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Modelling human power and endurance

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Abstract. A generalised three component hydraulic model has been proposed to represent the human bioenergetic processes relating internal energy stores to performance during exercise, and into recovery. Further development of the model allows testable predictions to be made. In particular in this paper I examine certain hypotheses of chemical fuel shortage as a subgroup of the potential causes of fatigue, and their implications for maximal power and for endurance. The assumption that the limitation to sustainable power is direct proportionality to the glycogen store remaining, appears the most feasible. Based on this assumption, equations for the decline in maximum attainable power over time, the endurance at fixed workrates and the endurance at incremental tests (as a function of the increment slope) are obtained. Using published data for fit males, the maximum exertable power declines after about 6 s at 972 W to very low levels after about 2 min. For constant powers selected between 208 and 927 W, endurance declines from ad infinitum to only 6 s. Endurance at $\dot{V}_{O_2max}$ is predicted to be about 9 min. For incremental exercise tests of slope ranging from 30 W/min to 60 W/min, endurance lessens from 14 to 9 min. In these tests the anaerobic threshold is reached in times between 6 and 3 min. Although the power at termination of a test increases with incremental slope, terminal oxygen consumption is effectively constant. Almost all these model predictions are observed to correspond well with published experimental findings. These results suggest that the model can be used to represent an adequate overview of the operation of the human bioenergetic system.

Key words: Anaerobic threshold — Energy metabolism — Exercise performance — Exhaustion — Fatigue — $\dot{V}_{O_2max}$

1. Introduction

In a previous paper in this journal (Morton 1986a) I have proposed a generalised linear three component hydraulic analogue systems model for human bioenergetics called the M–M (Margaria–Morton) model. Figure 1 gives a diagrammatical representation of the model.

Vessel O, of infinite capacity, representing the oxidative energy source is connected to vessel P, representing the alactic energy source, through a tube R_1. R_1 has a maximal flow M_O, known as the maximal oxygen uptake, frequency denoted $\dot{V}_{O_2max}$. Vessel P has an assumed height $H = 1$ arbitrary unit, a volume V_P and a cross sectional area A_P arbitrary units.

The height of the base of vessel O above the base of vessel P is denoted φ, and hence the constant height of fluid in vessel O above R_1 is $1 - \varphi$. A tap T, at the base of vessel P regulates the net outflow W from the system, where W represents the measured energy expenditure, power, or workload. Vessel G, representing the glycogen store, is connected at its base to P, by a one-way tube R_2 at a height λ above the base of P. Vessel G has a finite fluid volume V_G and R_2 has a maximal flow M_G. The top of vessel G, except for a very narrow extension tube B, is at a level θ below the fluid level in vessel O. The fluid in B represents resting blood and tissue glycogen and does not contribute in measur-

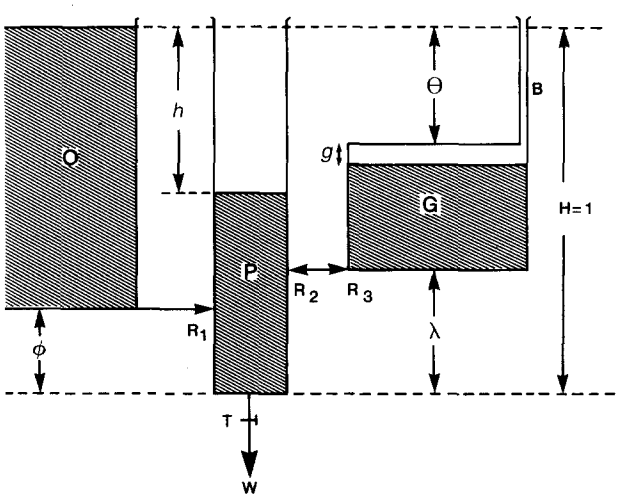

Fig. 1. The generalised M–M hydraulic model. This figure shows the layout, parameters and variables of the generalised hydraulic systems model analogue for human bioenergetics. O, G and P are interconnected vessels representing the oxidative, glycolytic and phosphagen based internal energy stores. O is regarded as very large relative to G and P, which are both finite. H, θ, λ and φ are the structural parameters of the system. The total fluid outflow W from the system is controlled by tap T, and the possible directions of flow are shown by R_1, R_2 and R_3. The situation depicted in the figure is as follows: T is open and the fluid level in P has dropped by an amount h which has induced a flow through R_1. Since $h > \theta$ a flow through R_2 has also been induced and the fluid level in G has dropped by an amount g

able amount to the net flows in the system. Vessel G therefore has a height of $1-\theta-\lambda$, and a cross-sectional area A_G arbitrary units.

The model operates as follows. Suppose tap T is opened to allow a net outflow W. This induces a drop, h, in the level of fluid in vessel P. This in turn induces a flow from O to P through R_1. This flow, representing a rise in net oxygen consumption, is in accordance with the ratio of h to $1-\varphi$, equalling the maximum M_O when h equals (or exceeds) $1-\varphi$. If W is small, then h will reach an equilibrium position, no greater than θ. This corresponds to a steady state oxygen uptake, $\dot{V}_{O_2SS}$. If T is closed at any time, the level in P will return to its resting level, by virtue of a decreasing flow through R_1. This flow ceases when h equals zero, and corresponds to the repayment of the alactic oxygen debt.

If W is of sufficient magnitude, greater than a threshold value W_θ known as the anaerobic threshold (AT), then after a while h will exceed θ, in which case a net flow from G to P is then also induced. This flow which represents glycogen depletion and the anaerobic production of lactic acid by the working muscles is in accordance with the difference in levels between vessel G (an amount g below the top), and vessel P, with the level in G dropping also but lagging behind the level in P. If T is closed, P will be refilled, initially from both O through R_1 and from G through R_2. This continues only until the lag in levels between G and P has been eliminated. This brief phase represents partial repayment of the alactic oxygen debt by contracting an increased lactic oxygen debt, known as delayed or post-exercise lactate formation. Thereafter P refills by a decreasing flow through R_1 and G in turn refills by a flow through the one-way return tube R_3. The flow through R_3 is also in accordance with the difference in levels between P and G. The maximal flow through R_3, M_R, is very much smaller than M_G or M_O. Ultimately both the lactic and alactic oxygen debts will be repaid. Once again, if W had not been too great, an equilibrium level with $h \leqslant 1-\varphi$ and $\dot{V}_{O_2} \leqslant \dot{V}_{O_2max}$ could have been achieved, by which time the early lactate flow through R_2 would have ceased. If T is closed after equilibrium has been reached, there would be no delayed lactic acid formation and both P and G would be refilled immediately, though R_1 and R_3, respectively.

If W is of even higher magnitude, demanding an energy expenditure in excess of $\dot{V}_{O_2max}$, then after a further while h will exceed $1-\varphi$. In this event, $\dot{V}_{O_2}$ will remain constant at $\dot{V}_{O_2max}$ and the flow through R_2 will persist. Since G is of limited capacity, it will later become empty, and so too will P. The subject would then have depleted his energy stores, and would no longer voluntarily be able to maintain exercise at this level. Once T is closed, repayment of the lactic and alactic debts will be very similar to that described above, except for the absence of post exercise lactate formation and for the fact that initially $\dot{V}_{O_2}$ will be constant at $\dot{V}_{O_2max}$ until such time as $h < 1-\varphi$.

I have argued (Morton 1986a) that the sixteen configurations of the generalised model could be reduced to four, by eliminating those inconsistent with known physiological facts. Since, in addition, it is well known that subjects can deplete their glycogen store through prolonged exercise at less than $\dot{V}_{O_2max}$ (Costill et al. 1973; Newsholme 1986), what marathon runners refer to as

"hitting the wall", the configuration of Fig. 1 has been adopted. That is θ, φ and λ all non-zero, $\lambda > \varphi$ and $\theta < 1 - \varphi$.

The purpose of this paper is to make some predictions derived from the model and to contrast these predictions with reported experimental observations on exercising subjects. In this way the model may be accepted as a satisfactory representation of the real system, or it may need to be revised in the light of contrary evidence. In particular in this paper I investigate the implications of certain hypotheses concerning the shortage of chemical fuel as a subgroup of the possible causes of fatigue, on the time course of maximal power and on endurance at various workloads. Some simpler aspects of these two questions (Morton 1986b, 1987) are indicative of the potential of this approach.

2. Maximal power

Let W_m denote the maximal power or workload that the system could attain at any time. In terms of the hydraulic model this is represented by the potential flow through T when the tap is fully open. The time course of W_m in exercising humans is dependent on the physiological mechanisms of neuromuscular fatigue and exhaustion. These mechanisms are complex and not fully understood, though it is relevant to consider the matter briefly (Porter and Whelan 1981; Lamb 1984; Brooks and Fahey 1985). The limitation to the ability to maintain muscular contractions at a given level of power could conceivably lie in (a) the central nervous system, (b) the final motor neuron, (c) the neuromuscular junction, or (d) the muscle.

Evidence from experiments involving electrical stimulus of muscle after voluntary fatigue, as well as from some EMG studies, implicate the central nervous system as a potential site for neuromuscular fatigue. Other EMG studies implicate the neuromuscular junction as another potential site of fatigue. On the other hand, experiments seem to have ruled out the motor nerve as a potential site of fatigue.

By far the majority of evidence implicates the contractile process in the muscle as the most probable single site of fatigue. For very short term supramaximal exercise it seems likely that the rate of ATP utilisation is simply too great to be met by resupply from muscle stores of ATP and creatine phosphate. For maximal exercise that can be sustained for longer than about 15 s but less than about 3 min, it seems that creatine phosphate depletion may limit the ability of muscle to sustain activity at these high loads. The accumulation of lactic acid, the by-product of anaerobic glycolysis, appears also to be implicated in this time frame. Intracellular pH of the muscle can as a result drop to values low enough to inhibit the replenishment of ATP by glycolysis through reduction in the activity of the enzymes glycogen phosphorylase and phosphofructokinase. Lactic acid accumulation is also implicated in heavy exercise lasting about 3–15 min, but without necessarily severe depletion of creatine phosphate or of glycogen. As well as reduced enzymatic activity, the lowered pH lessens calcium binding thereby reducing the activation of cross-bridges in muscle contraction. Further-

more, the administration of sodium bicarbonate as a pH buffer has been shown to increase endurance. For exhaustive exercise lasting 15 min to 1 h, there is no obvious single factor closely associated with the onset of fatigue. Some combination of factors may be operative. For endurance activities that can be sustained for between 1 and 4 h, it seems very likely that exercise is limited by the capacity of glycogen stores. The complete utilisation of these stores is a phenomenon (hitting the wall) well known to marathon runners, and numerous studies have shown that the manipulation of glycogen stores by diet and exercise can have a substantial effect on endurance.

Thus, given the brief nature of this overview and noting the construction of the hydraulic model in Fig. 1 with its energy storage vessels, the most obvious examinable hypothesis of power limitation during exercise appears to be one concerning the depletion of available energy stores. This section examines two such hypotheses.

2.1. A phosphagen based limitation

The trivial case, constant $W_m = M_P$, the maximum alactic power available, is not considered realistic in any way, and is not considered further. A more plausible hypothesis is the suggestion that W_m is proportional to the PC store remaining (Miller et al. 1987; Sargeant and Dolan 1987). That is:

$$W_m = M_P \frac{A_P(1-h)}{A_P H} = M_P(1-h). \tag{1}$$

The differential equation governing the system

$$W = \dot{V}_{O_2} - \dot{V}_P - \dot{V}_G \tag{2}$$

now becomes

$$W_m = \frac{M_O h}{1-\varphi} + A_P \frac{dh}{dt}, \tag{3}$$

which when combined with Eq. (1) and solved assuming $h = 0$ at $t = 0$ yields

$$W_m = \frac{M_O M_P}{M_O + M_P(1-\varphi)} + \frac{M_P^2(1-\varphi)}{M_O + M_P(1-\varphi)} \times \exp\left(-\frac{M_O + M_P(1-\varphi)}{A_P(1-\varphi)} t\right). \tag{4}$$

This model hypothesis therefore predicts an immediate exponential decline in all-out power once exercise commences, although Eqs. (3) and (4) are only valid during a first phase up until h reaches the value θ. There is considerable evidence (Di Prampero 1981; Hirvonen et al. 1987; Jones et al. 1985; Margaria et al. 1966; McCartney et al. 1983; Newsholme 1986) that maximum power output during all-out effort is constant for about the first four to six seconds; a situation quite unlike that described by Eq. (4). The first hypothesis, that W_m is proportional to

the remaining PC store, must therefore be rejected, even though changes in these muscle metabolites do tend to correlate with the power decline (Cheetham et al. 1986; Hooper et al. 1985; Jones et al. 1985; Miller et al. 1987).

2.2. A glycogen based limitation

Let us consider then as a second supposition that the maximum power available through M_P is governed in some way by the amount of glycogen store remaining (Conlee 1986; Green et al. 1987).

The first phase of activity, of duration until $h = \theta$, is very simple; $W_m = M_P$, constant. Equation (2) now becomes

$$M_P = \frac{M_O h}{1-\varphi} + A_P \frac{dh}{dt}, \tag{5}$$

and Eq. (5) has solution given by

$$h = \frac{M_P(1-\varphi)}{M_O}\left(1 - e^{-M_O t/A_P(1-\varphi)}\right),$$

and the phase ends when the anaerobic threshold is reached; a time $t = t_1$ given by

$$t_1 = -\frac{A_P(1-\varphi)}{M_O} \ln\left(1 - \frac{M_O\theta}{M_P(1-\varphi)}\right). \tag{6}$$

During the second phase of activity we hypothesise (as intimated above) that W_m is now proportional to the remaining glycogen store, thus

$$W_m = M_P\left(\frac{1-\theta-\lambda-g}{1-\theta-\lambda}\right), \tag{7}$$

and Eq. (2) now becomes

$$M_P\left(\frac{1-\theta-\lambda-g}{1-\theta-\lambda}\right) = \frac{M_O h}{1-\varphi} + A_P\frac{dh}{dt} + A_G\frac{dg}{dt}. \tag{8}$$

Now the fluid flow assumption through R_2 leads to

$$A_G\frac{dg}{dt} = M_G\frac{h-g-\theta}{1-\theta-\lambda}. \tag{9}$$

The solution for h, together with its derivative, from Eq. (9) are utilised in Eq. (8) to yield

$$\frac{d^2g}{dt^2} + a_1\frac{dg}{dt} + b_1 g = c_1. \tag{10}$$

Its solution

$$g = \frac{c_1}{b_1} + k_1 e^{-r_1 t} + k_2 e^{-r_2 t} \tag{11}$$

is therefore found in the standard manner (Golomb and Shanks 1950) and together with Eq. (7) leads to an equation of form:

$$W_m = M_P(\alpha + \beta_1 e^{-r_1 t} + \beta_2 e^{-r_2 t}),$$

where the constants are determinable making use of the fact that

$$g = \frac{dg}{dt} = 0 \quad \text{at } t = t_1.$$

This phase ends at time $t = t_2$ when $h = 1 - \lambda$. Thus utilising Eqs. (11) and (9), t_2 can be determined. It cannot however be expressed simply in any explicit form as was t_1 above. At time $t = t_2$ therefore, we suppose that $g = g_2$ say, utilising Eq. (11).

A third active phase now takes place in which Eq. (8) now applies but it becomes redundant since Eq. (9) is replaced by

$$A_G \frac{dg}{dt} = \left(\frac{1 - \theta - \lambda - g}{1 - \theta - \lambda}\right). \tag{12}$$

Equation (12) whose solution must satisfy $g = g_2$ at $t = t_2$ can easily be solved, and leads to an equation of form:

$$W_m = M_P \beta_3 e^{-r_3 t}.$$

This phase ends as g tends to $1 - \theta - \lambda$. Although W_m has not dropped absolutely to zero, in practical terms a point of exhaustion is reached after about 2 min. The glycogen store is almost completely empty and the individual is virtually compelled to stop exercising. This prediction represents a realistic time frame (Vandewalle et al. 1987).

The above phases of an all-out effort can be illustrated graphically. No estimates of θ, λ or φ are available, so suppose $\theta = 0.65$, $\lambda = 0.1$ and $\varphi = 0.05$. Following an earlier example (Morton 1986b) let

$$A_P H = 1646.4 \text{ ml } O_2, \qquad M_P = 972 \text{ W } (194.4 \text{ ml } O_2/\text{s}),$$

$$0.25 A_G = 3780 \text{ ml } O_2, \qquad M_G = 478 \text{ W } (95.67 \text{ ml } O_2/\text{s}),$$

$$M_O = \dot{V}_{O_2 max} = 2.8 \text{ l/min } (46.67 \text{ ml } O_2/\text{s}).$$

We can determine firstly that $t_1 = 6.0$ s, during which time

$$W_m = M_P = 972 \text{ W}. \tag{13}$$

Secondly we can determine that for $t_1 \leqslant t_2 = 50.3$ s

$$W_m = 972(0.1187 - 1.0935 e^{-0.233t} + 1.5971 e^{-0.0545t}), \tag{14}$$

during which time g increases from 0 to $g_2 = 0.1946$. Thirdly we can determine that for $t > t_2$:

$$W_m = 769 e^{-0.02531t}, \tag{15}$$

during which time g increases further, and tending to 0.25 for larger values of t.

Equations (13), (14) and (15) can now be graphically examined together with some experimental data for subjects exercising all-out for relatively short periods of time (Jones et al. 1985; McCartney et al. 1983, 1986). This information is presented in Fig. 2.

The correspondence between theoretical and experimental data is clearly evident, even though no attempt has been made to adjust the model parameters in order to optimise the fit. This could be achieved most obviously by reducing θ so as to shorten t_1 to say 4 s, and by making other possible changes also.

It appears therefore that this second supposition of fatigue provides a sufficiently realistic representation of actual events to enable some progress to be made on the second focus of this paper, the question of endurance at various workloads. Before doing so however, it is interesting to note several aspects of the M–M model predictions above and to compare them with available experimental evidence.

The value of t_1 determined from Eq. (6) is heavily dependent on the value of the structural model parameter θ, for which no estimates are available. Since the maximal workrate appears more or less constant for the first 4 to 6 s (Hirvonen et al. 1987; Ikuta and Ikai 1972; Jones et al. 1985; Margaria et al. 1971; McCartney et al. 1983; Murase et al. 1976), a value for θ in the range $0.45 < \theta < 0.65$ is suggested. Lower values of θ are associated with shorter t_1 time, lower anaerobic threshold, etc., and since our illustrative subject is a capable athlete, the value $\theta = 0.65$ is retained. Since t_1 is insensitive to a range of φ values, $\varphi = 0.05$ is also retained. Now as soon as t_1 has passed, lactate begins to appear, first in the working muscle, then into the bloodstream. Thus according to the model after 6 s, and certainly by 10 s, a significant lactate buildup should be observable, and this has been a reported finding in the laboratory (Jacobs et al. 1983). Furthermore the model predicts this event occurring well before the phosphagen stores are completely or almost completely emptied, say when $h = 0.70$, i.e. 30% of the PC store still remains. This is a low, but not critically low value. Indeed this is also a reported laboratory finding (Jacobs et al. 1983). All these studies mentioned above on maximal exercise, (see also Cheetham et al.

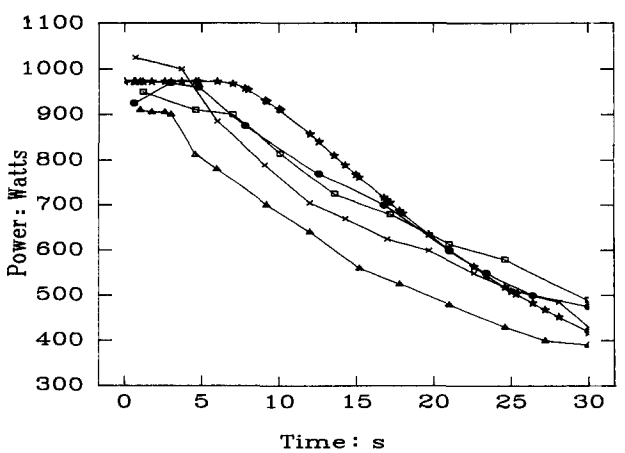

Fig. 2. Predicted and observed power outputs for continuous maximal work. ×, □, McCartney et al. 1983; △, Jones et al. 1985; ⊕, McCartney et al. 1986; ☆, model prediction

1986), limit the time of exercising to 30 s, perhaps because of the difficulty of motivating subjects to produce a genuine maximal effort beyond that time. In those studies in which power measurements are made (Jones et al. 1985; McCartney et al. 1983, 1986), the maximal power after 30 s has dropped to around 50% of its original highest value. This agrees very favourably with the curve of Fig. 2. Unfortunately the comparison cannot be extended beyond 30 s, and the 2 min to exhaustion indicated above cannot be confirmed, although it is suggested to be a reasonable estimate (Vandewalle et al. 1987).

The model makes an interesting prediction for maximal intermittent work. Due to the prior heavy work undertaken, the G vessel is partially emptied, not having had sufficient recovery time to refill. That is, the maximal workrate for subsequent exercise performance according to our assumption is reduced, and the structural parameter θ is increased above 0.65. When the subject is again called to work maximally, the initial flat portion of the power curve is therefore lower and extends for a longer period of time (i.e., t_1 appears longer). This prediction is confirmed by experimental evidence (McCartney et al. 1986; Sargeant and Dolan 1987).

Finally, since anaerobic glycogen depletion is concomitant with lactate buildup in the working muscle and thence the blood, the above adopted fuel shortage hypothesis for fatigue might be expected to yield very similar predictions to one based on lactate accumulation in muscle (Asmussen et al. 1948; Bang 1936; Karlson 1971; Tesch 1980). To investigate such a contingency would require extending the model to include the interconversion between glycogen and lactate and additional compartments for lactate circulation between muscle and blood, and lactate degradation.

3. Exhaustion and endurance

The simplest approach to the question of exhaustion and endurance is to suppose that an exercising subject becomes exhausted, and that the limit of his/her endurance has been reached at that time when the body can no longer maintain the workload demanded of it. Thus in reference to the previous section, the time-point of onset of fatigue during work, that is the endurance, can be determined as the time when the workload demanded just equals the maximum that the body could exert utilising the energy stores as they exist at that moment. Thus work at a fixed level W can continue as long as $W < W_m$, where W_m is calculable in the manner of the previous section as a function of changing body stores. Exhaustion occurs at a time t when $W = W_m$. Derivation of t as a function of W, or vice versa, is as follows.

3.1. Endurance at constant power

Firstly, since endurance at maximal constant workrate, M_P, has been already determined as t_1 seconds, from Eq. (6), the notion of exhaustion in less than t_1

seconds is vacuous. We must therefore consider $W < M_P$. However W is bounded below also, by that workrate which is just low enough to be sustained in the steady state ad infinitum. That is:

$$W > M_P\left(\frac{1-\theta-\lambda-g}{1-\theta-\lambda}\right) = \frac{M_O h}{1-\varphi} = \frac{M_O(g+\theta)}{1-\varphi}$$

$$> \frac{(1-\lambda)M_O M_P}{(1-\theta-\lambda)M_O + (1-\varphi)M_P}. \tag{16}$$

For W in this interval, Eqs. (2) and (9) yield another second order differential equation, once again of the same form as Eq. (10), which is solved for g in the same manner. The coefficients (but not the exponents) in the solution are simple linear functions of the constant workrate W. This aspect of the solution, together with Eq. (7) putting $W = W_m$ and eliminating g, yields a relationship between W and t. That is, given the endurance time, the maximum constant maintainable workrate for this endurance can be calculated. Doing so yields an equation of form

$$W = \frac{k_3 + k_4 e^{-r_4 t} + k_5 e^{-r_5 t}}{k_6 + k_7 e^{-r_4 t} + k_8 e^{-r_5 t}}, \tag{17}$$

and note that k_3/k_6 is given by the right hand side of Eq. (16).

Following on the illustrative example above we obtain the limiting lower value of W, k_3/k_6 as 208 W, which is at a level 89% of $\dot{V}_{O_2 max}$. Although a little high, this corresponds reasonably to the sort of workrate maintained for prolonged periods, say by marathon or ultramarathon runners (Costill et al. 1973; Maron et al. 1987; Maughan and Leiper 1983; Wells et al. 1981).

This maximal sustainable workrate also reveals some information about the structural parameters θ, λ and φ. From Eq. (16), we discover that the sustainable workrate increases with increasing θ and φ, being less sensitive to the former, and decreases with increasing λ, to which it appears the most sensitive. For example an increase in λ from 0.1 to 0.2 and a drop in θ from 0.65 to 0.55 while maintaining $\varphi = 0.05$, yields a sustainable workrate of 79% $\dot{V}_{O_2 max}$, which is more in line with published findings. However, to maintain consistency, the originally chosen values are retained.

We have for Eq. (17):

$$W = \frac{4.5 + 0.1695\, e^{-0.2849t} - 3.333\, e^{-0.00265t}}{0.0216 + 0.00106\, e^{-0.2849t} - 0.0209\, e^{-0.00265t}} \tag{18}$$

which since it would be preferable to be given W and then predict t, Eq. (18) is plotted in this way in Fig. 3.

This figure has the immediately recognisable typical features of an endurance curve, such as the collection presented by Wilkie (1960, 1980, 1981) and others (Clarke 1986; Gleser and Vogel 1973; Harman et al. 1987).

The familiar shape of Fig. 3 is insufficient on its own for acceptance. There are other characteristics which distinguish this model equation from most others. Firstly, note the behaviour as W becomes large. Wilkie's curves, which have

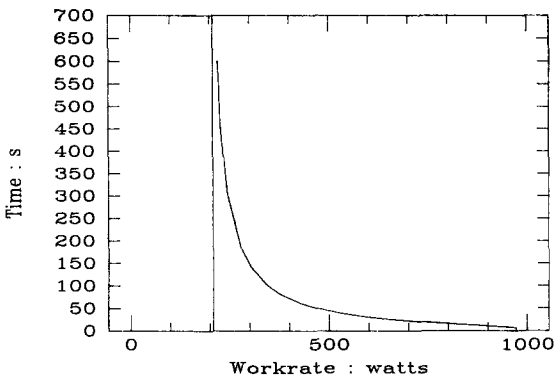

Fig. 3. Predicted endurance times at various constant workrates. These model predicted results assume that the limitation to sustainable power is directly proportional to the glycogen store remaining

some theoretical background, show t tending to zero asymptotically with W; while other examples (Clarke 1986; Gleser and Vogel 1973; Harman et al. 1987) cut off at a maximal instantaneous effort but have uncertain or no theoretical background. Figure 3 both cuts off and has a substantial model theoretical background. Secondly, note that in Wilkie's model t becomes infinitely large as W approaches 100% $\dot{V}_{O_2max}$, almost surely an overestimate. In two of the other studies (Gleser and Vogel 1973; Harman et al. 1987), this event occurs only as W tends to zero, almost surely an underestimate. The Fig. 3 value of around 89% $\dot{V}_{O_2max}$ (or perhaps down to 79% as indicated above) is much more reasonable. A third confirmatory point for Fig. 3 is to note that an endurance of 9 min is predicted at $\dot{V}_{O_2max}$, as has been established experimentally (Gleser and Vogel 1973).

3.3. Endurance at incremental power

A further type of endurance worthy of consideration is that related to the familiar $\dot{V}_{O_2max}$ ramp or incremental exercise tests. Let us therefore consider an incremental ramp test of slope s in W/min.

During the first active phase, Eq. (2) becomes

$$st = \frac{M_O h}{1-\varphi} + A_P \frac{dh}{dt},$$

with solution given by

$$h = \frac{s(1-\varphi)}{M_O} t - \frac{sA_P(1-\varphi)^2}{M_O^2}(1 - e^{-M_O t/A_P(1-\varphi)}).$$

This phase ends when the anaerobic threshold is reached, a time t_1 when $h = \theta$ given by the solution to

$$s = \frac{\theta}{(1-\varphi)t_1/M_O - A_P(1-\varphi)^2(1-e^{-M_O t_1/A_P(1-\varphi)})/M_O^2}. \tag{19}$$

Equation (19) can therefore be used to predict the time just after which a discernable rise in blood lactate can be expected, for any given incremental slope s.

During the second phase of activity, Eq. (2) becomes

$$st = \frac{M_O h}{1-\varphi} + A_P \frac{dh}{dt} + A_G \frac{dg}{dt},$$

which together with Eq. (9) yields another second order differential equation of form:

$$\frac{d^2g}{dt^2} + a_2 \frac{dg}{dt} + b_3 g = m + nt. \tag{20}$$

The solution to Eq. (20) can also be found similarly, also subject to the same conditions on g and dg/dt at time t_1.

The mathematics now involves a number of transcendental equations. An equation for g can be obtained as a function of s, t_1 and t, which can in turn be used in Eq. (7) with $W = W_m$ enabling the time of exhaustion to be determined as a function of s. This equation is only applicable up to that time when $h = 1 - \lambda$. That is, provided exhaustion occurs in the second phase of activity. This time is predicted to be 681 s (11.35 min), with the corresponding incremental rate of 42.87 W/min. At lesser incremental rates, and with exhaustion occurring later, the third phase of activity must be considered.

In this instance Eq. (12) applies with similar requirements on g at the time when $h = 1 - \lambda$. Required also therefore for the solution is a version of Eq. (2)

$$st = \frac{M_O h}{1-\varphi} + A_P \frac{dh}{dt} + A_G \frac{dg}{dt},$$

except that it now becomes necessary to consider each value of s separately in order to proceed. Thus, knowing the time of exhaustion by equating W given by st with W_m given by Eq. (7), we can find the value of W at exhaustion, and also the value of h and hence $\dot{V}_{O_2}$ at exhaustion also.

The results of these calculations are demonstrated graphically for our hypothetical subject as Figs. 4 and 5.

Figure 4 shows, for given incremental slopes in the commonly recommended range 30 to 60 W/min (Buchfuhrer et al. 1983; Davis et al. 1982; Shephard 1984; Whipp et al. 1981), both the time to anaerobic threshold and the time to exhaustion. Time to AT lessens from 6 min at slow ramps to just over three at fast ramps, and endurance lessens from 14.5 to just over 9 min. Although I have been unable to find published times to AT in incremental tests, the times that can be deduced from the work of Davis et al. (1982) agree very well with Fig. 4. The endurance times in Fig. 4 are perhaps a little longer than in published studies (Buchfuhrer et al. 1983; Davis et al. 1982; Shephard 1984; Whipp et al. 1981), but not unduly so.

Figure 5 shows terminal workrate and terminal $\dot{V}_{O_2}$ levels predicted as functions of the incremental slope over the same range. The former shows a noticeable rise with increasing slope, but the latter is almost absolutely constant. As occurred with Fig. 4, these two predictions are entirely consistent with

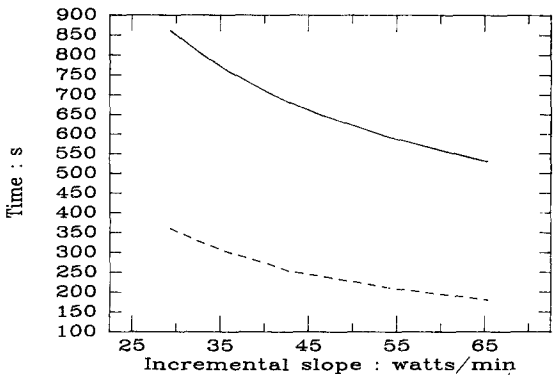

Fig. 4. Predicted endurance at incremental exercises. The figure shows model predicted endurance (*solid line*) and time to AT (*dashed line*) as functions of the ramp slope in incremental exercise testing

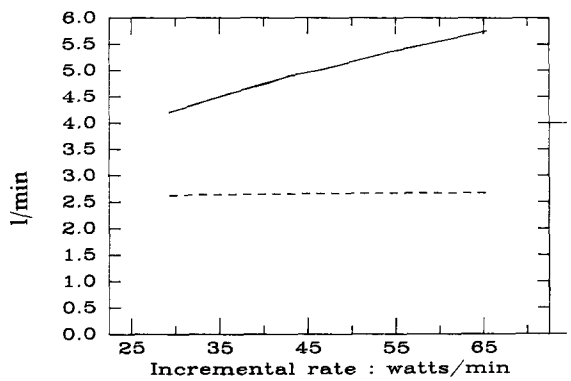

Fig. 5. Predicted terminal workrate and $\dot{V}_{O_2}$ levels. The figure shows model predicted workrate (*solid line*) and $\dot{V}_{O_2}$ (*dashed line*) at the termination of an incremental exercise test, as functions of the ramp slope. Workrate is expressed in hundreds of watts and $\dot{V}_{O_2}$ in litres/min

experimental findings on maximal work rate and $\dot{V}_{O_2max}$ (Buchfuhrer et al. 1983; Davis et al. 1982; Hughson and Green 1982; Shephard 1984).

It is very reassuring to note the evident correspondence between the general pattern of the model predictions illustrated in Figs. 4 and 5, and reality. In particular, the time predicted to AT is almost identical to the times calculable from Table 2 in Davis et al. (1982), whose subjects have the same $\dot{V}_{O_2max}$ as assumed for this model. The constancy of the terminal $\dot{V}_{O_2}$ is another particularly appealing prediction. On the other hand, although predicted endurance times at incremental tests for a subject with a $\dot{V}_{O_2max}$ of 2.8 g/min are only a little longer than have been found experimentally (Davis et al. 1982), the predicted terminal workrates appear too high. Workrates of 260–280 W might be expected, (Davis et al. 1982), whereas the predicted 420–520 W are more associated with a subject whose $\dot{V}_{O_2max}$ would be about 5 l/min. Since all these model predictions are so closely linked, it is difficult to see why manipulation of parameters in order to improve one poor prediction would not necessarily also worsen three good predictions.

There is a disguised prediction inherent in Fig. 5 which raises a definitional question. It is that the predicted terminal $\dot{V}_{O_2}$ does not equal the assumed $\dot{V}_{O_2max}$ of the model, but is a value around 94% $\dot{V}_{O_2max}$. That is, do we define $\dot{V}_{O_2max}$ as

that terminal $\dot{V}_{O_2}$ value measured by means of the conventionally accepted standard incremental test protocol? Or, do we define $\dot{V}_{O_2max}$ conceptually as a theoretical maximum $\dot{V}_{O_2}$ value which could be achieved if only exhaustion could be prevented or delayed in some way? In itself, this is an intriguing question, and the model theory above suggests that the conceptual $\dot{V}_{O_2max}$ may be unachievable in practice, at least by means of the incremental test procedure.

4. Conclusion

The purpose of this paper has been to demonstrate that the construction of an acceptable mathematical model of a physiological system (Morton 1986a) is only a starting point for more thorough study. Such models allow quantitative predictions to be made, based on various assumptions about the behaviour of the system. These predictions can then be tested experimentally. If they pass, then the model and assumptions are retained and further predictions can be made and tested, and so on. If a contradiction is established, then the model (in part or even as a whole) and/or the assumptions, must be revised before the prediction and testing cycle commences again. In this instance, the M–M model remains standing and a fresh more detailed cycle may begin. Developing laterally, the model may be extended by adding further components, allowing a more broad cycle of predicting and testing. These are therefore two future avenues of study for the M–M model.

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