end{aligned} \tag{34}$$

together with the terminal conditions

$$\psi_0 \geqslant 0, \qquad \psi_1(T) = \psi_2(T) = \psi_3(T) = 0 \tag{35}$$

and the jump conditions

$$\begin{aligned} \psi_2(t_1) - \psi_2(t_1+) &= -\alpha_1, \qquad \psi_3(t_1) - \psi_3(t_1+) = -\alpha_2 \\ \psi_2(t_2) - \psi_2(t_2+) &= -\alpha_3, \qquad \psi_3(t_2) - \psi_3(t_2+) = \alpha_3 \\ \psi_3(t_3) - \psi_3(t_3+) &= -\alpha_4 \end{aligned} \tag{36}$$

or

$$\psi_2(t_1) - \psi_2(t_1+) = -\alpha_1, \qquad \psi_3(t_1) - \psi_3(t_1+) = -\alpha_2$$
$$\psi_2(t_2) - \psi_2(t_2+) = -\alpha_3, \qquad \psi_3(t_2) - \psi_3(t_2+) = -2\alpha_3. \tag{37}$$

μ is a nonpositive function, which differs from 0 only when the constraint (31) is active. Moreover

$$\left(\psi_1 + \psi_2 \cdot \frac{v}{\eta A_P} + \mu \frac{v}{\eta}\right)(g - f) \leqslant 0 \qquad 0 \leqslant g \leqslant F. \tag{38}$$

With the Hamiltonian $\mathscr{H}(f) = \mathscr{H}(v, h, l, \psi_0, \psi_1, \psi_2, \psi_3, f)$

$$\mathscr{H} = \psi_0 v + \psi_1(f - r) + \psi_2\left(\frac{fv}{\eta A_P} - \frac{M_O h}{(1-\phi)A_P} - \frac{m}{A_P}\right) + \psi_3 \frac{(1-\theta-\lambda)}{V_L} m \tag{39}$$

the Pontryagin maximum principle becomes

$$\mathscr{H}(f) = \max_{g \in U(t)} \mathscr{H}(g)$$

where $U(t) = \{g \in [0, F] \mid gv/\eta \leqslant M_P(1-h)/(1-\theta)\}$. By using

$$\psi = \psi_1 + \frac{v}{\eta A_P}\psi_2 \tag{40}$$

this can be simplified to

$$\psi f = \max_{g \in U(t)} \psi g.$$

Thus ψ is a switching function, i.e.

$$f = \begin{cases} 0 & \text{if} \quad \psi < 0 \\ \min \left(F, \dfrac{1-h}{1-\theta}\dfrac{\eta}{v} M_P\right) & \text{if} \quad \psi > 0. \end{cases} \tag{41}$$

For the moment f is undetermined if $\psi = 0$. This condition determines the singular arc. If $\psi \leqslant 0$ then (38) shows $\mu = 0$ and for $0 < f < F$ (38) gives

$$\mu = -\psi \frac{\eta}{v} \quad \text{whenever} \quad f < F \quad \text{and when} \quad \frac{f \cdot v}{\eta} = \frac{M_P(1-h)}{1-\theta}. \tag{42}$$

Thus it is advantageous to replace the conjugate variable ψ_1 by ψ, where

$$\begin{aligned} \psi' = &-\psi_0 + r'\psi - \frac{1}{A_P}\left(r\frac{v}{\eta}\right)'\psi_2 + \frac{v}{\eta A_P}\frac{M_O}{(1-\psi)A_P}\psi_2 + \frac{v}{\eta A_P^2}(\partial_h m)\psi_2 \\ &- \frac{v}{\eta A_P} V_L^{-1}(1-\theta-\lambda)(\partial_h m)\psi_3 - \mu f\left(\frac{v}{\eta}\right)' - \mu \frac{v}{\eta A_P}\frac{M_P}{1-\theta}. \end{aligned} \tag{43}$$

The analysis of our problem is thus largely reduced to a study of ψ based on (34), (35), (36), (37) and (43).

Since the problem is autonomous, $\mathscr{H}$ is constant. This will give us some control of the jump conditions at the phase boundaries. We add that the jump constants $\alpha_1, \ldots, \alpha_4$ occur in our problem as Lagrange multipliers for the constraints (32) and (33) respectively. In order to use the constancy of $\mathscr{H}$ at the phase boundaries we write in the case of (36)

$$0 = \mathscr{H}(t_3) - \mathscr{H}(t_{3+}) = \psi(t_3)(f(t_3) - f(t_{3+})) - \alpha_4 \frac{(1-\theta-\lambda)}{V_L} M_L.$$

If $\psi(t_3) = 0$ we get $\alpha_4 = 0$. If $\psi(t_3) \neq 0$ we have either

$$f(t_3) = f(t_{3+}) = F \quad \text{or} \quad f(t_3) = f(t_3+) = M_P \frac{1-h}{1-\theta} \cdot \frac{\eta}{v}(t_3).$$

In both cases we obtain $\alpha_4 = 0$. Similarly one shows $\alpha_1 = \alpha_3 = 0$. Thus ψ_1, ψ_2 and ψ are continuous across the phase boundaries.

In order to analyze ψ we integrate (34) backwards from T by using the initial conditions (35). In this process we use that the (ψ_2, ψ_3) system is linear with piecewise constant coefficients and $-\mu(M_P/(1-\theta))$ as a possible inhomogeneous term. Thus this system can be solved in closed form, in principle at least.

This and (34) show that $\psi_0 \neq 0$, because otherwise the uniqueness of the solution of (34) would imply $\psi(t) = \psi_2(t) = \psi_3(t) = 0$ near T. This is impossible and we may therefore assume $\psi_0 = 1$.

Since the case $\psi(t) > 0$ almost everywhere leads to an all effort sprint, we may assume $\psi(t) = 0$ for some $t \in (0, T)$. This and the integration of (34) from T backwards imply that the run must close with a power constraint (31) on some final interval $[t^*, T]$ with $t^* < T$. Thus ψ_2 and ψ_3 are negative on (t^*, T). Solving the (ψ_2, ψ_3) equations for all phases separately shows then that ψ_2 and ψ_3 are negative in $(0, T)$ and (t_1, T) respectively.

The analysis of the (ψ_2, ψ_3) system shows moreover that ψ_2 is never strongly negative throughout all phases. This in turn can be used to deduce that ψ is nonnegative. Though this result is intuitively quite obvious, it requires a detailed analysis of the (ψ_2, ψ_3) system for all phases and cases. It is even more remarkable because we have not used that an $f = 0$ phase is associated with "negative work". The most complicated subcase in this analysis occurs at the boundary of phase 1 and phase 2.

This result implies that all phases are passed through in their proper order and that each phase boundary is traversed only once.

An interval $[r, s]$ with $\psi(r) = \psi(s) = 0$ and $\psi(t) > 0$ for $t \in (r, s)$ will be called a positive semiarc of ψ. By using $\psi \geqslant 0$ and properties of the (ψ_2, ψ_3) system as well as (43) one can deduce then that ψ has only finitely many, in fact in most cases only one semiarc, i.e. f is piecewise continuous. Moreover each semiarc is connected with a power constraint. Thus there is no semiarc in phase 1. If phase 3 is present one can show in addition that it does not contain a proper interval where ψ vanishes. Thus such runs are performed with maximal power, with the possible exception of a short time interval in phase 1 and phase 2. We can now summarize our results as follows:

Lemma. *The optimal control problem* (29), (30), (31) *possesses a maximizing solution with a piecewise continuous control variable f which satisfies $f > 0$. Moreover we have $f = F$ on some initial interval $[0, t']$ and $f = M_P(1-h)\eta/((1-\theta)v)$ on some final interval $[t^*, T]$.*

For runs, in which the maximal glycolytic rate M_L is attained, this has the following consequences:
The runner starts with maximal force $f = F$ in the initial time interval $[0, t']$, $\psi(t) > 0$ for $t < t'$. This is followed by a $\psi \equiv 0$ phase until $t = t^*$. For $t' < t < \min(t^*, t_1)$ the motion is governed by

$$\left[\left(r\frac{v}{\eta}\right)'' - \left(\frac{v}{\eta}\right)' \cdot \frac{M_P}{(1-\phi)A_P}\right](f-r) = -\left[\left(r\frac{v}{\eta}\right)' - \frac{v}{\eta}\frac{M_O}{(1-\phi)A_P}\right]\frac{M_O}{(1-\phi)A_P}$$

i.e. v decreases. In order to extend the $\psi \equiv 0$ condition into phase 2, we need

$\psi(t_{1+}) = \psi(t_1) = 0$ and $0 = \psi'(t_1) = \psi'(t_{1+})$. While the first condition holds because of the continuity of ψ, the second condition determines the jump of ψ_3 at t_1.

For short distance runs, $D \leqslant 320$m, this intermediate phase will be absent and one has $\psi(t) > 0$ for all $t < T$. Such runs will be called sprints.

Only if phase 3 is followed by phase 4 can we expect such an intermediate phase, if at all. It should be noted that h' is almost constant during phase 3 so that in runs of this type v is almost constant for a long intermediate interval. The velocity profile is thus almost the same as that obtained by Behncke [3] and Keller [12], though the underlying mathematical reasons are quite different.

In medium distance runs, $D \geqslant 600$m, phase 3, i.e., the maximal glycolytic rate is not attained any more, and phase 2 plays the role of phase 2 and 3 of the shorter runs. For this reason we shall first study runs, which are based on phase 2 and 4 alone, because any optimal solution restricted to (t, T) will be of this form. In long distance runs phase 2 intervals with $\psi(t) = 0$ play a dominant role. For such intervals the (ψ_2, ψ_3) system becomes homogeneous. The solution is therefore a superposition of two exponentials

$$\begin{pmatrix}\psi_2\\ \psi_3\end{pmatrix}(t) = A\, e^{\lambda_+ t}\begin{pmatrix}1\\ a_+\end{pmatrix} + B\, e^{\lambda_- t}\begin{pmatrix}1\\ a_-\end{pmatrix}$$

where $\lambda_\pm$ and $\binom{1}{a_\pm}$ are the eigenvalues respectively eigenvectors of the characteristic matrix. Since $\lambda_+ \approx 0.7$ and $\lambda_- \approx 0.0013$, A must be very small. Thus $-\psi_2(t)$ increases very little, and $\psi' = 0$ shows

$$\frac{d}{dv}\left(r\frac{v}{\eta}\right) \propto e^{-\lambda_- t}.$$

Therefore v will decrease slowly. This is valid until the power constraint (31) sets in, where we have again a short term increase of v. An analysis of the inhomogeneous (ψ_2, ψ_3) system indicates, that there is only one positive semi-arc of ψ during phase 2 and 4. Thus f will in general have at most three discontinuities.

The slow decay of v during phase 1 and phase 2 can be explained as follows. Since the breathing rate of the athlete is tied intimately, via h, to the initial energy expenditure, the athlete will start with a relatively high initial velocity to attain a large W_O, which is for free in this model, since we have neglected k. This is paid for by a higher energy expenditure $r(v)(v/\eta)$. Thus this solution is a sort of compromise between these two conditions. This indicates also that the optimal D, depends only very little on (x, v, h, l, f), i.e. we have a "flat" optimum.

The general solution can now be obtained by joining it with a corresponding solution for phase 1.

This however will in most cases lead to a coefficient A, which is small compared to the other coefficients but still too large. Thus the $\psi = 0$ phase might be even shorter than expected. This could be resolved by an additional boundary condition $v_1 = v(t_1)$ or by smoothing $\partial_h m$. The reasons for this difficulty arise because h controls the breathing rate W_O too strongly. In particular W_O grows too little when a large part of the power is derived from glycolysis. These difficulties can be resolved if one decouples W_O completely from h and l and replaces it in all equations above by

$$\sigma(t) = \sigma_0(1 - e^{-t/30}). \tag{44}$$

The resulting model is then a hybrid between the hydraulic model and that of

Behncke [3]. This model can be studied along the lines above, because the equations for the conjugate variables ψ_0, ψ_1, ψ_2 and ψ_3 are the same as above, only M_O has to be replaced by 0. Thus the lemma and its consequences also holds in this case. As expected v decreases less in the $\psi = 0$ phases in this model than in the previous one. In fact $\psi = 0$ leads to a constant v in phase 1 and a very slowly decreasing v for phase 2. The joining of the solutions for phase 1 and 2 can also be arranged easier in this case. For both models we expect only one power constraint phase at the end of the run.

In (44) σ_0 can also be considered time dependent in order to model long time fatigue, for this see e.g. [3]. The form of the optimal solution is not affected by this modification of σ.

For the long distance model we shall neglect all short time effects due to phosphagen depletion. Thus we combine (L) and (P) into a single vessel $(\mathscr{L})$ with volume V_L. The oxydative processes will be described by the breathing rate σ. The vessel (O) with volume V will be used to describe glycogen and lipid reserves. The state of the system is then defined by v, l and E, where l and E describe the energy reserves in $(\mathscr{L})$ and (O) respectively. In order to model the initial increase of breathing, long time fatigue and the effects of acidosis we shall assume that σ is roughly of the form

$$\sigma \approx \frac{E}{V}\sigma_0(t, l)(1 - e^{-t/\tau}), \quad \tau \approx 30. \tag{45}$$

The first factor indicates that σ declines proportionally to the remaining glycogen stores E. The last factor models the initial increase of σ. Fatigue and the effects of an acidosis imply $\partial_t\sigma_0 \leqslant 0$ and $\partial_l\sigma_0 \geqslant 0$. Of course $|\partial_t\sigma_0|$ and $\partial_l\sigma_0$ will be small. Our assumption $\partial_l\sigma_0 \leqslant 2 \cdot 10^{-4}$ for example means that a maximal acidosis will reduce σ by at most 40%.

In this model the total power is derived from oxydation of glycogen and lipids, σ, and phosphagen and glycolysis P_L. Thus we have

$$\frac{vf}{\eta} = \sigma + P_L.$$

Since glycolysis also uses glycogen as a fuel we see

$$E' = -\sigma - \delta P_L, \quad \delta \approx 19$$

where δ is the energy equivalent of the oxidation of glucose versus glycolysis. The depletion of the glycolysis-phosphagen store finally leads to

$$l' = -P_L.$$

This system can now be rewritten as

$$\begin{aligned} v' &= f - r(v) \\ E' &= (\sigma - 1)\sigma - \delta\frac{vf}{\eta} \\ l' &= -\frac{vf}{\eta} + \sigma. \end{aligned} \tag{46}$$

The constraints corresponding to (30) and (31) are

$$0 \leqslant f \leqslant F, \qquad \frac{vf}{\eta} - \sigma \leqslant P(l). \tag{47}$$

The power constraint function $P = P(l)$ will be a monotone sigmoidal shaped function of l. It may be approximated by

$$P(l) = \min\left(\frac{l}{\varrho}, P_0\right).$$

In this form ϱ is the time constant with which the energy reserves in $(\mathscr{L})$ are depleted [3]. On the basis of (24) one would expect $\varrho \approx 18$ or $P' \approx 0.05$. In this case the optimization of

$$D = \int_0^T v\, dt$$

defines an optimal control problem with a phase inequality constraint; which is obviously an extension of the model in [3]. The qualitative discussion of the solutions of the conjugate variable system proceeds as above and leads to.

Lemma. *The optimal control problem possesses an optimal solution with a piecewise continuous f. An optimal solution has the following form: A short $f = F$ phase is followed by a phase of very slow increase of v, until finally the force and power constraints take over.*

Remark. As in the previous two models, the phase where v is almost constant, corresponds to the $\psi = 0$ domain of the switching function ψ. On this domain the (ψ_2, ψ_3) system is governed by the eigenvalue 0 and $-((\delta - 1)\partial_E \sigma + \partial_l \sigma) = \lambda_-$. Thus this domain is shorter and v increases more strongly if $|\lambda_-|$ is larger. This means the power constraint takes over faster the stronger the effects of the acidosis are felt.

The solution has thus almost the same form as that in [3]. The slow increase of v is a consequence of the long term negative effect of glycolysis. In fact if the glycolytic stores are reduced v becomes more and more constant. It should be noted that the final power and force constraints lead initially to an increase of v. Then v decays slowly with a time constant of 20 to 30 seconds. Thus this also explains the final kick.

We have seen above that for longer distances the Pasteur effect reduces glycolysis. For this reason one should also consider an optimization model for longer distances based on phosphagen (P) and oxydation alone. The state equations for this model are then

$$\begin{aligned} v' &= f - r(v) \qquad h' = \frac{1}{A_P}\frac{fv}{\eta} - \frac{M_O}{A_P}\frac{h-k}{1-\phi} \\ k' &= \frac{M_O}{(1-\phi)A_O}(h-k) \qquad v(0) = h(0) = k(0) = 0. \end{aligned} \tag{48}$$

In addition the constraints (30) and (31) have to be satisfied. In order to apply the Pontryagin maximum principle as above, we introduce the adjoint variables ψ_0, ψ_1, ψ_2 and ψ_3, which satisfy equations, which are similar to (34)

$$\begin{aligned} &\psi_0' = 0, \qquad \psi_1 = -\psi_0 + r'\psi_1 - \frac{1}{A_P} f\left(\frac{v}{\eta}\right)'\psi_2 - \mu f\left(\frac{v}{\eta}\right)' \\ &\psi_2' = \frac{M_O}{A_P(1-\phi)}\psi_2 - \frac{M_O}{A_O(1-\phi)}\psi_3 - \mu\frac{M_P}{1-\theta} \qquad \psi_0 \geqslant 0 \\ &\psi_3' = -\frac{M_O}{A_P(1-\phi)}\psi_2 + \frac{M_O}{A_O(1-\phi)}\psi_3 \qquad \psi_1(T) = \psi_2(T) = \psi_3(T) = 0. \end{aligned}$$

Proceeding as above one determines the properties of the switching function $\psi = \psi_1 + (v/A_P\eta)\psi_2$ by integrating the ψ system backwards from T. With parameters in the range considered above one shows that ψ is nonnegative and has at most one positive semiarc in $(0, T]$. Thus f is piecewise continuous with at most two discontinuities. Contrary to the other two cases one does not have an interval where v is almost constant. The optimal strategy is rather: Attain a high initial velocity and breathing rate, let v decay a little for about 30 seconds and run the remaining time under power constraint conditions with breathing at nearly $VO_{2\max}$. Thus long distance runs are closely correlated to $VO_{2\max}$, as is well known.

So far it has not been investigated in the literature, whether fatigue, caused by lack of substrates, operates mainly via the force or the power of the muscles. In fact, so far the distinction between force and power constraints has not been considered at all.

In my opinion fatigue operates mainly through the power. Intermediate spurts of runners and cyclists on mountain roads seem to support this claim.

IV Discussion

The complete model for the force and energy in competitive running is now the synthesis of these three submodels. Thus the biomechanical part is determined by the starting phase (7), Newton's law (4) for $v \geqslant 6$ as well as (5), (6) and the constraint $0 \leqslant f \leqslant F$. It is connected to the energy submodel via $W = (f \cdot v)/\eta$. The energy model itself is determined by the hydraulic Eqs. (14), (18) or (22) along with the geometric parameters θ(13), ϕ(17), λ(20) and constraints (22), (23) and (28). On the basis of these equations and constraints each runner tries to minimize T given D respectively maximize D given T while using f as a control variable. Thus the running strategy and with it the dynamics is determined by the optimization model.

The application of the optimization models to our problem is complicated by the fact that the initial conditions for the systems equations are given for $t = 0$, whereas the initial conditions for the conjugate system are defined at T. In addition some of the parameters are still undetermined, which makes the optimization even more complicated.

The biochemical submodel extends previous models [12] and [23] in several respects. Firstly it considers a separate starting phase and secondly it uses a more general and realistic resistance expression. By choosing the free model parameters F, b and τ it yields a good fit of the data of Ballreich [1] and of the sprint world records. For good athletes these parameters lie in the ranges $5.5 \leqslant F \leqslant 7.0$, $0.68 \leqslant b \leqslant 0.8$ and $0.10 \leqslant \tau \leqslant 0.25$. The fit of the sprint world records for women can be improved a little if a smaller b and larger τ are chosen. However since it is not clear whether this represents a real effect, we have chosen a common b.

The energy submodel extends the hydraulic model of Margaria [16] and Morton [18, 19] in several respects. Firstly it uses a finite and layered oxygen-glycogen-lipid compartment. Secondly it allows a maximal glycolytic rate. Thirdly the use of the substrate glycogen for glycolysis is built in and finally the model is constrained by (22), (23) and (28).

While the finiteness of the O-compartment is clearly necessary, other models for the glycogen-lipid metabolism are possible. This model with a layered O-compartment is based on the following physiological facts [10, 11]. Glycogen

and lipids use oxygen as a common substrate to produce ATP. Both processes run largely in parallel, though the oxydation of glucose is initiated first. The oxygen equivalent of glucose is higher than that of fat. While local glycogen depletion is important for longer distance runs, the lipid reserves are almost unlimited.

A more serious objection to the present model is the fact that glycogen and even more lipids are released from their depots during activity. While the criticism addresses the applicability of the hydraulic model as such, this object is not as series as it seems at first glance, because the resulting exponential decay of W_0 can also be interpreted as increasing fatigue.

It is possible to model this latter situation by introducing a fourth lipid-glycogen depot vessel (D); which is coupled to (O) hydraulically but which is not coupled to (P) directly. In this case (O) should have a constant cross-section A_O. If the ceiling of (D) is at μ, the dynamics of this new semihydraulic system are unchanged for $k \leqslant \mu$. For $k \geqslant \mu$ one will have the constraints (22) and (23) and thus this system is, after a short intermittent period, again governed by one exponential. Thus for longer distances this new model is equivalent to the model described in Sect. II.

That the maximal glycolytic rate is attained rapidly with maximal workloads does not only follow from experimental results [16] but is also a consequence of theoretical studies [14]. For similar reasons power constraints are necessary. In nature these constraints follow from the kinetics of a large number of chemical reactions and the dependence of the enzyme activities on the substrate concentrations [9, 15, 16]. Thus these constraints should rather be described by sigmoidal functions. In the absence of further results and data however, a linear approximation (22) and (23) is justified. This has the advantage that no additional parameters are needed.

The function of the Pasteur effect is well established experimentally via the measurement of lactate concentrations for athletes [9, 10, 11]. Likewise it has been analyzed theoretically [9, 15] and its importance in the framework of a hydraulic energy model has been demonstrated above. Nonetheless it is extremely difficult to take it into account for a hydraulic model, and (28) should be considered only as a first attempt to do this.

In essence the Pasteur effect leads to two different models for competitive running. In the model for short distances, $D \leqslant 400$m, glycogen depletion is not important and one may set $k = 0$ throughout. For the long distance model $D > 2000$m, the glycolytic compartment should be neglected, i.e. $V_L = 0$. Thus the application of this model to middle distance runs seems more problematic than for the other distances. Another reason for this difficulty is the fact that the hydraulic model ties the breathing rate too strongly to h.

The model stills contains a number of parameters, which will have to be fixed depending on the special case under consideration. These are F, b, τ and M_P, M_L, M_O, V_P, V_L, A_O, A_F and μ. For good athletes these parameters will lie in the ranges

$$5.0 \leqslant F \leqslant 7.0 \qquad 0.68 \leqslant b \leqslant 0.8 \qquad 0.12 \leqslant \tau \leqslant 0.25$$

$$70 \leqslant M_P \leqslant 96 \qquad M_L \approx .4 \cdot M_P \qquad 0.25 \cdot M_P \leqslant M_O \leqslant M_P$$

$$700 \leqslant V_P \leqslant 1200 \qquad V_L \approx 1.4 M_P \qquad A_O \approx 90 \cdot A_P.$$

In addition we will also have $A_P \approx F(v_\infty)\tau(v_\infty)/\eta(v_\infty)$. Even though we have fixed θ, ϕ and λ, these parameters may also vary by $0.55 \leqslant \theta \leqslant 0.65$, $0.15 \leqslant \phi \leqslant 0.3$ and $0.05 \leqslant 1 - \theta - \lambda \leqslant 0.1$.

Acknowledgement. The author thanks the anonymous referee for a number of helpful suggestions and comments.

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