A MATHEMATICAL AND COMPUTER SIMULATION MODEL
OF THE RUNNING ATHLETE

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SIGNED: R. Hugh Morton — OCTOBER, 1984

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SAMPLE COMPUTER OUTPUTS IN SEPARATE BINDER

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ABSTRACT

This thesis describes the construction, computerisation and simulation of a mathematical model of the running athlete. The model is in part biomechanical and in part biochemical, in part theoretical and in part empirical.

A three variable (force, velocity and distance) Newtonian biomechanical model of Keller (1973, 1974) is examined, and extended to include a fourth variable, power developed. This model segment is feed-forward linked by equating mechanical power developed to bioenergetic power supplied. A proposed three component bioenergetic model of Margaria (1976) was examined in detail (Morton 1984), but found to be unsuitable. Thus an empirical three component model segment was developed. Bioenergetic power is supplied by three variables, oxygen uptake, and glycolytic and alactic body energy store depletions. The glycolytic process is of particular interest, since the accumulation of lactic acid in the working muscle can induce fatigue. A two compartment, working muscle and blood volume, physiological model segment originally developed by Freund and Zouloumian (1981a, b) for post exercise is examined for use during exercise. This segment takes as input the lactate produced by the glycolytic energy process and circulates it between compartments, or removes it by biochemical breakdown. Lactate concentrations in the two compartments are the modelled variables in this segment. Finally a negative feedback link in the whole model is provided by a fatigue equation, where the maximum muscular force exertable is constrained inversely by the lactate concentration increase in the working muscles. The athlete of course can by choice operate at a force below the constraining level, in which case the feedback link does not operate. Sooner or later however, except for the lower workloads, muscular lactate will build up to such a level as to invoke the feed-back.

Parameters of the whole model include initial body energy stores, maximal muscular strength, resistance to motion, diffusion constants for lactate circulation, bioenergetic parameters of the oxidative, glycolytic and alactic energy processes, biomechanical energy equivalents, body mass, fatigue coefficients, etc, twenty four in number. These parameters bind the relationships between the nine variables of the whole model, expressed as nine simultaneous differential equations with respect to time. Parameter values were in some cases obtained by estimation from data on exercising subjects, specially collected for that purpose. In other cases values determined experimentally by other researchers and published in the literature were utilised.

Simulation was performed using numerical integration methods provided by a computer programme from the NAG library of routines (NAG, 1983). Simulated results on all nine variables are realistic, conforming well to those observed in the laboratory on exercising subjects. There remains scope however for future refinements, in the main improving the theoretical content of the whole model and in the process extending it to include the recovery period after exercise.

CHAPTER 1

INTRODUCTION AND OVERVIEW

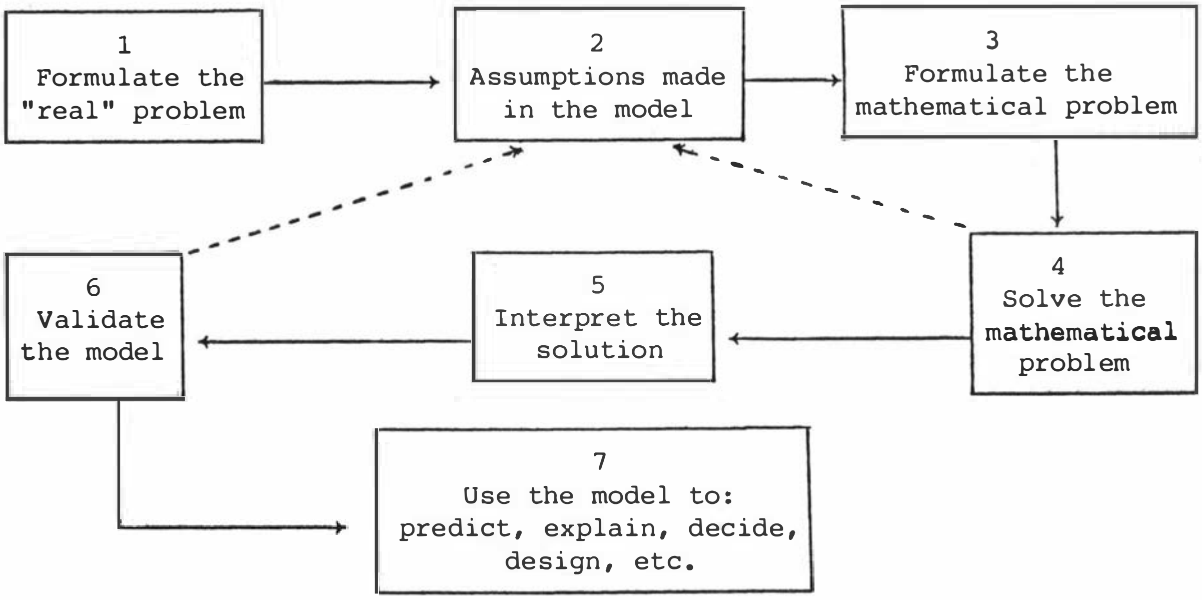
INTRODUCTION

This study began with the delightfully vague title: "The Application of Quantitative Methods to the Study of Biological Movement". Thus it would initially seem to be a study in the area known as biomechanics; that is the application of the principles of mechanics and dynamics to the study of biological movement. However, as my early work progressed and the ideas crystallised, it became the application of those techniques more specifically referred to collectively as modelling. Furthermore, their application was extended beyond biomechanics, to include physiological modelling (bioenergetic and biochemical), but restricted to human locomotion. Hence the final title: "A Mathematical and Computer Simulation Model of the Running Athlete". This provided the most interesting and challenging scope for investigative research.

The general interpretation of the word 'model' is: an imitation of the original, often in miniature, and bearing a strong resemblence to that original. In the scientific context, a model is an abstraction which faithfully captures the essential features of the original, but not necessarily the details of lesser importance. More specifically a 'mathematical' model is an abstraction expressed in mathematical terms. In the biological context, it becomes a mathematical description of a biological system, in this case, the running athlete. Adding the term 'computer', implies that a computer can, and has been, programmed with the mathematical equations and relationships comprising the model. Adding also the term 'simulation', implies that the results obtained by the computer when instructed to invoke the programme, simulates the behaviour of the real process being described; at least to a reasonably accurate degree. Indeed, it is this latter property which gives rise to the most common use of mathematical models: simulating and then studying the behaviour of a system without the necessity to obtain real data through actual observation and experimentation. This is not to say that experimental data is in any way unnecessary.

Many authors have already criticised modelling and simulation, favourably and otherwise, for example Standing and Tidball (1977). Nevertheless it is still useful to consider some of the features of mathematical modelling. The mathematical model has the advantages that mathematics is more efficient and less bulky than the written word; that it is especially suited to the quantitative elements of a system; that it is more difficult to falsify a conclusion in a mathematical argument; and that optimal solutions are possible through analysis. By using the abstraction, we are able to consider certain effects, the object being to see which of these account for given observations, and which effects are immaterial. One serious criticism of the combination is that no new information is obtained, for we can only obtain what is implicit in the concepts, equations and numerical data incorporated in the model.

Burghes and Wood (1980) have provided a useful introduction to models and modelling. They describe the process of modelling by the following diagram:



Arrows indicate the flows of progress, including likely circulation through various stages, though it is possible to bypass one or more stages. For example, in a very simple situation we may pass directly from stage 1 to stage 7. In general, stages 1, 2 and 3 are referred to as formulation, 4 as solution, and 5, 6 and 7 as application of the model respectively.

The mathematical equations involved in stage 3 can be arrived at through two possible routes, the empirical and the theoretical, and the two can, and often are, mixed in a many-equation model. The empirical approach can be characterised by fitting curves to experimentally obtained data. The equation is chosen for the simple expedient that it fits the real situation. These equations often have no theoretical foundation and their parameters have no meaningful biological interpretation. The theoretical approach can be characterised by the use of differential equations based on theoretical principles or assumptions. Though mathematically elegant, solutions to such equations may not be applicable to experimental situations. In most such cases this leads to a rejection of the differential equation, and its replacement by one which does yield an applicable solution. Such is the case in this study and in other examples cited below. A working model is usually a blend of both approaches, for some workers approach from one direction and others from another, and the two meet. Any given model may contain from as few as one, to a very large number of equations. The former are simple to understand, but lack detail, and vice versa for the latter. Once again a working model is a blend.

Mathematical modelling in biology is an expansive area of study, and interesting though it is, it is necessary to curtail a review of the literature to those of more obvious relevance. The following have provided insight but have been of little direct assistance: a model of walking (Mochon and McMahon 1980); a model of swimming (Seireg and Baz 1971); a model of pole vaulting (Walker and Kirmser 1973); models of long jumping (Brearley 1972, Hatze 1981). All these are biomechanical models of human movement. There are many models of human physiological systems which fall into the same category: a model of human respiration, modified to exhibit the effects of exercise, known as Macpuff (Dickinson 1977, Jones 1980); ventilatory control (Stegeman et al 1975); heart rate regulation during exercise and recovery (Hajek et al 1980); cardiovascular response to exercise (Stremel et al 1975).

There have of course been a large number of studies which have been of considerable assistance in the construction of my model of the running athlete. These are considered in depth in the later chapters. I have not been able to find reference to any study which has been both biomechanical and physiological. This construct of mine may therefore be the first such model.

Thus in this study, a complex series of mathematical equations are developed to describe the biomechanical, bioenergetic, physiological and biochemical components of the system. Some of these equations are empirical, but most theoretical. Some are borrowed from elsewhere, amended in some cases, and others are newly created. Their combination represents a unified new whole. A computer is utilised to provide simulated results, statistical procedures based on the analysis of experimental data providing an input to the programming. A series of simulated results under differing conditions, when compared to the actual behaviour of running athletes, provide the basis for validation of the model and define the boundaries of its area of applicability.

OVERVIEW

There have been many attempts to model running or running performance, and these are reviewed briefly in Chapter Two. With one exception, none contains more than a single equation though it may have alternative forms, and many attempts are solely graphical. One or two of the equations, are nevertheless quite comprehensive, but even these are of little more than historical relevance. The one exception, which appears to be the only attempt to model a runner himself, contains just three equations. It is based on simple Newtonian mechanics; force, velocity, power, distance and time. It is considered as a problem in the context of optimal control theory, however, the ideas are there and thus Chapter Three considers this model as a starting point. There it is critically examined, generalised and extended in a number of ways.

The major extension, the subject of Chapter Four, is the replacement of a single simple energy balance equation by three new equations : one related to the oxidative energy source, one to the glycolytic source (glucose → glycogen → lactic acid), and the third to the phosphagen based source (alactic, based on the conversion of high-energy phosphates). A three-compartment hydraulic model of these energy flows has been proposed, but no mathematical solution offered. Once I obtained the solution (the subject of Appendix 1), I found not only that the suggested graphical solution offered with the model was incorrect, but that the actual solution and some of its implications, were not sufficiently true to life to utilise. Thus I have resorted to some empirical modelling of my own.

Chapter Five considers an existing two-compartment lactic acid (lactate) circulation model. This physiological model is an excellent example of one arrived at from sound theoretical principles and whose mathematical solution fits observed data very well. The two compartments represent the (previously) working muscles and the remainder of the body. Flows of lactate into (production), between (transfer), and out of (utilisation or breakdown) the compartments are considered for an athlete at rest, but starting immediately after the cessation of exercise. Careful examination of the model suggests no reason for supposing any of the assumptions of the model to be invalid for an athlete at work, i.e. running. All that is necessary therefore is to replace one equation, the production of lactate by the (now) working muscles, by another, which is the one relating to the glycolytic energy source mentioned above. This one of the three bioenergetic equations is the 'feed-forward' link between the two biomechanical and physiological components of the whole model.

The 'feedback' link in the model is the subject of Chapter Six. Fatigue after prolonged exercise is primarily caused either by depletion of body reserves, which is characteristic of long-term exercise of low intensity, or by the **accumulation** of 'poisonous' catabolities, which is characteristic of shorter term exercise of higher intensity. Since this study considers athletes performing towards the limits of their capabilities, it is the latter form that is relevant. It is the accumulation of lactic acid in the working muscle, which, since the body cannot transfer or break it down fast enough, causes the muscle poisoning pains so well known to athletes. Thus a rise in the lactate concentration in the working muscle compartment, will tend to inhibit the muscular force which the athlete can exert through his legs. A large enough rise will override the athlete's own willpower and will compel him to slow down. This means a lesser energy requirement, hence a lesser lactate production. This is the self-correcting feedback.

Chapter Seven then considers the model as a linked whole, where each segment fits into the whole, the relationships between them, etc.

Chapters Eight and Nine cover the collection of data and its use in parameter estimation. The influence of much of this work is evident in various segments of the previous five chapters. The most available and suitable facilities for such a detailed study of exercising athletes are in the Respiratory Laboratory at the Princess Margaret Hospital (P.M.H.), Christchurch, New Zealand, and it was there that the data were collected. Six athletes volunteered for the study and all completed a series of tests. In addition I subjected myself to a series of tests. It was inconvenient to have to go to Christchurch, money for the study was limited, and I was working on my own, all of which reduced the scope of the experimental study. Nevertheless I was well satisfied with the support obtained at the P.M.H.

Once the whole model was constructed and suitable parameters chosen, it was implemented on the computer and an extensive set of simulation runs performed. These are the subject of Chapter Ten. They were designed with two main purposes in mind. Firstly they served as a source for the validation of the model; that is to ensure that given a normal set of parameters, the simulation results conform to what would actually occur to a real athlete under those conditions. Secondly they served to define the boundaries of normal performance expectation, and hence across the boundaries, to identify abnormal or unrealistic outcomes or combinations of parameter values. They also served to identify the sensitivity of athletic performance to changes (small or large) in parameter values.

These results were of great interest and are considered in a discussion in the whole-model context in Chapter Eleven. Included also in this thesis is a bibliography and attached are a selection of computer outputs pertaining to various segments of the study.

Lastly, since the athlete has the ability to wilfully control the muscular force exerted through his legs, and thereby influence all the other variables in the system, at least up until the time he is overtaken by fatigue, one is naturally led to consider some form of optimisation. It is acknowledged to be of great interest to discover optimum performances or optimum strategies for athletes in races, and also acknowledged that such discoveries are possible with this model. This aspect however has not been a driving force in the study. Had this been the case, a considerable reconstructing of the computer programming, and maybe of the model itself, may have been necessary. This would probably have detracted from, or at least altered, the flavour of the whole study.

CHAPTER 2

PREVIOUS ATTEMPTS AT MODELLING RUNNING

Performances in athletic running events are recorded over set distances. The relationship that is usually adopted between the distance run d and the time t it takes to cover the distance, is the average velocity $\bar{v} = d/t$. The locus of all average velocities is called the running curve. A wider interpretation of the term running curve is more usually understood to include velocity/time and distance/time relationships. A number of investigators have examined the behaviour of the running curve over the last 78 years with the aim of deriving an adequate model to fit the behaviour of the running curve.

The first recorded investigation was made by Kennelly (1906) who studied horses and humans, in various forms of locomotion. His approach was to plot the data on semi-log paper, and derived for the distance events

$$\bar{v} = 17.01/8\sqrt{d}$$

which, because fitted over a variety of standard and non-standard distances for both amateurs and professionals, generated a large average relative deviation.

Meade (1916) and Hill (1925) present graphical studies of the running curve. Kennelly (1926) followed up his earlier article, again using the same equation, but once again fitted graphically. Meade (1926) followed up also, with another graphical analysis, predicting that the existing 1500m world record should be about 1½ to 2 seconds less than it was at that time. Shortly afterwards it was lowered by 1.6 seconds!

Hill (1927a) used graphical analysis to discuss Kenelly's earlier work and concluded it to be inappropriate. He later (1927b) made a detailed analysis of sprint running and derived an exponential equation of form

$$v = fg\,a\,(1 - e^{-t/a})$$

In this equation, v is the instantaneous velocity of the runner, f is a dimensionless constant characteristic of the build, strength, skill and fitness of the subject, g is the acceleration due to gravity, and a is a constant with the dimensions of time and is linked to the "resistance" or "viscosity" of muscular contraction. This is the first occurrence of an equation of motion for a runner, which will recur a number of times. Its derivation, in somewhat simpler terms will be discussed in more detail in the next chapter.

Wissman (1929) developed three equations which fitted world records of the time quite well.

$$t = 2\,t_o\,d\,\sqrt[10]{(2d)^4} \quad - \quad \text{sprints}$$

$$t = 2\,t_o\,d\,\sqrt[10]{(2d)^2} \quad - \quad \text{middle distances}$$

$$t = 2\,t_o\,d\,\sqrt[10]{2d} \quad - \quad \text{long distances}$$

The model was cumbersome and, for a more useful treatment, one equation only would be much better.

Meade (1934), Allen (1936) and Teichs (1939, 1941) all made graphical studies of world records. Francis (1943) derived a hyperbolic equation claimed to be a good fit to mid- to long-distance world records of the time.

$$(\log d - 1.5)(\bar{v} - 3.2) = 6.081$$

The horizontal asymptote at 3.2m/sec was claimed to be the velocity that could be maintained indefinitely without tiring, "if it were not for lack of sleep, nourishment, etc". Horvath (1948) used the Francis hyperbola to analyse the performances of the Swedish athlete Gunder Hagg.

Ulbrich (1950) developed two different models for the running curve. In his first model he developed a set of parabolas: one for the sprints, one for the middle distances, and one for long distances. These equations are distance/time relationships, specifically

$$t = 96.7d^2 + 71d + 2.2$$
$$t = 8.82d^2 + 148.41d - 14.7$$
$$t = 17.23d^2 + 172.07d - 33.0$$

He of course realised that it would be more desirable to formulate a single equation to model all distances. His second model was empirical and when fitted by least squares to world record data yielded

$$t = 17.23 - 91.785\sqrt{d} + 223.983d - 10.7496d\sqrt{d} + 1.65595d^2$$

which described the data very well.

Henry and Trafton (1951) used the basic formula proposed earlier by Hill (1927b) to analyse the sprinting portion of the running curve. They obtained

$$v = v_{max}\,(1 - e^{-kt})$$

Lietzke (1952) generated a graphical solution for the running curve by passing a smooth curve through the running world records at the time. His derived equation for middle distances took the form

$$\log d = \log m + n \log t$$

though he did not solve the equation for the constants m and n. For shorter distances, and for very long distances, the equation is no better than that derived by Francis (1943).

Henry (1954a, b) published a two- and a five-term exponential model for the running curve, without derivation. The two term model

$$\bar{v} = -a_1 e^{-k_1 t} + a_2 e^{-k_2 t}$$

applied to distances up to 300 metres. Its expanded form

$$\bar{v} = -a_1 e^{-k_1 t} + a_2 e^{-k_2 t} + a_3 e^{-k_3 t} + a_4 e^{-k_4 t} + a_5 e^{-k_5 t}$$

adequately described all racing distances up to the marathon. The derivation of this equation was subsequently published (Henry 1955).

The intent was that each term in the model should represent a physiological parameter. The first was a subtractive exponential term to represent the acceleration factor. The second and third terms were to represent alactic and lactic oxygen debt incurred during short and middle distance running. The fourth term was to represent glycogen depletion and the fifth term to represent all other metabolic rates. He evaluated the exponents from graphical analysis, obtaining half-lives for each of the physiological rates. He stresses that each term does not in itself account for its associated metabolic process, but rather is a term employed specifically for describing the running curve, and has a time constant of approximately the same order as the associated metabolic parameter. His equation adequately describes world record data of the time.

Other authors; Leitzke (1956), Meade and Lietzke (1956), Frucht (1960) and Amado (1962), described the running curve by graphical analysis but did not derive any mathematical formulations.

Lloyd (1966), following the ideas of Hill, has closely examined the running curve, dissecting it into six segments, the sprints, plus five groups of distance events. The all-out effort is represented by

$$d = S_0(1 - e^{-gt})/B + (R - A)(t - \Delta t)/B - \alpha m(\dot{d})^2/2B$$

where S_0 is an initial energy store, R and A represent oxygen uptakes, B is a power-efficiency factor, Δt is a delay factor and the last term represents a kinetic energy factor. For longer distances the exponential term drops to zero, giving

$$d = S_0/B - (R - A)\Delta t/B - \alpha m(\dot{d})^2/2B + t(R-A)/B$$

as a version of the simple equation

$$d = S_0/B + t(R - A)/B$$

he derived earlier, where the differences represent delays due to the rise in oxygen uptake and the acceleration of the runner.

Garcia (1968, 1969) made extensive use of the parabola, taking the approach that only portions of the running curve can have individual equation solutions. His equation was estimated as

$$t = .7714d^2 + 168.818d - 33.428$$

applicable to the middle and longer distances.

Zatsiorsky and Primakov (1969) utilise Hill's (1927b) exponential equation, but modify it to include v_{in}, the runner's speed when crossing the start line, thus

$$v = v_{in} + v_{asy}(1 - e^{-kt})$$

where v_{asy} is the asymptotic velocity reached over and above the initial velocity.

Rumball (1970, 1973) and Coleman developed an expression for mid and long distances

$$1/\bar{v} = f \ln d + c$$

The reciprocal of velocity is usually referred to as pace and the constant f as the "fatigue coefficient", since on a semilog scale it represents the change in pace. They utilised the coefficient to suggest a means of discovering for which distances athletes are most suited, and for which they can train appropriately. Their relationship breaks down at a hint of 'anaerobic running'.

Gardner and Purdy (1970) developed the following equation

$$t = t_1 + c_1 + c_2 v + c_3 f(d) v^2$$

where t_1 is a reference time and f(d) is the fraction of the event distance run on the curved part of the track. It was used as part of an algorithm for the generation of running event scoring tables, and was derived from some earlier scoring tables of Amado (1962).

Keller (1973) formulated a 3- equation system for the running curve in terms of various physiological parameters. He later published a mathematically optimal solution to his formulation, giving the strategy required of the runner in order to complete any given distance in minimum time.

The method of solution is an example of optimum control theory. This formulation, and its solution, represents the most soundly based approach to the analysis of the running curve that I have been able to locate. His solution prescribes;

i) Maximal effort for all races less than a critical distance $d_c = 291$ m, or

ii) Maximal acceleration for one or two seconds followed by constant velocity throughout the race and finally a slight slowing down at the end of the race, for distances longer than 291 m.

Keller's system has been both criticised and applauded. This system has formed the starting point for the biomechanical segment of my model for this study, and as such forms the basis for the next chapter. The derivation, analysis, this criticism and my own further investigation of Keller's system are fully discussed there.

Purdy (1974) has reformulated Henry's five term exponential equation with d rather than t as the independent variable.

$$\bar{v} = -b_1 e^{-r_1 d} + b_2 e^{-r_2 d} + b_3 e^{-r_3 d} + b_4 e^{-r_4 d} + b_5 e^{-r_5 d}$$

and this he has fitted to 1970 world record data by non-linear least squares. The description of three data sets of world records at various times, is very good.

Reigel (1977, 1981) examined distance/time reltionships, and for middle to long distances used the equation

$$t = a\ d^{\,b}$$

for running and other activities. He also examined the effects of age on speed, and the question of specific endurance.

Volkov and Lapin (1979) introduce a new instrument, the speedograph, for measuring sprinters' times and velocities, but still use graphical analysis to fit Henry's (1954) bi-exponential model.

Henstock (1979) examines the equation

$$\bar{v} = a + b/d$$

for world records at distances from 400-5000 m and it fits well. He then derives a second equation, based on physiological arguments

$$\bar{v} = \frac{aE}{d} + \sqrt{\left(\frac{aE^2}{d}\right) + bq}$$

where E is an energy store and q is a rate of energy supply, and a and b are parameters. This equation, with chosen values of a, E and q fits reasonably well to world records, but less well than the simpler equation. He makes no effort to fit the equation to data and estimate a and b though he does make some assumptions of the values of q and E.

Alexandrov and Lucht (1981) used the, by this time *accepted* , exponential velocity equation for sprinting derived originally by Hill (1927b), and found excellent fits. Of interest was their modification to the equation for sprints run on a curved track, and they evaluated the relative advantage gained by the runner on the outer lane over the runner on the inside lane. Senator (1982) has extended the theory used originally by Hill (1972b) and considered the ways in which the maximum asymptotic velocity developed is limited. In so doing he obtains a composite picture of the velocity curve, for indoor and outdoor sprinting.

In summary therefore, the collective literature on the study of running curves has certain general characteristics:

(a) Earlier work was characterised by graphical analyses.

(b) More recent work has been characterised by empirical curve fitting, which seems to have been developed to about the limit of plausible sophistication.

(c) The most recent work, with the exception of Hill (1927b) who was well ahead of his time, is characterised by a willingness to investigate from theoretical principles, and to attach physiological relevance to the solutions so obtained.

(d) With the exception of Keller (1973), none of the studies could be classified as a model system, for only his has more than a single equation.

(e) Throughout these studies there has been a heavy reliance on world record data available at the time of publication. Such data leads the model to represent a composite individual, rather than a particular one. The latter is a more helpful approach.

It follows then, that modelling the running athlete as a more complex biological system rather than a mechanical one, is a study topic well suited for development.

CHAPTER 3

NEWTONIAN BIOMECHANICS

BACKGROUND

This chapter deals with those aspects of the model commonly described as Newtonian mechanics, but applied to a biological entity. It deals with the inter-relationships between the following variables:

f(t) or f: the horizontal propulsive force at time t which the runner can exert through his legs. This is the control variable, since it is under the wilful control of the runner, (except when he becomes fatigued). Its influence permeates the whole system of equations to be developed in this and the next four chapters,

m: the mass of the runner, in kilograms (kg), regarded as a constant for a particular individual, (at least for a reasonable period of time),

v(t) or v: the instantaneous velocity of the runner, in metres per second (m/s or $m.s.^{-1}$),

dv/dt: the acceleration, i.e. the derivative of v with respect to t, in metres per second per second (m/s^2 or $m.s.^{-2}$),

D(t) or D: the distance travelled by the runner, in metres (m),

P(t) or P: the power developed by the runner, in watts (w), or equivalently the rate of energy consumption, dE/dt in joules per second, where E is the energy store in joules. P is referred to as external power, to distinguish it from internal (i.e. bioenergetic) power.

Firstly consider the relationships between some of these, as postulated originally by Hill (1927b), later by Henry and Trafton (1951), then by Lloyd (1966) and more recently by Keller (1973).

Hill's (1927b) formulation was as follows:

Suppose a runner to be of mass m and his weight is therefore given by mg, then since the maximum force he can exert is proportional to his weight, let $F = fmg$, where f is a propelling force to body weight ratio.

Hill supposed the limiting factor to continually increasing velocity to be the "viscosity" of the muscles, that is their resistance to even fairly slow contraction. This viscosity he assumed to be proportional to the speed of contraction and to the mass. He further assumed the velocity of the runner to be directly proportional to the speed of contraction of the muscles.

Thus $$R = \frac{mv}{a}$$

where R is resistance and a is a muscle viscosity coefficient.

By the second law of motion, Force = Mass x Acceleration, and since the force exerted in part goes to accelerate the runner and in part to overcome the resistance, we can deduce

$$fmg = mA + \frac{mv}{a}$$

where $A = dv/dt$ is the acceleration.

$$\text{i.e.} \quad \frac{dv}{dt} = fg - \frac{v}{a} \qquad \text{i.e.} \quad v = fga(1-e^{-\frac{t}{a}})$$

$$\text{and} \quad \frac{d^2y}{dt^2} + \frac{1}{a}\frac{dy}{dt} - fg = 0 \quad \text{i.e. } y = fga[t-a(1 - e^{-\frac{t}{a}})]$$

where y is the distance covered.

The main result is that v approaches its limit along an exponential curve.

Hill acknowledged that at least two other factors may give rise to resistance; unevenness or friction on the ground and air resistance. The former he reckoned to be negligible on a good track. At very low speeds, and even at higher speeds for streamlined bodies and laminar airflow, air resistance is proportional to velocity. For the sprinting athlete he considered air resistance proportional to $(\text{velocity})^2$. He regarded this resistance to be negligible also, though gauged it to be of importance in record breaking. Thus neither of the above factors were taken into account in the above equation.

Hill collected data from some runners; over 60 yards for one group, timed at 1, 3, 6, 10, 15, 20, 30, 40, 50 and 60 yards, and over 200 yards for a second group, timed at 1, 3, 6, 10, 15, 20, 40, 60, 80, 100, 120, 140, 160, 180 and 200 yards. The first data set fits the equation well, though no statistical analysis was performed. The f's are deemed remarkably similar for dissimilar individuals, but the a's differ noticeably. For the second group the fit is not so good as there is evidence of a decline in velocity after about 100 yards or so. This, he argues, is due to the onset of fatigue in the muscles, since fatigued muscles have been shown to have higher "viscosity".

The second piece of substantive work was due to Henry and Trafton (1951). By this time Hill's idea of the 'viscosity" as a limiting factor to speed of muscle contraction was no longer accepted and had been replaced by a "tension loss". Regardless of how it is described the postulation of some limiting factor proportional to velocity seems a reasonable assumption.

In an analagous development Henry and Trafton derived an equation of the same form as Hill's,

$$v = v_m (1 - e^{-kt})$$

Henry and Trafton's study was to answer the questions:

i) does the curve really fit statistically?

ii) are there individual differences in curve constants?

iii) what is the relative importance of the curve constants in determining the speed of a run?

iv) is Hill's viscosity constant k related to metabolic efficiency?

v) what is the role of the constant k?

They collected data on sprinters and analysed these data. In doing so they note (inter alia); little or no intercorrelation of errors; that the rate of attaining an increased O_2 supply is slow, with a ½ time of about a minute, so the acceleration curve is not determined by O_2 availability; that reaction time of about .13 sec. (back foot first, front .05 sec. later) is of little importance except over very short runs of 10 or 15 yards at most; that since $v_m = fg/k$, v_m and k should be negatively correlated, but this is not found, and concludes that the force must drop off as v_m is approached.

In answer to the five questions raised, they conclude:

(a) the correlation between observed and predicted times is very good and deviations are random; except perhaps that times are slower than predicted for the first 15 yards but then faster at 25 yards; the fit of the formula is inappropriate over about 50 yards and suggests a second subtractive exponential term.

(b) there are decided individual differences in k and v_m, but although the v's are more reliable, no differences are statistically significant.

(c) the roles of k and v_m in prediction depend on the distance; less than 10 yards k is the important factor, from 10 to 20 yards v_m is more important, and over 20 yards v_m is the only determining factor.

(d) discarding Hill's viscosity concept, they conclude that the velocity constant a is unrelated to any general factor that influences the efficiency of muscular work.

(e) the coefficient k, unrelated to other measured metabolic coefficients, is to be interpreted as a measure of the rate of attaining speed, and corresponds in time sense only to metabolic measures (i.e. has a half-life of about the same order).

The added exponential term (a fatigue factor) suggested above was examined by Henry (1954a), as follows

$$v = v_o(e^{-k_2 t} - e^{-k_1 t})$$

and the ideas were extended (Henry 1954b, 1955, Purdy 1974) to include several metabolic-related fatigue factors so that the resulting equation applied over all distances, sprint to the marathon. Specifically

$$v = a_1 e^{-k_1 t} + a_2 e^{-k_2 t} + a_3 e^{-k_3 t} + a_4 e^{-k_4 t} + a_5 e^{-k_5 t}$$

a_1 is negative and represents the acceleration factor; a_2, k_2 represents the alactic O_2 debt; a_3, k_3 represents the lactate O_2 debt; a_4, k_4 is a glycogen depletion factor; and a_5, k_5 represents other fatigue factors. These fatigue factors however are all contrived and though they find a place in my model, it is not in this fashion that they earn it.

The third substantive piece of work was due to Lloyd (1966). He approached the derivation of the equation of motion differently. Starting from Hill's earlier work the athlete has an initial store of energy S_0 and more energy is supplied by breathing and circulation at some constant rate R, thus the available energy E is given by

$$E = S_0 + Rt$$

We further suppose S_0 runs down exponentially and that there is a delay in the rise of the rate of energy supply from its resting level A, to R. Thus

$$E = S_0(1 - e^{-gt}) + Rt - (R-A)\ \Delta t \quad ....(3.1)$$

where Δt is the effective delay.

Now the power, P or rate of energy utilisation by the runner is given by

$$P = \frac{E}{t} = \frac{S_0}{t} + R$$

and since the power is some function of velocity, say f(v), we can deduce that

$$\frac{1}{t} = \frac{f(v)-R}{S_0}$$

Plotting v against $\frac{1}{t}$, a series of very nearly straight lines emerge, each set being races having S_0 and R in common. This implies that f(v) is a linear function of v, say A+Bv. Thus

$$E = Pt = At + By$$

where y is the distance covered. But since the runner in an actual race has to accelerate himself as well as cover a specified distance, his actual energy requirement is

$$E = At + By + \frac{1}{2}\alpha m\dot{y}^2 \qquad \text{..............(3.2)}$$

where the latter is a kinetic energy term and α incorporates factors for mechanical efficiency and interconversion of units.

Thus eliminating E from (3.1 and 3.2) we get

$$y = S_0 (1-e^{-gt})/_B + (R-A)(t-\Delta t)/_B + \frac{1}{2}\alpha m\dot{y}^2/_B$$

"... a differential equation which seems difficult to integrate ...", which it is!

However, for a short sprint we may assume R = A (and t = Δt) thus reducing the equation to

$$\left(\frac{dy}{dt}\right)^2 - \frac{2B}{\alpha m} y = \frac{2S_0}{\alpha m} (e^{-gt} - 1) \qquad \text{..................(3.3)}$$

Analytically it appears intractible because of the exponential term on the RHS, and this term cannot be omitted. Thus we may attempt a numerical solution.

Lloyd gives values for B = 64.8 calories/metre or 271.25 newtons, m = 72 kg, S_0 = 10,627 cal or 44,485 joules and g = .04 S^{-1}. It can easily be deduced that α is about 3, thus we consider

$$\left(\frac{dy}{dt}\right)^2 - 2.5y = 410 (e^{-.04t} - 1)$$

It would appear at first sight that a solution could fairly readily be obtained, but this is found to be not so, because of the non-existence of the derivative at zero. This required a little manipulation to produce the following graphical solution, Figure 2.1.

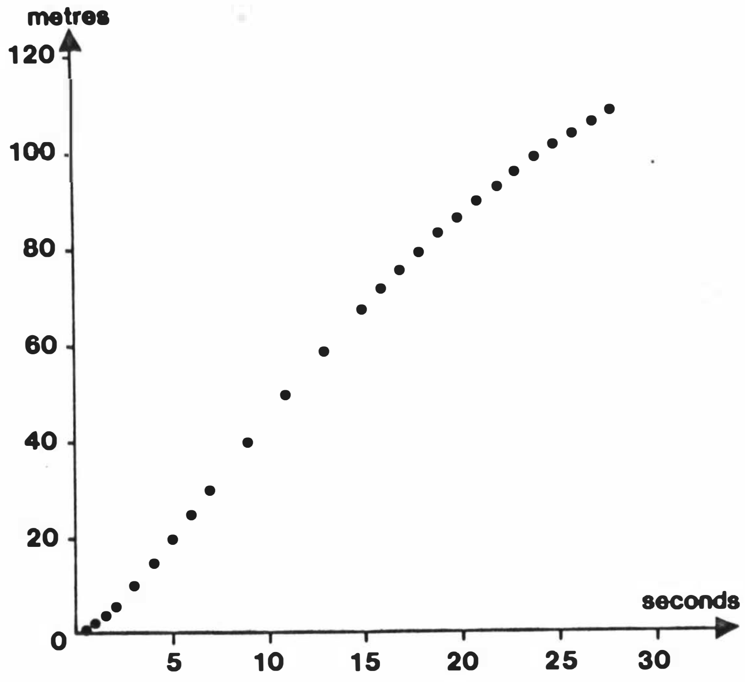


FIGURE 2.1: Graphical Solution to Lloyd's Equation

Though starting in the expected fashion as can be seen, the curve soon flattens out and later curves downwards, suggesting the runner is going backwards. It appears that the flaw is most likely caused by the error in multiplying P = A + Bv by t particularly since in sprint running v is changing quite rapidly. Of course y is the integral of v dt, not simply the product vt, but this complicates the whole procedure.

The whole approach however suffers from the same major drawback as before; the fatigue factor is contrived, albeit cleverly, and as such is not appropriate for this study.

The lesson from these studies is clear and can be very simply stated: the most appropriate solution to obtaining a comprehensive representation of the running curve, is as a solution to a model system of equations, and not as an ingeniously contrived single equation. Therefore consider the only such example, due to Keller (1973, 1974).

The total distance or length of a race D is related to the corresponding total time T taken to run it, by the equation

$$D = \int_0^T v(t)\, dt \quad \text{........................}(3.4)$$

where v(t) is the instantaneous velocity of the runner at time t.

This velocity v satisfies the equation of motion

$$\frac{dv}{dt} + \frac{v}{\tau} = f(t) \quad \text{........................ } (3.5)$$

where f(t) is the propulsive force per unit mass and is controlled by the runner. Part of it is used to overcome the internal and external resistive force per unit mass, v/τ, and it is assumed that this resistance is a linear function of v and that the damping coefficient τ is a constant. The remainder of course produces the acceleration of the runner dv/dt. Initially the runner is at rest, and so we require

$$v(0) = 0 \quad \text{...........................}(3.6)$$

The runner must adjust f, so that T, determined from equation 3.4 is as small as possible when v is the solution to equations 3.5 and 3.6. There are two restrictions on f. Firstly there is some maximum force per unit mass that the runner is able to exert, say F, and thus we must have

$$f(t) \leq F$$

Secondly, the rate of doing work per unit mass, fv, must equal the rate at which the body supplies energy, which in turn is limited by the oxygen supplied by energy-releasing reactions, which can be considered as follows.

Initially there is some quantity of available oxygen in the muscles, denoted E_0, and more is supplied by the respiratory and circulatory systems. Denote also the energy equivalent of the available oxygen per unit mass at time t by E(t), and the energy equivalent of the rate at which oxygen is supplied per unit mass over and above the non-running metabolism by σ. The equation of energy or oxygen balance is thus given by :

$$\frac{dE}{dt} = \sigma - fv \ ,$$

or perhaps more conveniently by :

$$fv = \sigma - \frac{dE}{dt} \ .$$

It is established that $E(0) = E_0$,

and it is clear also that $E(t) \geq 0$,

and these four equations are indirectly the second restriction on f.

Keller sought to find v(t), f(t) and E(t) satisfying the above equations, so that T determined from equation 3.4 was as small as possible. The four physiological constants τ, F, E_0 and σ are given, and of course D is known also.

Keller's system has been criticised, though it is well formed and does follow the observed racing data. One criticism (Purdy, 1974) seems to be that it has a discontinuity that is neither exhibited by the facts nor is suggested from a physiological basis. It could be argued however, that the discontinuity is only in the acceleration, nowhere else, and that this discontinuity is a property of a mathematically optimum solution only, and cannot be achieved in practice. There is no observed data anywhere near the location of the discontinuity, so it cannot be said to be "not exhibited by the facts". If the data did exist, it might be. It could also be argued that on a physiological basis, fairly abrupt changes in acceleration are in fact suggested, though the magnitude of such changes might not be large. Athletes do make sudden changes in their acceleration, though of course they cannot be instantaneous.

A second criticism is that the system takes no account of varying tactics used by runners to win races (Carey 1974). This of course is true, but the stratagem to win a race is not necessarily the same as the one required to cover it in the least time. A third, more relevant criticism (Osterle, et al 1974) concerns the equivalence (or not) between the physics and the physiology. For example they calculate that a 70 kg runner at 8 m/s has a power output of about 7000 watts based on Keller's figures. They argue that work physiologists would not accept such a high figure, having estimated about 1600 w. However since it can be deduced, and has been shown empirically (Fukunaga, et al 1980), that the power is proportional to the square of the velocity, this figure equates to a power of 1417 watts at 3.6 m/s. This value is within the range observed by Williams and Cavanagh (1983). It does appear on the high side however, and will be investigated later. Prothero (1974) however does find all Keller's parameter estimates to be of the right order of magnitude on independent grounds.

EXTENSIONS

In considering extensions or alterations to Keller's model, it should be noted that the basic exponential velocity equation fits the data very well, and that apart from the empirical formulae suggested by some authors (which often do not fit as well) there has been no change in this form of the equation. Thus the importance of a resistive force proportional to velocity seems firmly established, but it shall not be omitted from further scrutiny.

Firstly however, equation 3.4 above must be retained, which of course holds by definition, though in the context of a model consisting of a system of differential equations, it is better written as

$$\frac{dD}{dt} = v \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad A$$

with the obvious initial condition on that

$$D(0) = 0$$

Secondly let us consider the equation of motion for the runner. An observation of Henry and Trafton (1951) that "there is a tendency for the runners to be ... slower ... in the first 15 yards, and faster in the region of 20-25 yards. This may represent chance variation, or it may reflect the observed fact that the track was ... downhill ... in the first 15 yards and then uphill to 30 yards", may suggest a possible extension. It is unlikely to be chance variation, though it may be, and since their reasoning on the second suggestion appears perverse, it may be worthwhile to examine some plausible alternatives. Thus the simple exponential equation was fitted to some of the data in Hill (1927b) mentioned previously. This data is four replicates on the same runner, sprinting to 60 yards, timed at 1, 3, 6, 10, 15, 20, 30, 40, 50 and 60 yards. Exactly the same observation can be made on this data; a fact not reported by Henry and Trafton, though they do mention some of Hill's other data. In order to assess this observation, a complete ANOVA of Hill's data was obtained, to investigate this lack of fit. The following is the resulting table.

TABLE 2.1: AoV of Hill's Repeated Data

Source of Variation	d.f.	Mean Sq.	F-ratio
Exponential Regression	2	6434.65	120,803***
Lack of Fit	7	.17255	6.785***
Pure Error	30	.02543	
Residual	37	.05327	
Total	39		

Thus, though the exponential equation fits very well ($R^2 > .999$) the lack of fit term is significant beyond the .001 level ($F_{7,30,.001} = 4.82$). Henry and Trafton do not reproduce their data so it is not possible to subject it to the same analysis, though it seems the conclusion is likely to be the same. Hill's other data is for ten athletes, timed once each, sprinting to 200 yards. The exponential equation has been fitted to all of these and the residuals examined. Some show the observed lack of fit pattern, some show no pattern at all, and some show an opposite pattern; an inconclusive result. It might be pointed out that the residuals may well be expected to be autocorrelated, since an athlete ahead of prediction at one timing station, is quite probably ahead at the next also, and vice versa. Thus the addition of a new term in an attempt to explain the observed pattern can be considered. Different individuals having different parameter values in this term may explain its non-appearance in some cases.

The first line of thought is as follows: There could be an additional resistive force, the motion of the limbs, which depends on the stride rate. Since speed increases are usually achieved by increasing stride length, this resistive force is independent of v. Thus it should rise from zero with time, to a constant value. That is a force of form

$$a\ (1 - e^{-bt})$$

Thus the basic differential equation becomes

$$\frac{dv}{dt} + a\ (1 - e^{-bt}) + c\ v = F$$

with a and b > 0.

This equation has solution

$$v = \frac{dy}{dt} = \frac{a}{c-b}\ (1 + e^{-bt}) + \frac{F-a}{c}\ (1 - e^{-ct})$$

i.e. $$y = \frac{a}{c-b}\ (\frac{1}{b} + t - \frac{e^{-bt}}{b}) - \frac{F-a}{c}(\frac{1}{c} - t - \frac{e^{-ct}}{c})$$

this equation has been fitted to the four replicates, and it also describes the data very well. Examination of the residuals reveals the previously observed lack of fit pattern has been removed, and they are no longer autocorrelated. However the estimated values of a and b are both negative, and inconsistent therefore with the above reasoning. Estimated values of F and c are little changed. The equation, for what it is worth, is

$$v = 9.39 - .34\ e^{.125t} + 9.73\ e^{-.816t}$$

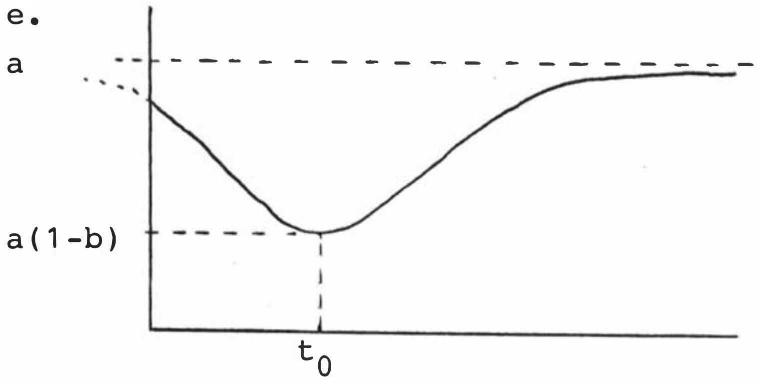
It should be noted that this does not stand up to extrapolation for large t, since after some time v declines, becomes zero and then negative. It should be asymptotic to some positive non-zero value. Thus this line of thought has not been pursued.

A second line of investigation might be as follows. It has been observed that athletes tend to settle into an almost constant stride rate, which for themselves is individually optimum, this rate is probably some function of leg length. Variation in speed is affected by variation in stride length, as before. It seems reasonable to suppose that it takes some short time to get to this optimum stride rate, and further to suppose that the resistive force of limb motion is in some way inversely proportional to the extent to which the actual rate differs from the optimum. That is, the force is minimal at $t = t_0$ and larger away from t_0.

For example, the force could be of form

$$a \{ 1 - b\, e^{-c(t-t_0)^2} \}$$

i.e.



$a > 0$

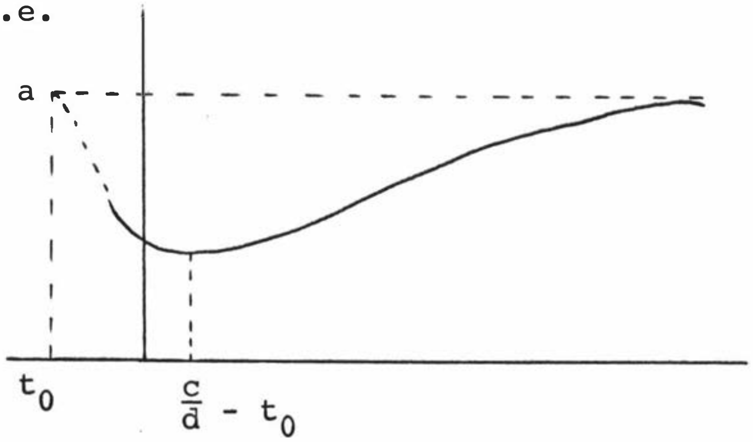
$0 < b < 1$

$c > 0$

but this is a symmetrical situation, which need not be the case. Alternatively we might have a force of form

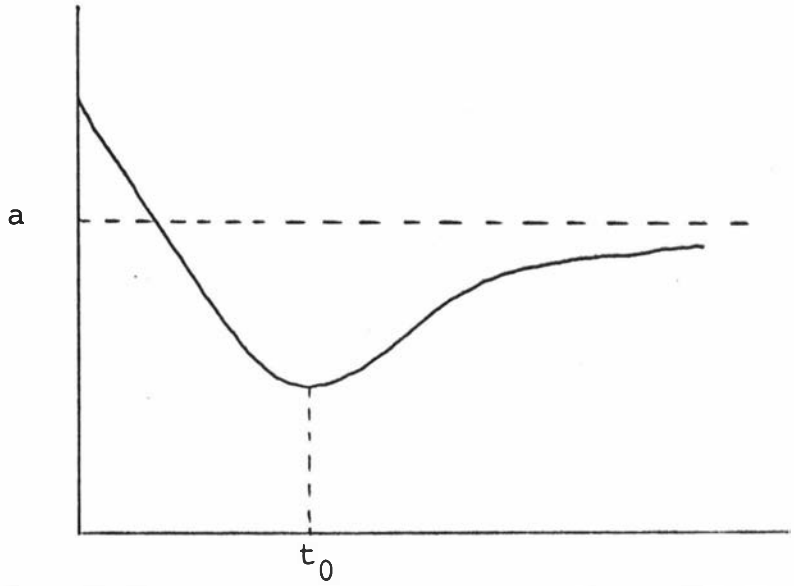
$$a \{ 1 - b(t+t_0)^c\, e^{-d(t+t_0)} \}$$

i.e.



Note t_0 has changed

In both these cases these forms are empirical in nature, and have no physiological basis, which basis might be difficult to establish. In both cases also they lead to a differential equation which is not easily soluble. Furthermore in both cases it is physiologically quite possible that the magnitude of the force at zero (or just after) be higher than a, i.e. graphically



neither of which is allowed by the two forms mentioned. Thus it would seem that such an explanation of the lack of fit, while conceptually plausible, cannot very easily be mathematically represented.

There is one other angle worth pursuing. It has been established (Pugh 1970, 1971, Shanebrook et al 1976 and Davies 1980) that the aerodynamic drag experienced by runners is not as small as Hill (1927b) originally thought. The agreed value now seems to be that for sprinters about 14% of the energy cost of running is expended overcoming wind resistance. For middle distance runners it is of the order of 7%, and for marathon runners, 3%. It had also been clearly established that this resistance is proportional to the square of the athlete's velocity. Thus we might consider a differential equation of form

$$\frac{dv}{dt} + av + bv^2 = F$$

This is a Riccati equation, and the solution, though involved, can fairly readily be found, it is

$$v = \frac{2F\,(e^{\sqrt{A}t} - e^{-\sqrt{A}t})}{(2\sqrt{A}+a)e^{\sqrt{A}t} + (2\sqrt{A}-a)e^{-\sqrt{A}t}} \quad ....(3.7)$$

where $\sqrt{A} = \sqrt{Fb - \frac{a^2}{4}}$

Now the data of course is in terms of distances and times, so this equation must be integrated. This is not so simple. Alternatively, Hill's data can be used to estimate velocities for the four replicates by interpolation and equation 3.7 fitted. By way of a check on the suitability of this approach, the simple exponential equation was fitted to these velocities, and next to no change in the estimates of parameters was observed from those estimates obtained previously from the distance data.

Equation 3.7 has therefore been fitted to these interpolated velocities. This set of data can of course no longer be regarded as four replicates, since the observations were taken at equal distance intervals, not equal time intervals and the distance variable has been eliminated. The expectation was that in view of the good fit of the simple exponential equation, the inclusion of a term for v^2 would be unlikely to cause much change. But this was not so. The changes can be summarised as follows:

(i) The parameter F dropped from 6.51 ± .20 to 4.90 ± .09.

(ii) The parameter a dropped from .735 ± .054 to zero. (It should be noted that the parameters a and b were forced to be ≥ 0 in order to have them retain a meaningful physical interpretation).

(iii) The estimated b value is .0660 ± .0019.

(iv) R^2 has increased from .986 to .993. The reduction in residual sum of squares from 4.4076 to 2.2001 on 1 d.f. is significant at the .1% level ($F_{1,36} = 36.12$).

(v) Removing the restriction on parameter a resulted in a slight reduction of F; an a estimate of -.395 ± .17 and an almost doubling of the b estimate.

Thus the conclusions seem to indicate that the original postulate of a resistance proportional to v, could perhaps be replaced by a resistance proportional to v^2. The former has always fitted the data well, perhaps too well for anyone to question it, but the latter appears to fit the data better. The muscle viscosity was the original "explanation" of the resistive force, but it was later shown that it could only be a component of the resistance. No other accepted suggestions have been put forward to explain the remaining major component. Secondly the accepted situation is that the wind resistance is proportional to v^2, but it is equally clear that this is also only a component. It remains to be seen what might explain the remaining component(s) of this resistive force.

Thus if we put a = zero, equation 3.7 simplifies to

$$v(t) = \sqrt{\frac{F}{b}}\ \text{Tanh}\ \sqrt{Fb}\ t$$

with an ultimate velocity limit of $\sqrt{\frac{F}{b}}$. Furthermore, this is quite simple to integrate to give the result (requiring D(0) = zero)

$$D(t) = \frac{1}{b}\,\ell n\ [\text{Cosh}\ \sqrt{Fb}\ t] \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots(3.8)$$

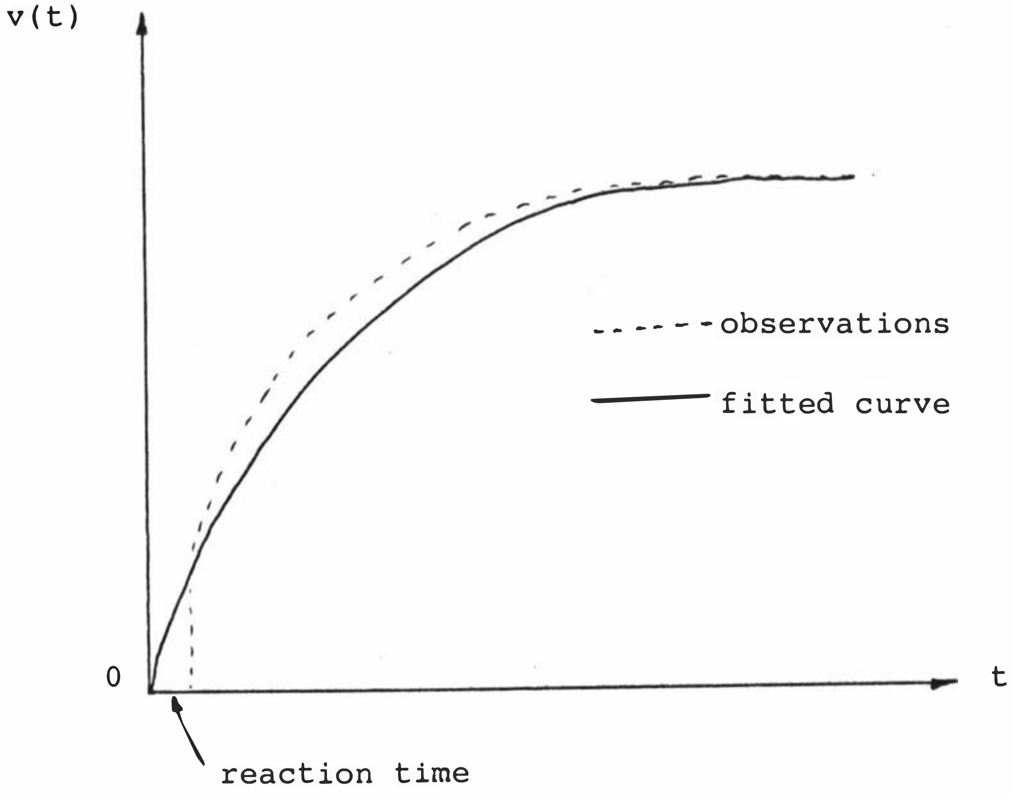
Now a much better comparison between the simple exponential model and equation 3.8 can be performed without the need to interpolate velocities, and using all the distance/time data available. The results of the comparison are as follows.

With Hill's replicated data set, the fit has improved, R^2 has increased from .999847 to .999930. The parameter F has decreased significantly from 6.682 ± .072 to 5.044 ± .032. The estimated value of b is .0682 ± .0007. The lack of fit pattern has lessened, but there may still be a significant factor. Hill's data set on ten individuals shows mixed conclusions, in most of them R^2 has decreased slightly, but in a few it has risen. In all cases the estimate of F has decreased significantly. No consistant lack of fit pattern is evident. Henry and Trafton's data on mean times has also been examined. The conclusion is similar to Hill's replicated data, a slightly improved fit. The lack of fit pattern is not really evident at all.

It has fortunately been possible to obtain a copy (by personal communication with Franklin M. Henry himself) of the 25 data sets of runners obtained by Henry and Trafton. Duplicated runs were made for all runners, timed at 5, 10, 15, 20, 25, 30, 35, 40, 45 and 50 yards. All 25 data sets have been analysed, as well as the pooled set. The new equation fits better in a total of 18 of the 25 sets, as was expected. The improvements are not great, for there isn't a great deal of room, but they cannot unfortunately be tested for statistical significance. On the other hand, when the fit is worse, it is not by much either, nor can it be shown statistically inferior. The analysis of the data in bulk, yields an overall improved fit for the new equation. It can be said though, that based on a simple binomial hypothesis of equality of fit, this result is significant at 2½%. That is for this group of runners, a resistance to motion which is proportional to v^2 only, represents a better fit.

It is interesting to note that Senator (1982), in allowing the resistance to motion to be proportional to any power of v, finds that he cannot reasonably estimate this power with available world record data. His methods are complex, and apply to maximal sprint efforts, but might be able to produce a result based on Henry and Trafton's data.

Returning for a moment to the lack of fit pattern, it is clear from the following exaggerated diagram that a simple plausible explanation for the observed pattern could be the reaction time of the runner.



Thus the integral form of the simple exponential equation can be modified to include a reaction time, t_0.

$$D(t) = F\tau(t - t_0) + F\tau^2 e^{-(t - t_0)/\tau} - F\tau^2$$

and fitted this to Hill's replicated data. A significantly better fit is achieved. The reduction in residual sum of squares of 1.058 on 1 d.f. is significant beyond the .1% level, ($F_{1,36} = 41.73$). The estimate of t_0 is .126 seconds

(SD = .019), a value consistent with observed reaction times of athletes. The lack of fit term which can be extracted has a sum of squares of .16 on 6 d.f. and yields a non-significant $F_{6,36}$ value of 1.06.

This equation has also been tried on Hill's second set of data, the ten athletes timed once each. The results are mixed, as before. A couple of the runners appear to have successfully jumped the gun by as much as .16 second, but in most cases t_o is not significantly different from zero.

Adding the reaction time and fitting to Henry and Trafton's 25 data sets, gave fairly consistent results, with most reaction times about .13 seconds, and most significantly different from zero. A few are not significant, and only one seems to have jumped the gun. It is interesting to note that 21 of the 25 have higher R^2 values than with the simple exponential equation. But in comparison to the v^2 resistance equation it is only 14 to 11.

The conclusion therefore, having examined the available data, is that though there is some evidence to suggest the inclusion of a reaction time in the simple exponential equation, or a change to the v^2 resistance term, it is not very strong. Thus the appropriate resistance form can be taken as proportional to v, as has been done all along.

However, the form must be modified slightly to include the mass, m, of the runner in kg. Thus with f(t) now being the total propulsive force exerted by the runner, we have

$$f(t) = m\frac{dv}{dt} + a\,v$$

with the constant of proportionality a now in units of kg/sec.

$$\text{i.e.} \quad \frac{dv}{dt} = (f - av)/m \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \text{B}$$

which is the second equation of my model system, having the initial condition of course that

$$v(0) = 0$$

Keller's energy balance equation

$$fv = \delta - \frac{dE}{dt}$$

must now be examined.

As far as the LHS is concerned, P = fv is true by definition in Newtonian mechanics, but the criticisms mentioned earlier must be examined. Now the solution to equation B for a constant force f is given by

$$v = f(1 - e^{-at/m})/a$$

and allowing for acceleration, $v \to f/a$, constant also. That is, since $av \to f$, $av^2 \to fv = P$. It was remarked earlier that Keller's equation led to rather high values of P, of the order of 7,000 watts when v = 8 m/sec, and that a figure more like 1600 watts is appropriate, a fraction between 1/4 and 1/5. As remarked also, Fukunaga et al (1980) has found experimentally that the power of a runner is proportional to v^2, with the constant of proportionality about .436 on a per kilogram basis, and with R^2 about .96. This for a 75 kg sprinter such as Keller considered leads to an estimated P = 2093 watts at 8 m/sec, which seems a more reasonable figure, a reduction between 1/3 and 1/4. Converting to a speed of 3.6 m/sec this yields an estimated P = 424 watts, which is more like the reasonable sorts of figures Williams and Cavanagh (1983) suggest.

Independent of this, there is the additional complication, that the power developed by running is much higher than that developed on a cycle ergometer. Many estimates, and a great deal of study are based on cycle ergometry, so this must be considered also. Jones et al (1975) have provided a conversion applicable to equating work on a treadmill, to work on a cycle ergometer. It provides a conversion from mph to watts, from which it can be deduced for example that a speed of about 3.6 m/sec is equivalent to about 250 w on the ergometer.

The appropriate action it seems is to use Keller's formulation for the external power developed by the runner and apply a conversion factor when considering the bioenergetics in the next chapter. The above arithmetical considerations suggest the factor of $\frac{1}{4}$ to be an approximate figure, and we shall return to this in the next chapter.

Thus for the external power developed by the runner, since P = fv by definition, we have

$$\frac{dP}{dt} = f\frac{dv}{dt} + v\frac{df}{dt} \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad C$$

with the initial condition that P(0) = 0. This now is the third and last equation of the biomechanical segment of the model, and we can shortly proceed to the bioenergetics.

In response to criticism by the examiners of this thesis, an addendum to this chapter (pp. 40A - 40F) has been appended inside the back cover.

CHAPTER 4

MODELLING BIOENERGETICS

BIOENERGY COMPONENTS

Keller's (1973) model assumed a two-compartment energy supply for the runner. Firstly a store in the body at the start of the race, of amount E_0. This seems intuitively obvious, but it isn't quite as simple as it sounds, and Keller makes no mention of the nature of this store. To this store is added a constant rate of supply, σ, during the course of the race; its nature being through the respiratory and circulatory systems. The external power demand given by the product of force and velocity, equates the internal power supply given by

$$-\frac{dE}{dt} + \sigma$$

$$\text{i.e.} \quad \frac{dE}{dt} = \sigma - f\,v$$

with the initial condition $E(0) = E_0$.

As will be discussed in detail later in this chapter, there are good reasons for rejecting this equation and replacing it with three others, for it is too simple to be sufficiently realistic. In the first place, the body store consists of two components; the phosphagen (high-energy phosphates) and glycolytic (glucose) sources. Physiologists know quite a lot about them. There are others, but these are only of importance in excessively prolonged exercise. Secondly, it is well known that the rate of oxidative energy supply is not constant. It can take up to two minutes to build up from the resting level to a more stable supply, and this level reached is dependent on the power demand. Let us therefore examine these bioenergy sources, without getting involved to any extent in the biochemical details.

The most obvious bioenergy source is oxidative energy, characterised by an increased oxygen consumption in response to exercise. The response, as just remarked, is quite slow and its extent depends on the severity of the exercise. The oxidative source is of course not limited in quantity, though the rate of supply is; a quantity regarded as import= ant by exercise physiologists, known as $\dot{V}O_2$ max.

The second source is the glucose → glycogen → lactic acid mechanism, also known as the lactic source. It is the latter conversion which is of major interest, for while providing energy, the lactic acid is a harmful by-product. It tends to accumulate in the muscle tissue, where, if not removed fast enough by diffusion into the bloodstream, it will cause muscular poisoning; the fatigue pains well known to athletes. The mechanism is a limited source, and also has a maximal rate of utilisation, which is higher than the maximal oxidative rate.

The third, and most important source since it is available on demand, is provided by the conversion of various high-energy phosphate compounds. This phosphagen source is also known as the alactic source. The by-products of these conversions are not harmful and are recycled by the oxidative and/or glycolytic mechanisms. This third source is limited in quantity and rate of utilisation also, though the maximal rate is quite large.

The usual investigative approach to these matters taken by exercise physiologists, has been to exercise subjects at a known workrate (power) on a cycle ergometer (or treadmill). The total rate of energy usage is therefore known. The subject breathes through tubes which measure the oxygen consumption, as well as other respiratory parameters. The energy equivalent, and the biomechanical efficiency of the oxidative mechanism are known and so the oxidative component of the workload could be calculated, and the sum of the other two components determined by subtraction. The modelling procedure has been to fit a curve to the oxygen consumption over time. Historically this was first done about 30 years ago (Henry, 1951) and the curve fitted very well. Countless equally good, or better, fits have been obtained since (Linnarson, 1974; Fox et al, 1980; Whipp et al, 1981) and this model is now in such widespread usage that it appears to be taken as fact. Of relevance to the mathematician is that it never had, nor has it since been given, any published independent theoretical basis.

During light to moderate exercise, below what is referred to as the "anaerobic threshold", it has been established that the glycolytic mechanism does not contribute to the energy supply. This is ascertained by inserting a small needle in a vein and drawing small blood samples every minute or so during exercise. The blood is analysed for the presence of lactic acid; none above normal resting levels having been found in such circumstances. Thus any difference determined above by subtraction, can be attributed to only one source, the alactic, and so the empirical modelling can be done.

In heavy exercise, all three mechanisms contribute, and the difference referred to has both lactic and alactic components. It might be thought that measuring blood lactic acid as described would provide one of the two, and the alactic component derived by a second subtraction. Here we run into a number of difficulties; there is a well-established but quite variable time-lag between muscle and blood lactic acid levels; blood flows around the body in a complex though known, manner; where should the needle be inserted, etc. For these reasons and others, blood measurements though frequently taken, do not provide a very useful indication of the rate of lactic acid production in the working muscle. The muscular concentration can be accurately determined, by a needle biopsy, that is by inserting a special needle into the muscle tissue and withdrawing, by powerful suction, a small piece of the tissue itself for subsequent analysis. Clearly this process cannot be done repetitively, nor while the muscle is actually in use.

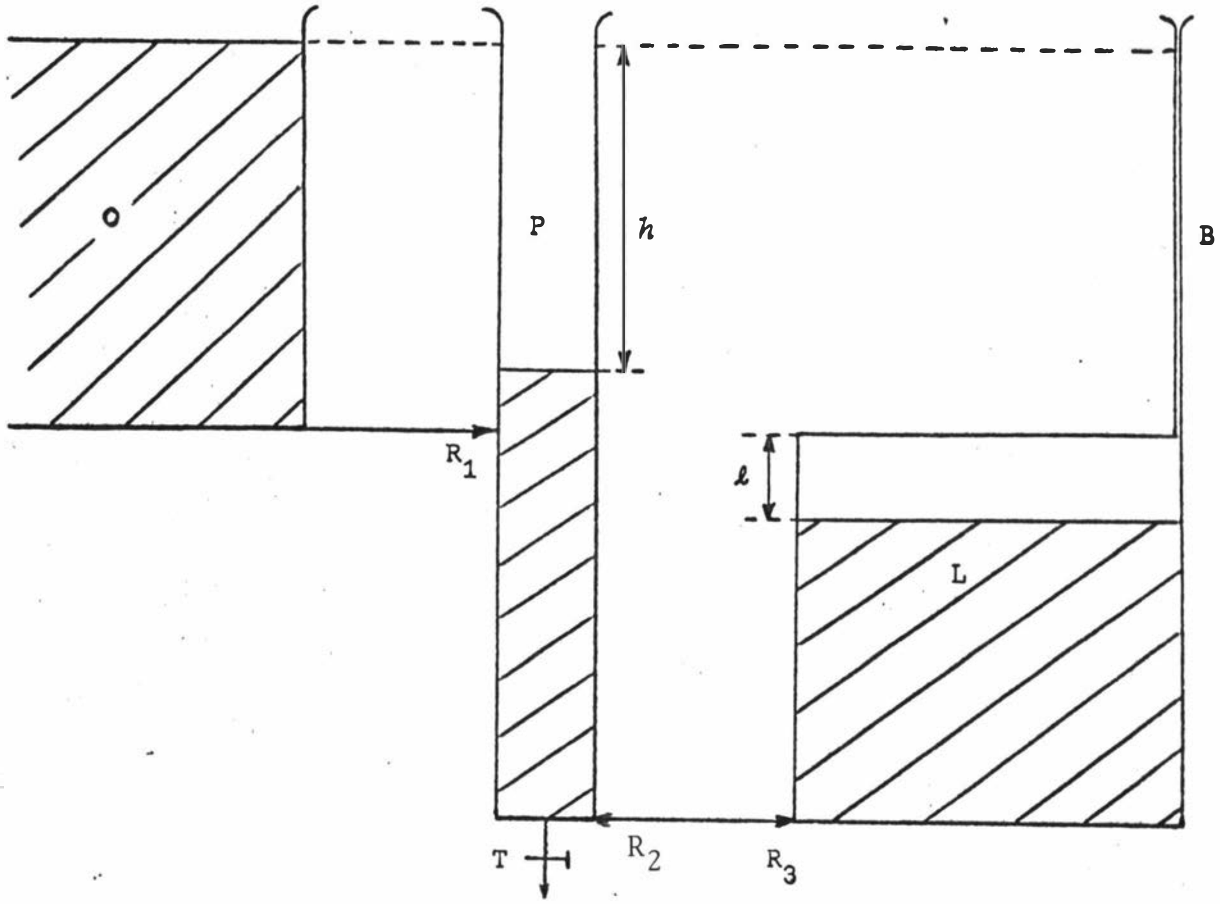
Thus physiologists have done a great deal of work on the oxidative mechanism and because of the above difficulties, less on the other two. Because of much empiricism it would seem that some theoretical means of making progress in modelling might be tried. A 3-compartment hydraulic model, postulated by Margaria (1976), might provide this progress.

Margaria's Model

This is the only attempt I have found to comprehensively model the energy processes involved in muscular exercise. The model is only conceptual, though diagrammatically described and there is no detailed attempt to quantify it. A diagrammatic part-solution is offered, but likewise no attempt made to solve the model analytically at all.

The hydraulic model is shown in Figure 4.1 below:

FIGURE 4.1: Margaria's Hydraulic Model



The fluid in vessel P (representing phosphagen) is directly connected with the outside through the tap T, which regulates the flow (total energy expenditure). At rest, with T closed, the upper level of fluid in P is the same as in the communicating vessel O (representing the oxidative source). The vessel O is of infinite capacity and is connected through tube R_1. The second communicating vessel L (representing the glycolytic source) is of finite capacity, with upper level the same as the bottom level of vessel O, apart from a *very* narrow extension tube, B. The fluid in B, corresponding to the resting blood lactic acid, is of *very* small volume relative to L, and does not contribute to any flows in a measurable amount. L is connected to P through a wider, but one-way tube R_2, and P is connected to L by another, but very much smaller one-way tube, R_3.

If T is partly opened, corresponding to a workload W, the level in P falls, inducing a flow through R (oxygen consumption, $\dot{V}O_2$) in accordance with the difference in levels, h, between the two vessels. This induced flow slows the rate at which the level in P falls, and provided W is not too large, an equilibrium will be reached at a level above the outlet R_1. The level in P is below the resting level, and fluid flows continuously from O to P and out through T. If the equilibrium level is exactly at the level of R_1, then the oxidative mechanism is at its maximum, denoted $\dot{V}O_2$ max. Once the equilibrium is established, the only energy mechanism contributing is the oxidative; the exercise is purely aerobic, and in theory could continue indefinitely. Prior to equilibrium of course, P has contributed some of its supply, and the empty volume in P above the equilibrium level is known as the alactic oxygen debt.

If T is now closed, i.e. exercise ceases, P will begin to refill through R_1, but at a slower and slower rate as the level in P returns to normal. When it does so, the flow in R_1 ceases and the subject is said to have repaid his oxygen debt during this recovery period.

If T had been widely opened, (severe exercise), the initial situation would be as described above, but the level in P would fall below R_1. This happens after about 50% of the fluid in P has been utilised, and the subject is said to have crossed his anaerobic threshold. As soon as this happens, two things occur; the flow in R_1 has reached and continues at its maximum determined only by the height of the vessel O; and a flow through R_2 is induced. This flow is in accordance also with the difference in levels between vessels L and P, (the level in L lagging behind the level in P). The flow through R_2 will slow the fall of level in P, but since the flow through R_1 is insufficient and the capacity in L is limited, the levels in both L and P will continue to fall. If exercise is prolonged, L and P will be emptied and the subject will become exhausted.

If T is closed at or before exhaustion, P will again be refilled. Initially it will be filled through R_1 at the maximal rate, and through R_2 until the lag in levels between vessels L and P has been eliminated. This latter flow is a delayed lactic acid formation which has been experimentally observed to occur after cessation of exercise. Once the levels have been equated, P will fill through R_1, initially at the maximal rate and thereafter at a progressively slower rate as described previously. L will be refilled from P through R_3 at a rate in accordance with the difference in levels between the two. Because R_3 is so small, the level in L will lag behind the level in P; the repayment of this, the lactic oxygen debt, is very slow. Finally, both P and L are refilled and the subject has fully recovered.

The full mathematical solution to this model has turned out to be a major piece of work in its own right, and has been published separately (Morton 1984). This solution of course would have been required for components of my model system. It is included as Appendix 1. In addition to the solution, a number of other features of the model are considered, and the result of these considerations is the rejection of the Margaria model in its present form. In a nut shell, this was because the solution was not sufficiently realistic. In particular the behaviour of the predicted oxygen uptake and hence at least one (if not both) of the other two energy sources, is incorrect. Several structural changes to the model would be necessary to right these inaccuracies, and for at least one of them the required information is unknown.

The mathematical solution to the restructured model would undoubtedly be more complex than the solution to Margaria's original model.

Before progressing to the energy source modelling there is one further concept that must be explored, and its place (or lack of a place) in this study considered. That is the vexed question of the anaerobic threshold.

Anaerobic Threshold Difficulties

The anaerobic threshold (AT) is defined as that workload beyond which the blood lactic acid level begins to rise above normal. It has also been observed as that workload resulting in a changed pattern on oxygen uptake kinetics (Wasserman et al, 1973). It results from the failure of aerobic oxygen to supply all the energy needs of the exercise, the balance (apart from that supplied by the phosphagen source), is supplied through anaerobic glycolysis, which in turn results in accumulation of lactic acid in the blood and a marked increase in CO_2 in the expired breath. AT is thus a unique workload which unbalances the O_2 demand/supply relationship (Davis et al, 1976). AT is commonly adopted to describe the maximal intensity of exercise (or of $\dot{V}O_2$ level) at which a person can work for a sustained period of time without lactic acid build-up, (Rusko et al, 1980), i.e. the maximal aerobic capacity in some sense. However some studies have shown that subjects can maintain a slightly higher workload than their AT for an extended period (Miyashita et al, 1981; La Fontaine et al, 1981, Kindermann et al, 1979).

AT is commonly expressed in various ways: as a workload in watts, which is perhaps the best way for an individual; as a level of O_2 consumption generated by that workload (presumably the steady state consumption, though nowhere specifically stated); as a % of $\dot{V}O_2$ max, which is perhaps the best way for more general reference to AT. It can be detected by various means:

(a) by an increase in blood lactic acid beyond resting level
(b) by a decrease in arterial blood bicarbonate
(c) by a pronounced increase in the respiratory quotient, $RQ = CO_2$ production $\div$ O_2 consumption,
(d) by a steeper than linear change in minute ventilation, $\dot{V}E$, with increasing workload,
(e) a minimum level in the ventilation equivalent for oxygen, $VEO_2 = \dot{V}_E \div \dot{V}O_2$.

While method (a) and perhaps also method (b) are the appropriate methods in terms of the definition of AT, they are invasive. The latter three methods are bloodless and are better to use from the subject's point of view (and with modern equipment are easier also). These three have been found to be reliable measures of the former two (correlations of the order of .95), and there is therefore no need for invasive techniques, (Naimark et al, 1964; Davis et al, 1976; Renhard et al, 1979). They are reliable measures of each other also (Weltman and Katch, 1979), and do distinguish hyperventilation from hypoxia.

Of the three non-invasive measuring techniques the two involving $\dot{V}_E$ are easier to use, (d) being the most popular. The difficulty with using RQ is that its marked increase is transient (Wasserman et al, 1973) while the observed changes in $\dot{V}_E$

persist. Tests designed to measure AT by these means invariably involve incremental workload ergometer cycling. Workload is increased discretely by about 15 watts each minute (Wasserman et al, 1973; Davis et al, 1979; Reinhard et al, 1979; Weltman et al, 1978; Rusko et al, 1980) though some increase faster (Davis et al, 1976; Weltman and Katch, 1979) and some slower (Miyashita et al, 1981). Whipp et al (1974) tried a 15 watt increment every 5, 10, 15 and 30 seconds, 1 min. and 4 min. The short durations gave an overestimate of AT, while the 4 min. duration took too long. More recently a "ramp" test to measure AT and other important parameters of aerobic function has been devised (Whipp et al, 1981). Workload is increased continuously at the constant rate of 50 watts/min. In all the above tests, respiratory measurements are made on a breath-by-breath basis, and AT determined from these, by one of methods (c), (d) or (e). Recently a computer algorithm has been designed to evaluate the AT (Orr et al, 1982).

The typical AT range for normal healthy individuals is about 45 to 180 Watts on the cycle ergometer (Wasserman et al, 1973). Lower values in this range are typical of sedentary unfit persons, while higher values are typical of active physically fit persons. Values below 45 watts are typical of those with respiratory or cardiac illness (Wasserman & McIlroy, 1964), while very high values are obtained for exceptional athletes (cross-country skiers, who show an unusually high $\dot{V}O_2$ max; Rusko et al, 1980). For the 20-39 year age group of healthy (but not necessarily very fit) persons, 1 watt/kg is a reasonable rule-of-thumb to determine AT (Reinhard et al, 1979). Expressed as a % of $\dot{V}O_2$ max, AT is about 50% for untrained healthy individuals (Weltman et al, 1978, Reinhard et al, 1979), while for fit runners and cyclists it is of the order of 60% (Davis et al, 1976; Weltman & Katch, 1979; Miyashita et al, 1981). The AT can be increased by endurance training (Davis et al, 1979;

Rusko et al, 1980), by up to 44% in absolute $\dot{V}O_2$ or 15% of $\dot{V}O_2$ max (which itself increases also). AT is regarded as a better measure of fitness than is $\dot{V}O_2$ max (Weltman et al, 1978).

AT values correlate with a number of other variates. It declines with age. It correlates positively with $\dot{V}O_2$ max, but only when measured in absolute terms and not as a %. AT is higher in males than in females, but not when the AT workrates are weight-corrected. AT varies with the type of exercise being undertaken; for arm cranking 47% of $\dot{V}O_2$ max, running and cycling 60%, cross-country skiing 85%. AT also correlates with various muscle oxidative enzymes. The AT measured for an individual over time is fairly constant provided there is no significant change in life-style or health. (Wasserman et al, 1973; Davis et al, 1976; Weltman & Katch, 1979; Rusko et al, 1980; Reinhard et al, 1979; Davis et al, 1979). Recently the maximal steady state (MSS = O_2 uptake, heart rate and/or treadmill velocity at which lactate concentration is 2.2 mmole/ℓ plasma) has been used to determine the maximum sustainable workload in place of AT (La Fontaine et al, 1981). MSS correlates well with pace in long-distance running, in particular over five miles, and with AT.

The transition from aerobic to anaerobic metabolism has recently been reviewed by Skinner & McLellan (1980). In addition to the discussion of the sort given previously, they examine a second AT as defined by another "school" of researchers (McDougall, 1978; Green et al, 1979; Kindermann et al, 1979). This point, occurring at about 65-85% $\dot{V}O_2$ max, is marked by a further steeper change in ventilation and also in blood lactate, the % expired CO_2 now begins to drop, $\dot{V}O_2$ max has been reached, and there is marked hyperventilation. The major distinguishing feature is the sharp rise in blood lactate from a level of about 4 mM/ℓ. The controversy appears to be related to the choice of criteria and to the definition of anaerobiosis. (Scheen et al, 1981).

However, when lactate and the various ventilatory measures are considered together during progressive exercise from rest to maximal loads, it does appear that three phases can be distinguished. The transition from phase I to II at about 2 mM/ℓ lactate (twice resting level) is the AT discussed earlier, perhaps better referred to as the (upper) aerobic threshold (AeT). Phase II is a transitional stage. Its passage to Phase III at about 4 mM/ℓ lactate is the AT of McDougall & Green, the "real" anaerobic threshold in some sense (An T). Measurements of An T are almost exclusively based on invasive techniques rather than on a gas exchange basis, usually the steep lactate rise (Graham, 1978). But since lactate levels in the blood lag behind levels in the muscle, short workload durations should be used carefully as they are likely to lead to overestimates.

The whole process is of course a continuous one, and it appears that perhaps a little too much emphasis has been placed on the approximately linear rises of various respiratory measures, and the "kinks" therein that mark the changes from one phase to another. It is clear that lactate levels are the important determinant, and this is the consideration relevant to use as the muscle fatigue inducing factor in my mathematical model. Thus the controversy is of little significance to this work, as it is a formula or equation for lactate rise over the whole range that is required.

In summary therefore, in spite of the apparent simplicity of the notion of the anaerobic threshold, and the amount that is known about it due to the extensive research, the concept remains shrouded with difficulties, highlighted recently by Yeh et al (1983). Thus it would be difficult to see how it would find a place in my model in spite of its obvious importance. In mitigation also, it will be seen in the next sections that the bioenergy modelling can be accomplished fairly accurately without it. I believe though, that in the future, it will return to a deserved place, perhaps in a more thoeretical development than this segment of my study.

MODELLING OXYGEN UPTAKE

It has long been recognised that, within certain broad limits, the uptake of oxygen by a runner varies with time. Regarding some measure of workload (e.g. velocity) as constant, and ignoring acceleration , the uptake rises from its resting level to a steady state. This rise may take two or more minutes to achieve.

Henry (1951) first postulated an exponential form for his uptake, specifically the equation

$$a_0(1-e^{-kt}) \qquad \text{..........................} \quad (4.1)$$

which seemed to fit his data quite satisfactorily. The parameter a_0 is the steady state uptake level above resting, and k is a rate constant. In the ensuing years, this form of exponential equation has gained wide acceptance, (Cerretelli et al, 1966; Di Prampero et al, 1970; Henry and De Moor, 1956; Fox et al, 1980; Linnarson, 1974; Margaria et al, 1965; Volkov, 1966; Volkov et al, 1969; Wasserman and Whipp, 1965; Whipp and Wasserman, 1972). There has however more recently become evident a second component to the curve. Its effect is a statistically significant rise in uptake between the fifth and 20th minute, and there was an attempt to explain this observation by means of its covariates (Hagberg et al, 1978), but its nature remains uncertain. It is thought by some to be exponential, (Costill, 1970; Hagberg et al, 1978a; Henry, 1953; Volkov et al, 1969; Whipp and Wasserman, 1972), though more recently to represent some delay mechanism, (Whipp et al, 1982). Attempts were made in some of these instances to model this component, but the results await general acceptance. Graphically it appears to be linear, (Hagberg et al, 1978a; Whipp and Wasserman, 1972).

Considerable effort has gone into investigating the relationship between the workload as represented by the velocity of the athlete, and the parameter a_0. This parameter is of course bounded below by the resting O_2 uptake level, regarded conventionally as a zero point of reference. It is assumed to be bounded above by a maximal oxygen uptake level, denoted usually $\dot{V}O_2$ max. This upper bound is widely accepted as an important determinant in the capability of endurance athletes. (Miura et al, 1971; Henstock, 1979; Saltin and Astrand, 1967; Shepherd et al, 1968; Whipp et al, 1981). In fact a recent study has shown it to be considerably superior to a whole host of cardiovascular parameters (Crain et al, 1980), most of which show no significant correlation at all! In the main, these investigations (Henry, 1951; Costill and Fox, 1969; Costill et al, 1973a; Daniels et al, 1977; Dressendorfer et al, 1977; Mayhew, 1977; McMiken and Daniels, 1976; Pugh, 1970; Shepherd, 1969; Bransford and Howley, 1977) find that over a moderate velocity range, the relationship between a_0 and v is linear. It is remarkable the extent to which the slope coefficients in these studies agree amongst each other. The intercepts vary quite markedly, and are probably not significant. There have been a few studies which show a curvilinear relationship between a_0 and v (Daniels et al, 1977; Fellingham et al, 1978; van der Walt and Wyndham, 1973; Henry, 1953) but the curvilinear coefficients are quantitatively very small. In a recent attempt to resolve this question, Hagan et al (1980) concluded that when O_2 uptake is expressed relative to body mass (i.e. in ml/kg / min) it is linearly dependent on velocity only, but when expressed in absolute terms (i.e. in ℓ/min) it is dependent again linearly on velocity and on body mass. In their review however, these authors omitted to examine the question of equality of slope coefficients, nor did they test the intercepts for equality with the resting level as might have been anticipated. Two gaps clearly remain; examination of the relationship for a wider range of workload, and for values of t prior to the attainment of the steady state.

Secondly there is the question of the rate constant k in the equation above. This aspect of the kinetics has received less attention, and the results are conflicting rather than in general agreement. In the original article Henry (1951) argues that k is a constant independent of the workload. It is more common nowadays to use the $\dot{V}O_2$ half-time response as the parameter indicative of the speed of the increase in oxygen consumption. This half-time response is mathematically equal to .693/k. Some findings agree with Henry (Corretelli et al, 1966; Margaria et al, 1965). Several others however have shown k to increase with increasing workloads, i.e. a shorter half-time response. The steady state level can be reached after about one minute for very severe workloads, but might take as long as 6 to 8 minutes for light exercise, (Di Prampero et al, 1970; Astrand and Saltin, 1961; Astrand and Rodahl, 1977). Yet a third group suggest the contrary, a lengthening of the half-time response with increasing workload, (Henry and De Moor 1956; Whipp and Wasserman, 1972; Bason et al, 1973; Hagberg et al, 1978b; Hickson et al, 1978; Weltman and Katch, 1976; Fox et al, 1980). Of these, Hagberg et al (1978b) has found .693/k to increase linearly with increasing workload, particularly when the latter is expressed as a percentage of $\dot{V}O_2$ max. Whipp & Wasserman (1972) have suggested that the difference in findings might be accounted for by the duration of the exercise, or the length of time over which the data was analysed, or both. In either case however, almost all these studies found k is larger (or the half-time shorter) for trained athletes in comparison with untrained athletes. A recent study (Di Prampero et al, 1983) suggests that k depends in some way on the level of an oxygen store prior to exercise.

It comes as a surprise therefore, in view of the many studies quoted (and others) that there has been nothing published (that I can find) which attempts to model both these aspects of O_2 uptake kinetics together. That is, the oxygen uptake as a function of both t and v, or some other variable representing the workload. This requires to be done.

Following the above summary, the various aspects of the oxygen uptake, together with some intuitively obvious characteristics, can be assembled into the following:

(a) Let $\dot{V}O_2$ (v, t) denote the oxygen uptake as a function of both velocity and time, $v \geq 0$ and $t \geq 0$.

(b) $\dot{V}O_2$ (0, t) = U_r the resting level for all $t \geq 0$.

(c) $\dot{V}O_2$ (v*, t) is of form $U_r + f_1(v^*)\ (1 - e^{-f_2(v^*)t})$ for fixed $v = v^*$, and where f_1 and f_2 are positive functions of v.

(d) For large t, $\dot{V}O_2$ (v, t) is linear in v, and hence from (a) and (b), it follows that;

$f_1(v) = bv,\ b > 0$

Problems of gas collection and analysis have lead to $\dot{V}O_2$ readings being taken at fairly wide time intervals and no evidence has been reported on the form of $\dot{V}O_2$ when t is fixed at some small value. More recently $\dot{V}O_2$ measuring apparatus has enabled breath-by-breath analysis to be made and so it should be possible to gather data for small t and differing v. Such data does not appear as the subject of any published investigation that I can find, however, some can be gleaned from individual sources. Nevertheless, because of the discrete nature of the breath-by-breath analysis, $\dot{V}O_2$ cannot be measured for fixed t. Thus an area of uncertainty exists here. For small t, is $\dot{V}O_2$ still linear in v, or curvilinear? It might be better to adopt the latter in such a form that linearity is a special case.

(e) Resolving f_2 is a more knotty problem, in view of the conflicting results of various studies. The two views can be regarded as two special cases of

$$f_2(v) = c\, v^d \;,\; c > 0 \;,\; d \neq 0$$

where from equation 4.1, p. 52
k increases with v ($\frac{1}{2}$ time shortens) when $d > 0$
and k decreases with v ($\frac{1}{2}$ time lengthens) when $d < 0$.

One problem of course is when $d < 0$ and $v=0$ but then e^{-ctv^d}, as $t \to \infty$ and $v \to 0$, tends to zero anyway and the limit as $v \to 0^+$ is U_r which is the defined value for $v=0$, so the function is continuous.

(f) This leads to a mathematical model which seems to fit all the research observations, of form

$$\dot{V}O_2\ (v, t) = U_r + b\, v\, (1 - e^{-ctv^d}) \ \ldots\ldots\ldots\ldots\ldots \ (4.2)$$

and the only ambiguity is whether $d < 0$ or $d > 0$.

Astrand and Saltin (1961) have some data, in graphical form which may give a lead to fitting such a model. It needed to be extracted from the graph, and is indicated to give $d > 0$, but at least some insight can be gained (e.g. confirmatory values of the estimates of U_r and b, and R^2 values). Reading off the graph is no real problem, apart from the possibility of the introduction of errors (which may unconciously smooth the data). But there are other difficulties.

Now the Astrand and Saltin subjects were exercising on a cycle ergometer, not a treadmill, and the workload is measured in kiloponds. This figure must first be converted to watts and then, using the equations of Chapter 3 and ignoring acceleration, be converted to m/sec before equation 4.2 can be fitted. This was done with data on three of the six subjects and yielded encouraging results:

(a) R^2 values around .97

(b) U_r values very close to actual resting values

(c) Values of b which agree fairly well with other studies of the relationship between oxygen uptake and velocity (e.g. Mayhew, 1977).

(d) Values of $d > 0$, but which differ widely.

One disturbing fact is that the velocities are very low, 2.2 to 3 m/sec which is rather surprising since exhaustion occurred at 3 m/sec in about 2 minutes, yet such a pace is equivalent to about 9 minutes for the mile, an easy one for almost any fit individual. The cause must surely have something to do with differing biomechanical efficiencies between running and cycling. This can be investigated as follows. Since the efficiency of bipedal motion ranges from about 12 to 20% (Stuart et al, 1981; Zarrugh, 1981), it follows that w would increase by a factor of 5 to 8, and v by the square root. The higher efficiencies are typical of the faster speeds, thus I arbitrarily chose a factor of 2.4 for the increase in v (a 17.36% efficiency). This factor is of course analogous to the one considered at the end of the previous chapter, where a value of $2 = \sqrt{4}$ was indicated. Velocities now range from 5.3 to 7 m/sec, about right. As expected, the fit was as good as originally and the parameter estimates were almost exactly those which could have been obtained by simple algebraic substitution of 2.4v in place of v.

The assumption that for large t, $\dot{V}O_2$ is linear in v seems untenable over a wider range of v values. There are two reasons for this. Most obviously $\dot{V}O_2$ is bounded above by a horizontal asymptote at $\dot{V}O_2$ max. Secondly, in the nine studies quoted by Hagan et al (1980), there is quite a strong negative correlation between the slopes and intercepts of the nine straight lines quoted. This suggests curvature as they contain different ranges of v values. Furthermore, plots of the Astrand and Saltin (1961) data sets, at t=15, 45, 75 and 105 secs, suggest curvature even though there are only five points to plot in each case and these values of v are not well chosen to show this effect. An equation of sigmoid form may be suitable, as it has the necessary characteristics. This leads to an alternate to equation 4.2:

$$\dot{V}O_2 = U_r + e^{\alpha-\beta/v}\,(1 - e^{-ctv^d}) \quad \ldots\ldots\ldots\ldots\ldots\ldots(4.3)$$

This has been fitted to the Astrand and Saltin data (using the higher velocities reflecting 17½% efficiency) and obtained the highest R² values so far in all three sets. The only problem is that the α and β estimates do not appear to be the right order of magnitude.

As a final look at the Astrand and Saltin data, four individual sets have been standardized by subtracting the resting oxygen uptake U_r, and dividing by the difference ($\dot{V}O_2$ max-U_r) and converting to a percentage. (This has been done by other researchers.) Next, all four sets of data were pooled and now we can more reasonably compare the two equations, or rather a standardised version of them, on overall performance. The following were the results, over all 138 observations.

$$\dot{V}O_2 = -1.59 + 16.23\ v\ (1-e^{.0000635t\ v^{3.2}})$$

with R² = .9267. The first parameter (which should be zero) is in fact not significant (t = 0.66). And:

$$\dot{V}O_2 = -1.76 + e^{5.51\ -\ 5.59/v}\ (1-e^{.000049t\ v^{2.9}})$$

with R² = .9278, and the first parameter again not significant.

In sum, equation 4.3 is consistently better, for all four individuals and in aggregate, but not by a great deal. It is clear that a properly planned experiment is required to investigate how $\dot{V}O_2$ depends on v, for fixed t. There are problems with this approach since the discrete nature of the breath by breath analysis makes it impossible to fix t. An interpolated $\dot{V}O_2$ value may be the best approach.

There is another data set on O_2 uptake, (M. Miyashita et al, 1981), though only two subjects are represented graphically and not adequately at that. They are taken at workloads at an intermediate level so in conjunction with the Astrand and Saltin data could prove useful.

Looking at one of the sets, the earlier conclusion that equation 4.3 is better than equation 4.2, is upheld both for the raw data (R^2 = .9950 and .9456 respectively) and for the standardised form expressed as % of $\dot{V}O_2$ max (R^2 = .9949 and .9455 respectively). However, the interesting finding is that the power for v in the exponential oxygen uptake term of both equations is not significantly different from zero, at least over this range of workloads. This would appear to indicate that the uptake rate constant k in the simple expression of the form

$$a_0(1 - e^{-kt})$$

either depends on v in a complex manner, or not at all.

Pausing at this stage, much of the above is rather shaky and it ignores the second component of the oxygen uptake. This second component is clearly important in magnitude, and it seems linear; both facts being evident in the following plot of data from an exercising subject.

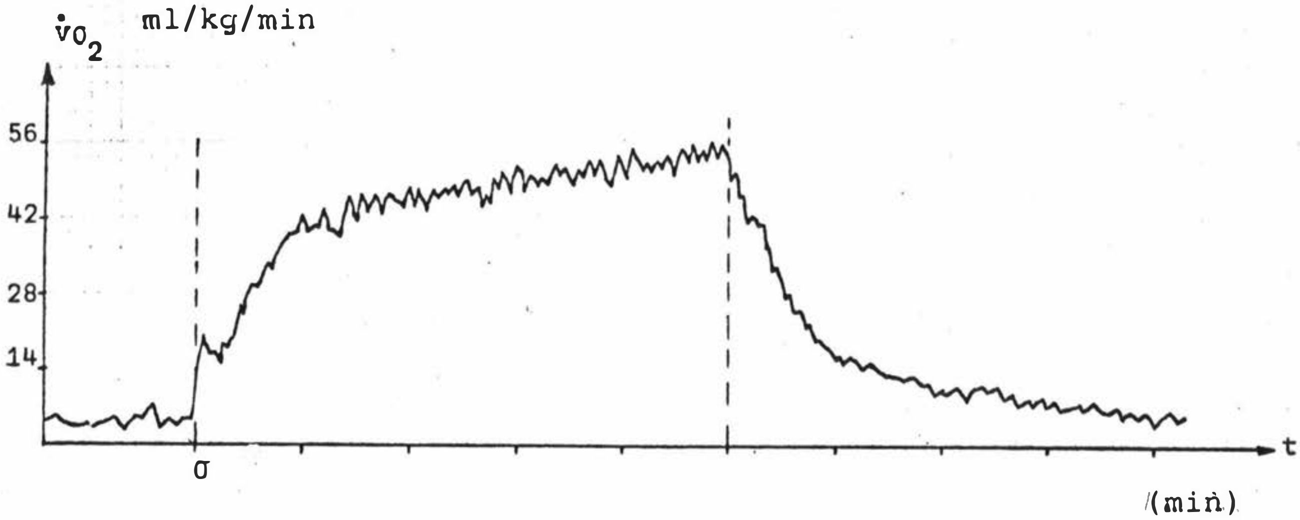


FIGURE 4.2: Oxygen uptake by Exercising Subject

Source: self exercising at 1700 kpm

Taking this into account removes the $\dot{V}O_2$ max bound on the linearity of $\dot{V}O_2$ with v. Generalising from v to a workload w, let us therefore postulate a model of form

$$\dot{V}O_2\ (w,\ t) = U_r + a(w)t + bw(1 - e^{-kt})\ \ldots\ldots\ldots\ldots\ (4.4)$$

where a(w) and b(w) are linear functions of w only. The exponent d is taken as zero and hence disappears from the equation. This seems to represent the best empirical compromise, so its justification must be considered, based on a properly selected data set.

I subjected myself, (a fit, healthy male, never a trained athlete, aged 36 years, height 1.78 metres, weight 72 kg) to a series of eight constant load exercise bouts on a cycle ergometer, with the workloads ranging from very mild, to quite severe (200 to 2000 kiloponds/min. or 33 to 333 watts). These exercises I performed under supervision in the Princess Margaret Hospital respiratory laboratory. Because I had believed the $\dot{V}O_2$ /workload relationship to be sigmoid, the workloads chosen were 200, 500, 800, 1000, 1200, 1400, 1700 and 2000 kpm/min. Each bout consisted of approximately two minutes of rest, followed by at least five minutes of exercise (or until exhaustion), followed by five minutes of recovery. Breath duration in seconds, oxygen uptake in $m\ell$/minute and other respiratory and cardiac data were recorded on a breath-by-breath basis during the entire bout. Exercise bouts were performed in a randomised order on alternate days.

The following table is summarised from the resulting eight computer files. $\dot{V}O_2$ at rest is an average over ten breaths immediately preceding the start of exercise. In all other cases it is the average of the three breaths straddling the time point for which the observation was required. Time is in seconds after the start of exercise, workload is in kpm/min and $\dot{V}O_2$ is in litres/min, * indicates missing data (exhaustion intervened).

TABLE 4.1: $\dot{V}O_2$ DATA SUMMARY w: kpm/min (hundreds)

		2	5	8	10	12	14	17	20
	rest	.511	.569	.627	.573	.531	.496	.474	.512
time (sec.)	10	.648	.666	.926	1.023	1.191	1.201	1.197	1.284
	20	.671	.731	1.112	1.132	1.218	1.454	1.599	1.735
	30	.798	.868	1.275	1.280	1.413	1.806	2.015	2.124
	45	.831	1.064	1.661	1.495	1.813	2.307	2.479	2.732
	60	.802	1.160	1.961	2.252	2.352	2.682	2.905	3.264
	90	.807	1.307	2.054	2.438	2.466	2.859	3.074	3.472
	120	.868	1.252	2.152	2.500	2.516	2.935	3.301	3.648
	180	.900	1.187	2.054	2.449	2.690	3.130	3.526	3.894
	240	.918	1.211	2.257	2.466	2.752	3.227	3.674	*
	300	.937	1.375	2.190	2.642	2.801	3.387	3.894	*

If we now start from the assumption that for fixed t (large), $\dot{V}O_2$ is linear in w, for which Whipp et al (1981) has suggested some theoretical justification, then for w=O we should find $\dot{V}O_2$=R, the resting level, and so on for w>o. Starting from t=300 and working back to t=10, we obtain for linear regressions across the table above:

TABLE 4.2: REGRESSING V AGAINST W

$t = 300$: $V = .500 + .202w$, $r = .995$

240 : $V = .496 + .192w$, $r = .990$

180 : $V = .554 + .175w$, $r = .992$

120 : $V = .678 + .156w$, $r = .984$

90 : $V = .706 + .146w$, $r = .981$

60 : $V = .661 + .137w$, $r = .982$

45 : $V = .591 + 110w$, $r = .980$

30 : $V = .558 + .081w$, $r = .981$

20 : $V = .518 + .063w$, $r = .987$

10 : $V = .588 + .039w$, $r = .942$

These results are encouraging.

Thus if we suppose that for fixed t, the $\dot{V}O_2$ function can be written as

$$\dot{V}O_2 = \alpha + \beta(t)w$$

we can investigate β(t) by plotting the slopes against t, for it seems that α is about the resting level, R, or maybe slightly higher. This plot is shown below

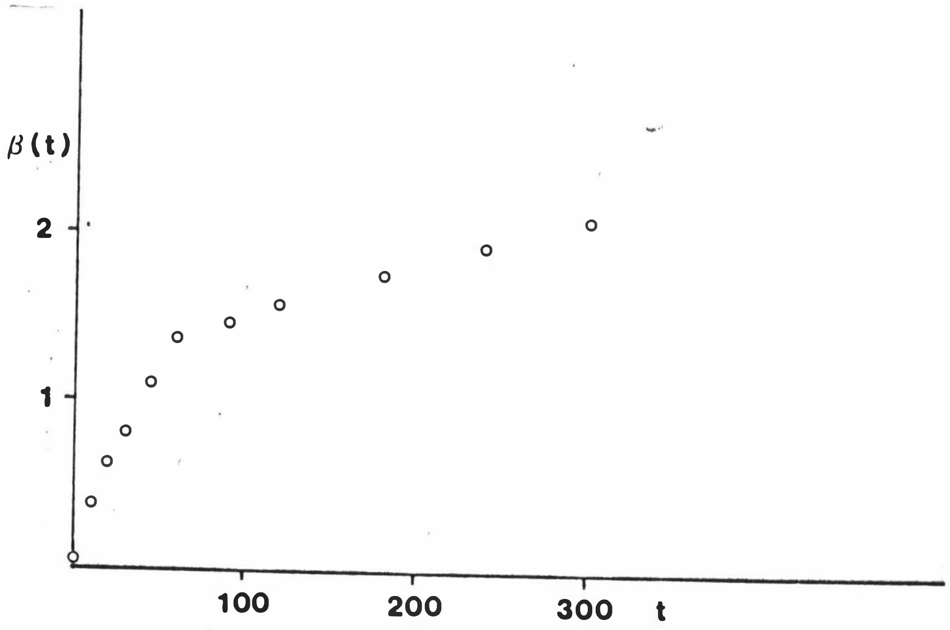


FIGURE 4.3: Slopes $\beta(t)$ versus t

In summary, at all non-zero values of t, a linear relationship between V and w is highly significant, and the slopes do certainly appear to be some function of t. The intercepts, with a mean of 564 and a standard deviation of 96.3 appear to accurately reflect the resulting value U_r, where a mean of 544 and a standard deviation of 116 were observed. At time zero, the slope coefficient is not significantly different from zero, as is to be expected.

It appears that an equation of form

$$\beta(t) = a + bt - ce^{-\lambda t}$$

would provide a suitable fit, and since $\beta(0)=0$, it simplifies to:

$$bt + a(1-e^{-\lambda t})$$

which over the eleven observed points fits well, with $R^2 = .942$.

This approach leads to the following

$$\dot{V}O_2 = U_r + bwt + aw(1-e^{-\lambda t})$$

as a two-dimensional expression of the relationship between V, w and t.

On the other hand, approaching from the other angle, where it was previously supposed that for constant workload w, an equation of form $R+h(1-e^{-\lambda t})$ had been generally accepted. To this we can add a slope, with t, to reflect the recently discovered rise, evident for larger t and larger w. That is, $U_r + g(w)\ t+h(w)(1-e^{-\lambda t})$, where it supposed that λ is constant, independent of w. (Some authors disagree, some saying it is a decreasing, others an increasing function.)

To examine g(w) and h(w), the previous table is not sufficient. Since λ is about .04, we can approximate $h(w)e^{-\lambda t}$ to zero for $t > 120$ say. Thus in this case $V = R+h(w) + g(w)t$. It is required that $h(0) = g(0) = 0$. Going back to the original eight computer data files and regressing, in each case, V on t every fifth breath from the end of exercise up to t=120, the following Table is obtained:

TABLE 4.3: REGRESSING V AGAINST t

w = 200 :	.929 + .000061t,	r = .157 ,	h(w) = .418
500 :	1.108 + .000656t,	r = .242 ,	h(w) = .539
800 :	1.835 + .00127t ,	r = .214 ,	h(w) = 1.408
1000 :	2.126 + .00148t ,	r = .257 ,	h(w) = 1.553
1200 :	2.327 + .001713t,	r = .329 ,	h(w) = 1.796
1400 :	2.766 + .011725t,	r = .397 ,	h(w) = 2.27
1700 :	2.961 : .002715t,	r = .569 ,	h(w) = 2.487
2000 :	3.264 : .003039t,	r = .5827,	h(w) = 2.752

Plotting the slopes g(w) and the values of h(w) against w, we get the following figure:

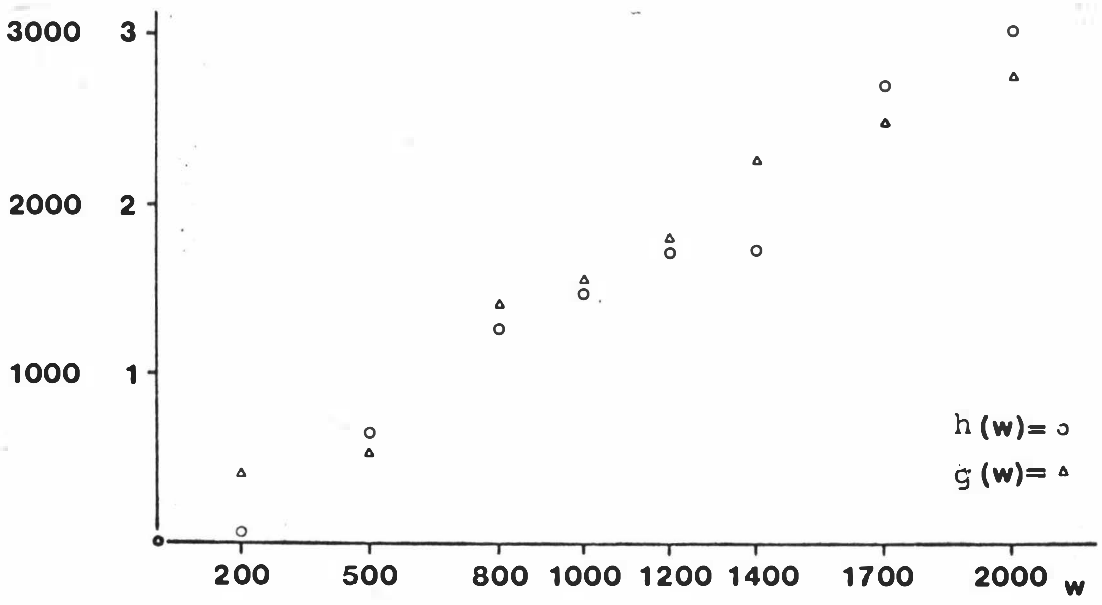


FIGURE 4.4: Plotting h(w) and g(w) against w

From these we obtain

$$g(w) = -.00114 + .000156w, \quad r = .988$$
$$h(w) = .067 + .143w, \quad r = .987$$

Linearity, through the origin, looks acceptable in both cases. Thus letting g(w) = bw and h(w) = aw, then we obtain as an expression for the relationship between V, w and t:

$$\dot{V}O_2 = U_r + bwt + aw(1-e^{-\lambda t}) \quad \ldots\ldots\ldots\ldots\ldots\ldots (4.5)$$

which is exactly as before.

The last step is to consolidate all the data into one large file and fit this equation. It would appear from the above that initial values for R, b, a and λ might be chosen as .5, .14, .16 and .03, with adjustments where necessary for decimal places.

This consolidation was performed as follows: Time zero (resting) values were determined by taking the average $\dot{V}O_2$ uptake over the last ten breaths immediately preceding the start of exercise, for each of the eight workloads. Thereafter an uptake was calculated at, or as near as possible to, 10, 20, 30, 45, 75, 90 ... every 15 seconds until the end of exercise. This was done by averaging over three breaths straddling each time point, and taking t as the mid-point of that interval, which did not exactly give 10 or 20 etc, but came very close. Again this was done for each of the eight workloads. A total of 168 observations were generated.

The last step is to fit the indicated equation to the whole data set of 168 observations, reduced in the manner described. This was done using the BMDP82 statistical package (Dixon, 1982). The resulting equation was:

$$V(t, w) = .541 + .000954\, wt + 1.623\, w(1 - e^{-.025t}) \qquad \text{..........(4.6)}$$

with $R^2 = .973$, which is most encouraging. R^2 is of course higher than would be the case on unreduced data because of the smoothing effect of the reduction process described. All the parameters have estimates of the right order of magnitude. This equation, therefore represents the best that can be currently achieved in the way of curve fitting. It is shown as a response surface below.

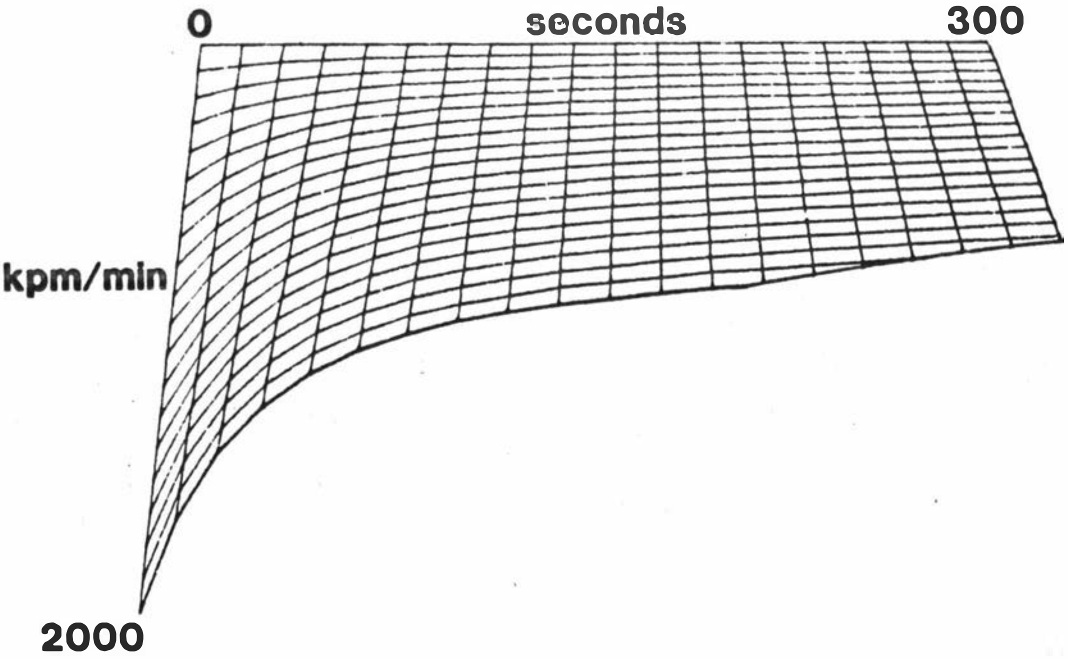


FIGURE 4.5: $\dot{V}O_2$ Response Surface

The major extension is of course the move to a second dimension which provides a true response surface. The other aspect of the extension is the inclusion of the linear term to reflect the second rising component of oxygen uptake mentioned previously. There are two questions to answer here; whether an additional term is warranted, and if so, whether that term should be linear, or exponential as some workers have suggested.

The base line from which to judge these inclusions is the 3-parameter equation obtained by omitting the linear term completely from equation 4.5 and fitting this. Next can be added a second exponential term of the same form as that already included, resulting in a 5-parameter equation, and fitting this also. An analysis of variance on the residual sums of squares relating to these changes is given in Table 4.4 below:

TABLE 4.4: ANALYSIS OF VARIANCE FOR ADDITIONAL TERMS

Source of Variation	Residual Sum of Squares	Degrees of Freedom	Mean Square	F ratio
5-parameter model	4.791	163	.0294	-
Difference	.013	2	.0065	0.22
Equation (4.5)	4.587	164	.0280	-
Difference	.217	1	.2166	7.74**
3-parameter model	4.804	165	-	-

It is seen that the inclusion of a second exponential term is not warranted, but that the inclusion of an extra linear term is statistically significant at the 1% level. It should be mentioned in passing, that the asymptotic level of the second exponential term is negative in the fit. This is contrary to expected and suggests convexity rather than the almost certain downwards concavity.

It follows therefore that the equation to adopt for the first (oxidative) component of the bioenergy source is of the form of equation 4.5 above. However we require some minor modifications to get it in the right form.

Firstly, since V is a bivariate function, its derivative with respect to t is of form

$$\frac{dV}{dt} = \frac{\partial V}{\partial w}\;\frac{dw}{dt} + \frac{\partial V}{\partial t}$$

and of course in this instance, the workload w is represented by the power developed

$$\text{i.e. } \frac{dV}{dt} = \frac{\partial V}{\partial P}\;\frac{dP}{dt} + \frac{\partial V}{\partial t}$$

Now $\frac{dP}{dt}$ is obtained from equation C in Chapter 3 (p 40) and the remainder from equation 4.5 above. We must however make the adjustment mentioned earlier, i.e. include the factor $\frac{1}{4}$ to account for the shift from running to the cycle ergometer whence equation 4.5 was developed. We must take care also to correct for the units of measurement. Equations 4.5 and 4.6 are based on w (or P) in thousands of kiloponds and t in seconds, and give V in l/min. It is more usual to have V in ml/kg/min and P (or w) in watts. These adjustments will be made later, in Chapter 7. Thus we obtain

$$\frac{dV}{dt} = \frac{b'}{4m}\left[t\frac{dP}{dt}+P\right] + \frac{a'}{4m}\left[\frac{dP}{dt} + e^{-\lambda t}\left(\lambda P - \frac{dP}{dt}\right)\right] \quad \ldots\ldots\ldots\ldots\ldots \quad D$$

with the initial condition that $V(0,t) = 6$ ml/kg/min say for any $t \geq 0$.

Note that embedded in adjusting for the change of units of measurement, is the energy equivalent of the oxidative energy source and its associated biomechanical efficiency. I have used the figures given by Margaria (1976) and confirmed by others, that 1 ml $O_2 \simeq 5$ cal $\simeq 21$ joules. At 25% efficiency this figure reduces to 5.25 joules/ml. Thus b' is in units of ml/60 joules and a' in units of ml.sec/60 joules.

LACTIC AND ALACTIC ENERGY SOURCES

I have already mentioned (Appendix 1, p 3), the difficulties in making lactate determinations, either concentrations or flows, during exercise. Even at rest, the techniques for the former are invasive, and alternate non-invasive techniques are indirect and unreliable. I cannot find reported any techniques to measure the flows. The situation in respect of the phosphagen based energy component is even worse. Apart from some invasive technqiues for measuring the body content of phosphagens, there is no other way of determining concentrations or flows at rest or during exercise. Since it is these quantities that are required for the model and so little is known, a heavy reliance must be placed on empirical modelling at this stage.

There is, however, an extensive literature on lactate accumulation and glycogen depletion, and it is consistency with these that will determine the adequacy of the empirical modelling. Apart from the biochemical aspects of the alactic energy source, which are not relevant here, there is no useful literature. Nevertheless, let us consider the information that is available.

Lactate formation occurs (Jacobs 1981a) when (i) at the onset of exercise the O_2 supply mechanism is accelerating, though hypoxia is not a prerequisite for lactic acid production (Graham, 1978; Jacobs et al, 1981a) and (ii) during the steady state if exercise is sufficiently intense. It is released (at least in part) into the blood where its detection is almost immediate (Jorfeldt, 1970). It may also accumulate in the muscle (Jacobs, 1981a) particularly at the early stages of severe exercise when production (and therefore concentration) is high, and exceeds release. Muscle lactate will only equate to blood lactate after several minutes (Graham, 1978; Jorfeldt et al, 1978) but it has also been found that blood lactate need not imply prior muscle lactate (Graham, 1978). Fast twitch (FT) muscle fibres have been found to accumulate more lactate than slow twitch (ST) fibres (Jacobs, 1981a; Tesch, 1980).

The general pattern of lactic acid accumulation at constant workloads seems to be well established, as follows:

(a) at light to moderate workloads of about 50% $\dot{V}O_2$ max: a rise in lactate concentration, then a return to about resting level (Jorfeldt, 1970; Scheen et al, 1981; Treffene et al, 1980; and others).

(b) at moderate workloads to about 70% $\dot{V}O_2$ max: a rise then a levelling off at an elevated, but not very high, level (Hermansen and Stensvold, 1972; Scheen et al, 1981; Treffene et al, 1980; and others).

(c) at severe workloads over 70% $\dot{V}O_2$ max: a rapid and sustained rise (Hermanson and Stensvold, 1972; Jorfeldt 1970; Scheen et al, 1981; Treffene et al, 1980; and others).

This latter rise is regarded by many as being exponential (Davis and Gass 1981; Farrell et al, 1979; Jorfeldt, 1970; and others), though some regard it as linear, i.e. a constant rate of production (de Bruyn-Prevost and Sturbois, 1980; Jacobs, 1981a; Margaria, 1976).

The dividing percentages in these phases were obtained for treadmill running and ergometer cycling, which two do not differ to a very great extent, though for swimming the percentages are noticeably higher (e.g. over 90% for phase c). At least one study (de Bruyn-Prevost and Sturbois, 1980) has noted a lag of an initial few seconds in lactate production, regarded as a role played by phosphagens. These three divisions seem roughly to correspond with the ventilatory and lactate anaerobic thresholds. The latter, often referred to as the point of onset of blood lactate accumulation (OBLA) has received considerable attention in the literature. It is clear from their lack of simultaneity (Davis and Gass, 1981; Scheen et al, 1981) that lactate concentration can not therefore be regarded as responsible for this observed ventilatory drive.

Results from OBLA include the following: Running performance correlates highly with the %$\dot{V}O_2$ and the treadmill velocity at which OBLA occurs (Farrell et al, 1979; Jacobs, 1981a; Jacobs et al, 1981b; Sjodin and Jacobs, 1981). OBLA occurs later for a fast/slow running protocol (provided it isn't too fast) than for a slow/fast or steady pace protocol

(Ariyoshi et al, 1979). $\dot{V}O_2$ rise is faster, lactate production is lower and the rating of perceived exertion is lower too, for the fast/slow protocol. OBLA is strongly related to the muscle respiratory capacity (Ivy et al, 1980; Jacobs, 1981a).

Specific findings with regard to glycogen depletion reveal the following: Glycogen depletion increases dramatically with increasing workloads (Gollnick et al, 1974a). At lower workloads the ST fibres are depleted first (Costill et al, 1973b; Gollnick et al, 1973; Gollnick et al, 1974a; Gollnick et al, 1974b); at moderate loads both ST and FT fibres are depleted together (Gollnick et al, 1974a); while at heavy loads the FT fibres become depleted first (Gollnick et al, 1973, 1974b).

Glycogen depletion studies have also been conducted with dietary induced depletion. (Asmussen et al, 1974; Jacobs, 1981b) which confirm some of the above findings. Glycogen can be depleted up to a point, yet still produce normal lactic acid levels when exercising (Jacobs, 1981a), but depletion beyond that point will result in significantly lower lactate production (Asmussen et al, 1974; Jacobs, 1981a; Jacobs et al, 1981b).

As I have already remarked, there is no relevant literature on phosphagen flows, depletion, etc, and thus some empirical modelling is required.

Margaria (1976) has provided the only quantitative hint for a possible start. He supposes the energy supply component attributable to the alactic source to be proportional to the amount of that store remaining. This is an intuitively reasonable supposition, and is common in a wide variety of other phenomena. Thus, knowing the power output of the runner, determined by equation C and the oxidative energy supply rate, determined by equation D, we can interpret his supposition as follows. The proportion of the net energy demand attributable to supply by the alactic source, is proportional to the amount of alactic source remaining. It is clear that at the start, since the oxidative component begins at zero (i.e. the basal metabolic rate), all the energy supplied is alactic. It follows then that the lactate based energy source can be determined by subtraction.

Thus for the alactic source, A, a differential equation of the following form would apply

$$- \frac{dA}{dt} / (P - \dot{V}O_2) = kA$$

where dA/dt is negative and k is the constant of proportionality. The initial condition is that $A(0) = A_0$, the normal resting value, and hence $k = 1/A_0$. This equation can now be tidied up, taking into account the units of measurement, energy equivalents and biomechanical efficiencies of the processes.

A is usually expressed in millimoles (mM), and the phosphagen content of skeletal muscle is about 30 mM/kg, (Bergstrom 1967; Karlsson, 1971; Margaria, 1976). Thus, since the muscles involved in running are about 40% of the body mass, the estimated value of A_0 is about 12m millimoles, where m is the mass of the runner in kg.[*] Wilkie (1968) and Margaria (1976) give the energy equivalent of phosphagen cleavage at about 11,000 calories per mole, or 46 joules/mM. Thirdly, since it is only the rise in $\dot{V}O_2$ above resting level that contributes to the energy supply, a correction must be applied, together of course with the associated energy equivalent of oxygen, both mentioned in the previous section.

Thus we can deduce

$$\frac{dA}{dt} = A(\frac{5.25m}{60} (V-6) - \frac{P}{4})/552m \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots E$$

with $A(0) = A_0 = 12m$ millimoles.

---

[*] Assuming uniform distribution through whole body mass.

The energy rate of supply from phosphagens is therefore given by 46 dA/dt joules per second, or watts.

It is now a relatively simple matter to deduce a differential equation for the lactic energy component. By difference therefore it is of the form

$$-\frac{dL}{dt} = P - \dot{V}O_2 + \frac{dA}{dt}$$

where L is the lactate store, and dL/dt is negative as well as dA/dt. The energy equivalent of lactic acid has been determined at around 240 calories per gram (Margaria, 1976; Cerretelli et al, 1969), which converts to 1 joule per milligram (mg. are used because one of the commonest expressions for the concentration of lactic acid is mg per 100 ml). The alternative is in mM per litre, the conversion factor being a division by nine). Thus we obtain

$$\frac{dL}{dt} = \frac{5.25m}{60}(V-6) - \frac{P}{4} - 46\frac{dA}{dt} \quad \text{.................F}$$

The initial condition, since the body contains (the equivalent of) about 1.12 grams of lactic acid per kg of body weight (Margaria, 1976) is thus L(0) = 1120m milligrams, where m is again the body mass of the runner.

COMMENT

Because of the empirical nature of equations D, E and F and of the earlier modelling aspects of the bioenergetic processes discussed earlier in this chapter, there are two obvious questions to consider. Firstly, there is the question of whether these three equations adequately describe what happens in reality. Secondly there is the question of whether some modification or development of the Margaria model might yield a more theoretical basis from which to develop three alternate equations.

In the first instance, I have fully discussed the adequacy of equation D in modelling the oxygen intake earlier in this Chapter, and so only equations E and F need be considered here. In regards to the latter, since no direct medical techniques are available for making measurements of lactate production in the working muscle, its adequacy cannot be directly assessed. However it is possible to gain an indirect assessment. This is done by the use of a two-compartment lactate circulation model, to be discussed fully in the next chapter. Compartment 1 is the working muscle where the flow or production is required to be measured but cannot be, and compartment 2 is the rest of the lactate space, represented by arterial blood, where sampling is relatively straight-forward (though invasive). It will be seen later, that equation F, when coupled to this model, produces simulated lactate concentrations in the blood which over time do adequately describe experimental observations on exercising subjects which have been made in the laboratory. Thus the adequacy of equation F can be inferred. It follows therefore, since the power requirement of the runner is known, that the adequacy of equation E is established by subtraction. This is not to say however, that any improvement is not possible or should not be sought; nor to say that the equations may not at some time be replaced by a more theoretically based set.

This raises the question in the second instance, of whether a development of the Margaria model is called for. This question is discussed more fully in the Appendix 1. On the face of it, a development is clearly possible but it is neither brief nor simple. Some aspects of the necessary development are fairly clearly indicated by the results of recent research. Other aspects must await future research findings. Thus an appropriate development is not possible at this time. I am confident however, that such an approach remains as a promising possibility for the future.

CHAPTER 5

LACTATE CIRCULATION MODELLING

The previous chapter has considered, amongst other things, aspects of lactate production, and it is relevant to consider now, what is known about its circulation, breakdown etc, around the body, and to develop equations to cover these aspects of the model.

It is clear that lactate produced in the anaerobic process may (i) be released to the blood, and/or (ii) be removed by the aerobic process by oxidation to H_2O and CO_2 with the resulting production of energy, and/or (iii) accumulate in the muscle resulting in fatigue (Graham 1978, Jorfeldt 1970). This latter consideration will be deferred to the next chapter. The rates of these processes are higher during exercise. The terms release and removal are both used, sometimes interchangably, in the literature, but I shall impose the interpretations in (i) and (ii).

Lactate release to the blood rises (approximately linearly) with muscle lactate concentrations up to an apparent limit (Graham 1978, Jorfeldt et al 1978, Woll and Record 1979). This limit, which has been estimated, is not attributable to inadequate bloodflow (Jorfeldt et al 1978, Woll and Record 1979). A small amount of this lactate is broken down in the liver and/or other organs (Belcastro and Bonen 1975, Woll and Record 1979). The remainder probably continues circulating or is returned for use in the aerobic mechanism (McGrail et al 1978) or reconverted to glycogen after cessation of exercise (Brooks and Gaesser 1980) or is removed by other inactive muscle, etc (McGrail et al 1978, Poortmans et al 1978). Removal from the blood is slower than removal from the muscle (Davis and Gass 1979, McGrail et al 1978), and it is the muscle which is regarded as the main site for lactate removal, (Hermansen and Stensvold 1972).

Lactate removal from the muscle via the aerobic mechanism is not related to lactate concentration (Hermausen and Stensvold 1972), but of course depends on the O_2 mechanism and its acceleration. In this mechanism, removal can apparently keep pace with production up to about 65 or 70% VO_2 max (Davis and Gass 1979, Hermausen and Stensvold 1972, Jorfeldt et al 1978) and hence this figure can be regarded in some sense as the true anaerobic threshold.

A fairly large amount of study has been devoted to the evolution of blood lactate after the cessation of exercise. In general it decreases to, or nearly to, the original pre-exercise level, and light to moderate workloads (< 65% VO_2 max) hasten the decrease if undertaken during the recovery period (Belcastro and Bonen 1975, Davis and Gass 1979, Freund and Zouloumian 1981, Hermansen and Stensvold 1972, Weltman et al 1979). The optimal removal rate occurs at around 30-40% $\dot{V}O_2$ max for running and cycling (Belcastro and Bonen 1975, Hermausen and Stensvold 1972, and others). In this form of active recovery it is probably improved circulation and raised aerobic function that hasten the decrease. However, it has been clearly demonstrated (Asmussen 1979, and others) that other forms of active recovery (e.g. mental activity) also hasten the decrease, and these factors cannot operate in such cases. The nature of this decrease is regarded by many as exponential, either mono, (Margaria et al 1933, Woll and Record 1979, and others) or double (Freund and Gendry 1978, Freund and Zouloumian 1981, Zouloumian and Freund 1981a). In the simple case the rate constant depends on previous peak lactate levels (Woll and Record 1979). In the double model the same is also true of both rate constants, which are also interrelated, (Freund and Zouloumian 1981).

In summary, lactate removal rate (above basal turnover) depends on the workload and on time, but also appears to be dependent on lactate concentration. After lactate elevation due to prior heavy exercise, the removal has been found (a) at rest to follow an exponential decline from a peak level (the peak usually occurs after cessation of exercise), this time course can be modified to include a rise to the peak, by inclusion of a second exponential term, (b) to show a higher removal rate at light to moderate exercise levels during recovery than at rest (or than at a more strenuous recovery regimen level), (c) to show a higher removal rate when the peak concentration had been higher, in each case for the corresponding workloads during recovery in (b).

The double exponential model is the solution to a theoretical construct by Freund and his co-workers, so it is appropriate to consider it in some detail.

THE TWO-COMPARTMENT MODEL

Freund and his co-workers have constructed and mathematically solved a two-compartment model for lactate circulation after exercise (Freund and Gendry 1978, Freund and Zouloumian 1981, Zouloumian and Freund 1981a,b). The model is based on the law of mass conservation in a volume, V. It allows for lactate production and utilisation, and lactate uptake from and release to other compartments, (i.e. lactate exchanges). Thus if the time varying concentration of lactate is denoted by C, we have

$$V\frac{dC}{dt} = \text{Production} + \text{Uptake} - \text{Utilisation} - \text{Release} \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ 5.1$$

which is a differential equation applicable to both compartments of the model at any time.

Diagramatically the model can be represented as follows:

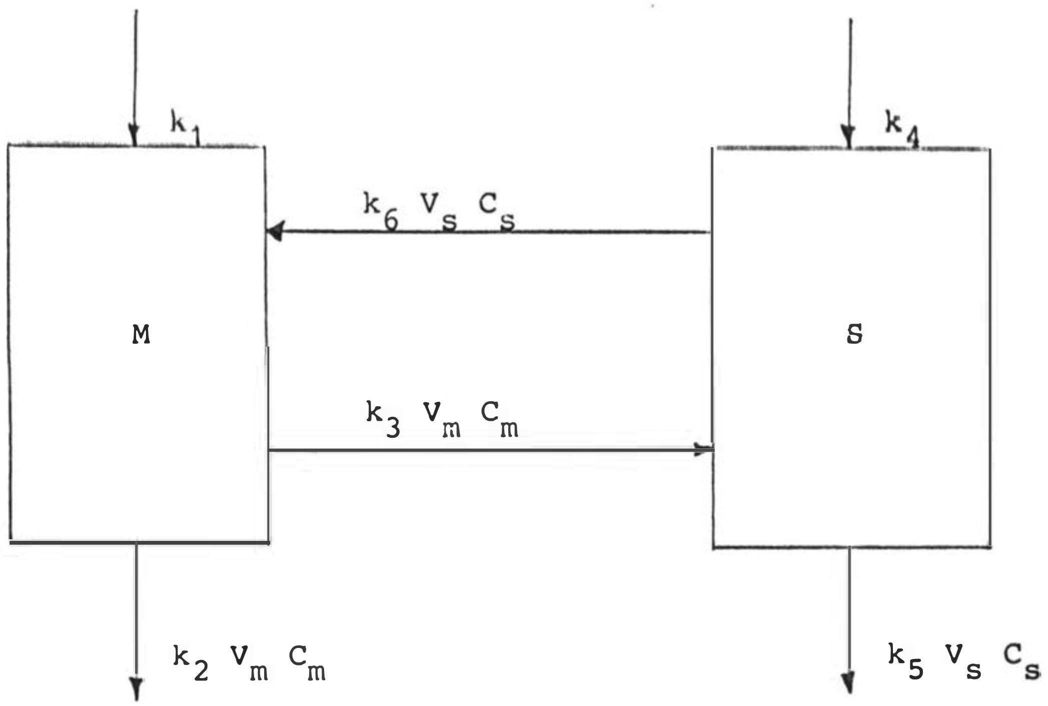


FIGURE 5.1: The Two-Compartment Lactate Model

The fundamental assumptions of the model are as follows: -

1. The total lactate distribution space is composed of two compartments; the previously working muscles M, and the remaining lactate space S; having respectively the constant volumes V_m and V_s, in which the time-dependent concentrations C_m and C_s prevail.

2. The rates of lactate release and utilisation in M and in S are proportional to the lactate contents

of these compartments, i.e. V_mC_m and V_sC_s. The coefficients of proportionality in all cases are positive constants.

3. The post-exercise lactate production rates in both M and S are positive constants.
4. Arterial lactate concentration represents the average in S ie. $C_s \simeq C_a$.

These assumptions are discussed and justified at some length in Zouloumian and Freund (1981a).

The purpose or values in having the model are given as: -

(i) It allows abstractions to be studied, using the mathematical relationships which bind them.

(ii) A variety of experimental situations which involve lactate accumulation or dissipation can be studied under varying conditions.

(iii) Simulation studies can be easily carried out.

(iv) The model can be inserted into a wider physiological scheme.

It is the last which applies in this study.

The mathematical solution, utilising Laplace transforms, is given in Zouloumian and Freund (1981a) and the properties of the model are investigated in detail in Zouloumian and Freund (1981b). None of this is of much concern in this study, except to note that the authors were not able to find any plausible assumptions on parameter values or signs, which invalidated the model.

It will have been noted of course that the model is proposed for lactate circulation <u>after</u> exercise, and the main purpose for which it is intended here, is for circulation <u>during</u> exercise. Thus the assumptions must be examined for their applicability during exercise.

DEVELOPMENT OF THE TWO-COMPARTMENT MODEL

Assumption 1 is unaffected, except to note that compartment M can be the working muscles or the previously working muscles, as the case may be. Assumption 2 is also unaffected, being independent of whether the subject is working or not. During work, the lactate contents (and concentrations) of the compartments, particularly of M, will of course be higher than when at rest. This in no way affects the assumption, only the consequences of that assumption. Assumption 3 on the other hand is in part very much affected; the rate of lactate production in compartment M is no longer constant (though it may be at various times). However, a production rate is obtainable from equation F of Chapter 4, where the three components of bioenergetic modelling were considered. As far as the lactate production in Compartment S is concerned, it seems plausible to assume constant production at the resting level all the time. This seems a necessary assumption, but one which could be questioned. To my knowledge there are no other demonstrated facts which can confirm or deny this assumption. Assumption 4, as with 1 and 2 is again unaffected, and holds during work or recovery. Thus the only change which must be made is to replace the assumed constant lactate production in compartment M, by one obtainable from equation F of Chapter 4. The remaining assumptions can be sustained and as will be seen later, no inconsistencies arise from application of the model to the exercise phase.

The work of Freund and his associates must therefore be adapted to derive two equations, for muscle lactate and for arterial (blood) lactate, from equation 5.1 above. Regarding the muscle compartment first, the production at any time is given simply by the basal metabolic lactate production, which is the constant post-exercise figure they assumed, plus that produced as a result of any exercise, from equation F in the previous chapter. Therefore

$$\text{Muscle lactate production} = k_1 - \frac{dL}{dt} \quad\ldots\ldots\ldots\ldots\ldots\ldots (5.2)$$

The rates of utilisation and release, from assumption 2, are readily obtainable as $k_2V_mC_m$ and $k_3\ V_mC_m$ respectively, while the uptake by compartment M is the release from compartment S. Regarding compartment S, the production is constant at k_4, and the utilisation and release are similarly $k_5\ V_s\ C_s$ and $k_6\ \ V_s\ C_s$ respectively. The uptake by S is the release by M. The $k_1\ .....\ k_6$ are the constants of proportionality. Thus applying equation 5.1 to the two compartments, and utilising 5.2, equations of the following forms are obtained:

$$V_m\frac{dC_m}{dt} = k_1 - \frac{dL}{dt} + k_6V_sC_s - (k_2 + k_3)V_mC_m \text{ .... } (5.3)$$

$$V_s\frac{dC_s}{dt} = k_4 + k_3\ V_mC_m - (k_5 + k_6)\ V_sC_s \text{ .... } (5.4)$$

A great deal of experimental work by Freund and his associates has gone into obtaining data to estimate the parameters in their model, though not necessarily the eight constants in the above pair of equations. An equivalent undertaking for this study would not have formed part of the main theme of the work, and in any case was beyond the scope of the experimental work able to be conducted.. The estimates obtained by Freund and his associates must therefore be used in this study also, modified only be conversion into appropriate units.

The two simplest are V_m and V_s, given as 13.5 and 28.6 litres respectively. These convert simply to 135 and 286 decilitres (i.e. units of 100 ml). Next, the two transfer coefficients k_3 and k_6 are estimated at .42 and .1983 min^{-1} respectively, which convert simply again to .007 and .0033 sec^{-1} respectively. Now at rest, the lactate concentration is the same throughout both compartments, C_r say, which is about 10 mg/100 ml (Margaria 1976). We note then that

$$k_3V_mC_r = k_6V_sC_r = 9.45 \text{ mg/sec}$$

which means that at rest (i.e. $t \rightarrow \infty$), the lactate transfers between compartments are in equilibrium. This seems eminently reasonable.

Thirdly, the utilisation coefficient in S is given a value of around 0.72 min^{-1}, i.e. $k_5 = .0012\ \text{sec}^{-1}$. We now note that only k_4 remains in equation 5.4. Thus at equilibrium when the concentrations are stable

$$0 = k_4 + (.007)(135)(10) - (.0012 + .0033)(286)(10)$$

i.e. $k_4 = 3.42$ mg/sec.

Fourthly, Freund and his associates give a figure of 2 millimoles per litre per minute as the lactate production in compartment M. This converts to $k_1 = 40.5$ mg/sec. This figure seems a little high, though it does seem reasonable to expect a larger value than $k_4 = 3.42$ mg/sec. Unfortunately there appears to be no other value in the literature to serve as a check.

Lastly, we can now apply equation 5.3 at equilibrium to obtain a value for k_2, i.e.

$$0 = 40.5 + (.0033)(286)(10) - (k_2 + .007)(135)(10)$$

i.e. $k_2 = .03\ \text{sec}^{-1}$

Once again a k_2 figure in excess of $k_5 = .0012$ might be expected, but this value does seem a little high. As above, there appears to be no other reported values to serve as a check.

Thus we can obtain the two differential equations for the lactate circulation component of the whole model. They are given by

$$\frac{dC_m}{dt} = (40.5 - \frac{dL}{dt} + .945\ C_s - 4.995\ C_m)/135 \ldots\ldots\ldots \text{G}$$

and

$$\frac{dC_s}{dt} = (3.42 + .945\ C_m - 1.287\ C_s)/286 \ \ldots\ldots\ldots\ldots\ldots \ H$$

These are the seventh and eighth equations of the system.

CHAPTER 6

FATIGUE FEEDBACK MODELLING

Since no subject can maintain a strenuous workload indefinitely, fatigue and exhaustion must enter the system as a feedback mechanism at some stage. It will be assumed that the fatigue mechanism acts in some way on the total force that the runner can exert through the legs. That is it acts on f, rather than on the power P. As the subject becomes affected by the onset of fatigue, the will power to maintain f at the chosen level (or to increase it) will be overridden by biochemical controls, and f will decline. This means in turn that the velocity will decline, and so will P and the metabolic energy demand.
Since the body could no longer sustain that metabolic demand, the change is self-righting. The subject can only maintain f at the level the body allows, and f will continue to diminish until the energy demand has been lessened to a level that the body can accommodate (such a level may be zero). The subject however could forestall the fatigue control, if he/she knows the indicators of its incipience, by voluntarily lowering f. This may be done a number of times, but unless f is radically reduced fatigue will eventually intervene. Alternately the subject may cease exercise all together, and so f = zero.

EXERCISE PRIOR TO THE ONSET OF FATIGUE

If the subject undertakes exercise at the maximal or "all-out" level, the onset of fatigue will be rapid; 20 or 30 seconds for a fit healthy individual. However, if he/she undertakes exercise at a lower level, say a constant load, then the endurance is increased many-fold. This sort of power-endurance relationship has been of some interest to physiologists (Tornvall 1963, Nordesjo 1974, Wilkie 1980), but is not of fundamental importance in this study. A curve depicting this relationship could be obtained as a particular set of solutions to the model system, if required.

Consider the second situation first (since it is simple to deal with mathematically). For constant workloads, or more correctly constant propulsive force, we have

$$\frac{df}{dt} = o \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad I(a)$$

with an initial value $f(0) = f_0$, selected by the subject. Now $0 \leq f_0 \leq F$, where F is the maximum possible in the resting state, and F seems to be about eight times the mass of the subject, in newtons, (Hill 1927b, Henry and Trafton 1951, Keller 1973, 1974).

It should be noted that equation I(a), together with equations B and C (allowing time for acceleration) imply that $\frac{dP}{dt} = 0$, i.e. constant workload. Many exercise testing procedures assume P is constant on the cycle ergometer (or that v on the treadmill is constant), and ignore acceleration. In a mathematical treatment, such a simplification cannot be made. If fatigue acts directly on P and not indirectly through f (which may be arguably inferior on physiological grounds), then for a constant workload equation I(a) should be replaced by

$\frac{dP}{dt} = 0$ with $P(0)$ set at the desired level. However, since $P = fv$ and the runner starts from rest, i.e. $v(0) = 0$, this implies $f(0) \rightarrow \infty$, which is clearly not possible. Thus it seems appropriate for constant load work prior to the onset of fatigue, to use equation I(a), with a chosen initial condition, f_0.

Prior to the onset of fatigue, there is no requirement for f (or the workload) to be constant, since f is under the wilful control of the subject. Indeed Equation I(a) could be replaced by any suitable alternative. For example, another common form used in exercise testing is the progressive, or ramp test, where $P = rt$. The parameter r is the ramp coefficient, values of $\frac{1}{2}$-1 watt per second being common on the cycle ergometer. (The situation is sometimes complicated mathematically by using discrete jumps, of say 30 watts per minute, rather than a continuous procedure.) In the true ramp test, we can deduce

$$\frac{df}{dt} = \tfrac{1}{2}ra/f \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad \text{I(b)}$$

but we must take care that the initial condition $f(0) = f_0$ does not include $f_0 = 0$. (a is the parameter from equation B in chapter 3, the resistance parameter in the equation of motion).

At the onset of fatigue there is a change, since equations I(a) or I(b) are replaced by one in which $df/dt < 0$. In order to derive such an equation we must first consider what is known physiologically about fatigue.

SOME PHYSIOLOGY OF FATIGUE

Asmussen (1979) differentiates between two basic mechanisms of fatigue; central (proximal to the motor neurons, i.e. mainly in the brain) and peripheral (distal to the motor neurons, i.e. mainly in the muscles themselves). It is the latter that is the major concern in this work. He regards this fatigue as being due to the depletion of metabolites (principally glycogen) and/or the accumulation of catabolites (principally lactic acid). The former is predominant in prolonged exercise of low to moderate intensities, and the latter in short-term high-intensity exercise. These views are shared and confirmed by many others (Farrell et al 1979, Karlsson et al 1975, Lindstrom et al 1970, Simmonson 1971, Tesch 1980, Treffene et al 1980, etc), and reflect the accepted state of belief at present.

There is all round agreement that heavy lactate accumulation leads to muscular fatigue, at least in the short to medium term, following heavy to severe workloads. Asmussen (1980) refers to lactic acid as a "fatiguing substance'. Muscle normally functions in an alkaline medium, and it has been long recognised that an acidic medium impairs the ability both to contract repeatedly and to maintain tension. Unfortunately there appears no published quantitative measure of the relationship between the degree of impairment and the concentration of lactic acid (or say the pH).

It is thought that the fatigue mechanism operates through a decrease in Ca^{++} ion availability (Tesch 1980). Several studies, while not directly quantifying the level of fatigue, have found, for example, that a rating of perceived exertion correlates strongly with both blood and muscle lactate (Ariyoshi et al 1979, Jacobs 1981a). Small to moderately elevated lactate levels have in some studies (Weltman et al 1977, 1979) been found to exert little or no demonstrable effect on subsequent performance of short-term maximal exercise, but in several others, the contrary result was demonstrated (Karlsson et al 1975, Stamford et al 1978, and others), particularly when the elevation was pronounced. It should be noted that, as with incomplete glycogen depletion, lactate level need not be unusually high when exhaustion after prolonged exercise occurs (Farrell et al 1979, Jorfeldt et al 1978). In such cases it is suggested that the mechanism may involve the inhibition of the free fatty acid metabolism (Ivy et al 1980, Jacobs 1981a).

Glycogen is of course the main precursor of lactic acid formation (Jacobs 1981a), and the impairment of strength and the endurance of subsequent exercise has been shown to depend on the pattern of prior depletion (Jacobs et al 1981a); i.e. on the level of prior exercise and the fibre-type distribution. However, depletion of glycogen is not a prerequisite for exhaustion, for exhausted runners have been found to still possess a significant, though reduced, glycogen level (Costill et al 1973b, Gollnick et al 1973, Gollnick et al 1974a), and this observation also implies that the remaining glycogen store does not necessarily therefore reflect the metabolic reserves of the working fibres (Costill et al 1973b, Gollnick 1973). Acute depletion however, will lead to impairment and reduced anaerobic capacity (Asmussen et al 1974), particularly in muscle rich in FT fibre (Jacobs 1981a). It has been noted that lactate accumulation matches glycogen depletion (Gollnick et al 1973, 1974b) but glycogen restoration does not match lactate removal (Brooks and Gaesser 1980, Gollnick et al 1974b).

It is a pity that, other than demonstrate some form of significant reduction in muscle performance, all the studies have failed to address or even answer the relevant question:

By what amount is the muscle strength reduced if the lactic acid concentration is X mg/100 ml?

Maybe they have tried but without success. One study (Tesch 1980) notes ... "In these experiments it was not possible to relate exhaustion time to a certain level of lactate concentration". I can see that I shall have to look hard at this last aspect of my modelling.

PROBLEMS OF FATIGUE EXPERIMENTATION

On the face of it, experimentation in order to relate the exhaustion times (or rather the constant forces being exerted until those times) to the levels of muscular lactate concentrations, seems feasible. This is only complicated by the invasive technique (needle biopsy) required to determine the muscular lactate concentration at the time of exhaustion. Alternately, perhaps the lactate circulation model could be used for backwards extrapolation after exercise, to estimate a C_m value at exhaustion for each workload. In either case a picture could be built up of the relationship between force f and muscular lactate concentration C_m. The curve denotes the boundary of a work area within which the subject can work at will without being overcome by fatigue. On the boundary, f is maximal for the associated C_m, and fatigue prevents the subject from crossing that boundary.

Based on the information in the previous section on the physiology of fatigue, the obvious features of such a boundary curve are as follows. It starts at the maximum propulsive force, F, about 600 newtons, at rest when the muscular lactate concentration is at the normal resting level of about 10 mg/100 ml. As Cm increases it is fairly flat, decreasing only slowly. It obviously drops fairly sharply nearing the 145 mg/100 ml mark (Karlsson et al 1972, Knuttgen and Saltin 1972). Thereafter it must drop as C_m increases further, and tend asymptotically towards a constant, probably zero. This suggests a sigmoid cúrve such as in Figure 6.1 below.

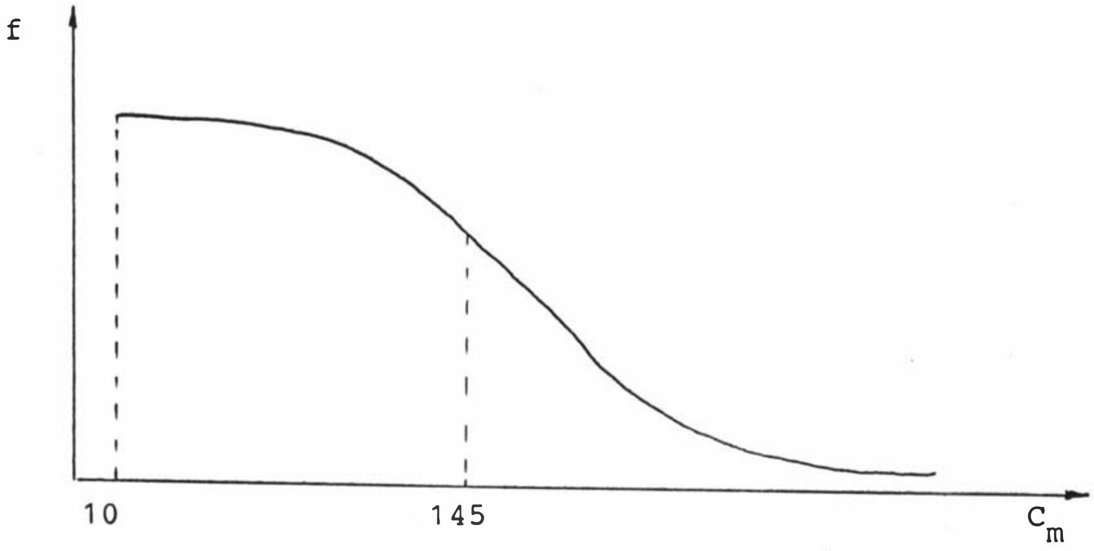


FIGURE 6.1: A Suggested Fatigue Curve

There is however an important difficulty in obtaining a full data set from any experimental procedure designed to estimate such a curve. It is well established (e.g. Scheen et al 1981, Treffene et al 1980, and others), that for any given workload, the lactate concentration in the blood or muscles will only rise to a certain peak value. This value may bring the onset of fatigue, in which case it represents a point on the required curve, or it may not, either maintaining a steady value or decreasing, as exercise continues. In both these latter cases, the points represented lie at all times below the required curve. This means that it is experimentally impossible to obtain some observations on the curve. This area falls below the peak-value curve which can be superimposed on Figure 6.1 and the area is shown shaded in Figure 6.2 below.

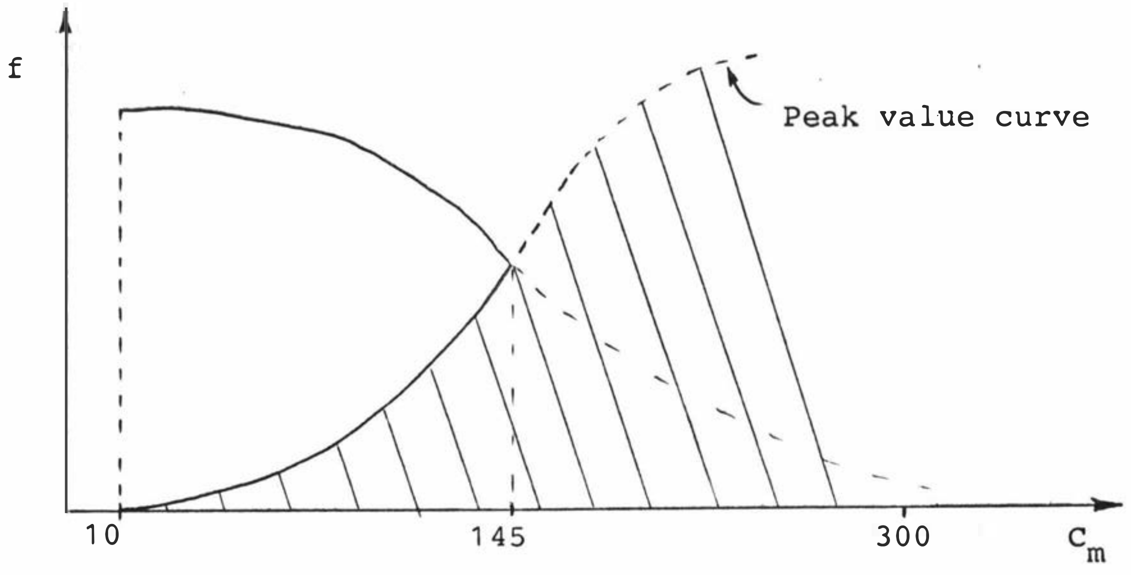


FIGURE 6.2: Fatigue and Peak Lactate Curves

Experimentally there seem only two ways around this problem. One would be to artificially induce a high muscle lactate concentration and then exercise the subject, in this way depressing the peak value curve (as it appears graphically in Figure 6.2) and so enlarging the experimentally feasible region. Just whether this approach is practicable is itself a difficult question. The second possibility would be to work the subject until exhaustion, and then, rather than cease exercise, let (or coerce) him/her to continue at the highest level the body will allow. In this way, the subject tracks down the boundary curve rather than "falls" off it as when exercise ceases. This approach has two requirements, some sort of continuously adjustable ergometer, and a continuous or at least frequent, monitoring of muscle lactate concentration. The practicability of this latter is open to question also.

Notwithstanding these difficulties, the resources for conducting such experimentation were unavailable. Thus some rather blind empirical modelling must be undertaken in a search for a suitable fatigue curve.

A FATIGUE FEEDBACK EQUATION

Since we are investigating a relationship between force f and muscle lactate concentration C_m, an expression for df/dC_m is required, to be used in

$$\frac{df}{dt} = \frac{df}{dC_m} \cdot \frac{dC_m}{dt} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (6.1)$$

where the latter derivative is obtained from equation G in Chapter 5.

There are two possible empirical approaches to obtaining a sigmoid curve to suit. The first is to use a relationship whose equation is known, from which df/dC_m can be obtained directly. The second is where df/dC_m is postulated to follow a certain form. In either case, appropriate parameter values and initial conditions must be utilised.

A candidate for the former approach might be an equation such as

$$f = e^{\alpha}[1 - \exp(- \beta/(C_m-10)^n] \ldots\ldots\ldots\ldots\ldots\ldots \quad (6.2)$$

Suppose for example F to be about 700 newtons, in which case α is about 6.55. As far as parameters n and β are concerned, note that n determines the gradient of the curve (in a general sense), and that n and β together determine the inflection point. Trying n = 1, 2 or 3, and assuming an inflection point at around the 145 mg/100 ml mark, determine β. Sketching those three leads to the following figure for equations 6.2.

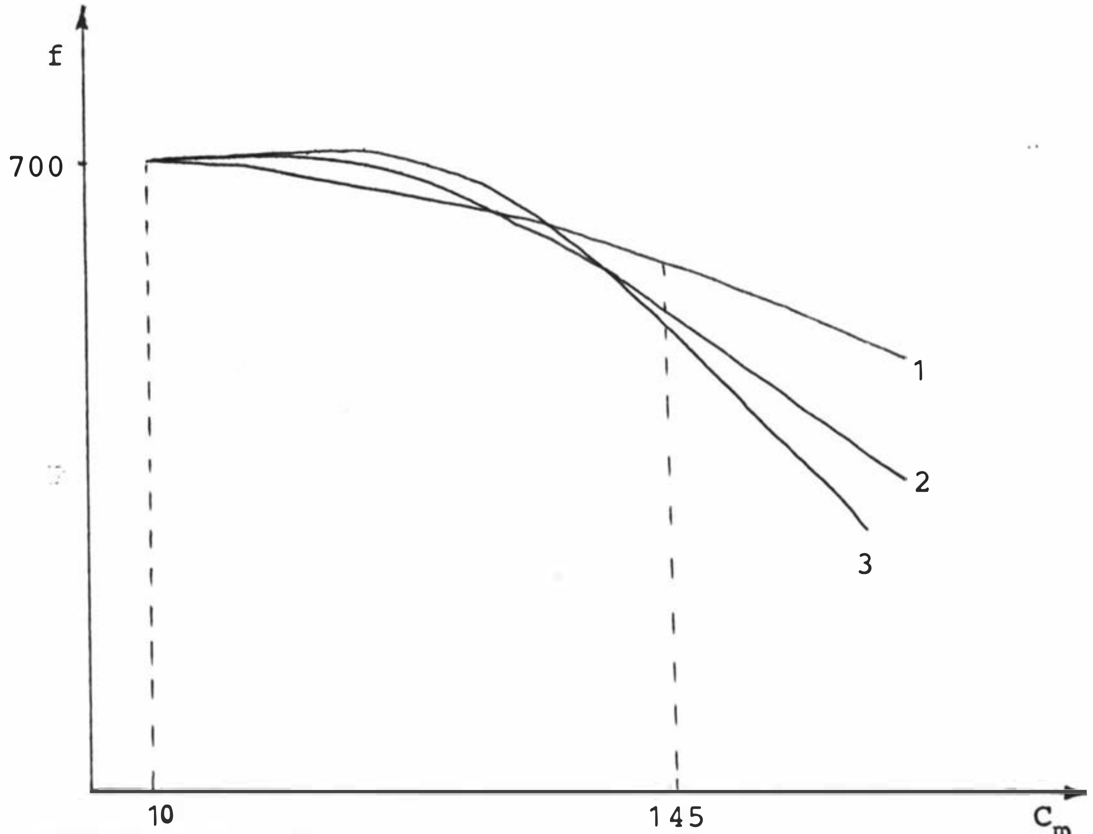


FIGURE 6.3: Sketches of Equations 6.2

Critical examination of these reveals that the cases for n = 1 and 2 do not drop sharply enough, and that n = 4 should perhaps be tried. Two other reasons however, suggest that this curve could not be manipulated to give the right sort of shape. Firstly, as n increases, the rate of curvature prior to the point of inflection is becoming noticably more rapid, and the segment leading up to it is becoming increasingly flat. For n = 4, the situation would be even more pronounced than in the above Figure. This would mean that increasing lactate concentration would have no effect, and then fairly suddenly, just prior to the 145 mg/100 ml level, the effect would be rather abrupt. Secondly, although f at the point of inflection is dropping, its value for n = 3 and probably for n = 4 also, is still too high, being about 2/3 of F. It would seem more appropriate to have a more gentle drop in f, but one which continues for a longer way before levelling off. That is, the value of f at the point of inflection would more appropriately be below say $\frac{1}{2}$F.

A candidate for the second empirical approach might be to suppose

$$\frac{df}{dC_m} = k_1 f^2 - k_2 f \;\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \;(6.3)$$

where k_1 and k_2 are suitably chosen constants and $f < k_2/k_1$. This is simply the decay version of a very common growth model, often used in population studies. Its solution is a hyperbolic tangent.

$$f = -\frac{k_2}{2k_1} \tanh\left(\frac{k_2}{2} C_m - C^*\right) + \frac{k_2}{2k_1}$$

Where C* is a constant to suit the initial conditions. The values of k_1 and k_2 can always be selected large enough to make the curve as steep as required, but the value of f at the point of inflection is always $\frac{1}{2}$ F by symmetry of the tanh function. This can be seen in the following figure.

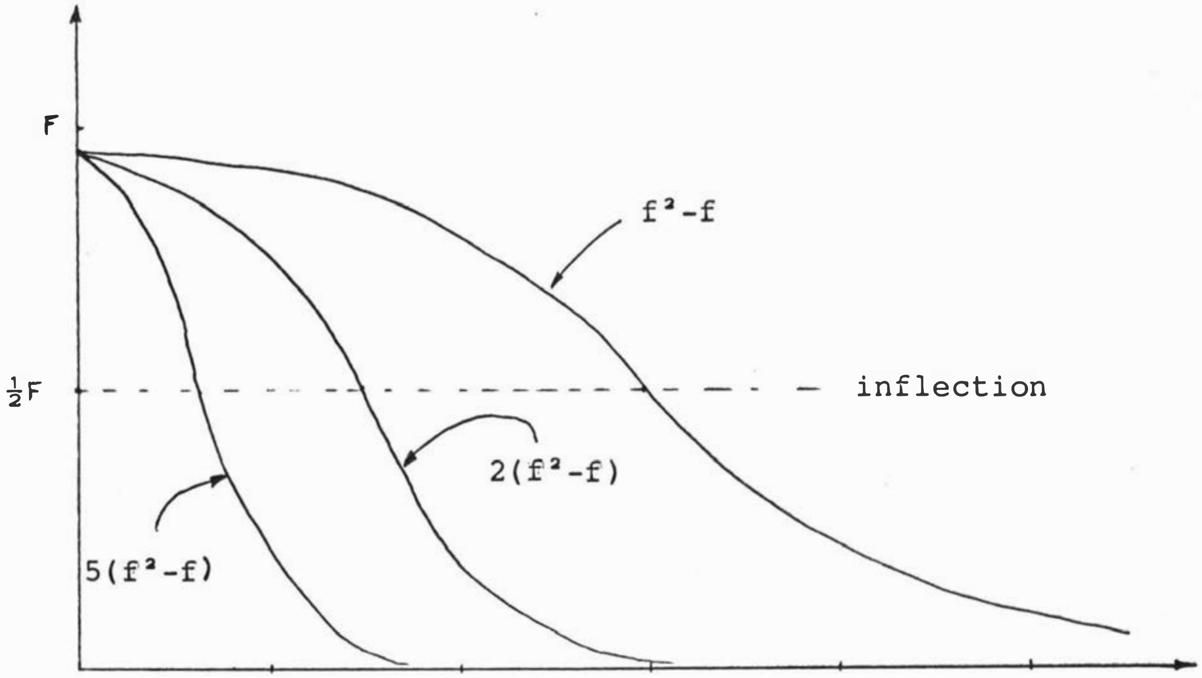


FIGURE 6.4: Sketches of Equations 6.3

If the form of the differential equation is changed to

$$\frac{df}{dC_m} = k_1 f^3 - k_2 f \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (6.4)$$

or $$k_1 f^4 - k_2 f \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (6.5)$$

the gradient is steepened (for the same k_1 and k_2), but the value of f at the point of inflection rises above $\frac{1}{2}F$, so changes in this direction are inappropriate. This can be seen in the following diagram.

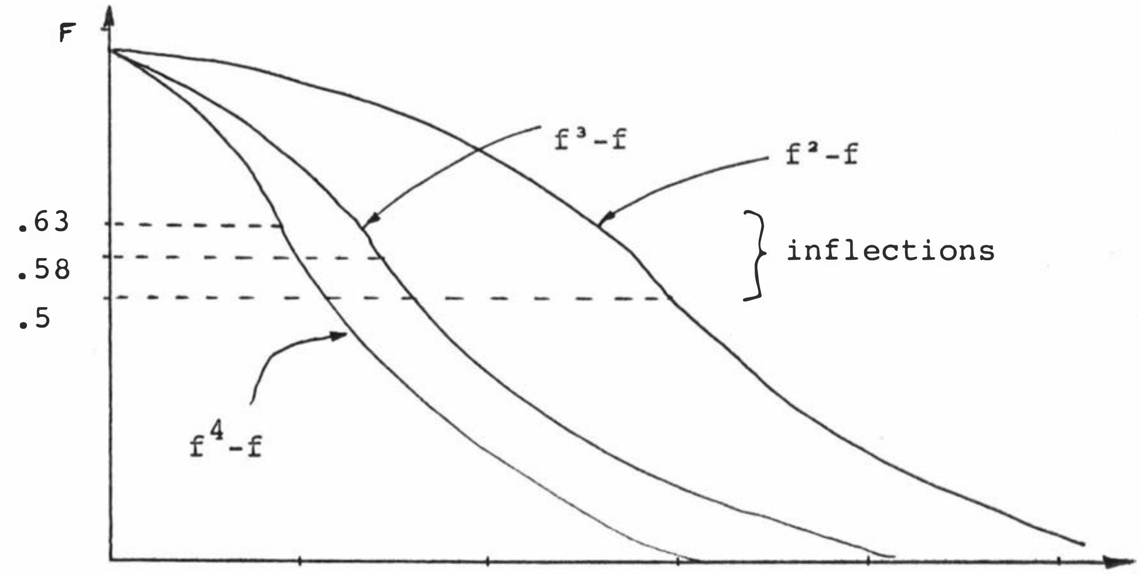


FIGURE 6.5: Sketches of Equations 6.3-6.5

A change in the other direction might be appropriate, i.e. df/dC_m of form $f - \sqrt{f}$, which can also be made as steep as required, but has its inflection point at f below $\frac{1}{2}$ F. A comparison with some of the previous alternatives is shown in Figure 6.6 below.

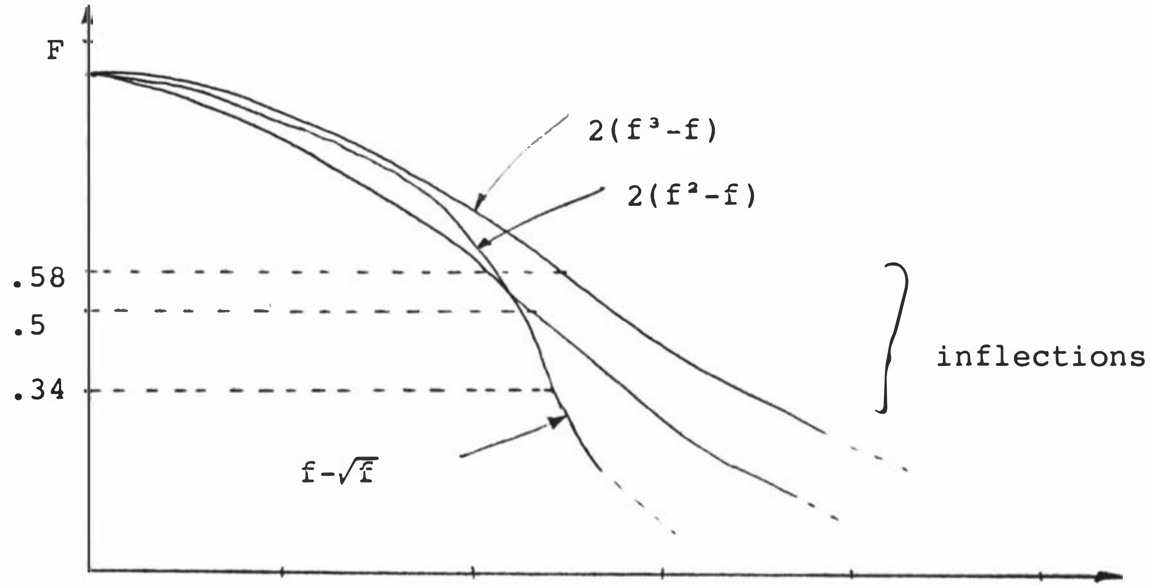


FIGURE 6.6: Comparison of Fatigue Curves

This suggests that a suitably chosen version of a differential equation of this form represents the preferred option. As will be seen later, a simulation of the full model with this and some of the previous alternatives, leads to the same preference.

There are two little difficulties with all these forms of the differential equation. The first is that we cannot set $f_0 = F$, for the derivative is then zero, and f never declines irrespective of the value of C_m. Secondly, if we start at some $f_0 < F$, then we find that the point of inflection where the drop in muscular power is steepest, is very dependent on the starting value chosen. If f_0 is not close enough to F, then it might occur at a value considerably lower than the "tolerance value" of about 145 mg/100ml suggested by the literature; and vice versa. These difficulties did not occur earlier when the actual equation was known.

This suggests we must solve the differential equation for its analytic solution to get around the problems. This can be done since the equation is simple. Thus suppose

$$\frac{df}{dC_m} = k_1 f - k_2\sqrt{f} \text{ with } f < \frac{{k_2}^2}{{k_1}^2} \quad \ldots\ldots\ldots\ldots \quad (6.6)$$

Putting $y^2 = f$ with $df/dC_m = 2\,y\,dy/dC_m$, then

$$\frac{dy}{dC_m} = \frac{k_1}{2}\,y - \frac{k_2}{2}$$

which is a simple first order line or differential equation with general solution

$$y = \frac{k_2}{k_1} + P\,e^{\frac{1}{2}k_1 C_m}$$

where P is a constant determined by the initial conditions.

Thus we obtain

$$f = \left(\frac{k_2}{k_1} + P\, e^{\frac{1}{2}k_1 C_m}\right)^2$$

and we must obtain values for k_1, k_2 and P.

We know that at rest, $C_m = 10$ and $f = F$, (= 600 newtons say, i.e. F is known)

$$F = \left(\frac{k_2}{k_1} + P\, e^{5\,k_1}\right)^2 \tag{a}$$

At inflection, df/dC_m is a minimum, which can easily be found to occur at $f = k_2{}^2/4k_1{}^2$. We may now suppose that this occurs at .4 F say, and hence

$$\frac{k_2}{k_1} = \sqrt{1.6\ F} \tag{b}$$

and hence from (a) and (b) we get

$$P = (1 - \sqrt{1.6})\sqrt{F}\ e^{-5k_1} \tag{c}$$

Thirdly we might suppose that at $C_m = 160$ say, f has dropped to 1/20 F. Hence

$$.05F = \left(\frac{k_2}{k_1} + P\, e^{80k_1}\right)^2 \tag{d}$$

and hence from (b) and (d) we get

$$P = (\sqrt{.05} - \sqrt{1.6})\ \sqrt{F}\ e^{-\ 80k_1} \tag{e}$$

Equating (c) and (e) and solving for k_1, gives

$$k_1 = .0184$$

Hence from (b), $k_2 = .0231 \sqrt{F}$

and from (c), $P = -.2416 \sqrt{F}$

$$\therefore \quad f = F(1.2649 - .2416\, e^{.0092\, C_m})^2 \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (6.6)$$

$$\text{or} \quad C_m = \ell n\, (5.2355 - 4.139 \sqrt{f/F})/.0092 \quad \ldots\ldots\ldots\ldots\ldots \quad (6.7)$$

and note that at $C_m = 10$, $f/F = .9999$, which is rather high.

Therefore, prior to fatigue, we can use I(a) or I(b) or some other suitable equation for df/dt. At fatigue there is an interrupt, as determined from equations 6.6 or 6.7. Thereafter if the subject rests, we must reset f to zero and continue with I(a). If the subject continues at a non-zero but lower value of f, then I(a) can be used again until the next interrupt, etc, with f being reset each time. If the subject wishes to track down the fatigue curve, i.e. to continue to the best of his/her ability, we must use equation 6.6 with $k_1 = .02$ and $k_2 = .023 \sqrt{F}$ in equation 6.1, i.e.

$$\frac{df}{dt} = (.02\, f - .023\sqrt{Ff}).\frac{dC_m}{dt} \quad \ldots\ldots\ldots\ldots\ldots \quad \text{I(c)}$$

In an "all-out" effort, where the subject begins with f_0 only just less than F, equation I(c) must of course be used ab initio.

CHAPTER 7

THE MODEL AS A WHOLE

Equations A to I, with appropriate selections for equation I, as described in Chapters 3 through 6, define the model system as a self-contained whole. Only variable f, the muscular force is exogenous to the system leading to the choice of form for equation I. The remaining eight variables are endogenous. Even variable f, if the subject is exercising "all-out", is endogenous, in which case the system, given appropriate initial conditions, is entirely self-determining. A re-ordering of the equations however is necessary before simulation.

Since each iteration in the simulation operates sequentially on the equations defining the system, we must take note of the following:

(a) df/dt and dv/dt are required in order to determine dP/dt.

(b) dP/dt is required in order to determine dV/dt.

(c) dA/dt is required in order to determine dL/dt.

(d) dL/dt is required in order to determine dC_m/dt.

(e) dC_m/dt is required in order to determine df/dt, unless a special form of df/dt is used prior to fatigue.

It follows that the system variables must be evaluated, through their derivatives, in the following order:

$$A,\ L,\ C_m,\ C_s,\ f,\ v,\ P,\ D,\ V$$

that is, the equations in corresponding order

E, F, G, H, I, B, C, A, D

The full mathematical description of the system is thus

$$\frac{dA}{dt} = A[5.25m(V-6)/60 - P/3]/552m \ \ldots\ldots\ldots \quad (7.1)$$

$$\frac{dL}{dt} = 5.25m(V-6)/60 - P/3 - 46dA/dt \ldots\ldots\ldots \quad (7.2)$$

$$\frac{dC_m}{dt} = (40.5 - dL/dt + .945C_s - 4.995C_m)/135 \ \ldots \quad (7.3)$$

$$\frac{dCs}{dt} = (3.42 + .945C_m - 1.287\ C_s)/286 \ \ldots\ldots \quad (7.4)$$

$$\frac{df}{dt} = (.0134f - .017\sqrt{Ff})dC_m/dt \ \ldots\ldots\ldots\ldots \quad (7.5a)$$

$$\text{or} \quad = 0 \quad \text{for constant load work} \ \ldots\ldots \quad (7.5b)$$

$$\text{or} \quad = \tfrac{1}{2}ra/f \quad \text{for ramp function} \ \ldots\ldots\ldots \quad (7.5c)$$

$$\frac{dv}{dt} = (f - av)/m \ \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (7.6)$$

$$\frac{dP}{dt} = fdv/dt + v\ df/dt \ \ldots\ldots\ldots\ldots\ldots\ldots \quad (7.7)$$

$$\frac{dD}{dt} = v \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (7.8)$$

$$\frac{dV}{dt} = b'(t\ dP/dt + P)/3m$$

$$+ a'[dP/dt + e^{-\lambda t}(\lambda P - dP/dt)]/3m \ \ldots\ldots \quad (7.9)$$

The parameters to be determined for use in these equations are:

(a) m: the mass of the subject in kg, obtainable simply by weighing.

(b) F: the maximal propulsive force for the subject in newtons obtainable from a fit to sprint data.

(c) a: the resistance to motion constant in kg/sec, obtainable also from the fit to sprint data.

(d) b' in ml/j x 60 and a' in ml/watts x 60, parameters of the empirical oxygen uptake equation, obtainable from a fit to $\dot{V}O_2$ data of the subject during exercise

(e) λ: the rate constant for the oxygen uptake adjustment in sec^{-1}, obtainable also from the $\dot{V}O_2$ data fit.

There are of course a number of other parameters in the system for which published experimental values have been assumed, as described in the previous Chapters. This was done because the experimental phases of this study did not extend to the necessary data for estimation. This does not mean such values cannot be modified.

The following two chapters will describe the experimental procedures used to gather data, and the techniques used to estimate the required parameters. More data than required for estimation was collected, much of the excess to provide supplementary information against which to judge the adequacy of the model.

CHAPTER 8

OBSERVED CHANGES IN ENERGY STATUS IN HUMAN SUBJECTS DURING DIFFERENT LEVELS OF EXERCISE

INTRODUCTION

This research thesis, not being a purely theoretical development, involves in part the collection and analysis of data on exercising subjects. The data itself is to be used for parameter estimation in the first instance as described in the next Chapter, and also for validation of the simulated results. This practical aspect of the theoretical work has been confined to the more easily obtainable data, since resources of time, equipment, expertise and finance were themselves limited. However, sufficient data has been obtained to provide a reasonable insight into the practical aspects of the model.

The required data fell into two categories, that which had to be assumed from published experimental results, and that which could be collected using the facilities in Christchurch. This latter itself fell into two categories also, the Newtonian biomechanical data, and the bioenergetic and biochemical data.

The biomechanical data included the subjects' weight, used in several of the system equations, together with sprinting data collected on the running track. The latter provided estimates for the resistance to motion coefficient for equations (7.5c) and (7.6), and the maximum propulsive force F for equation (7.5a). The resistance to motion coefficient is of particular importance in the model, since performance seems particularly sensitive to changes in its value. The bioenergetic data included detailed oxygen consumption data over a wide range of exercise levels; together with comprehensive data on oxygen consumption, blood lactate circulation and fatigue in exercising subjects.

The former is to elucidate the oxygen consumption function and establish the suitability of equation (7.9). The latter would be used in parameter estimation and model validation. It was not possible to obtain data on the other two bioenergetic functions as discussed in Chapter 4, and hence the empirical nature of the modelling. The biochemical (lactate) data was not utilised for parameter estimation; but for insight into fatigue modelling, and model validation.

The data obtained from published experimental results included such things as the energy equivalent of the bioenergetic processes, body energy stores, diffusion coefficients in the two-compartment model, etc, all discussed in Chapters 4, 5 and 6.

MATERIALS AND METHODS

Subjects: A single subject, myself; a fit, healthy, male never a trained athlete, aged 36 years, height 1.78 metres, weight 72 kg; performed the exercises to establish the oxygen consumption function defined by equation (7.9).

For the study as a whole, a homogenous group of athletes was required. Through the contacts made earlier, seven subjects (6 male and one female) were solicited, and an eighth agreed to act as stand-by. All seven attended a preliminary session at the Respiratory Laboratory together with most of the staff to be involved in the experiments. They were given a description of the proposed protocol and of the medical implications and ethics of the testing. They investigated the equipment and had all their questions answered. No subjects requested to withdraw from the programme as a result of these discussions.

All seven subjects were given a questionnaire to complete, Appendix 2.1. From their responses, a group of six were selected. All six were male, and five of them frequently trained and often competed together. The characteristics of all subjects are summarised in Appendix 2.2. All six were once again interviewed, and signed an informed consent form prior to taking part in their first testing session, Appendix 2.3 .

Experimental Design

The experiment proper was designed for six subjects each doing four workloads, and a schedule prepared for the 24 runs over the ensuing 16 days. The schedule was determined by the number of volunteers, the availability of the laboratory and its equipment, and the demand put on them by other users, etc. It was estimated that we could satisfactorily average three runs every two days. The design is a simple factorial experiment with respect to the workloads performed and oxygen uptake data collected. For blood lactate sampling since only two collections could reasonably be made (once in each arm), the design is a balanced incomplete block experiment. The design and schedule were as follows:

Design:

			Workloads			
		Low	Med	High	Max	Name
SUBJECTS	1	1*	7*	13	19	John K
	2	2	8*	14*	20	Bruce H
	3	3	9	15*	21*	James R
	4	4*	10	16*	22	Dave K
	5	5*	11	17	23*	Rob B
	6	6	12*	18	24*	Rob McG
kpm/min		1200	1600	1850	2100	÷6 for watts

Numbers indicate "run" number

* indicate blood sampling

Schedule:

May 1983

Sun	M	Tu	W	Th	F	Sat
			4th Arrival	and		
	Preliminaries		8*	1*	4* 5*	3 6
2 10	15* 12*	13	11	Spare	14* 7*	9
18	16* 23*	20	19 17	22	21* 24*	28th Return

This timetable was not exactly adhered to, but variations were minor, only a day or so either way in a few cases.

Laboratory Experimentation:

A detailed experimental protocol was submitted to and approved by the Ethical Committee of the North Canterbury Hospital Board, Appendix 2.4.

In the event, the actual experimental procedures were not as demanding as the protocol suggests, as was indicated in the design and schedule. Two "dummy" runs were performed prior to the actual commencement, as a check that experimentation could proceed.

At the time of first attendance of each subject, Dr Stewart went through the protocol once again, and after checking the subject's medical history, both signed the informed consent document, thus satisfying ethical requirements. Thereafter computerised spirometry was performed to measure lung volumes and lung function (details Appendix 2.5, p 200).

A typical one of the 24 experimental runs, with Dr Stewart, myself and others in attendance, followed a pattern thus:

(i) The subject presented at the laboratory and was allowed a warm-up of several minutes. During this time the equipment was checked and calibrated ready to start.

(ii) When ready, Dr Stewart attached ECG leads to the subject's chest and inserted a blood sampling needle with extraction tube and two-way tap into an arm vein near the wrist.

(iii) The subject then seated himself on the cycle ergometer in a comfortable position, inserted the breathing mouthpiece which was connected to the flow-meter and medical gas analyser. A nose clip was placed on his nose and the ECG leads plugged in to the heart-rate monitor.

(iv) When settled, recording commenced at rest (see example to follow). This was a period during which breathing rate, heart rate, etc were allowed to stabilise, and during which a resting blood sample was taken. At the first blood sampling run, a separate second sample was taken at this time from each subject, for a haemoglobin determination and blood count (details to follow). This period usually lasted two to three minutes.

(v) The subject was given the signal to start pedalling and the time noted. He was instructed to maintain a frequency of 60 r.p.m. (tachometer attached to cycle handle-bars with blue arc covering 55 to 65 r.p.m.), until told to stop (at eight minutes after start) or until overcome with exhaustion, if sooner. During this period of exercise, heart rate was monitored continuously, and on the first such bout a trace was actually taken for later examination by Dr Stewart (details to follow). During this time also, respiration was monitored on a breath-by-breath basis. Also, at 40 second intervals a sample of blood was taken, these samples labelled and kept for later lactate analysis.

(vi) At the end of exercise, all monitoring and sampling continued for a minimum of four further minutes, or until 12 minutes from the start of exercise if exhaustion had occurred.

(vii) At the end of this period of recovery the recording and sampling ceased, the mouthpiece and nose-clip were removed, and ECG leads unplugged. Dr Stewart then removed the needle and when ready, the subject dismounted from the cycle.

(viii) The subject wiped down with a towel, rested in a chair or on the bed, had the ECG leads removed and was briefly checked over by Dr Stewart. After several minutes (recovery was usually quite rapid) the subject was allowed to shower if desired, dressed again, and the run was complete.

(ix) The total duration of the run was from 40 to 60 minutes, a little less once a regular routine had developed.

Note: Not all the exercise bouts involved blood sampling, but apart from this aspect, they were no different.
Each subject performed four workloads (two with blood samples), and at least 48 hours were allowed between each, at least seven days being allowed between the blood collection runs.

At the completion of experimentation a letter of thanks was given to each subject for their assistance in the study.

Data recording was as follows: the ECG monitor provided a visual heart-beat trace on the VDU monitor and a digital rate read-out. The latter was also recorded on a disk file in the PDP11/34 mini-computer. The breath-by-breath analysis provided measurements of duration of breath, oxygen consumption, CO_2 production, expired volume, end tidal O_2 and CO_2, and time; all recorded along with the heart rate on the same computer disk. A real-time display on a separate VDU provided graphical representation, of O_2 consumption, of end tidal CO_2 and the respiratory exchange ratio, RQ. (see some sample copies attached). All the disk files were later recorded on magnetic tape and then read onto my user-code on the Massey Prime Computer.

The blood samples, all coded, were precipitated by perchloric acid in 2ml test tubes on ice, capped, shaken and frozen immediately after the end of the run. They were later analysed for lactate concentration in one large batch, by the Physical Education Department at Canterbury University.

Each subject was given a copy of the real-time VDU output for that run, and had it explained to him. At a later stage a data summary of his performances were sent by post (details to follow).

There were the occasional minor problems with the actual running of an exercise test. Twice the computer crashed, but was re-started, resulting in about a minute's gap of recorded data. The exercising and blood sampling were not interrupted. The ECG recording was not 100% reliable, but since it was not an essential requirement, no action was taken at those times when it was erratic, intermittent or just not recording. There were no malfunctions of the ergometer, gas-flow measuring device or mass spectrometer gas analyser or any other equipment - or their operators!

There were some problems with the blood samplings. On two occasions the needle became dislodged from the vein during exercise, resulting in an incomplete set of blood samples. In a third run, clotting took place shortly after the end of exercise, and no further blood could be drawn. Also, three individual samples were lost due to spillage. In all 201 of the 228 samples were sent for analysis, a further two being lost during that process.

In addition, as mentioned previously because of the absence of properly collected $\dot{V}O_2$ data, I subjected myself to eight bouts over a wide range of workloads. The protocol was exactly as above, but without blood sampling, and exercise only continued for five minutes, or until exhaustion, which occurred in only the severest exercise. I was clearly neither as fit nor as capable as the athletes, but in good general shape I was told! ECG data was only collected on one or two occasions.

Appendices 2.5, 2.6(a)-2.6(f), 2.7, 2.8(a),(b), 2.9(a),(b) summarise the laboratory collected data.

Running Experimentation:

During the preliminary visit to Christchurch, the Physics Department technicians at Canterbury University undertook to make a multiple-station infra-red timing device for running athletes. After some initial delays the equipment was functional and data collection could proceed.

The sprinting data was collected in the following manner: The subjects arrived at the running track together and the simple protocol was explained and discussed to everyone's satisfaction. They were familiarised with the equipment and its functioning. Time was allowed for all of them to warm up and make a few practice runs while the equipment was checked.

Each subject ran a 100 metre sprint in one straight lane of the running track. Timing to .01 second was made throughout the sprint from the start and at ten timing stations placed at 3, 6, 10, 15, 20, 30, 40, 60, 80 and 100 metres. The set of times for each run were recorded on file in the microcomputer. After each subject had made his run, duplicate runs were carried out. Twelve sets of printed timings, output from the microcomputer, were supplied and entered into the Massey Prime computer for subsequent analysis.

By way of a summary and an indication of their performances, the 100m times for the six subjects are as follows:

Bruce H : 14.17, 14.21
Rob McG : 14.25, 14.35
John K : 13.21, 13.31

James R : 14.17, 14.25
Rob B : 12.28, 12.34
Dave K : 12.60, 12.72

CHAPTER 9

PARAMETER ESTIMATION

The data gathered as described in Chapter 8 has enabled many of the parameters of the model to be estimated. For the most part these estimations have been as a result of computerised curve fitting. In a few cases the parameters were observed by direct measurement.

The Massey University Prime computer was utilised for all the curve fitting, using the routines provided by the BMDP-82 (Dixon, 1982) Biomedical Statistical Software package. Programmes AR (derivative-free nonlinear regression) and 3R (nonlinear regression) were used. Both routines provide least squares estimates of the parameters in a nonlinear regression function. The function itself must be specified in FORTRAN statements in both cases, and 3R requires in addition the specification of all derivatives of the function with respect to its parameters. AR is used if the derivatives are awkward or are costly to compute. Standard errors and correlation matrices (asymptotic in the case of AR) of the parameter estimates, predicted values, residuals, and various plots are available as output also. Both procedures require initial estimates of the parameter values as starting points in the minimisatin iterations, but these were not difficult to obtain. 3R uses a modified Gauss-Newton algorithm, and AR uses a Lagrangian technique for the minimisation.

The parameters requiring estimation from the collected data mass, were as follows:

body mass, m : for equations 7.1, 7.2, 7.6, 7.9
resistance coefficient, a : for equations 7.5b, 7.6
maximum force, F : for equation 7.5c
oxygen uptake parameters, a', b' and λ : for equation 7.9

In addition the following initial value estimates are required for the simulation procedure:

Body (blood) lactate concentration, $C_s(0)$: for equation 7.4
which is assumed equal to $C_s(0)$
muscle lactate concentration, $C_m(0)$: for equation 7.3
resting $\dot{V}O_2$, R_e: for equation 7.9

Estimates from Sprinting:

The body mass parameter m, relevant here for equation 7.6, was recorded for each subject by direct measurement as described in Chapter 8. It is assumed that these were the same at the time of the sprinting tests as they were at the time of the laboratory tests.

Consider then equation 7.6 for the runner's velocity, and suppose that starting from rest, the constant maximum force F is exerted, at least for the duration of the 100m sprint.

$$\text{i.e.} \quad \frac{dv}{df} = (F - av)/m$$

which yields as its solution

$$v(t) = F(1 - e^{at/m})/a$$

Considering next, equation 7.8;

$$\frac{dD}{dt} = F(1 - e^{-at/m})/a$$

which yields as its solution

$$D(t) = Fm(e^{-at/m} - 1)/a^2 + Ft/a \quad \ldots\ldots\ldots \quad (9.1)$$

Equation 9.1 can now be fitted, with the appropriate value of m inserted, to the duplicated distance-time data collected from each subject on the running track. This was done using routine BMDP-3R, allowing for the possibility of a consistent reaction time in equation 9.1 (as described in Chapter 3). The following table summarises the results.

TABLE 9.1: SPRINT FUNCTION PARAMETERS

Subject Initials	mass m kg	F newtons ± SD	resistance a ± SD	reaction time	F/a m.sec^{-1}	a/m sec^{-1}	R^2
BH	67	494.5 ±26.0	64.7 ±3.67	.12	7.64	.966	.9999
RMcG	65	454.7 ±11.4	60.1 ±1.95	not signif.	7.56	.925	.9996
JK	73	466.1 ± 9.5	55.3 ±1.59	not signif.	8.44	.757	.9997
JR	60	349.4 ±29.2	44.8 ±4.16	.12	7.80	.747	.9998
RB	69	526.1 ± 8.9	58.8 ±1.39	not signif.	8.94	.853	.998
DK	73	524.5 ±10.6	60.0 ±1.69	not signif.	8.74	.822	.997
Average	67.8	469.2	57.3	.	8.19	.845	.

These latter averages are taken as representative of this group of subjects, for the purposes of simulation.

Estimations from Oxygen Consumption

In Chapter 4, the derivation of equation 4. was detailed, based on the oxygen uptake data obtained exercising myself at eight different workloads. Data from the six subjects of the main study was utilised to estimate parameters of the same function, for each individually, using the computer routine BMDP-AR. The following table summarises the results ($\dot{V}O_2$ is recorded in litres/min and w in thousands of kilo-ponds), for the equation

$$\dot{V}O_2\ (t,w) = R_e + a'wt + b'w\ (1 - e^{-\lambda t})$$

TABLE 9.2: $\dot{V}O_2$ FUNCTION PARAMETERS

Subject	Re	a'	b'	λ	R^2
JK	.310 ±.0424	.00082 ±.000110	2.162 ±.0375	.0301 ±.00153	.9704
BH	.301 ±.0259	.00115 ±.000062	1.906 ±.0218	.0321 ±.00115	.9815
DK	.520 ±.0356	.00126 ±.000097	1.760 ±.0330	.0327 ±.00210	.9698
RB	.440 ±.0312	.00168 ±.000098	1.744 ±.0301	.0288 ±.00146	.9740
JR	.468 ±.0789	.00072 ±.000221	1.497 ±.0633	.0414 ±.00615	.8070
RMcG	.396 ±.0292	.00094 ±.000108	1.837 ±.0331	.0269 ±.00131	.9790
Average	.406	.00110	1.818	.0320	.

As above in Table 9.1, these averages will serve as representative of this group of subjects. However they must be converted such that $\dot{V}O_2$ is expressed in ml/kg/min and w in watts. This yields a resting $\dot{V}O_2$, the initial value for equation 7.9, of 5.94 ml/kg.min. Parameters a' and b' convert to .000106 and .161 respectively for the cycle ergometer, and λ remains unaltered at .032. Allowing in turn for the fraction conversion between power on the ergometer and power developed during running, the estimates for a' and b' for use in equation 7.9 become .000016 and .025 respectively.

There is one further interesting aspect to the analysis of the $\dot{V}O_2$ data. The six sets of residuals have been examined, and for one subject, J.K., a plot of average residual (in each 30 second interval) versus time yields the following figure:

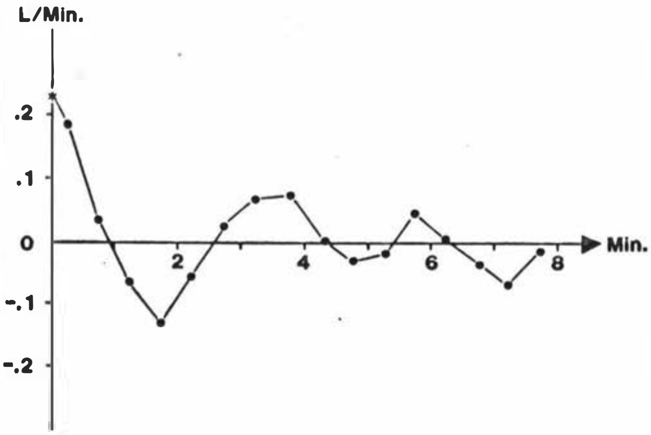


FIGURE 9.1: Residual Plot, Subject JK

This could be interpreted as a damped cyclic mechanism, with a periodicity of 2 1/2 to 3 minutes, with the amplitude reducing to that of residual variation over about two to three cycles. Such a cycle, if present, could speculatively be interpreted as evidence of some form of body response - control mechanism. The body initially over-reacts to a stimulus, then over corrects itself but by a lesser amount and so on in a convergent cyclic manner until equilibrium. In this way a cycle of diminishing amplitude is created as the body seeks dynamically to control its response to the stimulus, using a rather slow and coarse mechanism. Control processes of this sort are believed to exist as characteristics of other body responses to changes in the status quo, (Sollberger 1965, Bünning 1973).

The observation depicted in Figure 9.1 however could well be chance variation. There is insufficient evidence present in this data, and it cannot be further investigated here.

OTHER ESTIMATES

Estimates of blood and muscle lactate initial values are obtained directly from the resting determinations listed in Appendix 2.6. For the five subjects whose resting lactate concentrations were obtained, an average value of 9.4 mg/100 ml is taken as representative of the group.

There are several other parameter estimates which, as mentioned earlier, are assumed from other published experimental results. These include the various diffusion coefficients and compartment volumes of the two-compartment lactate circulation model, as used in equations 7.3 and 7.4. Likewise two initial values are assumed from published results, the body contents of phosphagens and of lactate equivalent for equations 7.1 and 7.2.

Almost pure empiricism was resorted to in Chapter 6 for the coefficients in the fatigue feedback equation 7.5c. The initial value for f in this equation is of course F as determined above.

Lastly there are two parameters of choice. They are the ramp coefficient for equation 7.5b, and the initial muscular force f(0) for equations 7.5a or 7.5b.

CHAPTER 10

SIMULATION RESULTS

KELLER'S MODEL AND EXTENSIONS

In order to check the feasibility of simulation at an early stage, Keller's simple model of the runner was simulated on a micro-computer, both in its original form and an extended form. The programme was written in BASIC and utilises a fourth order Runge-Kutta integration scheme. Programming was simple, and output was accurate.

The programme "RUNNER 1" functioned as follows:

A. Parameter Inputs
- (i) resistance constant a, where resistance to motion is proportional to velocity of motion.
- (ii) energy supply rate, σ, for the constant rate of supply during exercise.
- (iii) distance of the race
- (iv) maximum number of steps for iteration
- (v) time interval for each step, .1 sec for high accuracy - .5 sec for faster computation time.
- (vi) starting force exerted, f, maximal or submaximal
- (vii) energy store at start, E_0.

B. Running Simulation
- (i) prints time, force, velocity, energy store and distance at each iteration
- (ii) stops because: race completed and/or
  energy exhausted and/or
  final iteration reached and/or
  operator intervention

A batch of three groups of runs of "RUNNER 1" were completed as a check on the validity of the model and the simulation procedure.

(i) Generation of a running curve: An individual with the following characteristics was selected,

Resistance constant	a	$= 1.1 \text{ sec}^{-1}$
Energy supply rate	σ	$= 36$ watts
Maximum force	F	$= 10$ newtons/kg
Initial energy supply	E_0	$= 2000$ joules

and timed over both short and long distances. For all distances up to 300m, the subject is running with maximum exertion and the times cannot be improved upon. For the 400m and longer races the runner is not running with uniformly maximum effort. In this situation the choice of starting force is important. An approximation, ignoring acceleration can be obtained as follows:

Letting t be the time to exhaustion, $t = \dfrac{E_0}{fv - \sigma}$

Now $D = vt = \dfrac{E_0}{f - \sigma/v}$

Also the asymptotic velocity reached is $v = f/a$, which leads to a quadratic in f with roots

$$f = \frac{E_0 \pm \sqrt{E_0^{\,2} + 4aD^2\sigma}}{2D}$$

only one of which is realistic. Times for these races could be improved slightly by small changes in the starting forces selected.

All this leads to the following table, which when plotted (D versus $\bar{v}$) yields a running curve of the expected shape.

TABLE 10.1: SIMULATED DATA FOR THE RUNNING CURVE

Distance	Starting force	Time	Average Velocity
10	10	1.90	5.26
30	10	4.20	7.14
60	10	7.51	7.99
100	10	11.91	8.40
150	10	17.45	8.60
200	10	22.91	8.73
250	10	28.45	8.79
300	10	33.91	8.85
400	9.27	48.37	8.27
800	7.67	1 - 55.64	6.92
1000	7.41	2 - 29.69	6.68
1500	7.01	3 - 56.34	6.35
2000	6.82	5 - 23.49	6.18
3000	6.65	8 - 17.84	6.03
5000	6.50	14 - 7.13	5.90
10000	6.39	28 - 42.37	5.81

(ii) A designed experiment. Four responses were selected for modelling: 100m time, 400m time, 1000m time and the time to exhaustion. The independent variables are:

resistance constant, a, to vary between .8 and 1.2
energy supply rate, σ, to vary between 32 and 42
initial energy store, E_0, to vary between 1600 and 2400
starting force, f, to vary between 8 and 12.

A 2^{4-1} factorial, 1/2 replicate design of resolution IV was chosen, and for each of the eight simulations in this design, the four responses were measured. Analysis of each suggested the following:

100m time:	The resistance constant, a, and the starting force, f, were the only significant determinants, the former in the positive direction and the latter negative.
400m time:	The resistance constant, a, was the only significant factor, again in the positive direction.
1000m time:	The resistance constant, a (+), and the energy rate, σ (-), were significant, and the initial energy supply E_0 (-) lies just below the 5% significance level.
Endurance:	The starting force, f (-), and its interactions with the resistance constant and the energy supply rate were significant determinants.

Only one result is surprising, E_0 does not appear as a significant determinant of endurance. Of course the ranges and distances selected may have an effect, but these were chosen to be realistic for most good athletes.

To extend the response surface model a further nine simulations were added to the above eight, comprising the rest of a central composite design, allowing the fitting of quadratic terms. Generally, the results of these experiments are similar to the 1/2-replicate, though some more significances have been detected now that curvature has been more adequately allowed for. For example, the simplest case is the 100m time in which a surface given by the equation

$$t = 11.01 + 1.87\left(\frac{a-1}{.2}\right) - 2.08\left(\frac{f-10}{2}\right) - .419\left(\frac{a-1}{.2}\right)\left(\frac{f-10}{2}\right) + 4.11\left(\frac{f-10}{2}\right)^2$$

fits the responses well, with $R^2 = .996$. The other three surfaces have more complicated equations, with R^2 values of .945, .999 and .924 respectively. The resistance constant is always the most important determinant; f^2 appears as the only squared term in consideration and E_0 is noticeably absent.

(iii) Settings outside the normal limits: A large number of simulations with unrealistically high or low parameter settings have been tried in various combinations. In general, with the resistance constant too low, the athlete accelerates rapidly to a high velocity, covers 100m in a few seconds, but is burning up energy very rapidly and so expires very soon. E_0 and σ values outside the normal range have a less spectacular effect. One particular combination of relevance is how close the value of σ is to f^2/a. In general $\sigma < f^2/a$, but if very much less the athlete expires too soon, however, if $\sigma \geq f^2/a$ the athlete can run at top speed for ever! It should be noted also that it is possible to choose combinations of parameter values, some or all outside the normal ranges, and still get realistic times for runs at most distances.

This simple runners model can be extended in two main directions.

i) The first mathematical change is to modify the energy balance equation to include an exponential rise in energy supply. The simulation performs as expected, though it is not a simple matter to detect the changes directly from observing the output. The main result is that the endurance of the runner is shortened, since larger demands on the fixed energy store are being made at the start of the race during the build up of the supply rate to its asymptotic level. It follows that had estimations been performed on this modified model, they would have led to a larger estimated fixed energy store, E_0 . However, since E_0 is a parameter that can be set by the user, this presents no problem.

It is anticipated that in further developments of the programme, this energy supply wil be represented as the oxygen based component of supply, and will have a corresponding differential equation to describe its behaviour.

ii) The second and major mathematical change is to introduce a fatigue function. Unfortunately there had been little or no quantitative work in this area that can be used as a basis for deriving equations, so educated guesswork and an empirical approach of trial and error is required. As detailed in Chapter 6, fatigue is regarded as being due to the depletion of metabolites (principally glycogen, as represented by E_0) and/or the accumulation of catabolites (principally lactic acid). The former is predominant in extensively prolonged exercise of lower intensities, while the latter is important in shorter high-intensity exercise. Since a metabolite depletion process is already included in the model (depletion of energy E_0), this extension must include a lactic acid accumulation process. The fatique factor can then be based on a rise in lactic acid concentration (or possibly also on E_0 depletion if there is sufficient realism to extend the model to include very prolonged exercise)

Thus consider a new variable L: lactic acid concentration in grams/litre (of blood), and suppose simply that

$$\frac{dL}{dt} = Lp - Lr = (\text{production}) - (\text{removal})$$

(a) Production is simply the anaerobic component of energy use,

i.e. $-\frac{dE}{dt}$ converted to the correct units

i.e. $Lp = -k_1 \frac{dE}{dt}$, where $k_1 = \frac{1}{1210} = \frac{1}{220} \cdot \frac{1}{5.5}$

where 220 joules/gram is the energy equivalent of lactic acid

5.5 litres is the blood volume of an average male

(b) Removal is more "unknown", but studies have shown that it is positively related to the actual concentration and to some measure of the workload. Thus we might postulate that:

$$Lr = k_2 L fv$$ (the product fv is the power output)

However, there is no estimate for k_2 available and an educated guess is required. To this end, it could be argued that in the limit and in the absence of any fatigue, L tends to a constant, but elevated level, perhaps highly so.

i.e. $\frac{dL}{dt} = 0 = -\frac{\sigma - fv}{1210} - k_2 L fv$

If we assume that the asymptotic energy supply rate $\sigma = 32$, and the constant of resistance due to motion is $a = 1$, both of which are fairly accurate for trained athletes, then it can be argued as follows:

$f = 6 \Rightarrow v = 6 \Rightarrow fv = 36 \Rightarrow k_2L_1 = .00009183$

$f = 8 \Rightarrow v = 8 \Rightarrow fv = 64 \Rightarrow k_2L_2 = .0004132$

$f = 10 \Rightarrow v = 10 \Rightarrow fv = 100 \Rightarrow k_2L_3 = .0005620$

Since the highest observed muscle lactate concentrations are about 110mg/100ml or 1.1 g/l, this suggests k_2 approximately 2000. After allowing for a normal blood concentration at rest of .18 g/l, these yield equilibrium concentrations of .364, 1.01 and 1.30 g/l respectively. These will not necessarily be achieved, since fatigue will almost certainly intervene and induce exhaustion. (Note that the above suggests that for supra-maximal workloads, the highest equilibrium concentration is about 1.83 g/l, though this is probably beyond physiological limits).

Thus the following lactic acid accumulation equation has been introduced

$$\frac{dL}{dt} = - \frac{1}{1210} \frac{dE}{dt} - \frac{Lfv}{2000}$$

This approach has worked satisfactorily, and looks a promising start. However, lactic acid concentration rises too soon, and too steeply, in comparison to observed data. Also, no account is taken of the cleavage of high energy phosphates, a supply known to contribute to energy demands during the first several seconds of activity. Thus an amendment is immediately required to the above equation. Taking the phosphagen reaction into account may improve the situation.

Margaria (1976) regards the lactate production as zero until all (or very nearly all) the phosphagens have been utilised, whereupon it commences with a discontinuous jump. This seems unreasonable, and the lactate production must be lagged in some way to take into account these processes. This has been done in such a way that, depending on the workload, it rises slowly from zero, then more steeply, then levels off. This is illustrated in Fig. 10.1 below.

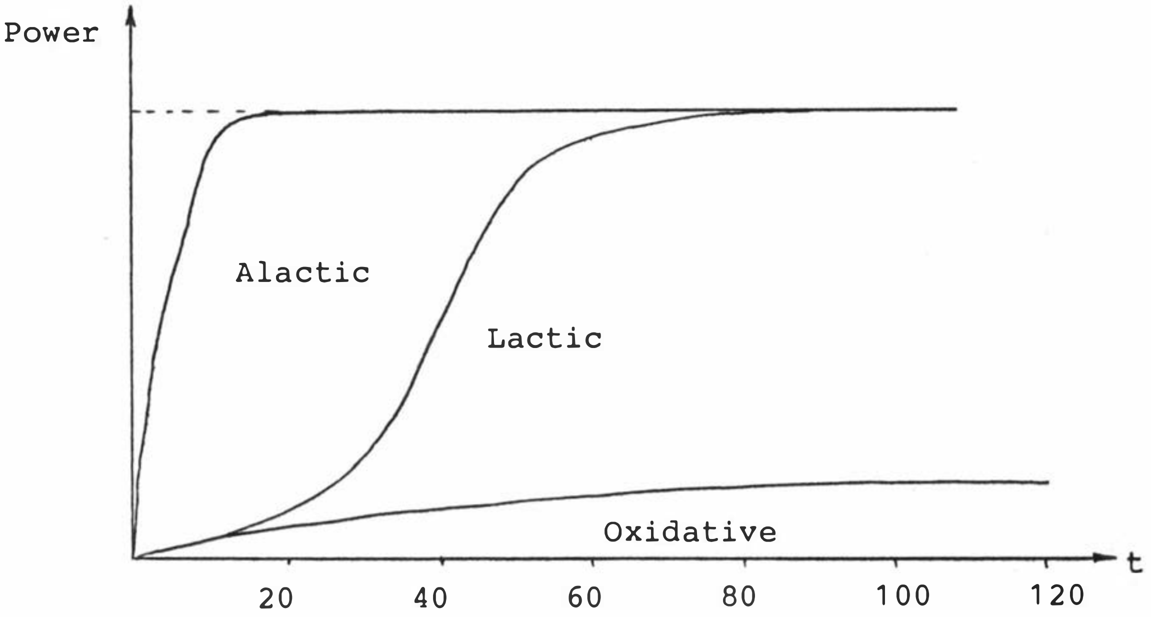


FIGURE 10.1: Power Components

The first and most obvious approach is to consider a simple variant of the law of mass action, where the rate of phosphagen based power supply is proportional to the phosphagen store remaining. The analytic form is a declining exponential. This approach can be formulated as follows:

$$\text{Power} = fv = 5.25\ O_2 + 1210\ Lp + 46\ Pc$$

i.e. Power required by Lp and Pc is $fv - 5.25\ O_2$

Thus if the proportion of the requirement supplied by phosphagen cleavage is proportional to the amount remaining

$$\frac{46\ Pc}{fv - 5.25 O_2} = k_3 \frac{P}{P_0}$$

where P is the phosphagen store (in m M) and $P_0 = 810$ mM is the initial store.

Since as t -> 0, all requirement is met by Pc, this suggests that $k_3 = 1$.

Also $Pc = -\frac{dP}{dt}$, thus

$$\frac{dP}{dt} = \frac{5.25O_2 - fv}{37260} \cdot P$$

Hence the original glycogen store E_0, from which the resulting lactic acid is produced to complete the power requirement above is obtained from

$$\frac{dE}{dt} = -\ 1210\ Lp = 5.25O_2 + 46\ Pc - fv$$

Hence the lactic acid accumulation equation, which remains unaltered, is given by

$$\frac{dL}{dt} = -\frac{1}{1210}\frac{dE}{dt} - \frac{Lfv}{2000}$$

(In addition, in order to generalise the simulation better, the mass of the runner is introduced as a further parameter. Also, as indicated earlier, the oxygen uptake has been introduced at this point, that is the energy supply rate is represented by its own differential equation. Some of the units of measurement have changed accordingly).

This approach, works well and looks promising. Figures 10.2, 10.3 and 10.4 are graphical illustrations of simulated outputs. In principle they conform to expectations based on research observations.

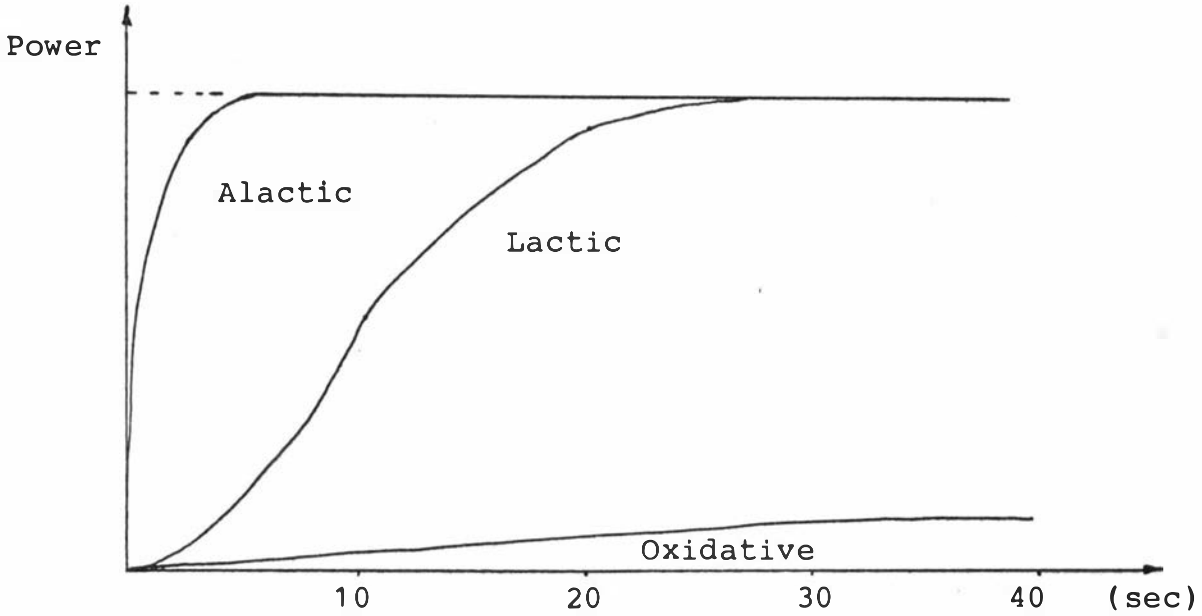


FIGURE 10.2: Power Components During Sprint

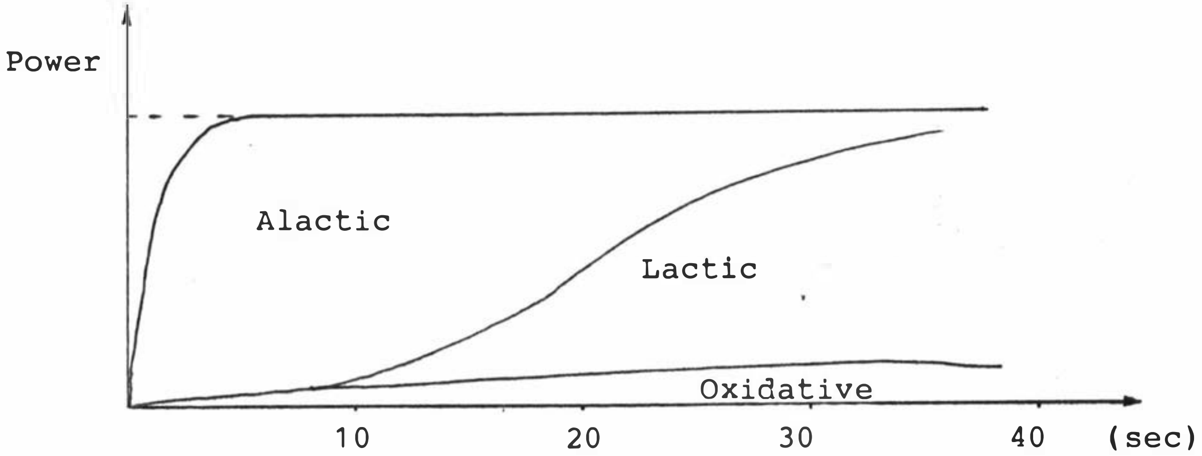
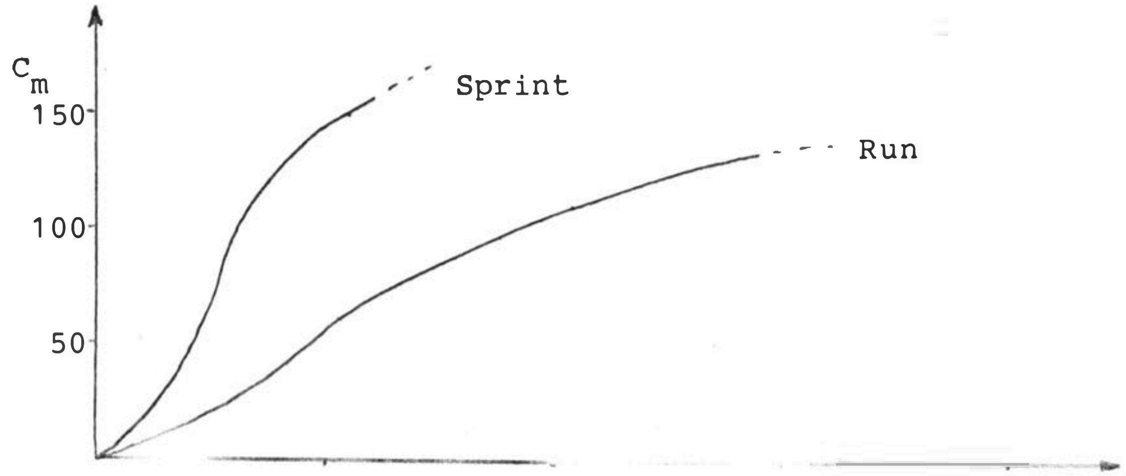


FIGURE 10.3: Power Components During Run



There remains the fault, though less marked, that the lactate concentration rises too soon (see previous page). There are several possible explanations:

(i) the initial phosphagen store is actually higher than 810 m M;

(ii) the mechanism for phosphagen cleavage is other than the modified law of mass action suggested;

(iii) the constant $k_2 = 1/2000$ for lactate removal is too low.

It is a simple matter to investigate (i) and (iii). This was done by setting the initial phosphagen store to 1200 m M, and/or setting $k_2 = 1/1500$. Two simulations were performed at maximal effort, and the output graphed in Figure 10.5, for comparison with Figure 10.4. Changing P_0 by + 50% had an effect in the right direction, but was insufficient. Changing k_2 by + 33% had a similar effect in the right direction, but its main effect was to lower the peak lactate.

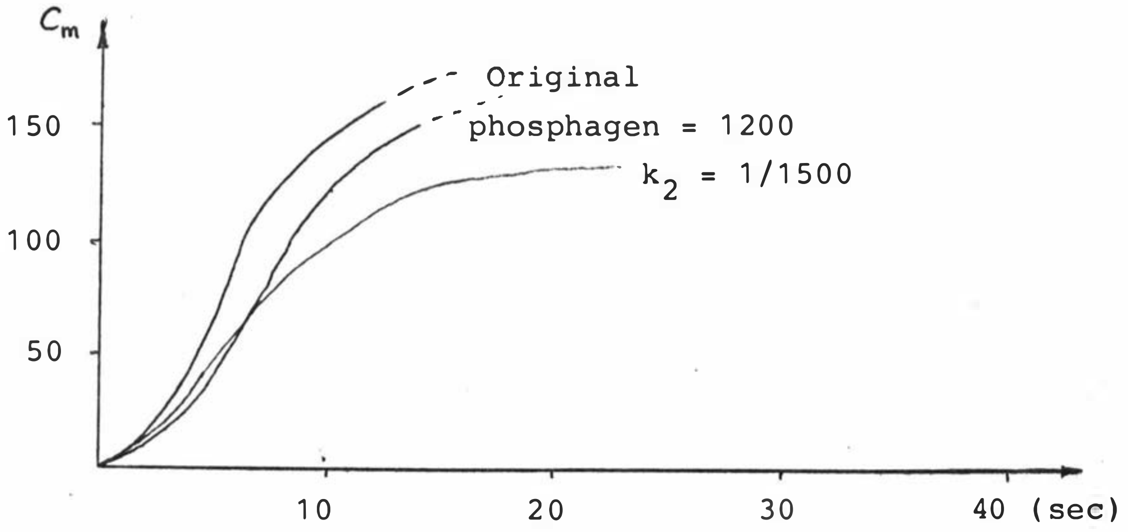


FIGURE 10.5: Muscle Lactate Concentrations

It is less simple to investigate (ii), for it is unclear what "law" to utilise. However a slightly different approach can be tried. Instead of modelling phosphagen, the lactate can be empirically modelled, to have a sigmoid shape of known form, similar to but steeper and to the left of those in Figures 10.2 and 10.3. The form of the lag is given by:

$$e^{-k_4/fvt}$$

and is applied to the former requirement of L_p and P_c, i.e. to $fv - 5.250_2$. It turns out that k_4 is quite large, of the order of 2×10^5. Some simulations of lactate concentration are attached as Figure 10.6, for comparison with Figures 10.4 and 10.5. These results are closer to reality, and k_4 could be adjusted further to this end.

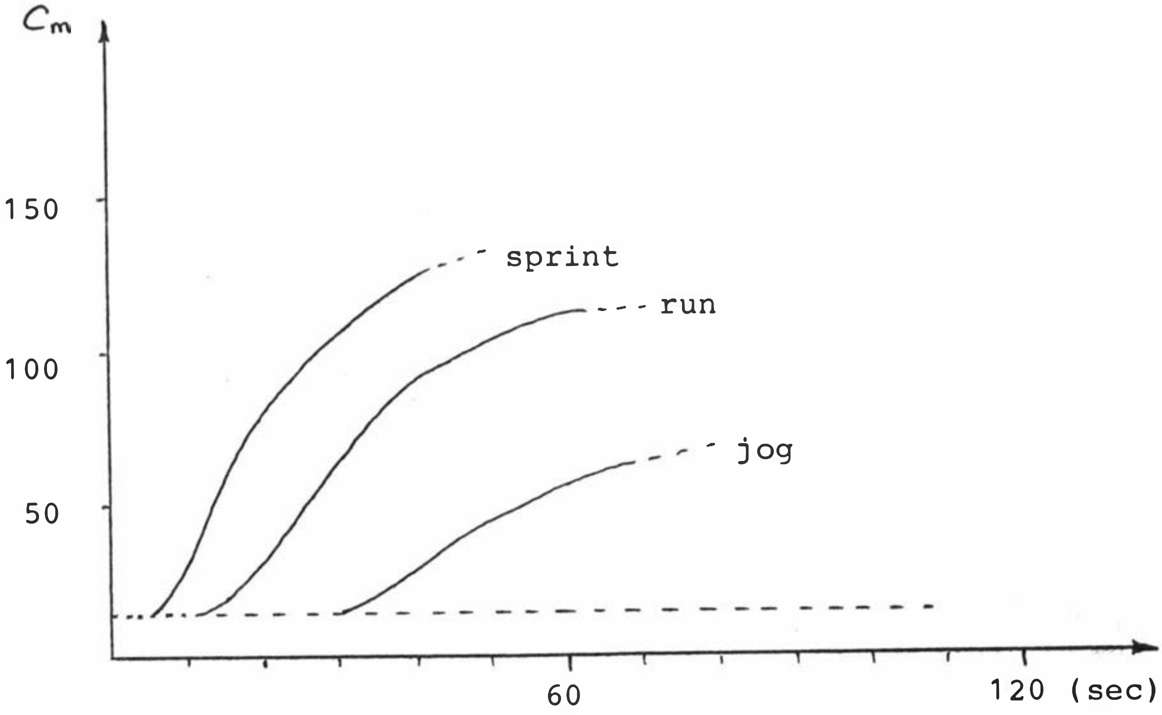


FIGURE 10.6: Muscle Lactate Concentrations

There is the danger however that ad hoc empirical modifications of this sort without knowledge of the biological basis, may give rise to a satisfactory solution, but which is trivial.

The next stage, which ought to resolve the above difficulties is to consider the two-compartment lactate circulation model, which has been discussed fully in Chapter 5, and to include earlier developments into the full model of Chapter 7.

THE NAG ROUTINE

The simulation of the whole model was performed using routine DO2PAF of the NAG FORTRAN Library of computer programmes (NAGFLIB , 1983). This routine advances the solution of a system of first-order ordinary differential equations:

$$Y'_i = F_i(T,Y, \ldots Y_n) \qquad i = 1 \; \ldots . \; n$$

over the range from T = X to T = XEND, with suitable initial conditions, using a number of steps of Merson's Runge-Kutta formula. The system of equations is defined by a subroutine and a variety of control options are available to the user. Full operating details are provided in the above reference.

Equations 7.1 - 7.9, together with initial values and other specifications, were prepared in accordance with Chapters 7 and 9. The resulting programme is FUMO1.F77, a copy of which is included in the accompanying computer output folder.

FUMO1.F77 was run with a wide variety of parameter settings, which, together with the results, are detailed in the next section. A selection of these results are included in the attached computer output folder also.

SIMULATION RESULTS

The first group of simulation results represent a preliminary check on the performance of the model. They comprise a period of rest (zero workload), eight minutes running (workload approximately 1200 watts), six minutes hard running (load approximately 1850 watts), two minutes maximum sustainable effort, and a period of ramp exercise.

At rest, the whole system is in stable equilibrium, as it should be, and there are no changes to any of the variable values.

Parameters for the equation of motion (and the variables with which it is associated) and the $\dot{V}O_2$ equation similarly were derived by curve fitting in Chapter 9, where good explanations of the data were achieved. That aspect of constant load work not considered so far is the lactate information. Observed blood lactates (averages over three subjects) at 1200 and 1850 are plotted together with simulated blood lactates in the following figure.

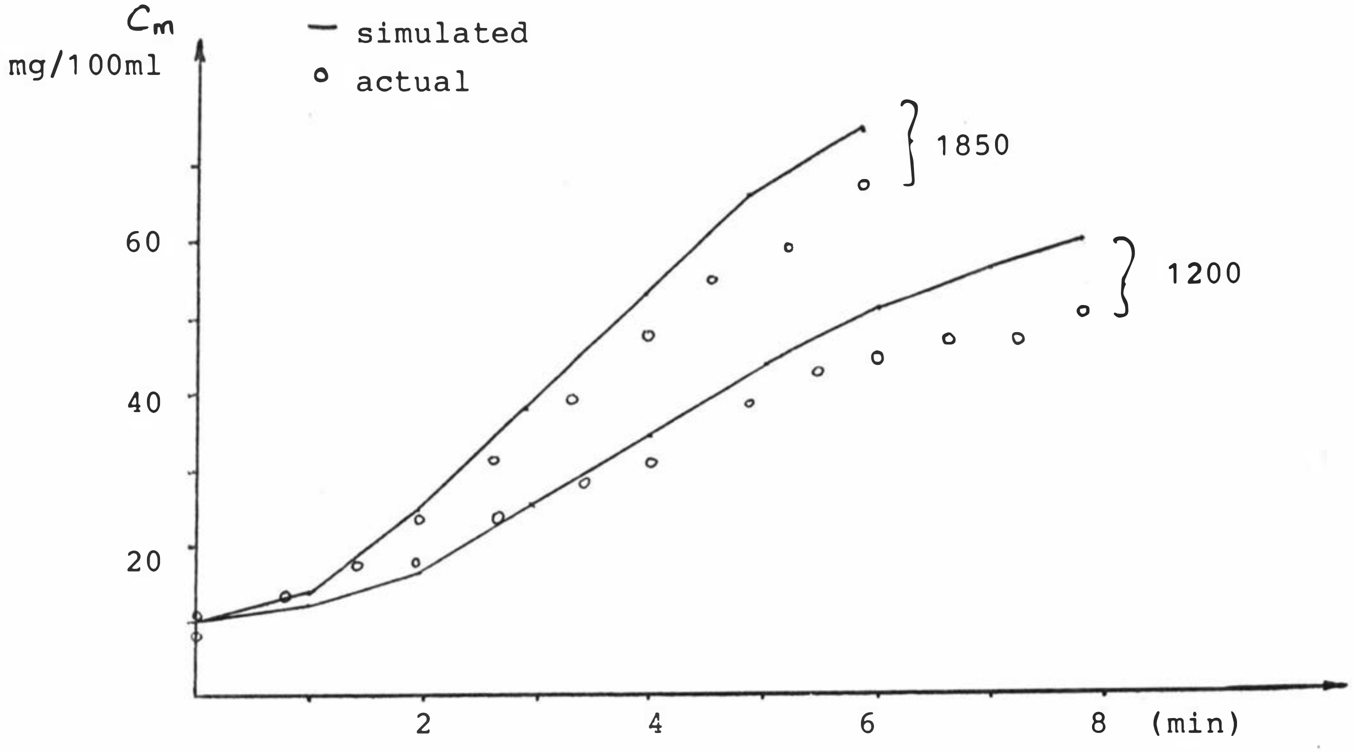


FIGURE 10.7: Simulated and Actual Blood Lactates

The general shapes are similar, but the actual lactates are below the simulated values. A comparison of simulated muscle lactates with the previously mentioned biopsy values indicate that actual muscle lactates are above simulated values. There are obvious reasons for this. The parameters of the two compartment model were obtained from published values of the original, and these may not suit the subjects of this study. Diffusion constants could be changed, and so could the compartment volumes. In so doing, simulated muscle lactates could be raised, and simulated blood lactates depressed, in such a way that a good match could be achieved. There appear to be no other estimates of these parameters available in the literature for the purposes of comparison, and so these ad-hoc adjustments were not made.

The all-out effort produces exactly the sort of power profile or velocity profile that is well-known to exercise physiologists, It is shown in the following figure.

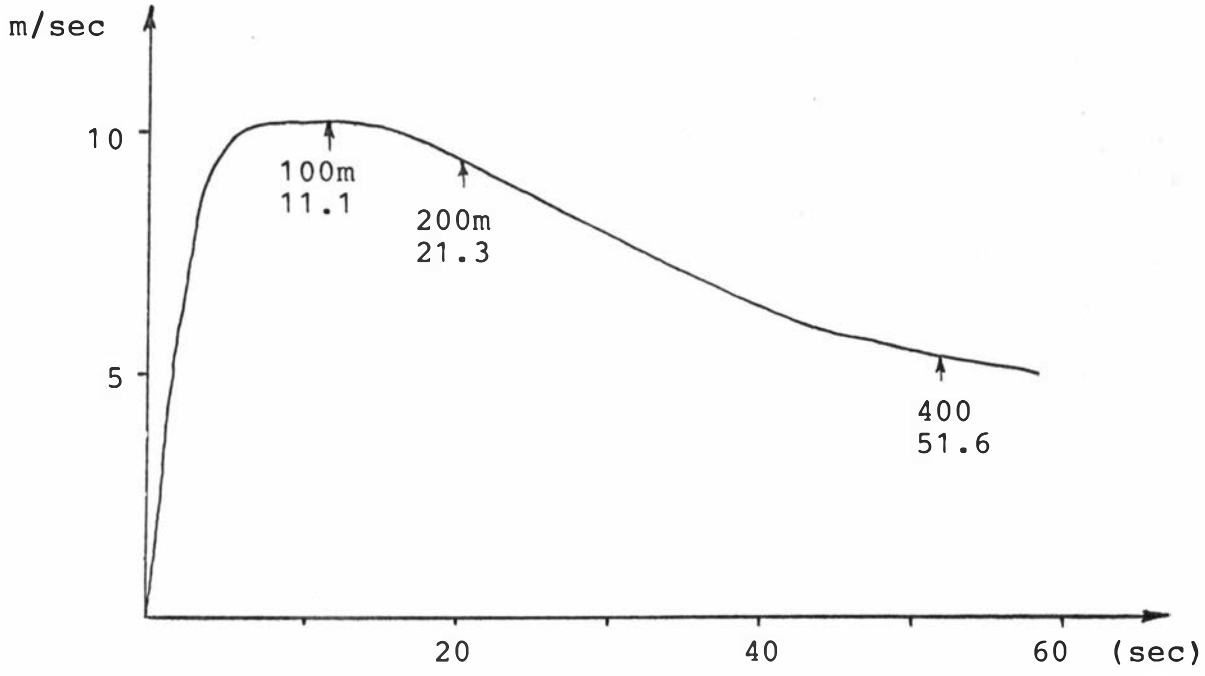


FIGURE 10.8: All-out Velocity Profile

More interesting is the ramp test, and its $\dot{V}O_2$ profile is plotted in Figure 10.9 below.

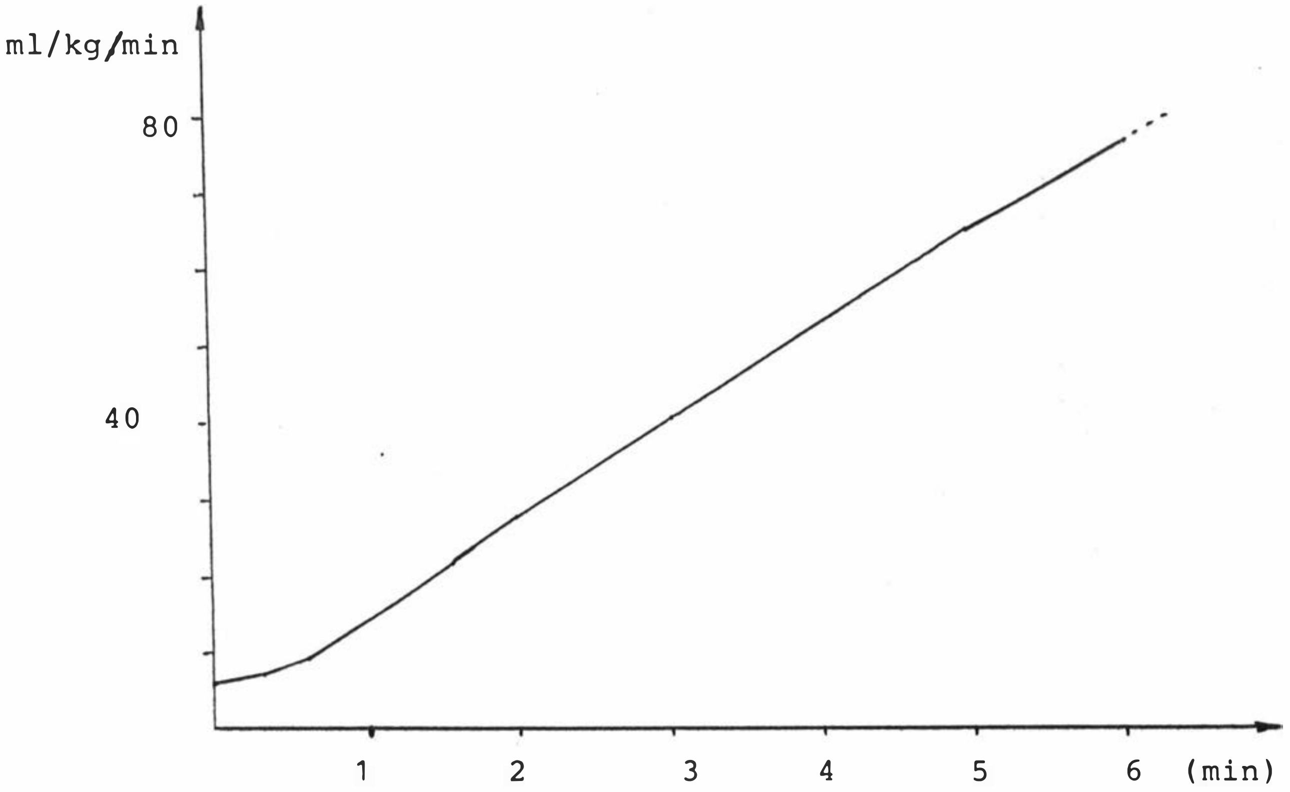


FIGURE 10.9: Ramp Test $\dot{V}O_2$

Once again there is a paucity of published data with which to compare the simulated $\dot{V}O_2$, but the pattern is as might be expected.

In general, this group of simulations has not lead to any real doubts about the model performance. They are included in the accompanying computer output folder.

The second group of simulation results represents a comprehensive investigation on the sensitivity of the model system to changes in parameter values. There are twenty-four parameters that could be examined. They are as follows:

Two energy stores, for the alactic and lactic energy processes,

Three energy equivalents, for the three energy components,

Two body compartment volumes,

Six parameters of the 2-compartment body model,

Two parameters of the fatigue feedback link,

Body mass, maximum force, resistance to motion coefficient,

Four VO_2 function parameters.

Two initial lactate concentrations.

This list can however be reduced considerably in the following manner:

(i) Since the biochemical energy stores can be converted to mechanical energy units by multiplication by the energy equivalents, this means that the lactic and alactic energy equivalents can be subsumed into the sizes of the original energy stores.

(ii) The volume of the lactate space compartment, V_s, in the two-compartment model can be regarded as a constant, since it is essentially a blood volume. It is V_m, the volume of exercising muscles which vary, not so much between individuals, but between types of exercise.

(iii) The production, diffusion and utilisation coefficients in the two-compartment model were obtained from Freund and Zouloumian (1981a) and not estimated in any way from collected data. Some doubt has been cast above on the magnitude of some of these, but without further evidence, they cannot realistically be changed, and are regarded as fixed.

(iv) Of the four $\dot{V}O_2$ parameters, only b' is really of relevance. The others, the resting level, a' and λ can be regarded as constant. b' corresponds fairly closely with the notion of $\dot{V}O_2$ max, considered to be the most important determinant of performance, and is therefore retained. The implication of this is that we can now also omit the energy equivalent of the oxidative process, since a higher equivalent has the same effect as a higher $\dot{V}O_2$ max.

This reduces the list of parameters which need further study to eleven.

The second group of simulations aims to identify which of these eleven variables are significant determinants of performance. Three aspects of performance were considered:

(A) 100m sprint time/sec
(b) Distance covered in 90 sec all-out run (metres)
(C) Endurance at 5m/sec constant velocity (mins)

The experimental design required for this type of variable screening procedure is one of the well known Plackett and Burman series, in this instance a 12-run design for screening eleven variables.

The eleven variables, with their nominated values, and chosen high and low values are as follows:

TABLE 10.2: Variables Investigated for Sensitivity

Variable	Name	(-)Low	Nominated	High (+)
A	Phosphagen store (mM)	810	1360	2000
B	Glycogen (lactate) store (g)	76	127	170
C	Working muscle volume (100 ml)	90	135	180
D)	Parameters of fatigue equation	.05	.08	.1
E)		+.001	+.002	+.003
F	Body mass (kg)	50	68	100
G	Maximum force (Newtons)	300	470	750
H	Resistance coefficient (kg/sec)	45	57	80
I	b' in Equation 7.9	.015	.025	.04
J	Initial muscle lactate (mg %)	8	10	30
K	Initial blood lactate (mg %)	8	10	15

The Plackett and Burman design is shown in the following scheme, together with the observations from RUN1A, RUN1B, RUN1C .... RUN12C in the attached computer output folder.

TABLE 10.3: Original Plackett and Burman Design

Run	A	B	C	D	E	F	G	H	I	J	K	Sprint (sec)	Dist (m)	Endurance (min)
1	+	+	-	+	+	+	-	-	-	+	-	17.05	545.9	9.2
2	+	-	+	+	+	-	-	-	+	-	+	16.21	526.0	6.9
3	-	+	+	+	-	-	-	+	-	+	+	27.25	329.2	0.0
4	+	+	+	-	-	-	+	-	+	+	-	7.15	810.5	12.4
5	+	+	-	-	-	+	-	+	+	-	+	28.01	330.0	0.0
6	+	-	-	-	+	-	+	+	-	+	+	11.91	487.4	1.05
7	-	-	-	+	-	+	+	-	+	+	+	8.72	716.3	20.0
8	-	-	+	-	+	+	-	+	+	+	-	27.66	335.7	0.0
9	-	+	-	+	+	-	+	+	+	-	-	16.27	367.7	.7
10	+	-	+	+	-	+	+	+	-	-	-	12.06	483.0	1.7
11	-	+	+	-	+	+	+	-	-	-	+	8.54	567.6	3.1
12	-	-	-	-	-	-	-	-	-	-	-	16.24	510.7	3.8

For each of the twelve selections, the three aspects of performance are considered. The first two can be obtained from a single simulation run, but the third aspect requires further attention.

The disadvantage of Plackett and Burman designs are that they are saturated, and of resolution III, that is, main effects are aliased with 2 factor interactions. The fold-over theorem however can be utilised to free this confounding and produce a resolution IV design. The corresponding scheme for the fold-over half of the simulation trials is as follows together again with the observations from RUN13A ... RUN24C in the attached computer output folder.

TABLE 10.4: Plackett and Burman Fold-over Design

Run	A	B	C	D	E	F	G	H	I	J	K	Sprint (sec)	Dist. (m)	Endurance (min)
13	+	+	+	+	+	+	+	+	+	+	+	12.12	576.2	20.0
14	+	-	-	+	-	-	-	+	+	+	-	27.13	336.5	0.0
15	-	+	-	-	+	-	-	-	+	+	+	16.11	517.6	3.7
16	+	-	+	-	-	+	-	-	-	+	+	17.19	575.6	6.3
17	+	+	-	+	-	-	+	-	-	-	+	7.36	597.9	3.5
18	+	+	+	-	+	-	-	+	-	-	-	27.46	321.9	0.0
19	-	+	+	+	-	+	-	-	+	-	-	17.27	554.7	20.0
20	-	-	+	+	+	-	+	-	-	+	-	7.85	517.0	2.0
21	-	-	-	+	+	+	-	+	-	-	+	28.64	293.8	0.0
22	+	-	-	-	+	+	+	-	+	-	-	8.43	783.3	20.0
23	-	+	-	-	-	+	+	+	-	+	-	12.60	491.2	1.8
24	-	-	+	-	-	-	+	+	+	-	+	11.66	495.2	2.6

The two halves of the experiment can be analysed separately, and the effect of each variable calculated. No analysis of variance is possible because of the saturated system, but the factors can be ordered in "significance" by the absolute magnitude of their effects. The foldover theorem does provide a "replicate" effect, which in this instance was merely a dummy effect, which can in a sense be used as a bench mark for the calculated effects.

An overall effect was calculated for each variable by adding the effects from each half. The following analysis resulted, (see page 140, Table 10.5).

100m Sprint Effects

With respect to the sprint times, it can be readily seen that the only significant effects are G: maximum force, which has a very strong negative effect (i.e. larger force means a shorter time) and H: resistance coefficient which has strong positive effect. That these are the only two seems hardly surprising. The only interaction indicated is the GH, with an effect of -37.5, which is probably just significant also.

Distance Effects:

As far as the all-out ninety second distance is concerned, the following four variables have obvious effects

H: resistance coefficient : -2374
G: maximum force : +1716
A: phosphagen store : + 678
I: "$\dot{V}O_2$ max" : + 628

A further four have effects which must be regarded as marginal.

F: body mass : +436
J: muscle lactate : +407
E:) : -390
D:) fatigue parameters : -382

H, G, A and I are much as expected, as are E and D, at least in sign, but F and J have sign opposite to expectation.

TABLE 10.5: Effects of all Investigated Variables on Responses

Variable Code	100m Effects (sec)			Distance Effects (m)			Endurance Effects (min)		
	P & B	Foldover	Overall	P & B	Foldover	Overall	P & B	Foldover	Overall
A	-12.3	5.6	- 6.7	356	322	678	3.65	19.7	23.35
B	11.5	-8.0	3.5	- 108	58	-50	- 8.05	18.1	10.05
C	0.7	-6.7	- 6.0	94	20	114	-10.65	21.9	11.25
D	-1.9	6.9	5.0	-73	-309	-382	18.15	11.1	29.25
E	-1.8	7.4	5.6	-349	- 41	-390	-16.95	11.5	- 5.45
F	7.0	-1.3	5.7	-53	489	436	9.15	56.3	65.45
G	-67.8	-73.8	-141.6	855	861	1716	19.05	19.9	38.95
H	49.4	45.4	94.8	-1343	-1031	-2374	-51.95	-31.1	-83.05
I	11.0	-8.4	2.6	162	466	628	21.15	52.7	73.85
J	2.4	-7.8	-5.4	440	-33	407	26.45	-12.3	14.15
K	4.2	-7.7	-3.5	-97	52	-45	3.25	-7.7	-4.45
Dummy	N/A	N/A	3.3	N/A	N/A	-51	N/A	N/A	21.05

The six indicated interactions between H, G, A and I have all been calculated. The three largest are:

GI = 582   HI = -559   GA = 489

In addition, seven others which intuitively might be of interest have also been examined, AB, BC, DE, DJ, EJ, FG and JK. The two largest are

BC = 505 and K = 376

Interaction effects generally are of much smaller absolute value than the main effects, and can thus be considered only as of interest, rather than significance.

Endurance Effects:

For endurance effects, running at a constant 5m/sec, which is a fairly brisk pace, there are some special considerations to note.

Firstly, since v is constant, we require df/dt to be zero, thus eliminating parameters D and E, except in as much as they affect the point of exhaustion. That is, the endurance test ends when that muscle lactate concentration is reached beyond which the force required to maintain 5m/sec cannot be exerted. This value of C_m must be determined in the four cases where D and E are low or high and low or high respectively. An interrupt must be inserted in FUMO1.F77 to cease calculation when muscle fatigue intervenes at these values. The values were extracted from among the previous twenty-four runs and used as appropriate as the interrupt.

Secondly, if muscle fatigue does not interrupt, the subject will run down his energy store until it is depleted fully. Thus an alternate interrupt is inserted to cease calculation when the glycolytic mechanism is empty of energy store. The programme will cease in fact whenever the first interrupt condition is satisfied.

Thirdly, when variable H (resistance constant) is high, a force of 400 newtons is required to reach 5m/sec, and this level cannot be achieved when variable G (maximum force) is at its low value. Thus 5m/sec cannot be reached in these six cases, and the endurance has been set to zero. (H is high and G is low in runs 3, 5, 8, 14, 18 and 21).

Lastly, since in some of the instances 5 m/sec can be achieved essentially aerobically, the endurance is very large (or even infinite). In such cases (usually when I: ($\dot{V}O_2$ max) is high and H (resistance constant) is low) the endurance has been set to 20 minutes, except in those cases where lactate tolerance was so low as to intervene beforehand.

The analysis table in respect of the endurance at 5m/sec reveals probably only the following real effects

H: resistance coefficient : -83.05
I: "$\dot{V}O_2$ max" : 73.85
F: body mass : 65.45
with G: maximum force : 38.95 as marginal.

The first two are as might be expected both in sign and magnitude, for we have previously seen the importance of H, and it has often been remarked in the sports science literature that $\dot{V}O_2$ max is one of the most important determinant of endurance running capability. The emergence of F is not altogether surprising, though one might have expected the sign to be negative, for distance runners are small in stature in comparison to sprinters.

The ten interactions indicated by the first five factors in the ranking were investigated also.

The largest four are IG = 51.25, FD = 50.15, IF = 41.95 and HI = -36.35. All must be regarded as marginal. (Note that the foldover dummy effect is itself 21.05).

A third group of simulations could be performed to push the model to the limits, that would be by selecting parameter values beyond the normal range. Those selected above are on the whole about the limits of the normal range. There is no doubt, judging by the indications above, that a variety of unrealistic simulations would have emerged. Thus this group has not been performed, for the effort would not be productive.

CHAPTER 11

CONCLUDING REMARKS

DISCUSSION

(i) Parameter Modification:

A number of simulations of the full model were **initially** required to get the whole system finely tuned. These fine tuning requirements resulted in minor modifications of some parameter values. The only changes implemented were for some of those parameters whose values had been "borrowed" from other sources. In the first instance, the initial body energy stores, both lactic and alactic, were raised above the value given in Margaria (1976). In various places in this study (e.g. Appendix 1 p.34), there were indications of this insufficiency and an increase of the order of 65% seemed to lead to about the right times for the known endurance capabilities of the subjects in the study. These raised levels were used as initial values for the body energy stores appropriate to equations 7.1 and 7.2. It was noted in Chapter 10 as a result of the simulation study that the system is not particularly sensitive to changes even of this magnitude in these two quantities. Thus a 65% rise does not have as great an effect as might be suspected. Margaria's original lower values were thus not the nominated figures for these parameters, but the lower ones listed in the sensitivity simulation study.

Secondly the question of the "equivalence factor" between the cycle ergometer workload and the power developed during running had to be resolved by fine tuning also. A factor of 1/3 to 1/4 had been indicated (p. 39/40), a figure inferred from various studies as an average among fairly wide ranging levels. For the subjects in this study it was found that a figure of 1/2 was much closer to the mark, and this value was used where applicable in the model equations. This figure of 1/2 corresponds more closely to the work of Fukunaga et al (1980) and the comments of Prothero (1974) in relation to Keller's (1973, 1974) formulation, than to some of the other studies investigated.

Thirdly, the fatigue feedback coefficients in the fatigue function derived in Chapter 6 were also modified as a result of fine tuning. These coefficients were originally chosen by empirical reasoning, so as to satisfy a basic constraint on the value of peak lactate. This value of 145 mg/100 ml (p.90) seems rather high for the subjects of this study, and a value around 110 mg/100 ml is more appropriate. Thus revision of these estimates towards the sorts of peak lactate and fatigue times recorded for the subjects resulted in those nominated values given in Chapter 10, and the higher and lower values used in the sensitivity simulations. There is the further relevant consideration that the form of the fatigue curve chosen may have been such that these changes became necessary. This choice was empirical, but it was exercised in good faith in the expectation that a reasonably realistic simulation would be achievable.

Lastly, a group of parameter modifications indicated during the fine tuning, but not undertaken, were some or all of the parameters of the two compartment lactate circulation model. With six parameters involved and with little or no physiological or experimental knowledge to make use of, the degrees of freedom for adjustment seemed too great. It would appear to be the case though, as mentioned in Chapter 10, that had such adjustments been made to some or all of the six parameters, some of the previously mentioned adjustments (e.g. fatigue equation parameters) might either not have been required, or need not have been made to the same extent.

(ii) Model Performance

Further fine tuning is possible clearly, but the marginal returns are diminishing. The performance of the model described in Chapter 10 had reached a stage where, with the nominated (estimated, borrowed or modified) parameter values, the simulated variable results were as close to real subject exercise data as could reasonably be expected.

Thus the first group of simulations were performed to assess the basic validity and realism of the model as a whole. Apart from examining these results in the accompanying computer output folder, and their discussion in Chapter 10, there is little further to note. Two small items which do come to mind for example however, are that in the simulation RUNFAST at a workload corresponding to 1850 kpm, exhaustion occurred at 6 min 40 sec, which is very close to the average endurance for the subjects on the cycle ergometer at this workload. Also, exhaustion in the ramp test occurred after about 7 mins, at a workload of around 2618 kpm which although not performed by the subjects, is probably an overestimate of their capability, especially with a predicted lactate of 160 mg/100 ml (itself an over-estimate), and since no fatigue feedback was allowed in that simulation.

The second group of simulations was designed for the two fold purpose of firstly testing the sensitivity of the system to changes in parameter values while at the same time ensuring that the resulting simulations remained realistic, and secondly for the purpose of considering boundary values, or those beyond normal physiological limits. Thus as presented in the previous Chapter with respect to the C group of simulations (endurance at 5 m/sec) these boundaries had been exceeded in a few instances, at both ends of the scale. At one end 5 m/sec was an unattainable speed, while at the other end, 5 m/sec could be maintained indefinitely. Thus the boundaries do seem to be fairly clearly indicated in respect of several of the parameters. Details of the sensitivity analyses are covered in Chapter 10.

In general these results do not lead to any unrealistic simulated outcomes, and the significant parameters that emerge from the analyses for the most part conform to expectations. The only exceptions seemed to be the perverse signs of the effects of F: body mass (+), and J: muscle lactate (+) in respect to the B and C simulation groups. These effects, if regarded as real (rather than marginal or not significant, which is a possibility), are difficult to explain. One possible explanation may be in the alternate view that several of the variables used in the screening experiment should perhaps have been taken on a relative rather than an absolute size. Thus for example the initial lactic and alactic body energy stores should have been measured on a per kilogram basis, and the absolute value adjusted according to the simulating of subjects of differing body masses. It is difficult to see how this suggestion might apply to muscle lactate, since it is already measured as a concentration, i.e. mg/100 ml. The explanation here might be the presence of an undetected or perhaps confounded interaction effect.

iii) Restructuring

Taking those remarks expressed during the development of the model as a whole (Chapters 3-6) together with the results detailed in Chapter 10, it is relevant to consider whether the model could be restructured in any way to improve its realism and perhaps its performance.

With respect to the Newtonian biomechanical model segment of Chapter 3, no restructuring is necessary, for Keller's original approach appears quite sound. The extension, adding a fourth equation for power developed, does raise the question of the measurement of mechanical power during running. There is debate in the literature, as has been discussed, and the calculated mechanical power does not seem to equate on a one-to-one basis with the calibrated power during cycle ergometry. There is therefore scope for further investigative work in this area to clarify the question.

The modelling of human bioenergetics is one segment of this runners model however which would benefit from restructuring based on a theoretical approach. Margaria's (1976) model was rejected, and while satisfactory equations can be empirically derived to describe the processes involved during exercise, they don't apply after the cessation of exercise. A generalisation of Margaria's model appears to currently offer the best scope for development of this model segment. However such a generalization will certainly be more complex than his original. Some ideas for such a development are presented in greater detail in Appendix 1, where the conclusion was reached that insufficient physiological information is currently available to make the generalisation with any reasonable degree of certainty.

The two compartment lactate circulation model seems to be basically quite sound in its originally proposed form. Its applicability in that form during exercise has not been directly tested, though the simulations in this study do not lead to any contradictory indications. Such direct testing ought not to be too difficult. Careful study of that model segment does suggest how an experiment could be designed to test the model validity during exercise, and to estimate its parameters. A time series of values on C_s (blood lactate) can be taken during exercise (and after), from which a time series of values on C_m can be imputed, from which in turn a time dependent function of lactate production in the working muscle during exercise can also be imputed. This may turn out to be a much better way of using blood lactate as an indirect means of estimating lactate production during exercise. (Some of the difficulties of this form of estimation were mentioned in Appendix 1, and there is no uniquely recognised standard or protocol. Researchers disagree as to the usefulness of such a procedure).

The major area for research and restructuring lies in the modelling of fatigue of the catabolite accumulation type. I am no more certain now of how to go about this, than at the start of Chapter 6. I do not feel that I know anything like enough about the biochemistry of fatigue to make any reasonable progress beyond that achieved in Chapter 6. I do feel confident however that a generalisation of Margaria's (1976) bioenergetic model will be able to incorporate some form of fatigue feedback based on the metabolite depletion principle. Likewise I feel confident that any fatigue mechanism based on lactate accumulation in the working muscle will exhibit the general behaviour assumed and modelled in Chapter 6. Apart from the sorts of approach adopted there, it is unclear how to do any substantial modelling of fatigue of this type.

CONCLUSION

The two broad conclusions to this study can be stated quite simply.

The project was embarked upon because of a quantitative interest in the analysis of sports and exercise and hence an area open to this sort of research was investigated. The project has been most interesting and has, I believe, made a contribution to the furthering of knowledge in modelling the human exercise processes. As is frequently the case in research projects, though many questions have been answered, the work itself has generated as many if not more further questions. This heightens the interest and the previous sections of this chapter have indicated various avenues where further development work can be undertaken. I expect to continue work in the immediate future by undertaking some of these developments.

Secondly, the project has achieved its objective of producing a working and realistic simulation model of a running athlete, by a combination of theoretical and empirical modelling. As a mathematician, it might have been preferrable in retrospect to have included more theoretical modelling, but the reasons for this not being possible are evident in Chapters 4 and 6. The empirical modelling of fatigue was unavoidable, since physiological knowledge of the processes involved is insufficient. The model as a whole was never intended to be THE answer. It is not so and the indications for restructuring have been discussed. Such restructuring will undoubtedly improve the model's theoretical content, though the simulated output will probably not reveal major changes except during the recovery after exercise, where a definite extension will have been made.

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Solution to Margaria's Model of the Energy Processes During Muscular Exercise

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Contents

ABSTRACT

In this monograph I examine a 3-compartment hydraulic model which has been proposed to represent the energy sources and flows during human muscular activity and recovery. Modelling work by exercise physiologists, in as much as it has utilised any mathematics and statistics, has been purely empirical. That is, there is an abundance of curve fitting, generally with no theoretical foundation. The only theoretical modelling approach to the bioenergetic processes is this hydraulic model, but it is only conceptual. Thus this monograph quantifies and solves the model mathematically, and illustrates the solution with a worked example using experimentally obtained data. In addition, implications of the model and its solution to maximum power, and to fatigue and exhaustion are considered. The model in part produces predictions consistent with observed results, and in part does not. It has some promise therefore, but needs further development.

1. Introduction

One can view the modelling of processes from two angles; the empirical and the theoretical. The former can be characterised by fitting curves to data, and while such curves may correspond well with experimental observation, their equations often have no theoretical foundation. The latter can be characterised by the use of differential equations based on theoretical principles, which though mathematically elegant, may have little suitability for application to experimental situations. One such process which illustrates these two angles is that of the energy processes of the human body during muscular activity. Exercise physiologists have taken to the empirical angle in large numbers, but mathematicians it appears have not taken to the theoretical angle in even quite small numbers. There is thus a noticeable gap to be filled, and the purpose of this paper is to assist in filling that gap.

The sources of energy for muscular activity in the short to medium term, are very well known. Without getting involved in the biochemical details, they are broadly as follows. The most obvious is the oxidative energy source, and is characterised by an increased oxygen consumption in response to exercise. This response is quite slow, for it can take up to two minutes after the onset of exercise for a steady state to develop. The oxidative source is of course not limited, though the rate of utilisation is. The second source is the glucose → glycogen → lactic acid mechanism, called the lactic or glycolytic source. It is the latter conversion that is of major interest, for the lactic acid is a harmful by-product. It tends to accumulate in the muscle tissue, where, if not removed fast enough by diffusion into the bloodstream to be broken down, it will cause muscular poisoning; the fatigue pains well-known to athletes. This mechanism is a limited source, and also has a maximal rate of utilisation, which is higher than the maximal oxidative rate. The third, and most important source, since it is available on demand, is provided by the conversion of high-energy phosphate compounds. This is referred to as the phosphagen or alactic source. The by-products of this conversion are not harmful, and are recycled by the oxidative and/or glycolytic mechanisms. This source is limited in quantity and rate of utilisation also, though the maximal rate is quite large.

The usual investigative approach to these matters taken by exercise physiologists, is to exercise subjects at a known workrate (i.e. power) on a cycle ergometer or treadmill. The total rate of energy usage is therefore known. The subject breathes through tubes which measure the oxygen consumption, as well as other respiratory parameters. The energy equivalent, and the biomechanical efficiency of the oxidative mechanism is known and so the oxidative component of the workload can be calculated, and the sum of the other two components determined by subtraction. The modelling procedure is to fit a curve to the oxygen consumption over time. Historically this was first done about 30 years ago (Henry, 1951) and the curve fitted very well. Countless equally good, or better, fits have been obtained since (Linnarson, 1974; Fox et al, 1980; Whipp et al, 1981) and this model is now in such widespread usage that it appears to be taken as fact. Of relevance to the mathematician is that it never had, nor has it since been given, any published independent theoretical basis.

During light to moderate exercise, below what is referred to as the "anaerobic threshold", it has been established that the glycolytic mechanism does not contribute to the energy supply. This is ascertained by inserting a small needle in a vein and drawing small blood samples every minute or so during exercise. The blood is analysed for the presence of lactic acid; none above normal resting levels having been found in such circumstances. Thus any difference determined above by subtraction, can be attributed to only one source, the alactic, and so the empirical modelling can be done.

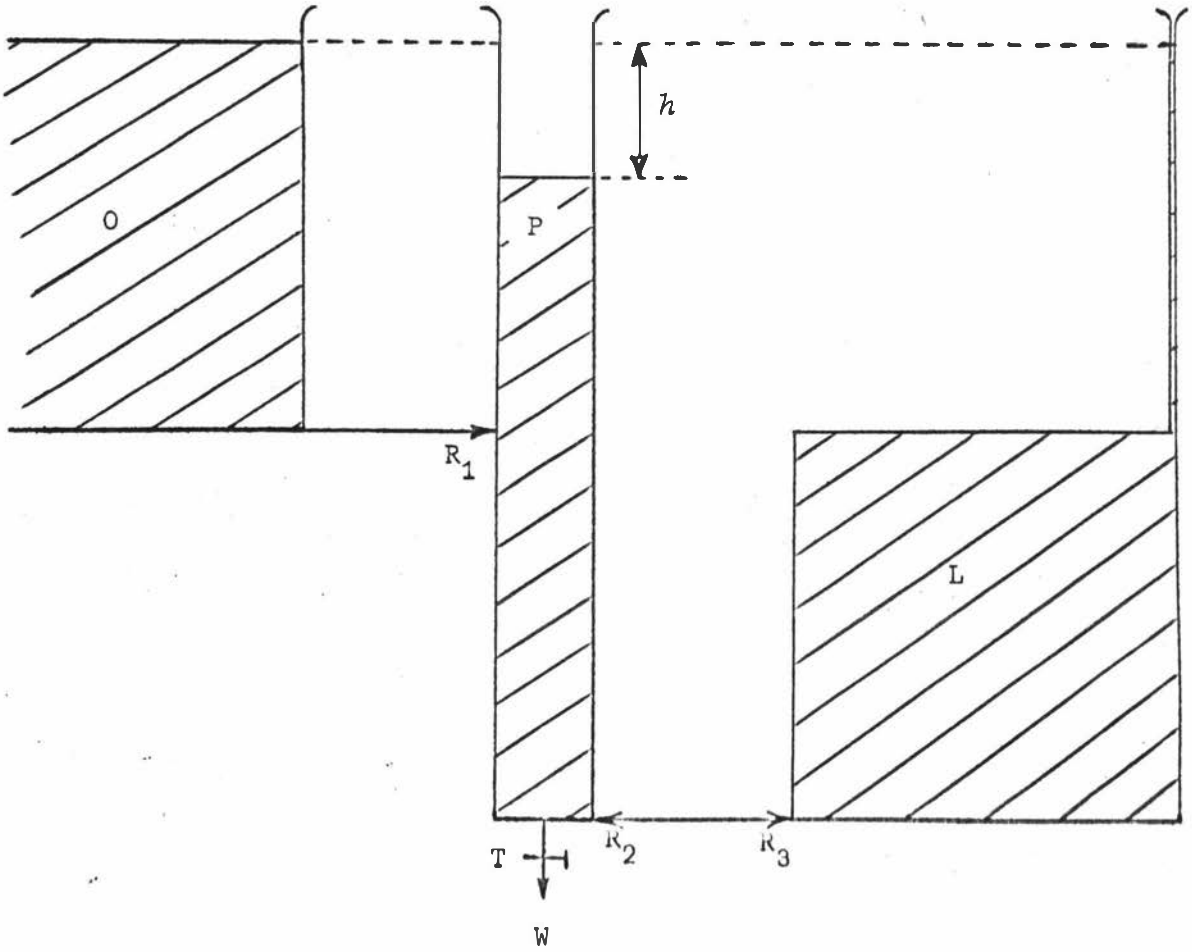
In heavy exercise, all three mechanisms contribute, and the difference referred to has both lactic and alactic components. It might be thought that measuring blood lactic acid as described would provide one of the two, and the alactic component derived by a second subtraction. Here we run into a number of difficulties; there is a well-established but quite variable time-lag between muscle and blood lactic acid levels; blood flows around the body in a complex though known, manner; where should the needle be inserted, etc. For these reasons and others, blood measurements though frequently taken, do not provide a very useful indication of the rate of lactic acid production in the working muscle. The muscular concentration can be accurately determined, by a needle biopsy, that is by inserting a special needle into the muscle tissue and withdrawing, by powerful suction, a small piece of the tissue itself for subsequent analysis. Clearly this process cannot be done repetitively, nor while the muscle is actually in use.

Thus physiologists have done a lot of work on the oxidative mechanism and because of these difficulties much less on the other two. As a means of progress therefore, it would seem that the theoretical angle might now be useful, but as I have mentioned, I have found very little such work. The only attempt to comprehensively model the processes involved is the hydraulic model, postulated by Margaria (1976). This model is only descriptive, with no detailed attempt to quantify it, and none to solve it analytically. No others it appears have made such attempts either. Specifically then, the purpose of this paper is to undertake these tasks.

2. Margaria's Model

The hydraulic model is shown diagramatically in Figure 1 below:

Figure 1: Margaria's Hydraulic Model, Phase 1A



The fluid in vessel P (representing phosphagen) is directly connected with the outside through the tap T, which regulates the flow (total energy expenditure). At rest, with T closed, the upper level of fluid in P is the same as in the communicating vessel 0 (representing the oxidative source). The vessel 0 is of infinite capacity and is connected through tube R_1. The second communicating vessel L (representing the glycolytic source) is of finite capacity, with upper level the same as the bottom level of vessel 0, apart from a very narrow extension tube, B. The fluid in B, corresponding to the resting blood lactic acid, is of very small volume relative to L, and does not contribute to any flows in a measurable amount. L is connected to P through a wider, but one-way tube R_2, and P is connected to L by another, but very much smaller one-way tube, R_3.

If T is partly opened, corresponding to a workload W, the level in P falls, inducing a flow through R_1 (oxygen consumption, VO_2) in accordance with the difference in levels, h, between the two vessels. This induced flow slows the rate at which the level in P falls, and provided W is not too large, an equilibrium will be reached at a level above the outlet R_1. The level in P is below the resting level, and fluid flows continuously from 0 to P and out through T. If the equilibrium level is exactly at the level of R_1, then the oxidative mechanism is at its maximum, denoted VO_2 max. Once the equilibrium is established, the only energy mechanism contributing is the oxidative; the exercise is purely aerobic, and in theory could continue indefinitely. Prior to equilibrium of course, P has contributed some of its supply, and the empty volume in P above the equilibrium level is known as the alactic oxygen debt.

If T is now closed, i.e. exercise ceases, P will begin to refill through R_1, but at a slower and slower rate as the level in P returns to normal. When it does so, the flow in R_1 ceases and the subject is said to have repaid his oxygen debt during this recovery period.

If T had been widely opened, (severe exercise), the initial situation would be as described above, but the level in P would fall below R_1. This happens after about 50% of the fluid in P has been utilised, and the subject is said to have crossed his anaerobic threshold. As soon as this happens, two things occur; the flow in R_1 has reached and continues at its maximum determined only by the height of the vessel 0; and a flow through R_2 is induced. This flow is in accordance also with the difference in levels between vessels L and P, (the level in L lagging behind the level in P). The flow through R_2 will slow the fall of level in P, but since the flow through R_1 is insufficient and the capacity in L is limited, the levels in both L and P will continue to fall. If exercise is prolonged, L and P will be emptied and the subject will be exhausted!

If T is closed at or before exhaustion, P will again be refilled. Initially it will be filled through R_1 at the maximal rate, and through R_2 until the lag in levels between L and P has been eliminated. This latter flow is a delayed lactic acid formation which has been experimentally observed to occur after cessation of exercise. Once the levels have been equated, P will fill through R_1, initially at the maximal rate and thereafter at a progressively slower rate as described previously. L will be refilled from P through R_3 at a rate in accordance with the difference in levels between the two. Because R_3 is so small, the level in L will lag behind the level in P; the repayment of this the lactic oxygen debt is very slow. Finally both P and L are refilled, and the subject is fully recovered!

3. Quantification and Solution

Figure 1 showed the measurements required to quantify the structure of the hydraulic model. Their units of measurement will be omitted until Section 4. The only assumption is the arbitrary one of convenience, that the height of vessel P is two units, and thus the height of both vessels 0 and L is one unit each. In addition the following are required to quantify the operation of the model:

maximal flow through R_1 = VO_2 max = M_0
maximal flow through R_2 = M_L
maximal flow through R_3 = M_R
cross-sectional area of P = A_P
" " " of L = A_L
volume of vessel P = $2 A_P$
" " L = A_L
anaerobic threshold = $\theta = M_0$

It is clear that a single differential equation, of conservation of energy, referred to as the equation of energy balance, is applicable to the model at all times. That is

$$W = VO_2 - \frac{dP}{dt} - \frac{dL}{dt} \qquad \text{- - - - - - - - - - -} \qquad ①$$

and it is this equation, in a variety of forms, which will be investigated. (P and L denote the volumes of fluid in vessels P and L respectively).

3.1 Phase 1A: Consider, as is shown in Figure 1, that shortly after T has been opened corresponding to a constant workload of W, the level in vessel P has dropped by a height h. The form of equation ① is now given by:

$$W = M_0 h + A_P \frac{dh}{dt} - 0 \tag{②}$$

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

$$h = \frac{A_P}{M_0}\left(\frac{W}{A_P} - e^{-\frac{M_0}{A_P}(t+c)}\right) \tag{③}$$

where c is an arbitrary constant 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

$$h = \frac{W}{M_0}\left(1 - e^{-\frac{M_0}{A_P}t}\right), \text{ for } t \geq 0 \tag{④}$$

Thus if $W \leq \theta = M_0$, then for large t, we see that an equilibrium level is reached with $h = W/M_0 \leq 1$. Of more interest to the physiologists is VO_2, which, using equations ①,② and ④, is given by

$$VO_2 = W(1 - e^{-\frac{M_0}{A_P}t}), \text{ for } t \geq 0 \tag{⑤}$$

which, provided $W \leq \theta = M_0$, tends to $W \leq M_0$ at equilibrium. That is, the workload is completely aerobic. The alactic debt at equilibrium is given by an amount $A_P W/M_0$. It is of interest to note that equation ⑤ is of identical form to the one previously mentioned as having been in very common use by physiologists for many years. Of interest also is the fact that the exponential rate constant M_0/A_P is independent of W, a subject of controversy among physiologists, (Whipp and Wasserman, 1972).

<u>3.2: Phase 1R:</u> Suppose now at time $t = t_1$, when $h = h_1 \leq W/M_0$ and $VO_2 = V_1 \leq W$, as given by the solutions of equations ④ and ⑤, we change W to W_1. Equations ② and ③ still apply but with W changed to W_1, and initial conditions $h = h_1$ or $VO_2 = V_1$ at $t = t_1$ respectively. Of most interest to physiologists is the particular solution for VO_2 when $W_1 = 0$, i.e. exercise has ceased and recovery commenced. This solution is given by

$$VO_2 = V_1 e^{-\frac{M_0}{A_P}(t-t_1)}, \text{ for } t \geq t_1 \qquad \text{- - - - - - - - -} \qquad \textcircled{6}$$

Again it is of interest to note that equation ⑥ is of identical form to the one which has been in common use by physiologists for the repayment of the oxygen debt, (Henry, 1951; Leger et al, 1980), for just as many years. The rate constant is independent of W and is the same value as in Phase 1A, as has been observed experimentally (Henry and De Moor, 1956). Phases 1A and 1R are shown diagramatically as part of a worked example in Section 4.

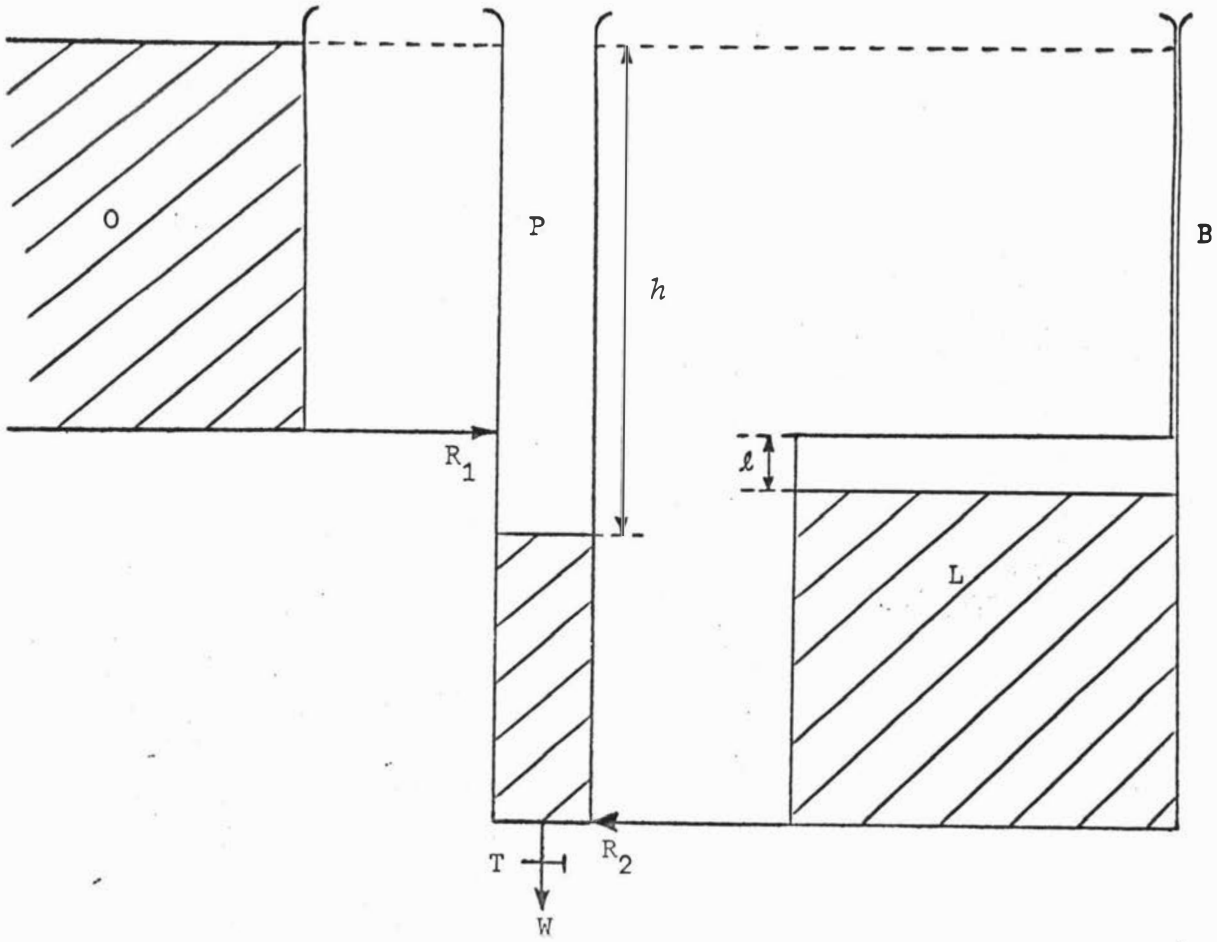
<u>3.3: Phase 2A:</u> Suppose that tap T was opened wide at the start, i.e. opened to some constant $W > \theta = M_0$. During Phase 1, equations ②, ③, ④ and ⑤ will apply, with appropriate initial conditions, but only up until some time t_2, when $h = 1$ and $VO_2 = M_0 = VO_2$ max. This time t_2 is obtained as the solution of equations ④ or ⑤ by putting $h = 1$ or $VO_2 = M_0$ respectively, viz

$$t_2 = -\frac{A_p}{M_0} \ln\left(1 - \frac{M_0}{W}\right) \qquad \text{- - - - - - - - - - -} \qquad \textcircled{7}$$

which is only defined for $W > M_0$.

Then consider the situation when $h > 1$, as shown below in Figure 2, but in addition we must consider the level in vessel L to have dropped say by an amount ℓ, where, due to the lag, $h > \ell + 1$.

Figure 2: Model Phase 2A



Equation ① now becomes:

$$W = M_0 + A_P \frac{dh}{dt} + A_L \frac{d\ell}{dt} \qquad \text{- - - - - - - - - - -} \qquad ⑧$$

and we note also that

$$A_L \frac{d\ell}{dt} = M_L(h-1-\ell)$$

since the flow in R_2 is in accordance with the difference in levels between vessels L and P.

i.e.
$$h = \frac{A_L}{M_L}\,\frac{d\ell}{dt} + 1 + \ell \qquad \text{- - - - - - - - - - - - -} \quad \textcircled{9}$$

and
$$\frac{dh}{dt} = \frac{A_L}{M_L}\,\frac{d^2\ell}{dt^2} + \frac{d\ell}{dt} \qquad \text{- - - - - - - - - - - - -} \quad \textcircled{10}$$

Now substituting equation ⑩ into equation ⑧ and simplifying, yields an appropriate version of equation ①, viz:

$$\frac{d^2\ell}{dt^2} + \frac{M_L(A_P+A_L)}{A_P\,A_L}\,\frac{d\ell}{dt} = \frac{M_L(W-M_0)}{A_P\,A_L} \qquad \text{- - - - - - - - -} \quad \textcircled{11}$$

This is a second order linear differential equation with constant coefficients, which has as its general solution

$$\ell = -\frac{c_1 A_P\,A_L}{M_L(A_P+A_L)}\,e^{-\frac{M_L(A_P+A_L)}{A_P\,A_L}t} + \frac{W-M_0}{A_P+A_L}t + c_2 \qquad \text{- - - -} \quad \textcircled{12}$$

with the two arbitrary constants c_1 and c_2, which can be determined by the initial conditions that at $t = t_2$, $\ell = 0$ and $d\ell/dt = 0$. This gives:

$$\ell = \frac{A_P\,A_L(W-M_0)}{M_L(A_P+A_L)^2}\left[e^{-\frac{M_L(A_P+A_L)}{A_P\,A_L}(t-t_2)} - 1\right]$$

$$+ \frac{W-M_0}{A_P+A_L}(t-t_2), \quad \text{for } t \geq t_2 \qquad \text{- - - - - - - - -} \quad \textcircled{13}$$

We note that as $t \to \infty$, so also does ℓ. That is, if severe exercise is prolonged, the vessel L will become empty, whereupon the subject can no longer call on the glycolitic energy source component. The question of when this occurs will be deferred for the moment, for it is not simply the solution of the transcendental equation ⑬ when $\ell = 1$.

Because of the lag in ℓ behind h, we must first consider when P becomes empty, because then equation ⑧ no longer holds and therefore neither does equation ⑬.

Thus let us consider the other energy sources in Phase 2A also, the behaviour of VO_2 and of h. The former is simple, it is constant at $VO_2 \max = M_0$. The latter can be obtained from equations ⑨ and ⑬, which yield, for $t \geq t_2$:

$$h = \frac{A_L^2(W-M_0)}{M_L(A_P+A_L)^2}\left[1 - e^{-\frac{M_L(A_P+A_L)}{A_P A_L}(t-t_2)}\right] + \frac{W-M_0}{A_P+A_L}(t-t_2) + 1 \qquad \text{- - - - - - - - - -} \quad ⑭$$

and we note that as $t \to \infty$, so also does h, and so vessel P will also become empty if exercise is prolonged. Just when this occurs must be deferred for it is not simply the numerical solution of the transcendental equation ⑭ for $h = 2$. We must consider the very real possibility that exhaustion may intervene beforehand. Such matters are discussed later, in Section 6.

<u>3.4: Phase 2R1</u>: However, suppose at time t_3, prior to exhaustion or to the emptying of vessels P or L, the workload is changed to W_3. At this time $\ell = \ell_3 < 1$ and $h = h_3 < 2$ obtainable from equations ⑬ and ⑭ respectively. Figure 2 and equation ⑫ will still apply for ℓ, with $W = W_3$ and appropriate initial conditions.

As before, of most interest to physiologists is the recovery of the subject, when $W_3 = 0$. At first equation ⑫ holds of course, with $W = 0$ and appropriate initial conditions, but only up until a time t_4, when the levels in vessels P and L equate. Vessel P of course starts to fill at t_3, but L will continue to empty until t_4, and this is the delayed lactic acid formation referred to earlier. Let us therefore consider this phase between t_3 and t_4. Equation ⑫ now becomes

$$\ell = -\frac{c_1 A_P A_L}{M_L(A_P+A_L)} e^{-\frac{M_L(A_P+A_L)}{A_P A_L} t} - \frac{M_0}{A_P+A_L} t + c_2 \quad \text{- - - - - -} \quad \text{⑯}$$

The first initial condition is that $\ell = \ell_3$ at $t = t_3$ as mentioned above. The second is that the derivative $d\ell/dt$ is continuous also at t_3, i.e. the derivative of equation ⑬ at $t = t_3$ equals the derivative of equation ⑯ at $t = t_3$. These two conditions lead to

$$\ell = \frac{A_P A_L (W-M_0)}{M_L(A_P+A_L)^2} e^{-\frac{M_L(A_P+A_L)}{A_P A_L}(t-t_2)} - \frac{W A_P A_L}{M_L(A_P+A_L)^2} e^{-\frac{M_L(A_P+A_L)}{A_P A_L}(t-t_3)}$$

$$+ \frac{W(t_3-t_2) - M_0(t-t_2)}{A_P + A_L} + \frac{M_0 A_P A_L}{M_L(A_P+A_L)^2} \quad \text{- - - - - - - -} \quad \text{⑰}$$

Hence from equations ⑨ and ⑰ we obtain

$$h = \frac{W A_L^2}{M_L(A_P+A_L)^2} e^{-\frac{M_L(A_P+A_L)}{A_P A_L}(t-t_3)} - \frac{(W-M_0)A_L^2}{M_L(A_P+A_L)^2} e^{-\frac{M_L(A_P+A_L)}{A_P A_L}(t-t_2)}$$

$$+ \frac{W(t_3-t_2) - M_0(t-t_2)}{A_P + A_L} - \frac{M_0 A_L^2}{M_L(A_P+A_L)^2} + 1 \quad \text{- - - - -} \quad \text{⑱}$$

During this time of course $VO_2 = VO_2$ max $= M_0$, and this, together with equations ⑰ and ⑱ describes the behaviour of the system between $t = t_3$ and $t = t_4$.

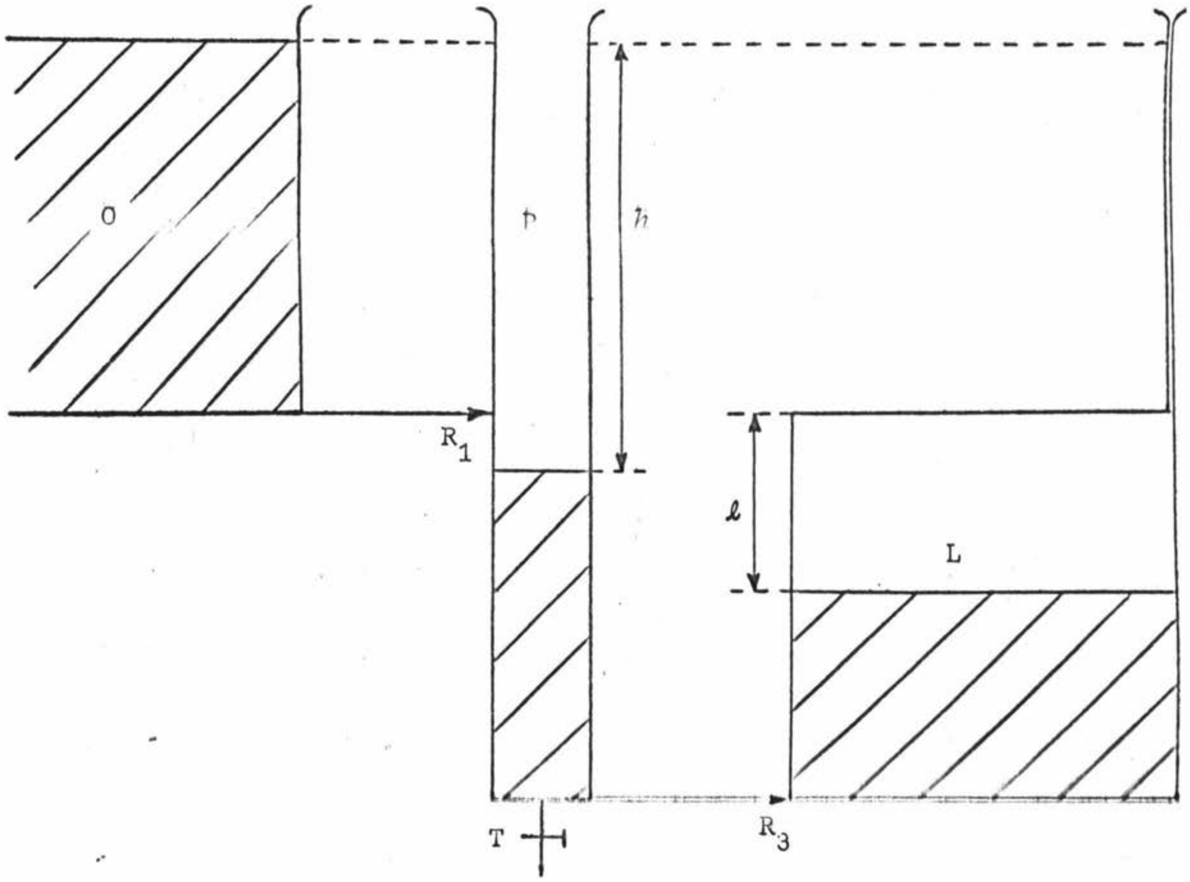
At $t = t_4$, the levels in L and P equate and the flow in R_2 will stop. Thus t_4 can be found as the solution for t of the equation $\frac{d\ell}{dt} = 0$ from equation ⑰. Hence we can determine the levels ℓ_4 and h_4 of the fluids in vessels L and P at time $t = t_4$. Of course $h_4 = \ell_4 + 1$ at this time.

The time $t = t_4$ marks the boundary between Phase 2R1 and the next, Phase 2R2.

3.5: Phase 2R2:

Once time t_4 has passed, the flow in R_2 has ceased, and a reverse flow, in R_3, has commenced, the lactic oxygen debt is now being repaid. Figure 3 below is appropriate.

Figure 3: Model Phase 2R2



During this phase the appropriate version of equation ① is

$$0 = M_0 + A_P \frac{dh}{dt} + A_L \frac{d\ell}{dt} \quad \text{- - - - - - - - - - -} \tag{19}$$

and we note that $A_L \frac{d\ell}{dt} = -\frac{M_R}{2}(\ell+1-h)$

i.e. $$h = \ell + 1 + \frac{2A_L}{M_R}\frac{d\ell}{dt} \quad \text{- - - - - - - - - - -} \tag{20}$$

and $$\frac{dh}{dt} = \frac{d\ell}{dt} + \frac{2A_L}{M_R}\frac{d^2\ell}{dt^2} \quad \text{- - - - - - - - - - -} \tag{21}$$

Hence from equations ⑲ and ㉑ we obtain

$$\frac{d^2\ell}{dt^2} + \frac{M_R(A_P+A_L)}{2A_P A_L}\frac{d\ell}{dt} = -\frac{M_0 M_R}{2A_P A_L}$$

which is another second order differential equation, with a general solution given by

$$\ell = -c_1 \frac{2A_P A_L}{M_R(A_P+A_L)} e^{-\frac{M_R(A_P+A_L)}{2A_P A_L}t} - \frac{M_0}{A_P+A_L}t + c_2$$

The two initial conditions requiring to be satisfied are that at $t = t_4$, we have $\ell = \ell_4$ and $\frac{d\ell}{dt} = 0$. These lead to the particular solution given by

$$\ell = \frac{2M_0 A_P A_L}{M_R(A_P+A_L)^2}\left[1 - e^{-\frac{M_R(A_P+A_L)}{2A_P A_L}(t-t_4)}\right] - \frac{M_0}{A_P+A_L}(t-t_4) + \ell_4 \tag{22}$$

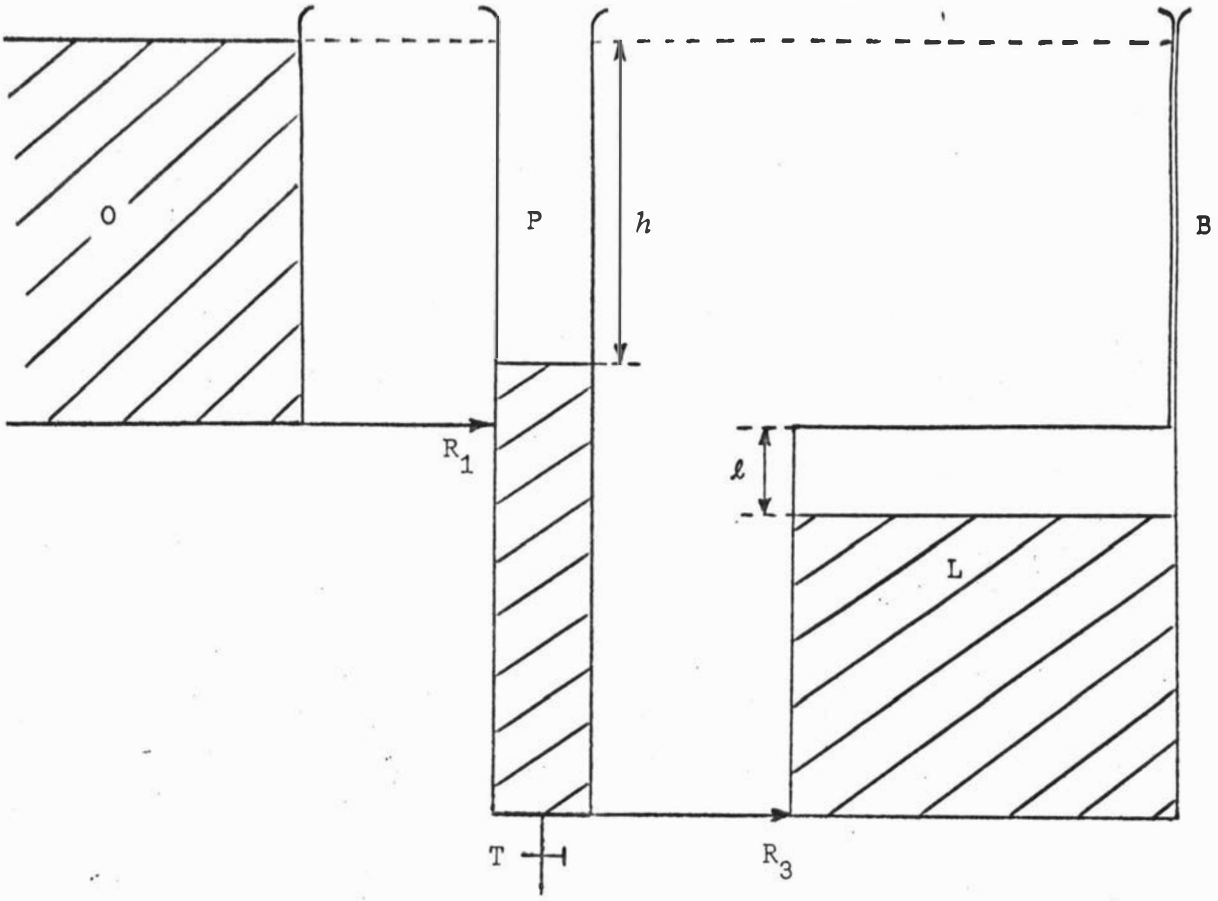
Hence from equations ⑳ and ㉒ above we can obtain

$$h = \frac{2A_L^2 M_0}{M_R(A_P+A_L)^2}\left[e^{-\frac{M_R(A_P+A_L)}{2A_P A_L}(t-t_4)} - 1\right] - \frac{M_0}{A_P+A_L}(t-t_4) + h_4 \tag{23}$$

These two equations describe the behaviour of ℓ and h, i.e. the levels in vessels L and P respectively during the second phase of recovery after strenuous exercise. During this time of course VO_2 is constant at $VO_2 \max = M_0$. This state of affairs will not last long, only until $h = 1$.

3.6: Phase 21R1: Suppose the previous phase ended at $t = t_5$ when $h = 1$, obtainable from equation (23) above. Once t_5 has passed, the following, Figure 4, is appropriate.

Figure 4: Model Phase 21R1



The appropriate version of equation ① in this phase is given by

$$0 = M_0 h + A_P \frac{dh}{dt} + A_L \frac{d\ell}{dt} \qquad \text{- - - - - - - - - - -} \qquad ㉔$$

and we note that equations ⑳ and ㉑ still hold, which together with equation ㉔ yields

$$\frac{d^2\ell}{dt^2} + \frac{M_R(A_P+A_L) + 2M_0 A_L}{2\ A_P\ A_L}\ \frac{d\ell}{dt} + \frac{M_0 M_R}{2A_P\ A_L}\ \ell = -\frac{M_0 M_R}{2A_P\ A_L}$$

This equation can be solved for ℓ utilising the following details:

(i) Auxiliary equation: $r^2 + \frac{M_R(A_P+A_L)+2\,M_0A_L}{2\,A_P\,A_L}\,r + \frac{M_0\,M_R}{2\,A_P\,A_L} = 0$ - - - ㉕

having two negative roots r_1 and r_2.

(ii) Complimentary function: $\ell_c = c_1\,e^{r_1 t} + c_2\,e^{r_2 t}$, with c_1 and c_2 arbitrary constants

(iii) Particular integral by inspection: $\ell_p = -1$

(iv) General solution $\ell = c_1 e^{r_1 t} + c_2 e^{r_2 t} - 1$ - - - - - - - - ㉖

Hence using equation ⑳ also, we can obtain

$\hbar = c_1 e^{r_1 t}\left(1 + \frac{2r_1\,A_L}{M_R}\right) + c_2 e^{r_2 t}\left(1 + \frac{2r_2\,A_L}{M_R}\right)$ - - - - - - - - ㉗

and $VO_2 = M_0 c_1 e^{r_1 t}\left(1 + \frac{2r_1\,A_L}{M_R}\right) + M_0\,c_2\,e^{r_2 t}\left(1 + \frac{2r_2 A_L}{M_R}\right)$ - - - - - - - ㉘

The constants c_1 and c_2 can be determined by setting ℓ and $\frac{d\ell}{dt}$ from equation ㉖ equal to ℓ and $\frac{d\ell}{dt}$ respectively from equation ㉒ when $t = t_5$; or similarly for $\hbar$ and $\frac{d\hbar}{dt}$ at $t = t_5$ in equations ㉓ and ㉗.

It is interesting to note that equation ㉘ is exactly of a form obtained by empirical means nearly thirty years ago by Henry and De Moor (1956).

This phase 21R1 is likely to last some time since the repayment of the lactic oxygen debt, i.e. the refilling of vessel L, is so slow.

It ends however at time t_6 when vessel L is filled. This time can be determined as the solution of equation ㉖ with appropriate c_1 and c_2, when $\ell = 0$. The corresponding value $h = h_6$ at $t = t_6$ can be obtained from equation ㉗ . Now that the lactic oxygen debt has been repaid, the last phase of recovery can commence.

3.7: Phase 21R2:

This phase is a repetition of Phase 1R described before, but with initial conditions for h obtained as above, i.e. $h = h_6$ when $t = t_6$, which from Section 3.2 yields:

$$h = h_6 e^{-\frac{M_0}{A_P}(t-t_6)} \quad \text{- - - - - - - - - -} \quad ㉙$$

and

$$VO_2 = M_0 h_6 e^{-\frac{M_0}{A_P}(t-t_6)} \quad \text{- - - - - - - - - -} \quad ㉚$$

with $\ell = 0$ after t_6 of course.

Phases 2A, 2R1, 2R2 and 21R1 are shown diagramatically as part of a second worked example in Section 4 to follow.

4. Worked Example

Let us now consider the realities of the previous section, by putting known numerical values to the various symbols, and comparing where possible, the mathematical solution with what actually happens to exercising subjects. Wherever possible I have taken Margaria's own values, obtained experimentally. In a few places I have used other values, deduced from known or normal values for adult humans.

Suppose we take a trained male distance-runner of 65kg in weight as the subject. He has a (usable) VO_2 max of about 65 ml/kg.min which has a workrate equivalent of 370 watts, i.e. $\theta = M_0 = 370$ watts.

The energy available from the phosphagen source is about 117.6 calories per kilogram of body weight, and thus the volume of vessel P in energy units is about 32,000 joules. Hence the cross-sectional area $A_P = 16{,}000$ units. There is a maximal rate of energy utilisation for the phosphagen energy source, nearly four times VO_2 max, about 1370 watts. This figure is really quite large, and is unlikely to be attained or nearly so, and thus it did not enter into the model solution in Section 3. However, some remarks are addressed to the question of maximum attainable power in Section 5. The energy obtainable from the glycolitic source is about 270 cal/kg, and so the volume of vessel L is about 73,465 joules, and the area $A_L = 73{,}465$ units. The maximal rate of energy utilisation of the glycolitic process is a little less than twice VO_2 max, $M_L = 715$ watts. As far as the repayment rate of the lactic oxygen debt is concerned, i.e. the flow through tube R_3, little is known and I can find no figures. Based on the assumption that a marathoner exhausts, or nearly exhausts, vessel L, and takes 24 hours to recovery fully, then it seems $M_R = 2$ watts is a rough approximation. As noted earlier this is very much less than M_0 or M_L.

4.1: Light Exercise and Recovery: Let us first consider a bout of (for the subject) light exercise, followed by recovery. Suppose $W = 300$ watts, which corresponds to a running pace of about 3.2m/sec or 11.5 k/hr. Since $W < M_0$, the equilibrium phase is aerobic, and the oxygen debt is alactic only. Thus we are only dealing with Phases 1A and 1R, and we shall examine the behaviour of all three energy sources, i.e. of VO_2, h and ℓ.

Equation ④ now becomes

$$h = .811(1 - e^{-.0231t}) \qquad \text{- - - - - - - -} \qquad ㉛$$

and equation ⑤ becomes

$$VO_2 = 300(1-e^{-.0231t})$$

when expressed in watts, otherwise

$$VO_2 = 52.7(1-e^{-.0231t}) \qquad - - - - - - - - \tag{32}$$

in units of ml/kg.min.

Note in passing that the exponential rate constant of -.0231 is of the right order of magnitude, (Margaria et al, 1965; Whipp et al, 1982).

Suppose the subject exercises for five minutes, followed by recovery for another five.

During recovery, from equation ⑥ we see that

$$VO_2 = 300\, e^{-.0231\,(t-300)}$$

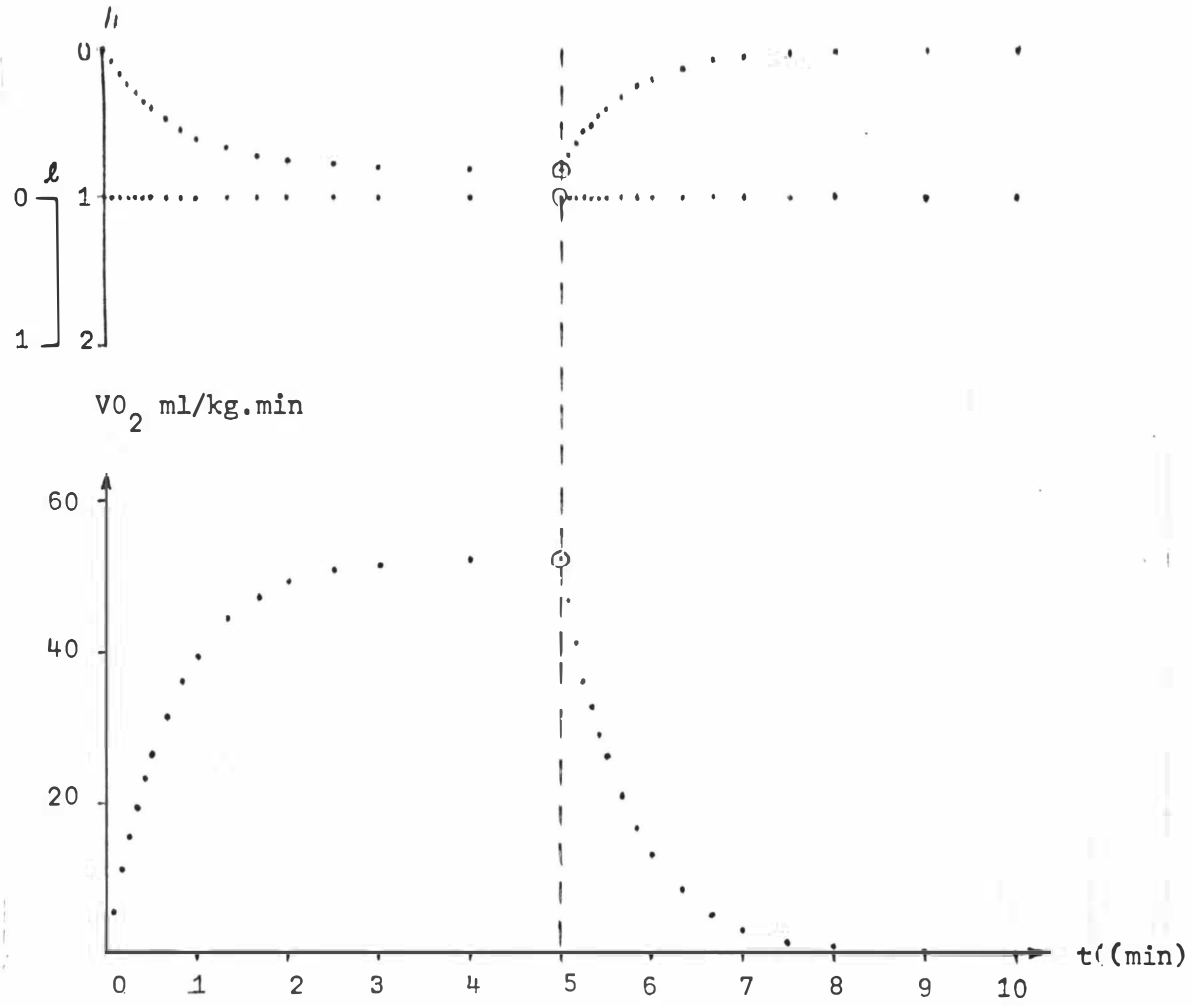
$$\text{or } = 52.7\, e^{.0231\,(t-300)} \qquad - - - - - - - - \tag{33}$$

and it can easily be shown that

$$h = .811\, e^{-.0231(t-300)} \qquad - - - - - - - - \tag{34}$$

During all this time, $\ell = 0$, since exercise is aerobic. Plotted graphically, equations ㉛ - ㉞ yield

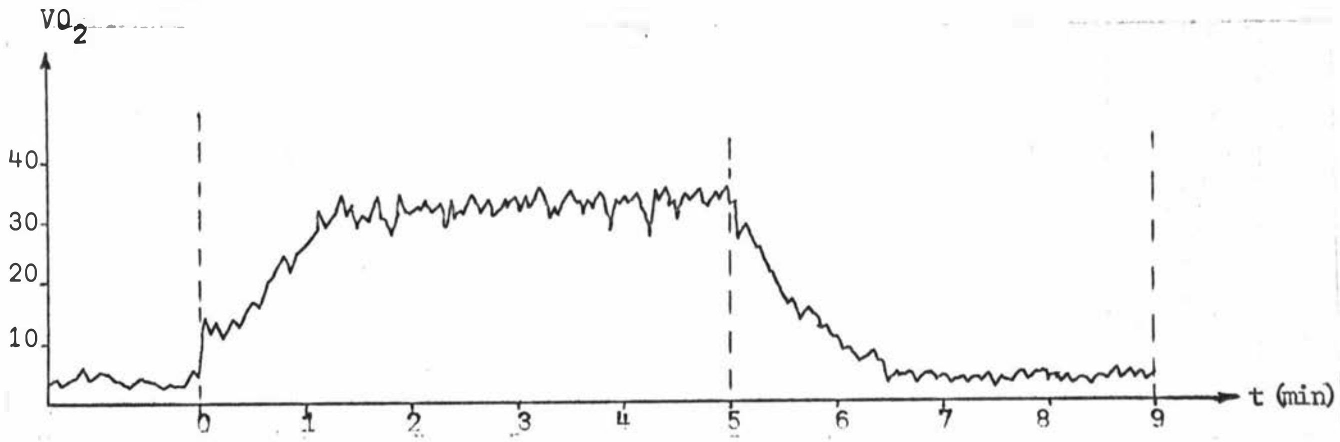
Figure 5: Model Solution Phases 1A and 1R



This figure is analagous to a part of Figure 1.27 in Margaria (1976) and reflects that model for oxygen uptake, which physiologists have used empirically for so many years.

Actual results for my own V0$_2$, which I have obtained experimentally in a respiratory laboratory are shown in Figure 6 below. The likeness to Figure 5 is excellent.

Figure 6: VO_2 for Exercising Subject (Light Load)



Note: This figure shows total VO_2, including the basal metabolic rate evident in the 1½ minutes prior to the start of exercise.

4.2: Heavy Exercise and Recovery: Suppose secondly that the subject undertakes a bout of more severe exercise, beyond the anaerobic threshold. Suppose $W = 2M_0 = 740$ watts, which corresponds to a running pace of just over 5m/sec, or a 1500 metre in just under 5 minutes, which is quite good going. Suppose he runs at this pace for 2½ minutes followed by a 9½ minute recovery period.

From equation ⑦ we find that the duration of Phase 1A is given by $t_2 = 30$ seconds, and hence from equations ④ and ⑤

$$h = 2(1-e^{-.0231t})$$

and $$VO_2 = 740(1-e^{-.0231t}) \text{ watts}$$

or $$130(1-e^{-.0231t}) \text{ ml/kg.min}$$

and $$\ell = 0$$

all for $0 \leq t \leq t_2 = 30$ seconds.

At time $t_2 = 30$, $h = 1$, $VO_2 = 370$ watts or 65 ml/kg.min, i.e. M_0, and $\ell = 0$, and after $t_2 = 30$, Phase 2A begins.

From equation ⑬ we obtain

$$\ell = .076(e^{-.0544(t-30)}-1) + .00414(t-30) \qquad ------ ㉟$$

and from equation ⑭ we obtain

$$h = .349(1-e^{-.0544(t-30)}) + .00414(t-30) + 1 \qquad ----- ㊱$$

and of course $VO_2 = M_0$, all for $30 = t_2 \leq t \leq t_3 = 150$. At the end of 150 seconds, we can see from equations ㉟ and ㊱, that $\ell_3 = .420$ and $h_3 = 1.845$. The latter is the first inkling that something might be awry, for a trained athlete should be able to manage this pace for at least twice this time, if not longer still. Such matters however will be deferred to Sections 6 and 7.

The subject has now ceased exercise and Phase 2R1 has begun. Equation ⑰ for ℓ yields

$$\ell = .076\,e^{-.0544(t-30)} - .152\,e^{-.0544(t-150)} - .00414(t-30) + 1.067$$

--- ㊲

while equation ⑱ for h yields

$$h = .698\,e^{-.0544(t-150)} - .349e^{-.0544(t-30)} - .00414(t-30) + 1.644$$

--- ㊳

and of course $VO_2 = M_0$, all for $150 = t_3 \leq t \leq t_4$. Time t_4 marks the end of this phase, when the levels in vessels L and P equate, and $\frac{d\ell}{dt}$ from equation ㊲ equals zero. From this derivative we find $t_4 = 162.7$ seconds. We obtain, from equations ㊲ that at this time $\ell = \ell_4 = .442$, and of course $h = h_4 = 1.442$. This marks the end of Phase 2R1.

Phase 2R2 now begins for $t \geq t_4 = 162.7$ seconds. During this time the system is described by equations ㉒ and ㉓ which for our subject are given by

$$\ell = 54.34(1-e^{-.0000761(t-162.7)}) - .00414(t-162.7) + .442 \quad \text{- - - - - - - - -} \quad ㊴$$

and

$$h = 249.5(e^{-.0000761(t-162.7)}-1) - .00414(t-162.7) + 1.442 \quad \text{- - - - - - - - -} \quad ㊵$$

Of course, $VO_2 = M_0$ still.

This phase ends when $t = t_5$, the time when $h = 1$. This time can be obtained from equation ㊵, yielding $t_5 = 181.8$, at which point from equation ㊴, $\ell = \ell_5 = .4415$, barely any change. During this phase, $VO_2 = M_0$ still. Phase 21R1 begins now at $t = t_5 = 181.8$, and to obtain the appropriate versions of equations ㉖ - ㉘, we must first solve equation ㉕ for roots r_1 and r_2. This yields

$$r_1 = -.02319 \text{ and } r_2 = -.00001358$$

Substituting these values and using the initial conditions as prevailing at $t = t_5$, we obtain

$$\ell = -.0397e^{-.0232t} + 1.446e^{-.0000136t} - 1 \quad \text{- - - - - - -} \quad ㊶$$

$$h = 67.56e^{-.0232t} + .00339e^{-.0000136t} \quad \text{- - - -} \quad ㊷$$

and

$$VO_2 = M_0h = 24997e^{-.0232t} + 1.25e^{-.0000136t} \text{ watts} \quad \text{- - - -} \quad ㊸$$

or

$$4391e^{-.0232t} + .22e^{-.0000136t} \text{ ml/kg.min} \quad \text{- - -} \quad ㊹$$

As remarked earlier, this phase continues for an extended period of time, until $\ell = \ell_6 = 0$. From equation ㊶ this occurs at $t = t_6 \approx 27140$, or nearly 7½ hours later, at which time $h = h_6 = .00235$ and $VO_2 = .87$ watts or .15 ml/kg.min. [For practical purposes, as far as h and VO_2 are concerned, we can see that approximations could be used. For h, an equation $67.56e^{-.0232t}$ would suffice, and the phase duration is only another 3 or 4 minutes. VO_2 can be similarly approximated from equations ㊸ or ㊹.]

The final phase, 21R2, is of the same ilk as Phase 1R, with initial conditions as prevailing at $t = t_6$. That is

$$h = .00235\, e^{-.0231\,(t-27140)} \qquad \text{- - - - - - - - -} \qquad ㊺$$

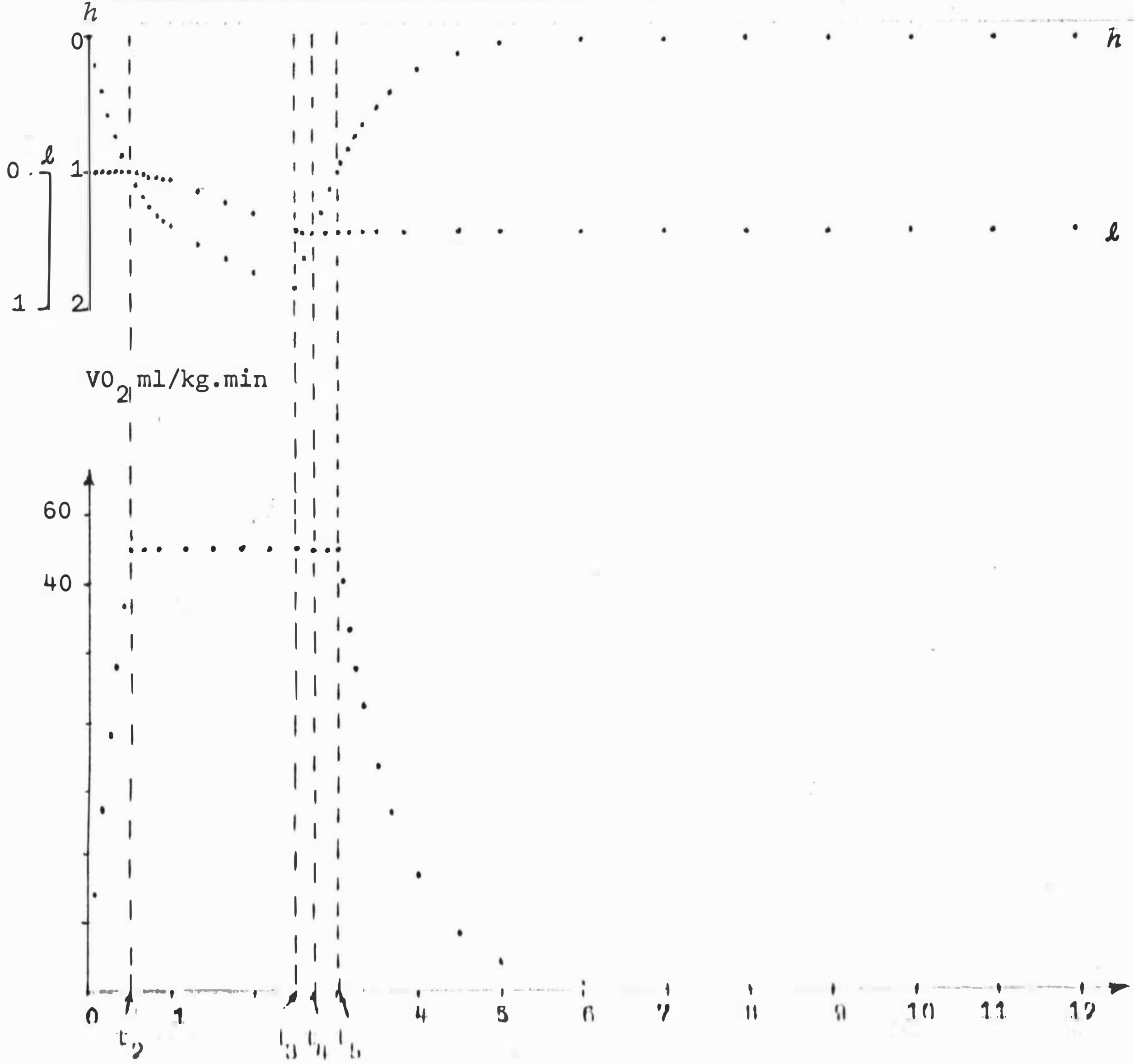
$$\gamma\alpha_2 = .87\, e^{-.0231\,(t-27140)} \text{ watts}$$

and $$\ell = 0.$$

Now as $t \rightarrow \infty$, the whole system returns to normal. [In practical terms, it has done so at t_6].

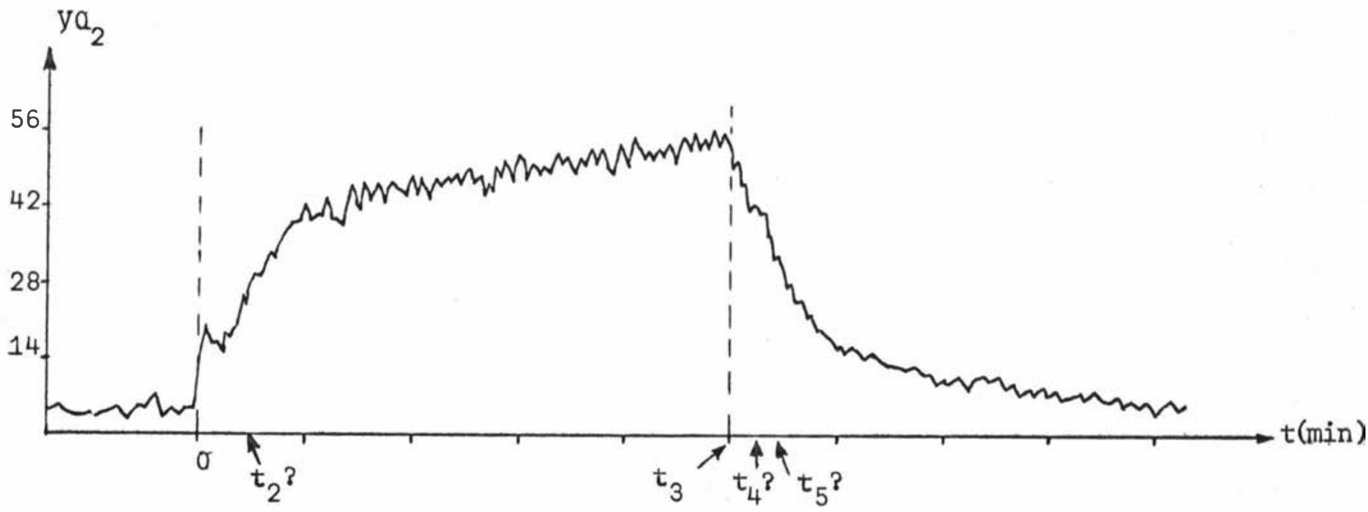
Now utilising all the above information, we can diagramatically represent the behaviour of our subjects energy utilisation and restorative processes during the 2½ minutes of exercise and subsequent recovery. This is shown in Figure 7 on the following page.

Figure 7: Model Solution Phases 1A, 2A, 2R1, 2R2 and 21R1



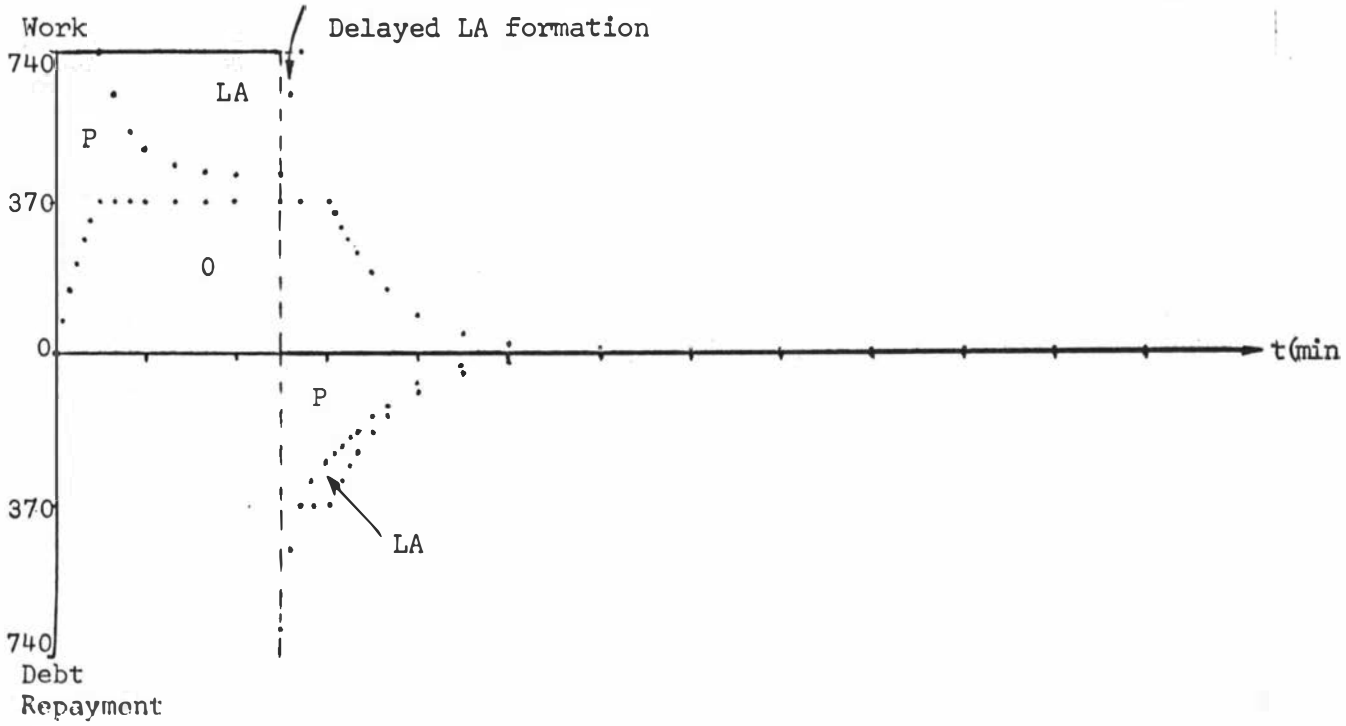
By comparison, Figure 8 following shows actual results for VO_2 obtained in an exercise laboratory. The likeness is not as good as [illegible]. This problem is discussed further in Section 7.

Figure 8: VO_2 for Exercising Subject (Heavy Load)



In addition it is interesting to compile a diagram to show the three energy components and their contributions to the total workload during exercise. That is, in addition to VO_2 we include $A_P \frac{dh}{dt}$ and $A_L \frac{d\ell}{dt}$, the alactic and lactic energy sources. Furthermore this is continued through the recovery period to show the oxygen debt repayment mechanisms as predicted by the model. This diagram, Figure 9 can be compared with Figure 1.28 in Margaria (1976).

Figure 9: Model Solution to Energy Components



It is found that a superficial resemblance exists, but that there are some important differences. These differences, discussed further in Section 7, mean that the graphical presentation given by Margaria (1968) in Figure 1.28, is not correct, for it does not represent the solution to his hydraulic model. Alternatively, if Figure 1.28 is taken to be correct, then his formulation of the hydraulic model is incorrect.

5. Maximum Power

It is of interest to investigate Margaria's model for the maximum attainable power at any instant, in the various phases discussed in Section 3.

We have made assumptions about the flows through R_1 and R_2, but we must also consider how the alactic flow is governed. Now it is well-known that the final sprint in a long race cannot be performed at the same speed as when starting from rest, as in a 100 metre race. Margaria et al (1971) have shown experimentally that the maximal anaerobic power due to phosphagen, as well as the total energy available from the alactic process, are decreased when short bursts of very strenuous exercise are performed starting not from rest, but from one of steady state exercise. This observation was not quantified in the terms required here, but its message is clear. In terms of the model, if P is partly empty, then the maximal power cannot be achieved. It would be consistent with the earlier approach to suppose that the maximal alactic power is proportional therefore to the fluid level remaining in vessel P, i.e. to $(2-h)$. With this assumption we can now turn to the determination of the maximum attainable power in any of the phases of activity or recovery.

During phase 1A when Figure 1 is appropriate, we see that the maximum power available, W_M, for given h, is

$$W_M = M_0 h + M_P\left(\frac{2-h}{2}\right) \tag{46}$$

During Phase 2A, when Figure 2 is appropriate, we can see that the maximum power available is given by

$$W_M = M_0 + M_P\left(\frac{2-h}{2}\right) + M_L(h-1-\ell) \tag{47}$$

Similarly, if we now apply these ideas to the phases of recovery, to see how the maximum power available returns to its resting level, we have

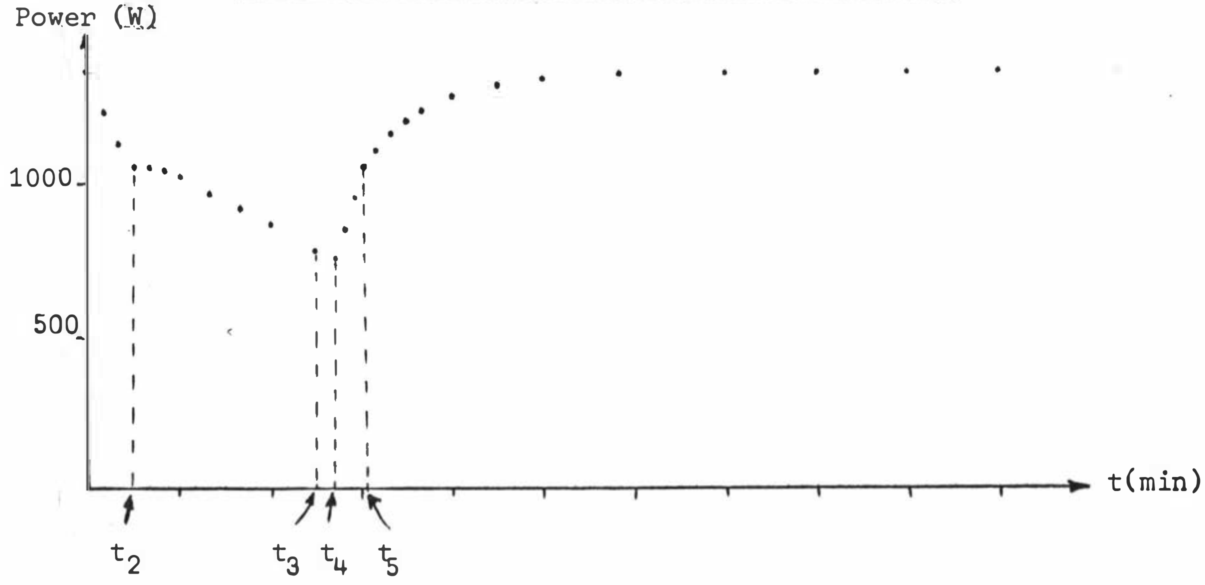
Phase 2R1 : $W_M = M_0 + M_P\left(\frac{2-h}{2}\right) + M_L(h-1-\ell)$, as equation (47)

Phase 2R2 : $W_M = M_0 + M_P\left(\frac{2-h}{2}\right)$ (48)

$$\left.\begin{aligned} &\text{Phase 21R1}: W_M = M_0 h + M_P\left(\frac{2-h}{2}\right) \\ &\text{Phase 21R2}: W_M = M_0 h + M_P\left(\frac{2-h}{2}\right) \end{aligned}\right\} \text{, as equation (46)}$$

It is of interest now to plot W_M for our subject during his bout of heavy exercise at 740 watts, and his recovery, for it reveals some interesting observations. This plot is shown below as Figure 10.

Figure 10: Subjects Maximum Available Power



It is interesting to note that at the point of cessation of exercise, W_M = 780 watts, a value not much in excess of the 740 demanded of him. This suggests that he is not far from exhaustion, as alluded to earlier, and this will be discussed in the next section.

Of interest also in this Section is to enquire of the solution to the model if the individual were to make an "all-out" effort, that is if he were to keep up as great a workload as he possibly can, for as long as he possibly can. We will consider the active phases only, that is phases 1A and 2A, for there is little interest in the question as it pertains to recovery (though it could of course be done). The solution is simply obtained by putting W_M for W in the two versions of equation ① and solving for h and/or ℓ and/or VO_2 as the case may be.

In Phase 1A, we obtain from equations ㊻ also

$$W_M = M_0 h + M_P\left(\frac{2-h}{2}\right) = M_0 h + A_P \frac{dh}{dt}$$

i.e. $$\frac{dh}{dt} + \frac{M_P}{2\,A_P}\,h = \frac{M_P}{A_P}$$ - - - - - - - - ㊾

which, since h = 0 at t = 0 gives as its particular solution

$$h = 2\left(1-e^{-\frac{M_P}{2\,A_P}t}\right)$$ - - - - - - - - ㊿

i.e. $$VO_2 = 740\left(1-e^{-\frac{M_P}{2\,Ap}t}\right)$$

Hence $$W_M = 2\,M_0 + (M_P - 2\,M_0)\,e^{-\frac{M_P}{2\,A_P}t}$$ - - - - - - - - (51)

This is a very interesting result, for if $M_P > 2\,M_0$ (which is believed to be true) then W_M is decreasing with t during Phase 1A. If the inequality is reversed, then W_M increases with t during this phase.

Now Phase 1A ends at $t = t_2$ when $h = 1$, with

$$t_2 = -\frac{2\,A_P}{M_P}\,\ln \tfrac{1}{2}$$

In Phase 2A, from equations ⑨ and ㊼ and simplifying

$$\frac{dh}{dt} + \frac{M_P}{2\,A_P}h = \frac{M_P}{A_P}$$

which is identical to equation ㊾, and therefore its solution, since the initial conditions match, is also given by equation ㊿. This means, that as far as the behaviour of h is concerned, the time t_2 when the subject crosses the anaerobic threshold, is of no relevance at all.

However, as far as VO_2 and ℓ is concerned, it is of relevance. During Phase 2A, $VO_2 = M_0$ of course, and we must now consider the behaviour of ℓ during this time.

From equations ⑨ and ㊿ we can obtain after simplification that

$$\frac{d\ell}{dt} + \frac{M_L}{A_L}\ell = \frac{M_L}{A_L}\left(1 - 2\,e^{-\frac{M_P}{2\,A_P}t}\right)$$

which, since $\ell = 0$ at $t = t_2$ as given above, has the particular solution

$$\begin{aligned}\ell = 1 &- \frac{4\,M_L\,A_P}{2M_L A_P - A_L M_P}\,e^{-\frac{M_P}{2\,A_P}t}\\ &+ \frac{A_L\,M_P}{2M_L A_P - A_L M_P}\,e^{-\frac{M_L}{A_L}(t-t_2)} \qquad - - - - - - - - \end{aligned} \tag{52}$$

Hence from equations ㊼, ㊿ and (52), we can obtain an expression for W_M, given by

$$\begin{aligned}W_m = M_0 &+ e^{-\frac{M_P}{2\,A_P}}\left[\frac{4\,M_L^2\,A_P}{2\,M_L A_P - A_L M_P} - 2\,M_L + M_P\right]\\ &- \frac{A_L M_P M_L}{2\,M_L A_P - A_L M_P}\,e^{-\frac{M_L}{A_L}(t-t_1)} \qquad - - - - - - - - \end{aligned} \tag{53}$$

We could now plot a power component curve, as in Figure 9 for our subject of the previous section.

6. Fatigue and Exhaustion

This is a rather difficult area to deal with, for there seems to be no uniquely accepted definitions among physiologists, though the general notions of fatigue and exhaustion are quite clear. The two terms are sometimes used interchangably, though in general the former is preferred. The situation is complicated by the recognition of two types of fatigue. Central fatigue (i.e. proximal to the motor neurons) is mainly of the brain and the body's message carrying mechanisms. Peripheral fatigue (i.e. distal to the motor neurons) is mainly in the muscles themselves. They can occur separately or together. For a full discussion see Asmussen (1979). It is the latter, muscle fatigue, that is of interest in this work.

The inability of the muscles to perform the work demanded of them can arise in two ways: either the depletion of body metabolites, principally glycogen; or the accumulation of catabolites, principally lactic acid. The former is predominant in prolonged exercise of lower intensities, and the body in effect exhausts its fuel supplies, or at least cannot supply fuel at a rate fast enough to maintain the demand. The latter is predominant in short-term high-intensity exercise, and is the muscle poisoning referred to in Section 1. Without getting engrossed in the physiological questions, the most suitable approach to take for this work is the former; that is to say that the subject has succumbed to fatigue at any time when his body cannot sustain the workload demanded of it at that time. This approach does not mean the inability to perform any work at all. Such a point might be described as "utter exhaustion"!

In quantitative terms therefore, and in reference to the previous section, the point of fatigue onset can be determined as that time when the workload demanded equals the maximum that the body could produce utilising the body stores as they exist at that time. We have seen in Section 5 how the maximum available power can be calculated in terms of the existing levels in the various compartments of the model during the various phases. Section 5 in fact went further and actually obtained the maximum power/time curve representing an all-out effort by the subject, through the various phases.

We now have a means of obtaining a prediction of the endurance of the subject from the model, that is the length of time for which the subject could continue at a given workload. This leads to an endurance curve of the type considered by Wilkie (1980), though a considerably more detailed one. All that is required is to express W_M from equation ㊼, substituting for h and ℓ from equations ⑬ and ⑭ , and solving (numerically) for t when $W_M = W$, where W is the given workload. This value of t determines the onset of fatigue.

Once the onset of fatigue has occurred, the subject is forced to lessen his workload, for it can no longer be maintained. He may either follow the maximum power-time curve, such as in Figure 11, from then on. On the other hand he may lessen the workload to a level below the maximum possible. This new level can then be maintained for a time until fatigue once more intervenes. If it is low enough, below the anaerobic threshold, the subject will commence an active recovery, to a steady state, though as in Phase 21R1 the repayment of the lactic debt takes a long time.

Thus it can be seen that the Margaria model provides a built-in fatigue feedback mechanism, of the longer term body metabolite depletion type.

This is an aspect of the model not considered by Margaria, and can perhaps be regarded as a bonus for him. We have seen previously that it is the depletion of vessel P that is the more relevant, rather than of vessel L as is suggested by Asmussen (1979). The Margaria model does not provide a fatigue mechanism of the short term catabolite accumulation type. Such a consideration is a possibility as it can be based on the flow in R_2 less some sort of diffusion out of the system. However this would create an inelegant appendage to the model.

7. Criticisms

This is a brief section, to highlight those obvious areas where the Margaria model predictions deviate from experimental observations on exercising subjects.

The first, the endurance of our subject in Section 5, can probably be dealt with fairly easily without structural change to the model. Margaria's estimates of the body phosphagen and glycolitic energy contents, from which the volumes of vessels P and L were obtained, are based on normal adults. So too are the estimates of biomechanical efficiency. For a fit trained athlete, a 60% improvement in body energy stores, plus a 25% increase in efficiency does not seem unreasonable, and this would lead to an effective doubling of the volumes in P and L. Furthermore a smoother and more efficient running style means a higher velocity is attained for the same power output. All these factors, together perhaps even with a higher VO_2 max, could well account for the earlier observation.

A fundamental area where observation differs from the model assumptions is that the anaerobic threshold θ, equals VO_2 max, M_0. It seems to be accepted now, that $\theta < M_0$. For trained distance runners θ is about 60 to 65% of M_0, but for the untrained it is less (Weltman & Katch, 1979; Reinhard et al, 1979). The only way this can be taken into account is by making a structural alteration to the model. That is by raising the top level of vessel L (refer Figure 1) to a height say 70% of the height of vessel 0. This alteration of course means a completely different solution to that obtained in Section 3, but one which can nevertheless be obtained analytically in the same manner.

A second fundamental area of difference is highlighted by Figure 8, a figure which is typical for an individual exercising to exhaustion. The illustration suggests that VO_2 max seems never to be reached. I do not mean VO_2 max as is defined in the exercise physiology literature, but VO_2 max as a concept, representing the maximum oxygen uptake the body could theoretically reach. What this observation means in terms of the model is that the level of tube R_1, the inflow from vessel 0 to vessel P is indeterminate. It is not opposite θ as has just been shown, nor is it at the half-way point. (Which also means θ is not necessarily 70 to 75% of M_0 at all, since M_0 is now unknown). Another structural alteration is required to account for this difficulty, but only the direction and not the extent of the alteration is known. No new solution could therefore be obtained until this indeterminancy is removed.

A confirmation of the suggestion that VO_2 max is never reached is evident from the recovery portion of Figure 8, for it can be seen that VO_2 commences an immediate decline. This is contrary to the model prediction, which showed that VO_2 max if reached, is maintained for some time (over half a minute for our subject in Section 4), before declining, as can be seen in Figure 7.

Thus it is clear, that although the model and its solution are a useful and interesting entity, the model requires several structural alterations to more properly represent reality. For at least one of these, the necessary information is as yet unknown, and so we must wait a while before making the attempt at improvement.

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APPENDIX 2

PROTOCOLS AND DATA SUMMARIES

North Canterbury Hospital Board

The Princess Margaret Hospital
Cashmere Road, Christchurch, 2. New Zealand. Telephone 39-169

Department of Respiratory Medicine
When replying please quote: Respiratory Physiology Laborato
"C" Block

APPENDIX 2.1

QUESTIONNAIRE

A) NAME: AGE: (Yrs)

ADDRESS:

PHONE:

HEIGHT: WEIGHT:

B) <u>Fitness Level</u>:

How would you rate your own present level of fitness, on a scale from 0 to 10; when zero represents utterly unfit and ten represents absolute peak fitness?

What do you regard as your present best race distance(s)?

What would you estimate to be your time for them if you were to run today under competitive conditions?

Briefly describe your current type of training schedule:
(e.g. number of kilometres per week, predominant form of training etc.)

R Hugh Morton

APPENDIX 2.2

Name Age	Address	Ht. Wt. Phone	Current Fitness	Distance Time	Current Training
Bruce HUNTER 32	26 Konini St Riccarton	174, 67 486-003	85%	800 1-55	110 k/wk mixed
Rob McGREGOR 25	70 Clyde Rd Christchurch 4	174, 65 487-565	70%	5k ?	60 k/wk
John KNIGHT 23	19 Rolleston Ct Cambridge Tce.	183, 73 796-149	85%	1500 4-05	120 k/wk mixed
James ROGAL 19	7 Julius Tce. Richmond	172, 60 853-749	60%	800 2-03	90 k/wk mixed
Rob BOYD 18	16 Belmont St Wainoni	176, 69 889-487	70%	400 51.5	70 k/wk Fartleks
Dave KETT 17	108 Warden St Richmond	177, 73 853-707	70%	1500 4-05	100 k/wk mixed
Hugh MORTON 36	18 Carlton Ave Palmerston Nth	179, 71 71-110	50%	N/A	3 games squash per week

TREATMENT TRIAL OR INVESTIGATION - PATIENT CONSENT FORM

APPENDIX 2.3

PROJECT TITLE:

LACTATE KINETICS AND EXERCISE TOLERANCE IN THE EXERCISING ATHLETE

INVESTIGATORS:

Mr. R. Hugh Morton	Massey University
Dr. Donald E. Stewart	The Princess Margaret Hospital

VENUE OF TRIAL:

University of Canterbury Running Track / Respiratory Physiology Laboratory TPMH

AIM OF TRIAL OF NEW TREATMENT OR INVESTIGATION

To gain an understanding of lactate kinetics and their relationship to exercise tolerance in the exercising athlete.

****SEE ATTACHED PROTOCOL****

DESCRIPTION OF NATURE AND DURATION OF PATIENT'S INVOLVEMENT:

A series of short sprints 2 - 3 hours duration

Seven sessions of exercise at TPMH on a bicycle ergometer, each on different days. 1 - 1½ hours per day

On two of the above occasions, blood will be taken from a lower arm vein

DESCRIPTION OF INCONVENIENCES OR HAZARDS WHICH MIGHT BE EXPECTED:

(1) Transient minor discomfort while intravenous needle is inserted into the skin. There after, blood tests will not cause further discomfort.

(2) Normal discomfort associated with strenuous effort.

STATEMENT BY PATIENT: (TO BE SIGNED IN THE PRESENCE OF A DOCTOR)

I HAVE READ THE ABOVE AND HAVE HAD THE OPPORTUNITY FOR DISCUSSION WITH A DOCTOR. I UNDERSTAND THAT THE PROCEDURES HAVE BEEN APPROVED BY A SPECIAL HOSPITAL COMMITTEE AND THAT I MAY WITHDRAW MY AGREEMENT AT ANY TIME. I UNDERSTAND THIS WOULD NOT AFFECT MY CONTINUING TREATMENT OR CARE AT THE HOSPITAL. I UNDERSTAND THAT MY DOCTORS WILL DISCONTINUE THE TREATMENT OR INVESTIGATION IF ANY HARMFUL EFFECTS APPEAR.
I AGREE TO TAKE PART IN THIS STUDY OR TRIAL OF TREATMENT.

Signature of Patient Date

APPENDIX 2.4: PROPOSED PROTOCOL

LACTATE KINETICS AND EXERCISE TOLERANCE IN THE EXERCISING ATHLETE

Introduction

The experimental work proposed forms part of the Ph D research project being undertaken by Mr R. Hugh Morton of the Mathematics and Statistics Department of Massey University. The project is titled "A Mathematical and Computer Simulation Model of the Exercising Athlete" and is jointly supervised by Professor Ron Munford and Dr Alex S. Davies of the Physiology and Anatomy Department, and Professor Brian Hayman of the Mathematics and Statistics Department.

In an athlete exercising at a workload exceeding his anaerobic threshhold (that is, a level at which the oxygen supply to the working muscle is insufficient to supply all of the necessary force), fatigue rapidly develops.

Current mathematical models of athletes under conditions of maximal exertion have no provision for the inclusion of fatigue in the feedback processes. A feedback process involving lactate poisoning of the muscle is proposed. Whilst some data exists on the kinetics of lactate following exercise, information has not been published for lactate kinetics during exercise.

Aims

To collect information regarding the parameters of maximal and near-maximal exercise in athletes and obtain information on lactate kinetics to allow inclusion of a feedback process in the models available.

Experimental Protocol

Subjects:	Eight to ten male middle-distance (800-3000m runners at a good level of fitness (slightly below peak condition).
Consent:	Subjects will be fully informed of the nature of the project and their involvement by way of discussion and a copy of this document. Written consent will be obtained on the official forms (enclosed) for the section of the protocol involving cannulation.

Exercise Regime

Initial estimates of maximum possible muscle performance will be obtained by accurate timing of short (50-70 metre) sprints on the running track at the University of Canterbury. These measurements will be under the supervision of Mr John Edyvean of the Department of Physical Education.

Data collection at the Respiratory Physiology Laboratory at The Princess Margaret Hospital will comprise the remainder of the study.

An initial session of exercise on a bicycle ergometer will be performed to ascertain the level of exercise at which anaerobic respiration within the exercising muscles becomes a significant factor. This session will involve short bouts of exercise at different workloads. During these bouts, oxygen consumption, carbon dioxide production, ventilation and hear rate will be monitored using the PDP11 computer system at The Princess Margaret Hospital. All such measurements are non-invasive.

Having determined the "anaerobic threshold" subjects will be subjected to six separate bouts of exercise on separate days at workloads at or above their anaerobic threshold. Exercise at each workload will continue for eight minutes or until the workload cannot be maintained, at which point exercise will cease. During exercise, and for four minutes subsequently, oxygen consumption, carbon dioxide production, ventilation and heart rate will be monitored by the computer. The order in which the different workloads are presented to a subject will be randomised.

In two of the six bouts of exercise blood lactate levels will be measured from venous samples (see below). Lactate analysis will be performed by the University of Canterbury Physical Education Department.

Blood Lactate Sampling

A 21 g intravenous "butterfly" needle will be inserted into a lower arm vein by Dr D. Stewart using standard sterile procedure. Venous blood will be withdrawn via an attached three-way tap and the needle will be periodically flushed using isotonic saline.

A 4 ml blood sample will be taken every twenty seconds from the commencement of exercise to four minutes post-exercise (a total of 12 minutes maximum). Total blood removal will be at most 144 ml (12 minutes x 3 samples per minute x 4 ml per sample).

Following exercise the needle will be removed.

Exercise bouts will be arranged so that the subject has at least one week between those bouts in which blood samples are taken.

Supervision

During all exercise at The Princess Margaret Hospital, technical staff trained in the exercise testing of patients will be present. During those bouts of exercise involving blood sampling, Dr D. Stewart will supervise.

R. Hugh Morton
Department of Mathematics and Statistics
Massey University
PALMERSTON NORTH

APPENDIX 2.5: SUBJECTS' INDIVIDUAL CHARACTERISTICS

		B.H.	J.K.	Rob B.	J.R.	D.K.	Rob M.	H.M.
Forced Lung Volume (l)		5.06	7.09	4.96	5.47	6.26	5.10	5.40
Predicted Volume Range {(l)		4.36 5.84	5.11 6.59	4.76 6.24	4.57 6.05	4.91 6.39	4.50 6.01	4.28 5.76
Predicted Max. Ventilation (l/min)		148	204	150	154	186	139	155
VO_2 max (ml/kg/min)		74	76	70	70	70	71	56
Blood Test	Haemoglobin	Normal	Normal	Good	Normal	Normal	Normal	-
	R & W Cell Volume	Normal	Normal	Good	Normal	Normal	Good	-
E.C.G.	Rest	Good	Good	Good	Good	Good	Normal	Normal
	Exercise	Good	Good	Good	Good	Good	Normal	Normal
Pred. Max. Heart Rate		189	195	198	198	199	194	189

APPENDIX 2.6a: Bruce Hunter : Exercise Performance

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	8	4-01
Peak VO_2 (l/min)	.27	2.82	4.11	4.76	4.92
Peak Ventilation (l/min)	7.6	49.2	89.1	120.4	140.9
O_2 Efficiency (%)	17	27	22	19	17
Peak CO_2 output (l/min)	.21	2.38	3.90	4.89	5.85
Peak Heart Rate	56	135	Variable	170	202
Blood Lactate (4 min.)(mM/l)	.9	-	Lost	8.2	-
Peak Muscle Lactate (mM/l)	-	-	Lost	23	-
Anaerobic Threshold			About 1700		

APPENDIX 2.6b: Rob McGregor : Exercise Performances

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	6-14	3-05
Peak VO_2 (l/min)	.55	2.81	3.91	4.64	4.06
Peak Ventilation (l/min)	11.9	48.8	101.2	123.4	106.9
O_2 Efficiency (%)	22	27	18	18	17
Peak CO_2 output (l/min)	.46	2.45	3.75	4.95	4.80
Peak Heart Rate	70	151	175	183	176
Blood Lactate (4 min.)(mM/l)	1.2	-	8.02	-	6.7
Peak Muscle Lactate (mM/l)	-	-	22	-	25
Anaerobic Threshold			About 1700		

APPENDIX 2.6c John Knight : Exercise Performance

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	8	4-12 *
Peak VO_2 (l/min)	.62	2.82	3.83	5.23	4.64
Peak Ventilation (l/min)	13.8	52.6	80.3	144.0	146.4
O_2 Efficiency (%)	21	26	23	17	15
Peak CO_2 output (l/min)	.47	2.42	3.47	5.04	4.95
Peak Heart Rate	54	116	149	173	Lost
Blood Lactate (4 min.)(mM/l)	.9	5.8	7.9	-	-
Peak Muscle Lactate (mM/l)	-	15	21	-	-
Anaerobic Threshold				about 1950	

* Would have been longer, pedals obstructed. Thus data are probably underestimates.

APPENDIX 2.6d James Rogal : Exercise Performance

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	3-20 *	4-06
Peak VO_2 (l/min)	.54	3.06	3.96	3.97	3.86
Peak Ventilation (l/min)	11.6	78.3	106.4	113.7	138.3
O_2 Efficiency (%)	22	19	18	17	13
Peak CO_2 output (l/min)	.42	2.70	3.77	4.49	4.34
Peak Heart Rate	58	150	176	164	185
Blood Lactate (4 min.)(mM/l)	1.2	-	-	7.02	Lost
Peak Muscle Lactate (mM/l)	-	-	-	25	Lost
Anaerobic Threshold			About 1650		

* Could have been longer, so figures are underestimates.

APPENDIX 2.6e: Rob Boyd : Exercise Performance

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	5-59	3-24
Peak VO_2 (l/min)	.49	3.40	4.47	4.65	4.55
Peak Ventilation (l/min)	12.4	79.7	123.9	165.2	153.9
O_2 Efficiency (%)	19	20	17	14	14
Peak CO_2 output (l/min)	.40	3.30	4.52	4.91	5.17
Peak Heart Rate	72	179	192	221	194
Blood Lactate (4 min.) (mM/l)	Lost	7.61	-	-	9.25
Peak Muscle Lactate (mM/l)	-	20	-	-	28
Anaerobic Threshold			About 1600		

APPENDIX 2.6f: Dave Kett : Exercise Performance

Workloads	Rest	1200	1600	1850	2100
Duration (min)	-	8	8	8	5-02
Peak VO_2 (l/min)	.47	3.07	4.12	4.88	4.87
Peak Ventilation (l/min)	10.2	67.0	107.2	135.1	168.0
O_2 Efficiency (%)	22	22	18	17	14
Peak CO_2 output (l/min)	.31	2.77	3.86	4.85	5.71
Peak Hea[illegible]	61	144	163	171	185
Blood Lactate (4 min.)(mM/l)	1.0	6.9	-	11.5	-
Peak Muscle Lactate (mM/l)	-	18	=	26	-
Anaerobic Threshold				About 1850	

APPENDIX 2.7

TYPICAL COMPUTER FILE RECORDS

Fortran format	8F8.2
Respectively:	duration of breath (sec)
	O_2 consumption (ml/min)
	CO_2 production (ml/min)
	expired volume (ml)
	end tidal O_2 (mm Hg)
	end tidal CO_2 (mm Hg)
	heart rate (beats/min)
	time of last event (sec)

The first block shows transition from a resting period of almost 3 minutes (178.04 secs), to constant workload exercise (1700 kpm 283 watts), covering just over one minute (61.24 secs)

Note: (i) Last event is actually timed at end of the breath during which the event occurred.

(ii) Note typical "bump" on first breath, but because of (i) above it is shown on last breath of rest.

(iii) Marked rise in $\dot{V}O_2$, CO_2 output and expired volume.

(iv) Heart rate monitor not recording.

Typical computer file records: Self @ 1700kpm/min.

```
3. 50   582. 70   460. 69   797. 71   100. 71    40. 88    -3. 20     0. 00
3. 52   522. 87   418. 13   835. 74   102. 44    40. 22    -3. 46     0. 00
3. 64   582. 97   482. 56   898. 96   104. 18    39. 35    -3. 46     0. 00
4. 08   429. 88   367. 48   722. 35   105. 56    39. 15    -2. 94     0. 00
4. 20   281. 00   236. 73   573. 06   103. 83    39. 66    -3. 73     0. 00
4. 34   274. 51   226. 73   557. 12   101. 40    41. 81    -3. 46     0. 00
4. 00   448. 47   365. 24   729. 84    98. 98    42. 75    -3. 46     0. 00
4. 34   549. 14   447. 28   899. 19   100. 02    41. 92    -3. 99     0. 00
4. 38   640. 65   520. 83  1054. 71   102. 44    40. 18    -3. 46     0. 00
4. 32   339. 20   273. 44   623. 69   100. 71    41. 46    -3. 46     0. 00
4. 26   290. 58   230. 39   544. 37   100. 71    41. 19    -2. 94     0. 00
5. 32   289. 22   228. 67   639. 82    99. 32    41. 40    -3. 46     0. 00
4. 98   297. 91   233. 11   585. 19    99. 32    41. 46    -3. 46     0. 00
4. 74   457. 05   354. 98   780. 83    97. 94    42. 19    -3. 46     0. 00
5. 32   228. 88   175. 94   523. 74    98. 28    41. 36    -3. 46     0. 00
5. 36   319. 77   247. 08   641. 15    97. 94    42. 02    -2. 94     0. 00
5. 48   303. 57   233. 89   646. 22    98. 28    41. 53    -3. 46     0. 00
5. 00   427. 74   326. 01   754. 78    96. 90    41. 85    -3. 46     0. 00
4. 88   450. 99   344. 18   790. 03    98. 28    41. 05    -3. 73     0. 00
5. 18   328. 40   252. 07   682. 82   100. 71    39. 94    -3. 46     0. 00
5. 84   325. 82   258. 20   741. 20   101. 06    40. 08    -3. 46     0. 00
4. 60   344. 36   273. 73   630. 02   101. 40    39. 94    -3. 46     0. 00
5. 00   293. 77   232. 95   643. 79   102. 10    39. 56    -2. 94     0. 00
4. 98   258. 74   206. 75   554. 30   102. 44    40. 15    -3. 99     0. 00
5. 06   266. 06   212. 88   568. 66   100. 02    41. 15    -3. 73     0. 00
4. 90   270. 77   212. 26   536. 55   100. 36    41. 12    -3. 46     0. 00
4. 98   314. 57   245. 67   608. 20   100. 02    40. 95    -3. 99     0. 00
4. 24   331. 52   256. 38   582. 90   100. 36    40. 70    -3. 99     0. 00
4. 52   292. 21   227. 23   579. 44   100. 71    40. 36    -3. 46     0. 00
5. 28   236. 99   186. 56   543. 96   100. 02    40. 67    -3. 20     0. 00
4. 90   419. 13   331. 66   758. 17    99. 32    41. 22    -3. 46     0. 00
4. 86   759. 75   610. 46  1224. 06    96. 90    42. 78    -2. 94     0. 00
6. 54   238. 01   186. 37   541. 75   100. 02    41. 81    -3. 20     0. 00
5. 02   346. 32   274. 55   689. 36    98. 63    42. 30    -3. 46     0. 00
4. 42   338. 95   262. 89   600. 06    97. 94    42. 78    -3. 46     0. 00
4. 32   362. 78   276. 52   639. 19    96. 55    42. 61    -3. 20     0. 00
4. 26   721. 77   551. 68   999. 31    95. 51    43. 58    -3. 46     0. 00
3. 48  1670. 36  1315. 80  1914. 89    99. 32    41. 33    -3. 20     0. 00
3. 22  1435. 26  1206. 94  1696. 82   103. 83    39. 84    -3. 46   178. 04
3. 82  1255. 24  1119. 39  1783. 47   104. 52    40. 50    -3. 46   178. 04
2. 74  1216. 90  1111. 46  1410. 84   107. 64    39. 39    -2. 94   178. 04
2. 84  1163. 91  1111. 39  1475. 35   109. 38    38. 90    -3. 20   178. 04
2. 94  1030. 96   973. 35  1307. 18   107. 30    40. 25    -3. 46   178. 04
2. 76  1575. 30  1424. 85  1660. 43   104. 18    41. 50    -3. 46   178. 04
2. 92  1359. 82  1176. 11  1489. 39   103. 14    41. 64    -2. 68   178. 04
2. 84  1862. 20  1569. 31  1807. 18   100. 02    42. 64    -3. 46   178. 04
2. 84  1657. 19  1342. 06  1544. 84    96. 90    44. 03    -3. 20   178. 04
2. 92  1901. 14  1513. 35  1785. 87    96. 55    44. 13    -2. 94   178. 04
2. 82  2072. 98  1641. 59  1773. 01    94. 12    46. 17    -2. 94   178. 04
2. 78  2071. 09  1627. 04  1704. 97    92. 74    46. 94    -3. 46   178. 04
2. 96  2156. 32  1696. 69  1867. 72    91. 35    47. 70    -3. 46   178. 04
2. 84  2396. 57  1882. 38  1896. 91    90. 31    48. 22    -3. 46   178. 04
2. 96  2335. 13  1841. 06  1900. 03    89. 27    49. 67    -3. 99   178. 04
2. 68  2484. 40  1976. 63  1855. 40    89. 62    48. 94    -3. 46   178. 04
2. 98  2617. 84  2123. 99  2156. 48    89. 96    50. 26    -3. 20   178. 04
2. 78  2720. 18  2254. 70  2175. 42    91. 70    49. 78    -3. 20   178. 04
2. 96  2818. 12  2365. 46  2349. 62    90. 66    50. 95    -2. 94   178. 04
2. 82  2863. 12  2474. 89  2395. 09    92. 74    50. 19    -3. 46   178. 04
2. 82  3059. 80  2718. 20  2616. 97    95. 16    49. 64    -2. 94   178. 04
```

The second block shows the transition from the end of 5 minutes of exercise (307.76 secs) to the first 53 seconds of recovery.

Note: (i) Breath duration much shorter at end of exercise, nearly constant.

(ii) High $\dot{V}O_2$, CO_2 and expired volumes to end.

(iii) $\dot{V}O_2$ shows rapid decrease, not matched by CO_2 and expired volumes.

(iv) End tidal CO_2 goes up, decreases later.

```
1.90 3938.59 4441.00 3304.64   110.41    42.23   -2.94  178.04
1.96 3761.14 4230.13 3317.73   109.38    43.27   -3.46  178.04
1.98 3655.51 4132.70 3324.45   110.41    42.37   -3.20  178.04
1.82 3807.67 4312.02 3173.78   110.41    42.85   -2.94  178.04
2.00 3538.32 4018.33 3178.51   109.38    43.51   -3.20  178.04
1.84 3769.60 4227.49 3086.29   109.72    42.95   -3.46  178.04
1.94 3651.83 4120.55 3224.61   109.38    43.65   -2.94  178.04
1.94 3671.03 4145.29 3201.48   110.07    42.78   -2.94  178.04
1.92 3830.63 4246.41 3261.13   109.03    43.20   -2.94  178.04
1.94 3728.92 4153.06 3224.47   109.38    42.99   -3.46  178.04
1.88 3861.66 4284.66 3227.37   110.41    42.23   -3.46  178.04
1.94 3663.13 4018.59 3125.96   109.03    42.92   -3.46  178.04
1.94 3707.74 4070.26 3091.01   109.03    43.09   -3.73  178.04
1.92 3924.69 4269.95 3184.69   107.99    43.82   -3.46  178.04
1.92 3985.78 4364.36 3354.93   109.72    42.37   -3.46  178.04
1.88 3802.93 4167.89 3180.34   108.34    43.65   -3.73  178.04
1.98 3752.61 4126.19 3286.09   109.38    42.30   -3.20  178.04
1.86 4088.37 4454.90 3342.59   109.03    43.27   -3.99  178.04
1.96 3796.74 4161.04 3313.55   109.03    42.75   -2.94  178.04
1.98 3922.92 4297.69 3402.09   109.03    42.92   -3.20  178.04
1.80 3660.71 4024.68 3049.77   110.76    41.26   -3.46  178.04
1.92 3859.23 4263.49 3346.26   110.07    42.43   -3.20  178.04
1.86 3688.62 4040.61 3076.17   110.07    42.09   -3.99  178.04
1.86 4052.12 4399.27 3339.29   109.03    42.33   -3.20  178.04
1.86 3828.48 4174.92 3203.46   109.38    42.57   -3.99  178.04
1.90 3957.84 4356.27 3361.25   109.38    42.57   -2.94  178.04
1.80 3956.80 4336.22 3203.47   111.11    41.15   -3.20  178.04
1.94 3826.25 4214.83 3311.06   109.38    42.92   -2.94  178.04
2.04 3885.13 4250.73 3488.78   110.07    41.92   -3.20  178.04
2.00 3610.81 3938.78 3376.49   111.11    40.60   -3.20  485.80
1.98 3548.40 3910.43 3437.31   111.80    40.36   -2.94  485.80
1.94 3325.70 3739.08 3240.52   113.53    39.21   -3.46  485.80
```

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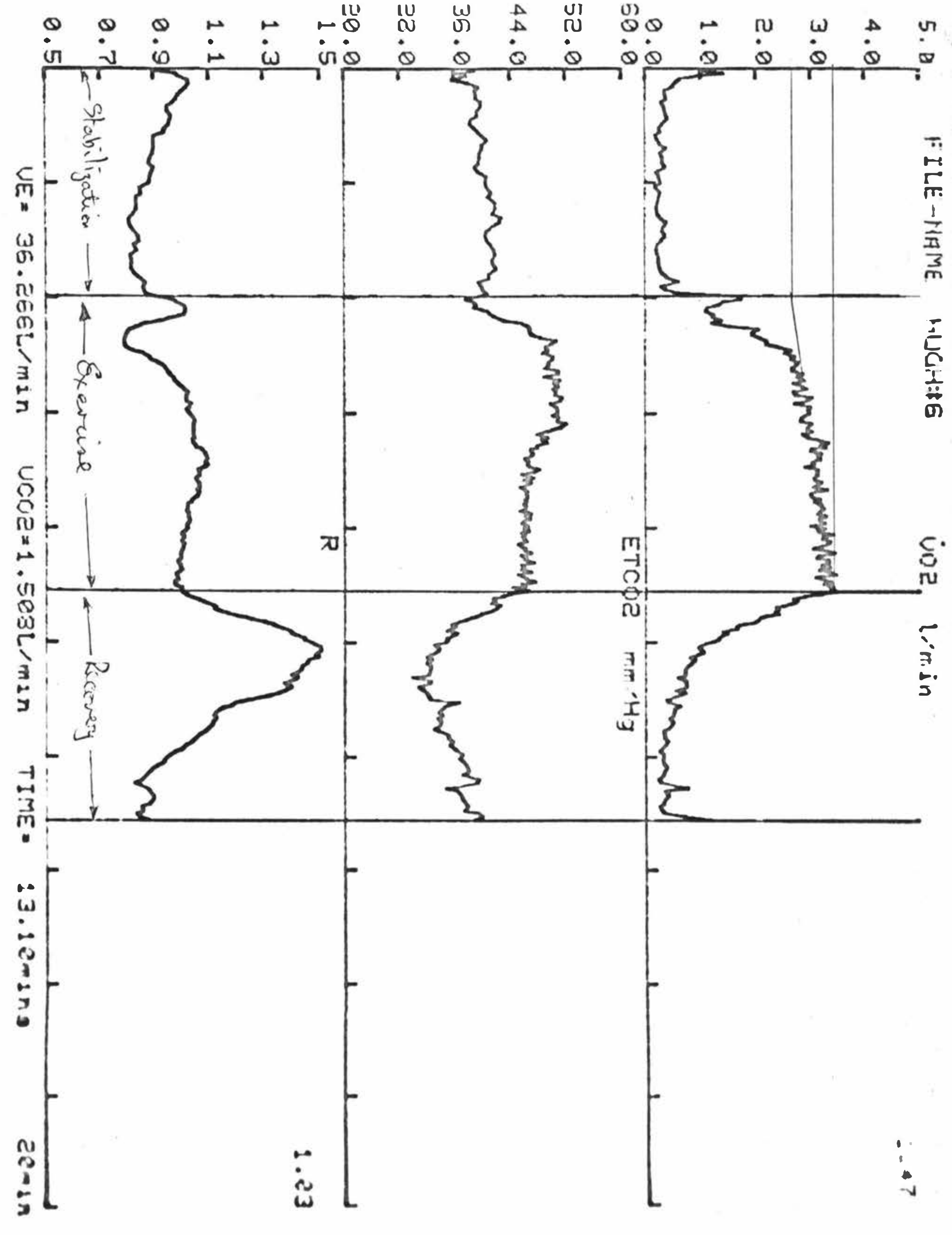
1.94 3358.20 3870.75 3366.51   113.88    39.60   -3.46  485.80
1.90 3116.49 3667.01 3222.66   114.92    38.69   -3.46  485.80
1.90 3015.61 3563.29 3054.89   114.57    39.70   -3.46  485.80
1.90 3066.66 3678.91 3056.34   114.57    40.08   -3.46  485.80
1.92 2998.60 3647.56 3068.17   114.92    39.80   -2.94  485.80
1.90 2936.67 3687.34 3106.52   115.96    40.22   -2.94  485.80
1.80 2926.67 3812.84 3081.71   117.35    39.32   -2.94  485.80
1.80 2639.68 3565.80 2917.89   118.73    38.83   -3.46  485.80
1.86 2574.36 3584.33 3039.80   119.43    38.73   -2.94  485.80
1.78 2325.73 3318.03 2829.61   120.47    37.34   -3.20  485.80
1.90 2389.71 3424.56 3004.84   120.12    38.56   -3.46  485.80
1.80 2282.25 3333.09 2841.16   121.16    37.45   -3.20  485.80
1.92 2147.63 3204.77 2902.77   121.85    37.31   -3.46  485.80
2.04 1973.61 2985.51 2875.43   121.51    38.04   -2.94  485.80
2.04 1987.52 3038.89 2940.99   122.55    36.93   -3.46  485.80
2.02 1927.64 2976.91 2924.70   123.24    36.58   -3.73  485.80
2.04 1752.80 2778.60 2801.88   124.28    35.54   -3.46  485.80
2.00 1777.55 2859.51 2884.11   125.32    34.68   -3.46  485.80
2.04 1715.49 2826.26 2941.52   125.67    34.47   -3.20  485.80
2.14 1562.11 2618.45 2838.51   125.67    34.26   -3.20  485.80
2.04 1559.37 2614.60 2804.63   126.36    33.88   -2.94  485.80
2.04 1504.06 2546.27 2767.98   127.05    33.22   -2.94  485.80
2.00 1423.69 2428.32 2646.99   127.75    32.19   -3.20  485.80
1.96 1381.90 2370.14 2547.68   128.09    31.63   -3.46  485.80
2.08 1314.73 2274.14 2630.69   128.44    31.49   -3.73  485.80
2.06 1300.76 2301.59 2665.90   128.44    31.67   -2.94  485.80
2.06 1250.10 2209.29 2572.10   128.44    31.32   -3.20  485.80
```

APPENDIX 2.8

(a) On-line $\dot{V}O_2$, $ETCO_2$, R



APPENDIX 2.8

(b) Summary of on-line run

```
NAME: Self.                        DATE: 21-MAY-83

AGE: 36          HEIGHT:                 WEIGHT:

FILE-NAME: HUGH#6
STAGE      MINS        VO2      VCO2       VE       ETO2      ETCO2    H.RATE
                      L/min    L/min    L/min      mmHg      mmHg    Beats/min

REST                   0.35     0.31     9.56       107        40        83

  1      2.0- 3.0     3.06     3.25    69.05       104        43       153
  2      4.0- 5.0     3.28     3.25    71.07       104        46       158
RECOVERY
         5.0- 6.0     1.92     2.35    63.94       118        37       148
         6.0- 7.0     0.72     1.01    33.05       125        32       118
         7.0- 8.0     0.39     0.42    14.50       117        35       108
         8.0- 9.0     0.40     0.34    11.66       109        37       101
?ERR 12 FLOATING ZERO DIVIDE
IN   ROUTINE "AUGE   "  LINE 28

FROM ROUTINE ".MAIN."  LINE 68
```

APPENDIX 2.9a

LACTAT

Losungen:

Tris-EDTA-Puffer:
26 g Tris (215 mM)
0,6g EDTA (1,7 mM) (DiNa-Salz/0,5g freie
1 l H_2O dest Komplexon III Saure)

K_2HPO_4 1,8 M: Kaliumhydrogenphosphat) 31,35 g/100 ml

Perchlorsaure 6%: 1 vol knoz. + 10,6 vol H_2O

Mischung Tris-Hydrazin-NAD: (halber Tag haltbar)

NAD 20 mg/ml (27 mM)	10 ml	
Hydrazinhydrat 80%	3 ml	pH 9,6*
Tris-EDTA-Puffer	100 ml	

* pH kann mit HCL korrigiert werden

Messung: Enteiweissung:

Perchlorsaure 0,6 N	0,500 ml	(0,100 ml)*
Blut	0,100 ml	(0,020 ml)*

sofort mischen, zentrifugieren 10min,3000r

Ueberstand 1	0,300 ml	(0,060 ml)*
K_2HPO_4 1,8 M	0,150 ml	(0,030 ml)*

Mischen, (bei sofortiger Verarbeitung 20 m
Eisbad) und einfrieren

zentrifugieren, (Ueberstand 2) (pH muss zwischen 6,8 - 7,5 liegen)

Analyse:

	Blindwert	Probe
dest	0,100 ml (0,050 ml)*	- -
Ueberstand 2	- -	0,100 ml (0,050 ml)*
Gemisch Tris-Hydr.-NAD	2,000 ml (1,000 ml)*	2,000 ml (1,000 ml)*
LDH 10 mg/ml 5 mg/ml	0,020 ml (0,010 ml)*	0,020 ml (0,010 ml)*

mischen, 40 min bei 25° stehen lassen, bei 366 nm gegen Leerwert messen (Absaug Kuvette)

Ausrechnung: E x F 57,816 = mM/1

Kontrollseren: Precinorm U / Precinorm S

( )* = fur 0,020 ml Blutmengen

PMH

STEP 3

Prepare supernatant 1.

- Take 20µl blood
  .1ml Perchloric Acid 0.6N ($HClO_4$)
- quickly mix
- centrifuge 10min at 3000rpm
- OR Freeze

Note

1. Adding blood to $HClO_4$ stops glycolysis
2. Usually freeze blood until such time as possible to analyze. When ready, thaw, then spin.
3. Require: 20µl pipettes, 2ml test tubes with cap, centrifuge, test-tube rack, pipette (.1ml), freezer.
4. Field kit for taking of sample: swabs, lancet, Finalgon, pipettes, t-tubes, marking pen, tt rack, chilly bin, ice packs, stop watches, paper, pencils.
5. Prepare supernatant in 2m test-tubes with cap.

216

auto pipetters

100µL 20µL
200µL 1ml = required + lots of tips

tubes + caps @ 2ml
+ 'idler tubes.
+ syringes (5ml)
+ heparinised saline
+ labels ?
+ tt rack
+ chilly bin

Paul Carpinter.

~~CHAPMAN~~

erchloric acid 0·6N ≈ 250ml. (stability of?)
ubes' + caps (? labels)

DUTTON

5ml syringes (400 sterile disposable)
~12 bags N. saline

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ADDENDUM

This chapter has taken a rather simplistic approach to modelling Newtonian biomechanics; the first instance of the general modelling philosophy I have adopted throughout the thesis. Such a simplified approach has been taken in the main so as to be able to piece the model segments more readily together into a coherent whole. Parsimonious motives also apply. Indeed, such characteristics are intrinsic to any model, but are not so severe as to have rendered the model as a whole down to a trivial exercise.

Firstly, I have applied Newton's laws in a rather uncritical way. I have simplified by regarding the runner's body, in reality a complex body with hinges, as a point mass. The human body during locomotion has been the subject of more detailed segmented kinematic studies, (e.g. Cappozza et al, 1976; Mansour et al, 1982; Winter et al, 1976). Such studies have as a prime concern, information relating to motion and energy changes of the various body segments. As such, variables of interest include internal and external forces, torques, angular velocities, and so on. This level of detailed complexity is not the intention of my thesis. Nevertheless, even in these and other similar studies, simplifying assumptions or restrictions are made: (i) the head, arms and torso are regarded as a single point mass, (ii) rotational kinetic energy of body segments is neglected, (iii) analysis is restricted to the sagittal plane, and (iv) symmetry of the left and right legs is imposed. In some instances, reasons other than for simplicity are given for such assumptions. These include: that the magnitudes of the effects of including more variables are only of the same order as the level of experimental accuracy; and that the results are not sensitive to the inclusion (or exclusion) of these variables.

Secondly, the overall force vector exerted through the legs has at least two components. In addition to the vertical and horizontal components, there is a small lateral component, noticable as the cause of a slight yawing motion of the hips. Couples acting in various planes are also possible.

Throughout this thesis I have taken the exerted force to refer to the horizontal component, and therefore consider horizontal motion only. The vertical and other components go towards keeping the centre of mass "bouncing", and towards the reciprocating motion of the arms and legs, elastic storage of energy, etc. These and other components are difficult to deal with. The simplified approach I have adopted to deal with the presence of these other components, is to subsume them within a mechanical efficiency coefficient. This is an approach adopted for example by Margaria (1976). That these components are in an assumed constant proportion of the overall force vector during the limiting or constant speed phase of motion is I believe a reasonable assumption. For example air resistance accounts for 7 to 14% of power expenditure (p33) at moderate to high running speeds. Although net vertical movements of the hips are slight in runners (McMahon and Greene, 1981), say 2cm, for a 75kg runner with a stride period of 0.2 sec, a power output of 75 watts can be estimated. This represents about 20% of the total power generated at 3.6 m/sec. Such considerations indicate how the total power can be divided into various attributable portions.

During the acceleration phase however, the situation is different; air resistance rises from zero, as does internal resistance, and so on. The force available through the runner's legs can therefore be expected to diminish through the acceleration phase, to a value sufficient to overcome the "steady state" resistances above. Let us therefore examine an alternative formulation.

In assuming above a constant (possibly maximum) force, f, and in so doing fitting the equation (p39)

$$v(t) = f(1-e^{-at/m})/a \qquad \ldots\ldots \qquad ①$$

a coefficient a so large that the power output at constant speed, f/a, is physiologically exhorbitant, is obtained. Instead let us consider a more natural process of force variation which takes some account of the runner's ability to "gear" his output. This is to assume constant power rather than constant force, but while continuing to assume a compound resistance proportional to speed.

Thus, since power is the product of force and velocity, and utilising equation (B) on p39:

$$p - av^2 = m\,v\,\frac{dv}{dt}$$

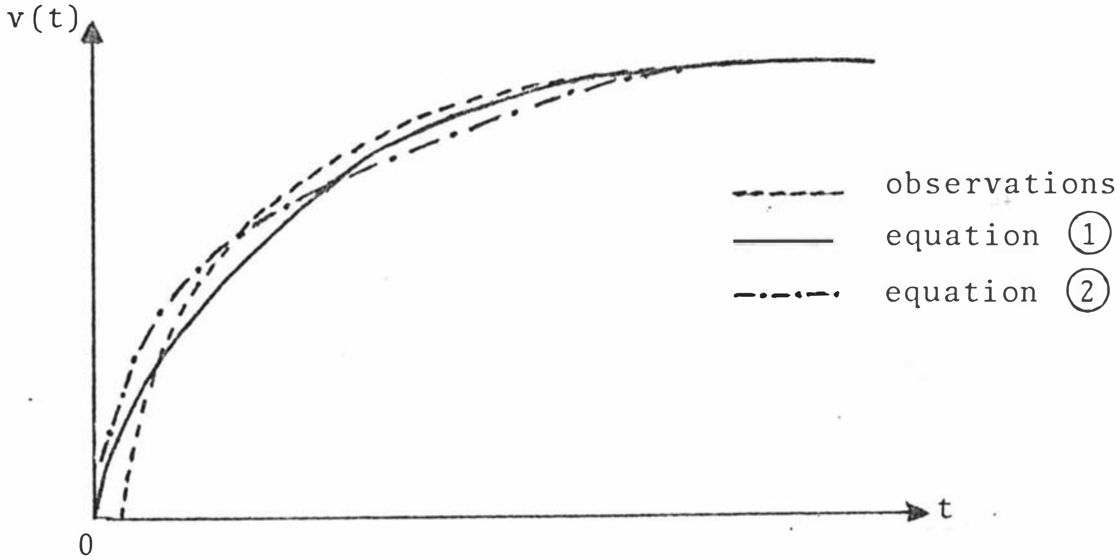
$$= \frac{1}{2}\;m\,\frac{d(v^2)}{dt}$$

which leads to the analytic solution:

$$v^2 = p(1 - e^{-2at/m})$$

i.e. $v(t) = (p/a)^{\frac{1}{2}}(1 - e^{-2at/m})^{\frac{1}{2}}$ ...... ②

Now obtaining p and a to suit typical sprint data and plotting as on page 37, we obtain:



The representation of the data appears as good by equation ② as it is by equation ①. Furthermore whereas $a/m \approx 0.5$ units in equation ①, it is now of the order of 0.15 units in equation ②. This latter occurrence has the immediate consequence that the power required to overcome the air and other resistances is under one third of that required by the Keller model. This may therefore be regarded as a possible solution to the 1/4 to 1/3 reduction factor mentioned previously, and the difficulties discussed on pages 28 and 39. In addition this approach permits initial force to be high, when velocity is low, and to decline as a "steady state" is approached.

While it might at first sight appear that the above alternative has gone a long way in meeting the criticisms of the Keller model I have used, there are nevertheless mathematical difficulties in its implementation. These are hinted at on page 86.

Specifically with $p = fv$ assumed constant, the assumption leads to a discontinuity or (perhaps more correctly) a vertical asymptote for $f(t)$ at $t = 0$. The reason is simply that a runner starting from rest must have initial velocity $v(0) = 0$, in which case the initial force $f(0)$ is infinitely large. This is not just physiologically exhorbitant, it is impossible. Two equivalent avenues out of this mathematical difficulty are possible, either set $f(0)$ at some very large but physiologically possible value and then calculate $v(0)$ for the chosen value of p; or vice versa. Without instituting the necessary equation changes and rerunning simulations, the results of the above approach must remain conjecture.

I believe however, that since the consequences of this alternative are likely evident in only the first say seven seconds or so, the advantages mentioned above will outweigh the contrived nature of the initial conditions. The exception to this belief is likely to be in the sprint mode.

Lastly, the impulsive nature of the force exerted has not been clearly discussed. I have assumed it to be continuous, though in reality its form is one of a large number of discontinuous successive thrusts.

No force at all can be exerted when the runner's foot is not in contact with the ground, and this leads directly to the problem of the meaningful inclusion of the derivative df/dt. The simplifying approach taken in this instance has been to approximate the step function profile of the horizontal force component by a smooth curve for which a continuous derivative exists. That this basic level of simplification is reasonable can be seen for example in the force and velocity traces produced by the speedograph (Volkov and Lapin, 1979) which though wavy, do have a smooth trend.

Acknowledgement: The matters discussed in the above addendum have been raised by the examining panel and its adviser. I would like to thank them for drawing these matters to my attention and suggesting some possible means of their rectification.

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