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A three component model of human bioenergetics

R. Hugh Morton*

Department of Biological Sciences, Cumberland College of Health Sciences, P.O. Box 170, Lidcombe, NSW, Australia

Abstract. The model described in this article is a generalised three component hydraulic model, proposed to represent net whole body bioenergetic processes during human exercise and recovery. During exercise, fluid flows from the three interconnected vessels in the system represent the breakdown of high energy phosphates (phosphagens), oxygen consumption and lactic acid production. During recovery, replenishment of the fluids represents the repayment of oxygen debt. The model is quantified and solved mathematically, and the solution compared with observed experimental data. Since currently known physiological facts are consistent with four configurations of this model, further experimentation is necessary.

Key words: Anaerobic threshold — Energy metabolism — Exercise — Lactate production — Oxygen uptake — Recovery

1. Introduction

There appears to be only one published theoretical model of whole body human energy processes during muscular exercise. This is the three component hydraulic model proposed by Margaria (1976). The three components represented are oxygen consumption ($\dot{V}O_2$), lactic acid (lactate) formation (glycolysis) and phosphagen breakdown (alactic energy). Their contributions towards the net total energy expenditure above resting levels are modelled by flows of fluid between (and out of) the three vessels comprising the system. Some models, both empirical and theoretical, exist for oxygen consumption only; for example Henry (1951), Volkov (1966), and Morton (1985a). No model for lactic acid production has been proposed, though Freund and Gendry (1978) and Zouloumian and Freund (1981) have proposed, and solved, a model for lactate kinetics after cessation of exercise. Likewise there is no modelling ancestry for alactic energy.

Unfortunately, Margaria's hydraulic model was not quantified, though sufficient information was available to do so. No mathematical solution was

* *Permanant address*: Mathematics and Statistics Department, Massey University, Palmerston North, New Zealand

offered therefore, though a suggested graphical solution (Margaria, 1976; Figs. 1.27 and 1.28) was presented. A mathematical solution to Margaria's model has now been obtained (Morton, 1984a). This solution conforms neither to Margaria's graphical presentation, nor to what actually happens under experimental conditions. These discrepancies, and directions for further work are discussed in Morton (1985b, 1986).

This paper presents the M-M (Margaria-Morton) model, a generalisation of Margaria's original. All but four of the sixteen forms of the generalised whole body model can be rejected on the basis of known physiological facts. These four are solved mathematically, and the solutions compared with some experimental data obtained from exercising subjects. Since a unique form of the M-M model is not yet indicated by physiological observations, further experimentation is necessary in the hope of identifying a unique model.

2. The generalised M-M model

Figure 1 below gives a diagrammatical representation of the generalised three component whole body hydraulic 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, frequently denoted $\dot{V}O_2$ max. 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* $1-\phi$. A tap T, at the base of vessel P regulates the net outflow W from the system, where W represents the measured energy expenditure, or workload. Vessel L, representing lactic acid, is connected at its base to P, by a one-way tube R_2 at a height λ above the base of P. Vessel L has a finite fluid volume V_L, and R_2 has a maximal

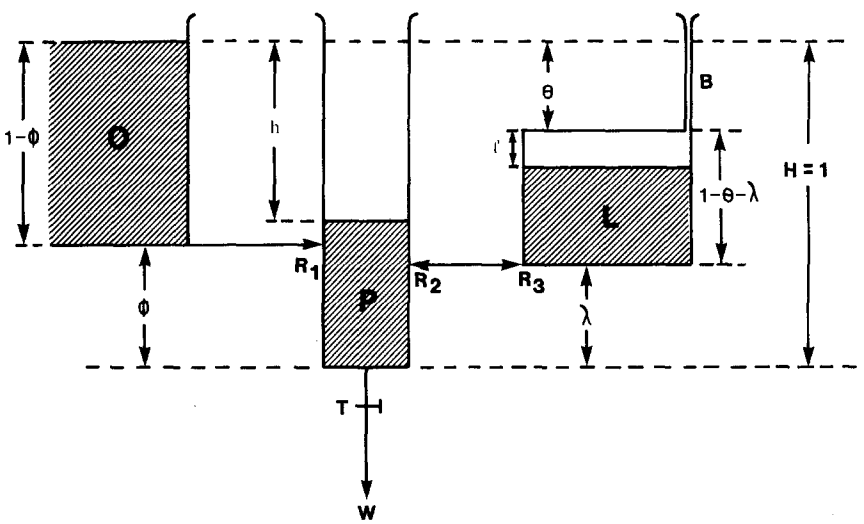

Fig. 1. The generalised M-M model

flow M_L. The top of vessel L, 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 lactate, and does not contribute in measurable amount to the net flows in the system. Vessel L therefore has a height of $1-\lambda-\theta$, and a cross-sectional area A_L .

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 $1-\phi$, equalling the maximum M_O when h equals (or exceeds) $1-\phi$. If W is small, then h will reach an equilibrium position, no greater than θ. This corresponds to a steady state oxygen uptake. 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, then after a while h will exceed θ, in which case a net flow from L to P is then also induced. This flow which represents the production of lactic acid by the working muscles is in accordance with the differences in levels between vessel L (an amount l below the top), and vessel P, with the level in L 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 L through R_2. This continues only until the lag in levels between L and P has been eliminated. This 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 L 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 L. The maximal flow through R_3, M_R, is very much smaller than M_L 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 $h \leqslant 1-\phi$ and $\dot{V}O_2 \leqslant \dot{V}O_2$ max 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 L 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 $\dot{V}O_2$ max, then after a further while h will exceed $1-\phi$. In this event, $\dot{V}O_2$ will remain constant at $\dot{V}O_2$ max and the flow through R_2 will persist. Since L 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. It is of course recognised that the causes of fatigue are not well understood, and that it may result from other causes, such as proton accumulation, neuromuscular junction breakdown, calcium depletion or an interaction of these or other factors. However, the model structure would have to be extended and additional assumptions made in order to incorporate any such factors. In any event, the assumption of depleted energy stores plays no part in the mathematical development to follow. It is therefore one of convenience in the general description of the model operation. Once T is closed,

repayment of the lactic and alactic oxygen debts will be similar to the delayed lactate production (provided L is not yet emptied) described above, except that initially $\dot{V}O_2$ will be constant at $\dot{V}O_2$ max until such time as $h < 1 - \phi$.

Since, as will be seen below, there are several different configurations of the model, the above description of the operation of the model serves as a general guide only. In addition, for the purposes of the mathematical treatment of the model later in this paper, the volumes or capacities of the three vessels of the system are all regarded as measured in energy or work units, while flows in the system are regarded as measured in units of power. The "fluid" is therefore not real, but it and its flow are regarded as analogous to work and power. However, since exercise physiologists frequently measure oxygen uptake in litres per minute for example, and glycogen stores in mM, etc., this latter measurement approach has been adopted in general. The two approaches differ only by the appropriate energy equivalent constant, for example 1 ml O_2 is equivalent to about 5 cal or 21 joules, and so on. These constants, see Margaria (1976) for details, are subsumed within the mathematical workings.

3. Model configurations

There are in fact sixteen configurations of the generalised M-M model, depending on whether ϕ and/or θ and/or λ are or are not zero; and whether λ is greater than, equal to or less than ϕ; and/or θ is greater than, equal to or less than $1 - \phi$. Margaria's (1976) original is the particular case when $\lambda = 0$ and $\phi = \theta = \frac{1}{2}$. The sixteen configurations can be reduced to four, by eliminating those inconsistent with known physiological facts.

Firstly, since it is very well known that light workloads, even up as high as 180 watts for active physically fit persons, can be performed entirely aerobically and without the production of any lactate, all those configurations, six in number, in which $\theta = 0$ must be eliminated. That is to say in normal individuals the anaerobic threshold is at a greater than zero workload. Secondly, since the anaerobic threshold, or the onset of lactate production, has been determined to be at workloads clearly less than the maximal oxygen uptake (Davis et al. 1976; MacDougall 1978; Weltman et al. 1978; Weltman and Katch 1979; Reinhard et al. 1979; Miyashita et al. 1981) those cases where $\theta \geq 1 - \phi$ can also be eliminated. Margaria's original is one of these further four cases. There are very many people however who believe that net anaerobic energy production at levels of $\dot{V}O_2$ below maximal does not truly exist in steady state conditions, (e.g. Cerretelli et al., 1979; Seeherman et al., 1981). That is, increments in blood lactate levels are only transients that disappear with sustained activity. This is of course consistent with the model configurations below and has been described in the previous section. It is detailed in Sect. 4.3 below. Thus if one were to define the anaerobic threshold as that workload beyond which increments in blood lactate levels ceased to be transient, then such an occurrence would indeed coincide with $\dot{V}O_2$ max. Even so, one would still have to define some model parameter (no longer called the anaerobic threshold), say θ, the level at which the onset of transient lactate production began, and clearly $\theta < 1 - \phi$ as indicated. Thirdly, in specially contrived experimental conditions, a real asymptotic $\dot{V}O_2$ max can

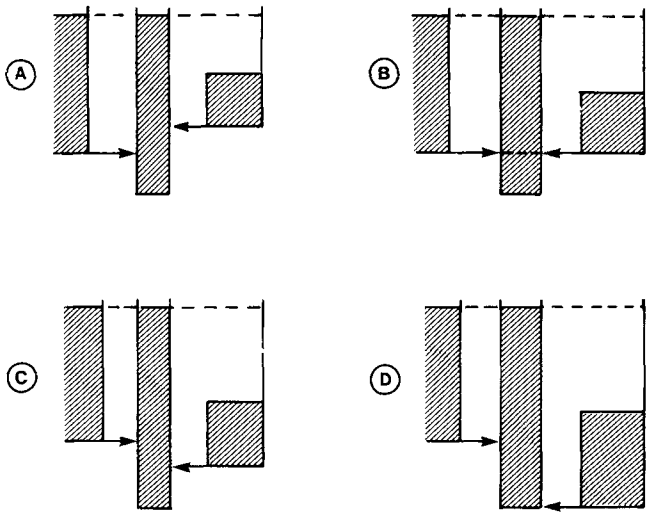

Fig. 2. Configurations of the M-M model

be achieved for very severe workloads, and oxygen consumption does not commence an immediate decline after cessation of exercise, (Katch 1973; di Prampero et al. 1973). This implies that h has exceeded $1-\phi$, that is $\phi \neq 0$, in which case a further two configurations can be rejected. This leaves four configurations, denoted *A*, *B*, *C* and *D* shown in the montage of Fig. 2 above.

These configurations are characterised by different combinations of phases of operation, such as were described in the previous section. Apart from the obvious flow from *P* through *T*, and the asymptotic equilibrium conditions, $\dot{V}O_2$ may be in a transient phase tending to a steady state, or in a phase constant at $\dot{V}O_2$ max; and lactate production may be in a phase depending on the difference between h and $l+\theta$, or in a phase dependent only on l. Denoting these phases of activity by $\dot{V}O_2$ tran, $\dot{V}O_2$ max, La_d and La_l respectively, then the model configurations and phases can be collated in Table 1 below, in which an * denotes the presence of that phase in the appropriate model configuration.

4. Mathematical solution

Since the net flow through *T* above resting level at any time is made up of the sum of flows through R_1, R_2 and the drop in level of vessel *P*, the behaviour of

Table 1. Phase characteristics of the M-M model configurations

Active energy	Energy source description	Model configuration A	B	C	D
A1	$\dot{V}O_2$ tran	*	*	*	*
A2	$\dot{V}O_2$ tran & La_d	*	*	*	*
A3	$\dot{V}O_2$ max & La_d			*	*
A4	$\dot{V}O_2$ tran & La_l	*			
A5	$\dot{V}O_2$ max & La_l	*	*	*	

the system is governed by a single differential equation, an equation of net energy balance given by

$$W = \dot{V}O_2 - \dot{V}_P - \dot{V}_L \tag{1}$$

and it is this equation in a variety of forms which will be investigated. In physiological terms, the total measured workrate or power output is given simply by the sum of oxidative power, alactic power and glycolytic power, all measured above resting levels.

4.1. Phase A1

Consider the first phase of activity, common to all four configurations, and suppose that in response to a constant workrate W, the fluid in P has dropped to a level $h < \theta$, (refer Fig. 1). The form of Eq. (1) applicable in this phase is given by

$$W = \frac{M_O}{1-\phi} h + A_P \frac{dh}{dt}. \tag{2}$$

This is a simple first order linear differential equation having as its general solution

$$h = \frac{A_P(1-\phi)}{M_O}\left[\frac{W}{A_P} - \exp\left[-\frac{M_O}{A_P(1-\phi)}(t+c)\right]\right] \tag{3}$$

where c is an arbitrary constant, its value to be determined by the initial conditions. These are simply that $h = 0$ at $t = 0$. Thus the particular solution, starting from rest, is given by:

$$h = \frac{A_P(1-\phi)}{M_O}\left[1 - \exp\left[-\frac{M_O t}{A_P(1-\phi)}\right]\right] \tag{4}$$

from which using Eqs. (1), (2) and (3)

$$\dot{V}O_2 = W\left[1 - \exp\left[-\frac{M_O t}{A_P(1-\phi)}\right]\right] \tag{5}$$

$$-\dot{V}_P = W \exp\left[-\frac{M_O t}{A_P(1-\phi)}\right] \tag{6}$$

both expressed in workrate units, and of course $-\dot{V}_L$ is zero.

Thus provided $W \leqslant W_\theta = M_O\theta/(1-\phi)$, then for large t, an asymptotic steady state is reached, when $h = W(1-\phi)/M_O$ and $VO_2 = W$, and exercise is completely aerobic. The alactic oxygen debt at equilibrium is given by an amount $A_P W(1-\phi)/M_O$, the empty volume in P above the steady state level of h.

It is of interest to note that Eq. (5) is of identical form to the empirical model proposed by Henry (1951), and has been used by physiologists to model oxygen kinetics for light workloads for many years. Of interest also is the fact that the exponential rate constant $M_O/A_P(1-\phi)$ is independent of W, a subject of controversy among physiologists, (Whipp and Wasserman 1972).

4.2. *Phase 1R*

To consider the recovery phase initiated during phase A1, denoted 1R, suppose that at some time t^*, when $h = h^* \leq \theta$, and $\dot{V}O_2 = V^* \leq W_\theta$, tap T is closed corresponding to the end of exercise and commencement of recovery. Equations (2) and (3) still apply, but with W changed to zero and initial conditions $h = h^*$ at $t = t^*$. We can then obtain:

$$\begin{aligned} h &= h^* \exp\left[-\frac{M_O(t-t^*)}{A_P(1-\phi)}\right] \\ \dot{V}O_2 &= V^* \exp\left[-\frac{M_O(t-t^*)}{A_P(1-\phi)}\right] \\ -\dot{V}_P &= -\frac{M_O h^*}{1-\phi} \exp\left[-\frac{M_O(t-t^*)}{A_P(1-\phi)}\right] \end{aligned} \tag{7}$$

where $M_O h^*/(1-\phi) = V^*$.

Again it is of interest to note that Eq. (7) is of identical form to the $\dot{V}O_2$ recovery curve in common use by physiologists for the repayment of oxygen debt (Henry 1951; Leger et al. 1980). The rate constant is independent of W, and is the same value as during exercise, as has been observed experimentally (Henry and de Moor, 1956).

4.3. *Phase A2*

Suppose however that tap T had been opened wider at the start, to some constant $W > \dot{W}_\theta$. During phase A1, Eqs. (2)-(6) will still apply, but only up until some time t_1, when $h = h_1 = \theta$, and which marks the end of phase A1. This time can be obtained as the solution for t in Eq. (4) when $h = \theta$, viz.

$$t_1 = -\frac{A_P(1-\phi)}{M_O} \ln\left[1 - \frac{M_O\theta}{W(1-\phi)}\right]$$

which is only defined for $W > M_O\theta/(1-\theta) = W_\theta$.

Thus consider phase A2, and suppose that fluid in P has dropped to a level $\theta < h < 1-\phi$, and that as a result of the induced flow through R_2, fluid in vessel L has dropped to a level $0 < l < h - \theta$, (refer Fig. 1). The form of Eq. (1) applicable during phase A2 is given by

$$W = \frac{M_O}{1-\phi} h + A_P \frac{dh}{dt} + A_L \frac{dl}{dt}. \tag{8}$$

Since the flow through R_2 is in accordance with the differences in levels between vessels P and L, we have

$$A_L \frac{dl}{dt} = M_L \frac{h - l - \theta}{1 - \theta - \lambda}$$

i.e.

$$h = \frac{A_L(1-\theta-\lambda)}{M_L}\frac{dl}{dt} + l + \theta \tag{9}$$

and

$$\frac{dh}{dt} = \frac{A_L(1-\theta-\lambda)}{M_L}\frac{d^2l}{dt^2} + \frac{dl}{dt}. \tag{10}$$

Now substituting Eq. (9) and (10) into Eq. (8) and simplifying, yields

$$\frac{d^2l}{dt^2} + \frac{M_O A_L(1-\theta-\lambda) + M_L(A_P + A_L)(1-\phi)}{A_P A_L(1-\phi)(1-\theta-\lambda)}\frac{dl}{dt} + \frac{M_O M_L}{A_P A_L(1-\phi)(1-\theta-\lambda)}l$$
$$= \frac{M_L(W(1-\phi) - M_0\theta)}{A_P A_L(1-\phi)(1-\theta-\lambda)}. \tag{11}$$

This second-order differential equation with constant coefficients is of form:

$$\frac{d^2l}{dt^2} + a\frac{dl}{dt} + bl = c$$

where a, b and c are constants and can be solved for l utilising the following details.

(i) Auxilliary equation: $r^2 + ar + b = 0$ which has two negative roots r_1 and r_2 obtainable by solution with coefficients a and b from Eq. (11).

(ii) Complementary function $l_c = c_1 e^{r_1 t} + c_2 e^{r_2 t}$ where c_1 and c_2 are arbitrary constants.

(iii) Particular integral: $l_p = c/b$ with coefficients c and b from Eq. (11).

(iv) The general solution is given by

$$l = c_1 e^{r_1 t} + c_2 e^{r_2 t} + c/b. \tag{12}$$

(v) Constants c_1 and c_2 can be obtained making use of the boundary conditions, that: at $t = t_1$

$$l = \frac{dl}{dt} = 0$$

by utilising Eq. (12) and its derivative at $t = t_1$.

Having now obtained the phase A2 applicable version of Eq. (12), it together with its derivative can be substituted into Eq. (9) to obtain the phase A2 applicable version of Eq. (9). This is of the form:

$$h = k_1 e^{r_1 t} + k_2 e^{r_2 t} + c/b + \theta \tag{13}$$

where k_1 and k_2 are constants.

By considering Eqs. (12) and (13) as $t \to \infty$, but provided $W \leqslant M_0$ or $\leqslant M_O(1-\lambda)/(1-\phi)$ then an aerobic steady state is possible during phase A2, with h and l tending to asymptotic levels in configuration A, differing only by an amount θ. It is relevant to note however that after t_1 but prior to the attainment of the steady state, there occurs a partially anaerobic period of exercise. This is the early or transient lactate production discussed by Cerretelli et al. (1979) and mentioned earlier. A second exponential component of $\dot{V}O_2$, originally derived empirically by Henry and de Moor (1956), has been attributed by Whipp and Wasserman (1972) to this early lactate production.

Lastly, using Eqs. (8), (12), (13) and its derivative, we can obtain equations for $\dot{V}O_2$, $-\dot{V}_P$ and $-\dot{V}_L$, the three energy components of the exercise.

4.4. *Phase 2R1*

Suppose that exercise was to cease at a time t^* prior to the attainment of a steady state during phase A2, when $\theta < h = h^* \leqslant 1-\phi$, (or $h^* \leqslant 1-\lambda$ for configuration *A*), $\dot{V}O_2 = V^*$ and $0 < l = l^* < 1-\theta-\lambda$, then Eqs. (8)-(11) would still hold, but with $W = 0$. So also would Eq. (12), but with $W = 0$ also and different values of c, c_1 and c_2; say c', c_1' and c_2' i.e.

$$l = c_1' e^{r_1 t} + c_2' e^{r_2 t} + c'/b \tag{14}$$

The constants c_1' and c_2' can be determined from the boundary conditions that at $t = t^*$, $l = l^*$ and dl/dt from Eq. (12) equals dl/dt from (14). Equation (14) together with its derivative can be substituted into (9) to obtain the equation for h, which is of the form:

$$h = k_1' e^{r_1 t} + k_2' e^{r_2 t} + c'/b + \theta \tag{15}$$

Once again, using (8) with $W = 0$, (14), and (15) and its derivative, equations for the three energy components during recovery phase 2R1 can be obtained.

During this phase, the delayed lactic acid formation occurs, (di Prampero et al., 1979). Phase 2R1 is of fairly short duration, ending when the levels in P and L equate, i.e. at a time when $h = l + \theta$. Recovery from this time point on, is equivalent to the situation which would have arisen if phase A2 had continued to a steady state before exercise ceased. Let us therefore consider this stage of recovery.

4.5. *Phase 2R2*

This phase is characterised by both dh/dt and dl/dt being negative, that is P and L are being refilled from O. In the typical situation $0 < l < 1-\theta-\lambda$ and $h < l + \theta$, and the level in L lags behind the level in P (this time both rising). Equation (8), with $W = 0$ once again applies, but a flow through R_3 must now be considered. This flow is determined by the difference in levels between P and L. *i.e.*

$$A_L \frac{dl}{dt} = -M_R \frac{l + \theta - h}{1-\lambda}$$

$$\therefore \quad h = \frac{A_L(1-\lambda)}{M_R} \frac{dl}{dt} + l + \theta \tag{16}$$

and

$$\frac{dh}{dt} = \frac{A_L(1-\lambda)}{M_R} \frac{d^2 l}{dt^2} + \frac{dl}{dt}. \tag{17}$$

Equations (16) and (17) can now be substituted into (8) (with $W = 0$), which yields another second order differential equation of the same form as (11), which therefore has another biexponential solution of the same form as (12). The general procedure for solution is therefore analogous to Sects. 4.3 and 4.4.

Phase 2R2, because of the very small magnitude of M_R, continues for an extended period of time, until $l=0$. However, the behaviour of h is dominated by the faster of the two exponential terms, and tends fairly quickly to a very small positive value almost indistinguishable from zero. Once L has been refilled, a final phase 2R3 occurs when h actually does drop to zero, (that is, the resting level). The characteristics of this phase are analogous to phase 1R.

4.6. Digression

The above described phases, both active and recovery, are all applicable to the four configurations of the M-M model shown in Fig. 2. However it has been noted above, and in Morton (1985b), that experimental observations on $\dot{V}O_2$ of exercising athletes have been unable to distinguish between configurations *A*, *B*, *C*, and *D*. In general this is because exhaustion or fatigue has led to early termination of the exercise periods of high workload necessary to distinguish these configurations. For this, and for parsimonious reasons, the remainder of this section will consider only the differential equations for the various active and recovery phases of the four configurations, without rigorous solution and with minimal comment. Whenever the necessary information becomes available, mathematically inclined physiologists can return here, extract the appropriate equations and solve them. The notation tacitly adopted above will be retained. That is, the remaining active phases of Table 1 are denoted A3, A4 and A5 respectively, and the recovery phases commencing at the end of one of these three active phases are denoted 3R1, 3R2, etc., 4R1, 4R2, etc. and 5R1, 5R2, etc. respectively. Relative positions of h and l can be deduced by referring to Figs. 1 or 2 as necessary.

4.7. Remaining phases

Consider firstly the third active phase, applicable to configurations *C* and *D* only, followed by a recovery. The following differential equations apply:

During A3:

$$W = M_O + A_P\frac{dh}{dt} + A_L\frac{dl}{dt}$$

$$A_L\frac{dl}{dt} = M_L\frac{h-l-\theta}{1-\theta-\lambda}$$

which leads to a solution of the form $\alpha e^{-kt} + \beta t + \gamma$ (18)

During 3R1:

$$0 = M_O + A_P\frac{dh}{dt} + A_L\frac{dl}{dt}$$

$$A_L\frac{dl}{dt} = M_L\frac{h-l-\theta}{1-\theta-\lambda}$$

which is a short lived phase of delayed lactic acid formation, and leads to a solution of the same form as (18).

During 3R2:

$$0 = M_O + A_P \frac{dh}{dt} + A_L \frac{dl}{dt}$$

$$A_L \frac{dl}{dt} = -M_R \frac{l + \theta - h}{1 - \lambda}$$

another phase of short duration during which (as in 3R1) $\dot{V}O_2 = M_O$, and which has a solution also of the form of (18).

During 3R3:

$$0 = \frac{M_O}{1 - \phi} h + A_P \frac{dh}{dt} + A_L \frac{dl}{dt}$$

$$A_L \frac{dl}{dt} = -M_R \frac{l + \theta - h}{1 - \lambda}$$

which is a phase of long duration, analogous to 2R2, with a similar biexponential solution.

During 3R4:

$$0 = \frac{M_O}{1 - \phi} h + A_P \frac{dh}{dt}$$

which leads to a single exponential as in 1R.

Note. I have assumed above that 3R1 ends when $h = l + \theta > 1 - \phi$. It is of course possible that $h = l + \theta = 1 - \phi$, in which 3R2 does not occur. It is also possible that $h = l + \theta < 1 - \phi$ in which case 3R1 ends when $h = 1 - \phi$. Phase 3R1 is then a second phase of delayed lactic acid formation analogous to 2R1, ending when $h = l + \theta$, then passing to 3R3.

Next consider the fourth active phase, A4, applicable only to configuration *A*, followed by recovery. The following differential equations apply:
During A4:

$$W = \frac{M_O}{1 - \phi} h + A_P \frac{dh}{dt} + A_L \frac{dl}{dt} \tag{19}$$

$$A_L \frac{dl}{dt} = M_L \frac{1 - \theta - \lambda - l}{1 - \theta - \lambda} \tag{20}$$

which pair of equations represent a different structure from previous cases, requiring (20) to be solved first, then substituting into (19) to give an equation of form

$$\frac{dh}{dt} = \alpha h + \beta e^{-kt} + \gamma \tag{21}$$

which solved for h gives an equation of form

$$(at+b)\,e^{-ct}+d \tag{22}$$

from which, provided $W \leqslant M_O$, it can be shown that h tends to an asymptotic steady state as $t \to \infty$.

During 4R1: Eq. (19) with $W=0$, and (20) apply, and this phase has similar solution to (22). It is a phase of delayed lactic acid formation, ending with $h=1-\lambda$.

During 4R2:

$$0=\frac{M_O}{1-\phi}h+A_P\frac{dh}{dt}+A_L\frac{dl}{dt}$$

$$A_L\frac{dl}{dt}=M_L\frac{h-l-\theta}{1-\theta-\lambda}$$

which is a second phase of delayed lactic acid production, a biexponential for l analogous to (14) in 2R1, ending when $h=l+\theta$.

During 4R3:

$$0=\frac{M_O}{1-\phi}h+A_P\frac{dh}{dt}+A_L\frac{dl}{dt}$$

$$A_L\frac{dl}{dt}=-M_R\frac{l+\theta-h}{1-\lambda}$$

which yields another biexponential solution, analogous to 2R2, a phase of long duration.

During 4R4:

$$0=\frac{M_O}{1-\phi}h+A_P\frac{dh}{dt}$$

a single exponential decline in h as in 3R4, 2R3 and 1R.

Lastly, consider the fifth active phase, A5, and recovery, applicable to configurations A, B and C. The following equations apply:

During A5:

$$W=M_O+A_P\frac{dh}{dt}+A_L\frac{dl}{dt} \tag{23}$$

$$A_L\frac{dl}{dt}=M_L\frac{1-\theta-\lambda-l}{1-\theta-\lambda} \tag{24}$$

which pair represent yet another structure. The solution for l in (24), differentiated and substituted in (23) yields an equation which can be integrated directly for h.

During 5R1: Eqs. (23) with $W=0$, (24) apply, and the solution is obtained as in A5. This is a phase of delayed lactic acid formation, terminating when $h=1-\phi$ in configurations A and B, but when $h=1-\lambda$ in configuration C.

During 5R2, etc: For configuration *A*, analogous stages to 4R1 to 4R4 apply. For configuration *B*, analogous stages to 2R1 to 2R3 apply. For configuration *C*, analogous stages to 3R1 to 3R4 apply (with the proviso mentioned therein).

5. Experimental verification

As evidence of verification to parts of the M-M model, consider firstly the following $\dot{V}O_2$ time series, fully described in Morton (1984b, 1985a). The subject exercised at a light workload (133 watts) for 5 min, followed by 3 min of recovery. Figure 3 shows this data, plotted breath by breath for the first 150 s of phases A1 and 1R; otherwise every third breath is plotted.

Jointly fitted curves using the *BMDP* package (Dixon, 1983) to the active and recovery phases are respectively:

$$\dot{V}O_2 = 0.527 + 1.559\ (1 - e^{-0.0244t}) \qquad \text{l/min}$$

and

$$1{\cdot}559e^{-0.0217t} + 0.527 \qquad \text{l/min}$$

and are plotted in Fig. 3 also. Coefficients of determination for the phases are respectively $R^2 = 0.867$ and $R^2 = 0.893$. These values may appear somewhat low, but note that breath by breath variability is relatively large, and there may also be a cyclic departure from regression (Morton, 1985a). The exponential time constants for the active and recovery phases do not differ significantly at the 5% level.

Consider secondly the same subject, exercising similarly at a severe workload (300 watts). The $\dot{V}O_2$ time series plot is shown in Fig. 4.

The anaerobic threshold for this subject is estimated to be at a $\dot{V}O_2$ of 2.835 l/min (2.430 l/min above the resting level). Active phase fitted curves for $\dot{V}O_2$ in l/min are as follows:

$$\text{A1:}\quad 0.405 + 3.329(1 - e^{-0.0221t}) \qquad \text{for } 0 < t \leq 59.3\ s$$

$$\text{A2:}\quad 4.213 - 0.105e^{-0.366(t-59.3)} - 1.27e^{-0.00546(t-59.3)} \qquad \text{for } 59.3 \leq t < 300\ s.$$

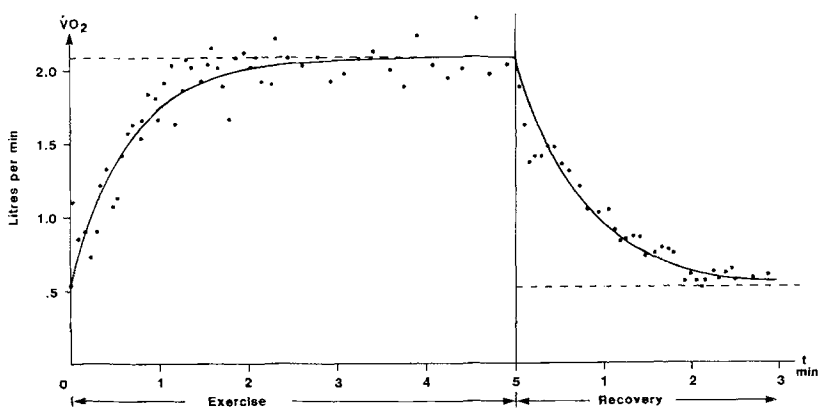

Fig. 3. $\dot{V}O_2$ for light exercise and recovery

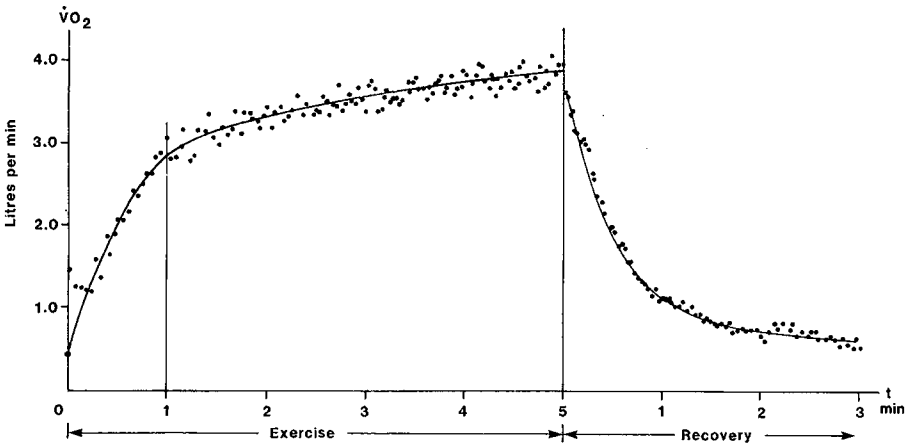

Fig. 4. $\dot{V}O_2$ for strenuous exercise and recovery

For these two phases the R^2 values were respectively 0.849 and 0.877. Fitting recovery curves is not quite so simple since the "break point" is not clearly defined, though it can be deduced from Fig. 1 that it must occur at some $\dot{V}O_2$ in excess of 2.835 l/min. A brute force approach such as that of Orr et al. (1982) has been adopted and the following curves obtained:

$$2R1: \quad -7.160+0.241\, e^{-0.366(t-300)}+10.79\, e^{-0.00546(t-300)}$$

with a phase duration of 10.9 s, and

$$2R2: \quad 0.437+2.491\, e^{-0.0272(t-310.9)}+0.083\, e^{-0.0000124(t-310.9)}$$

That is to say, the phase duration is determined so as to minimise the total sum of squared residuals over both line segments. The two line segments were fitted using the BMDP package, (Dixon 1983). Coefficients of determination were respectively 0.738 and 0.866. In phase 2R2 the second exponential term was only just significant at the 10% level, but the coefficients are of comparable magnitudes to their counterparts in Morton (1984a), which were derived from previously published values of the appropriate physiological constants.

It should be noted that these data were not collected for the purpose of elucidating this model, and the above evidence should therefore be interpreted accordingly. Furthermore these curves lend support only to one of the three bioenergetic components. Measurements equally in agreement with the theory would be needed on at least one of the other two components before the whole model could be confirmed.

The most useful such confirmatory experimental data would be some measure of the rate of lactic acid production by the working muscles. Lactic acid concentrations in the blood are routinely taken both during exercise and recovery, and from these, inferences on production are made. However, since the blood is neither the location of lactic acid production nor the location of any significant removal, such inferences must be regarded as hazardous. Needle biopsy techniques are available to obtain muscle lactate concentrations, from which lactate production can be calculated but these cannot realistically be performed on the

working muscles say every 10 or 20 s during the exercise itself. Until this measurement problem is solved, or until measurements on phosphagen breakdown become available, neither the unambiguous configuration nor the complete acceptance of the M-M model can be certain.

6. Comment and further development

It has been pointed out (referee communication) that the model is not biologically realistic in the sense that there do not exist separate aerobic and anaerobic fuel stores, at least as far as carbohydrate is concerned, (fat is only accessible aerobically). Perhaps therefore this should be modelled by a single compartment with two taps, one representing aerobic access via oxygen consumption, and the other anaerobic access via lactate production. On the face of it, this would seem appropriate, but the quantitative exploitation of such a change requires the very important assumption as to the operation of some governing mechanism which determines the balance of flows between the two taps. This is no simple matter, and for the present this paper chooses to exploit the properties of the general three compartment model to the full, before turning to such an alteration, which would of course represent progress of the sort I allude to below.

Nevertheless those two further developments of the M-M model, unambiguous configuration and acceptance by physiologists, must now become the focus of some research effort. In particular some method must be devised for continuously monitoring the production of lactic acid by the working muscles. Exercise physiologists are best suited to these methodological developments. On the other hand, a theoretical modelling and simulation approach may provide an alternate avenue out of those difficulties. Since the rigorous exploitation of conceptual models, at least in exercise physiology, lags behind detailed experimentally derived knowledge, there will undoubtedly occur future developments which render the M-M model obsolete, or at least inapplicable except in rather general circumstances. This is progress. This is not to say however, that models such as the one presented here, although not leading or suggesting new directions nor novel experiments, perform no useful role. Such a useful role for example lies in the teaching field and as a learning aid. Indeed, stochastically treated microcomputer simulations of certain configurations of this model produce "data" realistic enough for students to analyse as part of their assignment work in a course in human exercise physiology.

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