

Mathematical modelling in sport

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Abstract

We give examples of how mathematical modelling is used to model processes in sport. We start by giving a general overview of areas currently employing mathematical tools to analyze the processes involved. We then go on to look in more detail at the use of mathematics to model the physiological response to exercise. It should also be noted that this area is not only fundamental to the science of technique and training methodology in sport but are also fundamental areas of medicine. As a result improvements in the mathematical analysis of such problems will have far reaching benefits.

1 Introduction.

Mathematics has many uses in sport from the statistical analysis of games of football to the design of sports equipment. Here we will concentrate mainly on the use of mathematics to model and analyze the physiological response of the body to exercise. We will start however with a short review of the use of mathematics in biomechanics.

2 Mathematical modelling and analysis in biomechanics

Biomechanics is the science that examines forces acting upon and within a biological structure and the effects produced by such forces. A introduction to the biomechanical response to exercise can be found in Nigg and Herzog [47], see also Cavanagh [20] for a introduction to the biomechanics of distance running. Such types of analysis are used in many areas of sport to analyze technique so as to improve performance and also to investigate the causes of injuries and look for methods for how to prevent them. Another very interesting area of biomechanics involves looking at the motion of and forces applied to the foot during walking and running, see for example De Clerque et al [25], De Wit et al [24], Robbins et al [53, 54, 55] and Hennig et al [30]. Such studies investigate the biomechanical functions of the foot and can be used to identify unnatural motions likely to cause injuries in an athlete. Many of these studies also compare the motion of the foot when running barefoot and shod and hence can be used to help improve the design of running shoes and see whether the shoe is preventing or causing injuries.

Other areas of biomechanics involve the analysis of the time series of forces exerted on or by the body such as ground reaction forces. Stirling and Zakyntinaki [61] used tools from dynamical systems to model the ground reaction forces at the feet of a person standing on a force platform and hence show how an athlete regains stability following being pushed out of balance. Zakyntinaki and Stirling [72], and Gajewski [28] used Fourier analysis to analyze tremor during muscular failure. In this work Fourier analysis was used to analyze the frequency spectrum of the time series of the forces produced when carrying an exercise out until muscular failure. This work has practical uses in the prediction of imminent failure and hence maximal training loads. It can also be used to detect premature failure when testing for maximum load an athlete can cope with.

Animation tools are also of much use in biomechanics especially for the visualization and correction of technique. For example such tools can be used to look for and hence correct nonalignment of relevant parts of the body to the direction of motion. Improvements in this area can lead to a more efficient technique and hence a better performance and also a reduction in the number of injuries an athlete suffers. One such tool is that developed by Ng-Thow-Hing, V., Faloutsos [46].

3 Models of the physiological response to exercise.

We present a review of some of the models currently used to describe the physiological response to exercise (see Billat et al [11] for a review of some other models not presented here and also Stirling et al [60] for models using dynamical systems). We shall focus on the modelling of the heart rate $HR(t)$, volume of oxygen uptake (breathed in) per unit time $\dot{V}O_2(t)$, the lactate concentration levels in the blood, $BLa(t)$ and velocity, v as these are the most commonly used variables to define exercise intensity and fitness levels. For more details on exercise physiology see Astrand et al [1], Martin and Coe [41], Wasserman et al [65] and Wilmore and Costill [70], .

3.1 Heart rate kinematics

According to Martin and Coe [41] "If there is one single physiological variable that identifies the total stress load under which an athlete is performing, it is the heart rate". Heart rate monitors are now commonly used by athletes to monitor the training intensity (see Burke [19], Martin and Coe [41], Daniels [22] and Mille-Hamard et al [43]), the bodies response to training sessions with increased fitness levels and the over all health status of the athlete (ie. overtraining and excessive levels of tiredness) found for example via the resting heart rate levels. These monitors are capable of recording beat to beat intervals for the heart and allow one to down load this information for analysis as a time series to a computer, see figures 1 and 2.

From figure 1 we can see that assuming the distance covered in all of the 3 minute repetitions was the same and other conditions (see 3.1.3) were equal then there are two obvious conclusions. Firstly that as the training session progresses then it becomes more demanding for the body, this is reflected in the increase in both the peak heart rate attained during a repetition and the minimum heart rate achieved during the 3 minute recovery. Secondly as the second time series has a lower heart rate through out the whole training session than the first then the body was more able to cope with the exercise and hence the athlete is getting fitter. We can also see when we compare figures 1 and 2 that the time series for the heart rate reflects the particular training session, hence the differences in the two figures.

3.1.1 On the relationship between HR and $\dot{V}O_2$

Functionally we have two hearts, with the right heart delivering blood to the lungs and the left everywhere else. The two primary operating variables

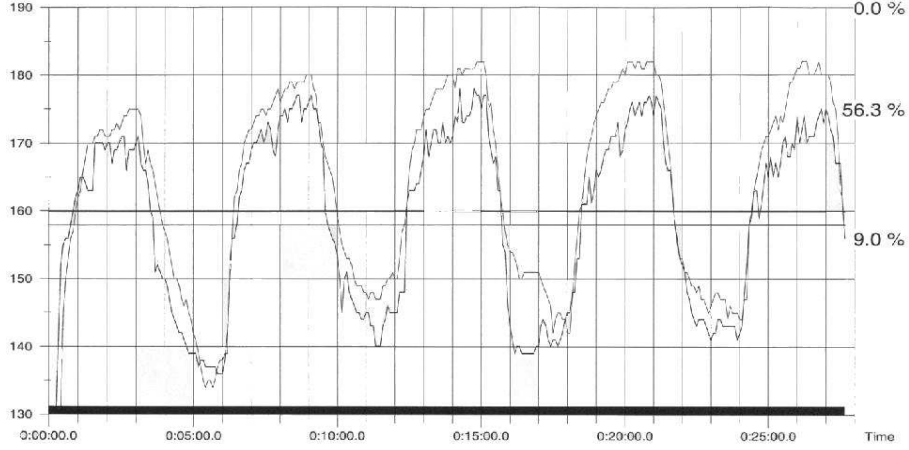


Figure 1: Heart rate data of the author, (5 second average) from two separate training session of 5 repetitions of 3 minutes at $v\dot{V}O_{2max}$ with 3 minutes recovery at $50\%v\dot{V}O_{2max}$ between repetitions.

of each heart are the heart rate, HR and the volume pumped per beat, the stroke volume, Q_s . The cardiac output is the product of heart rate and stroke volume

$$CO = HR \cdot Q_s \quad (\text{ml or L of blood}) \quad (1)$$

The cardiac output must equal venus return as the heart can only deliver to the arterial side of the body blood which returns to it from the venus side. At rest venus return is controlled by 4 principle factors: the tone or caliber of the venus vessels, the position of the body in space, the total blood volume and the depth of breathing. Upon exercising the milking action of the skeletal muscles help push blood through the veins towards the heart.

When blood is sampled from the coronary arteries and from the coronary venus sinus and analyzed for its O_2 content, we find a very large amount of O_2 has been removed, (termed the arteriovenous O_2 difference $a\bar{V}O_2^d$, ie. the difference between the mixed venus blood O_2 concentration and the arterial blood O_2 concentration).

It is found that the maximum heart rate HR_{max} is either unchanged or slightly reduced by endurance training, and the maximum value of the

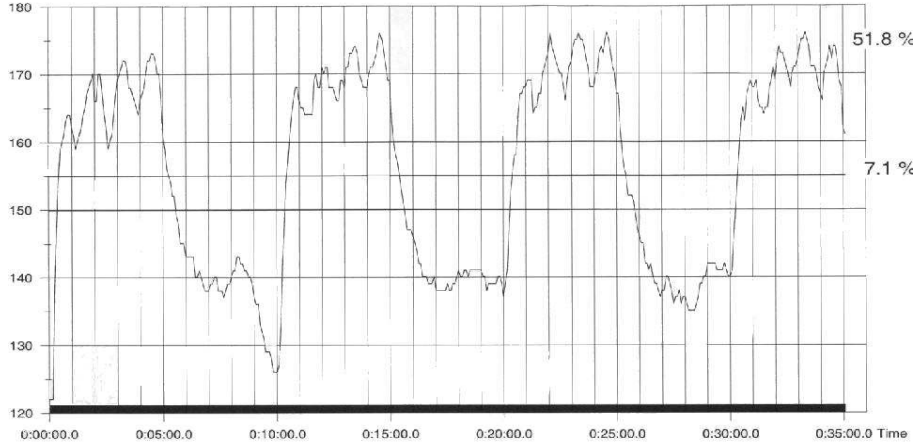


Figure 2: Heart rate data of the author, (5 second average) from a training session of 4 sets of 4 repetitions of 45s at maximum speed with 30 seconds recovery jogging between repetitions and 5 minutes recovery jogging between sets.

arteriovenous O_2 difference $a\bar{V}O_2^d$ peaks at about $16ml/dl$. It is observed that in the trained heart as a result of cardiac chamber enlargement, the stroke volume at any given work load is larger and the heart rate is lower, hence increasing the available perfusion time, resulting in an increase in the bodies aerobic power.

$\dot{V}O_2$ is equal to the product of cardiac output CO and the O_2 extracted from the blood, $a\bar{V}O_2^d$.

$$\dot{V}O_2 = HR \cdot Q_s \cdot a\bar{V}O_2^d \quad (2)$$

This relationship between the HR and $\dot{V}O_2$ is relatively simple, though not trivial as neither Q_s nor $a\bar{V}O_2^d$ are constant. Both Q_s and $a\bar{V}O_2^d$ are functions of exercise intensity, fitness and time. It is believed that the cardiac output, CO and heart rate HR normally increase linearly with $\dot{V}O_2$ during increasing work rate exercise. Notice this linear relationship can only be true if we reach HR_{max} at the same time as we reach $\dot{V}O_{2max}$, which isn't necessarily the case. Swain et al [62, 63] found the $\%HR^{res}$ is approximately equal to the $\%\dot{V}O_2^{res}$ on average for a large population of runners, (where

the reserve for the heart rate $\%HR^{res} = \frac{HR(t)-HR_{min}}{HR_{max}-HR_{min}} \cdot 100\%$ and $\%\dot{V}O_2^{res} = \frac{\dot{V}O_2(t)-\dot{V}O_{2min}}{\dot{V}O_{2max}-\dot{V}O_{2min}} \cdot 100\%$, with HR_{min} and HR_{max} being the minimum and maximum heart rates respectively and $\dot{V}O_{2min}$ $\dot{V}O_{2max}$ being the minimum and maximum $\dot{V}O_2$ respectively). This relationship however needs to be understood on an individual basis as the deviation from the $\%HR^{res}$ vs $\%\dot{V}O_2^{res}$ curve is large.

3.1.2 A model for $HR(t)$

It has been found that the heart rate HR , like the $\dot{V}O_2$, is an exponential like function of both the speed (or intensity) V , and the time t . The relationship is such that the HR at which we plateau out at (i.e. $\dot{HR} = 0$) given sufficient time is determined by the intensity of the exercise and an additional slow component and drift. In Engelen etal [26] (see also Linnarsson [39]) they model both the on and off kinetics for the $\dot{V}O_2$ and HR response to exercise. Here we look at the on/off response for the HR . The 'on' response corresponds to how the heart rate reacts to an increase in the intensity of the exercise to a new higher level intensity. HR_{on} is modelled as

$$\begin{aligned} HR_{on} = & HR_b + A_0(1 - e^{-\frac{t}{\tau_0}}) \quad \textit{phase1} \\ & + A_1(1 - e^{-\frac{(t-TD_1)}{\tau_1}}) \quad \textit{phase2} \\ & + A_2(1 - e^{-\frac{(t-TD_2)}{\tau_2}}) \quad \textit{slow component} \end{aligned} \quad (3)$$

The off kinetics correspond to how the heart rate reacts to a decrease in the intensity of the exercise to a new lower level of intensity. HR_{off} is modelled as

$$\begin{aligned} HR_{off} = & HR_{End_{ex}} - 0 \quad \textit{phase1} \\ & - A_1(1 - e^{-\frac{(t-TD)}{\tau_1}}) \quad \textit{phase2} \\ & - A_2(1 - e^{-\frac{(t-TD)}{\tau_2}}). \quad \textit{slow component} \end{aligned} \quad (4)$$

The parameters are very similar to those used used to model the on and off $\dot{V}O_2$ kinetics (see section 3.2.5 for a description). The magnitude of the

parameters reflects the intensity of the exercise. Note there is no rapidly decreasing phase 1 term unlike in the off kinematics for the $\dot{V}O_2$. However like in the off kinematics for the $\dot{V}O_2$ the time delay is common, for the same reasons as described there (see section 3.2.5).

3.1.3 Other factors effecting the heart rate

Heart rate can be effected by many other variables, other than the intensity of the exercise. Some of these variables can be controlled and some cannot hence to understand the heart rate dynamics one needs to understand how the following variables may effect the time series.

Temperature - High temperature and humidity causes an elevation in the heart rate. Heart rate shows the lowest value at an outside temperature of about 20 degrees centigrade. High surrounding temperate and air humidity place greater demands on the body during physical exercise. For equal work loads an increase in the surrounding temperature and or humidity will result in an increased heart rate. This is because the demands on the bodies heat regulation system are increased, requiring increased production of perspiration, and an increased blood flow in the capillaries of the skin. To do this we require an increase in the fluid loss. This decreases the circulating blood volume and diminishes the blood supply to the heart, which the body will compensate for by increasing the heart rate. This shows why during extended exercise efforts it is essential to intake fluids and cool the body sufficiently so as to prevent dehydration and hence enable peak performances. At equal sub maximal efforts it is often assumed that for each degree centigrade rise in temperature we have an approximate 10 to 15 beats per minute increase in heart rate. Obviously at maximal efforts when the heart rate is at HR_{max} we cannot increase the heart rate. It should also be noted that in hot and humid conditions the time an athlete can remain exercising at a particular intensity will also decrease.

Age - with age the HR_{max} gradually decreases.

Over-training, insufficient recovery and illness - Depending on the type of over-training the morning pulse may be higher or extremely low. As a result of over-training a lag in the increase of the heart rate HR with high intensity exercise may be observed. Another result of over-training is the fact that the maximum heart rate HR_{max} may no longer be obtainable. Over-training therefore results in a completely different heart rate response both during exercise and at rest.

Nutrition and hydration- Adequate nutrition and hydration can improve

performance in endurance events resulting in a lower HR at a given intensity.

Altitude - HR_{min} decreases upon the first few hours after an athlete arrives at high altitude. This is however followed by an increase, which is approximately 10% at 2000m and 45% at 4500m when compared with sea level values for HR_{min} . Depending on the altitude the HR_{min} will return to sea level values or lower after some days acclimatization. In fact this is used as a good sign of acclimatization.

Medication - Various medications can effect the heart rate (ie. beta blockers have been shown to decrease both the HR_{min} and the HR_{max}).

Infectious disease - A fever can lead to an increase in the HR_{min} of 10 to 15 beats for every degree temperature rise. Also during sub-maximal efforts the HR shows a different pattern. HR_{min} is also increased during recovery from infectious deceases.

Mental activity - Mental stress can effect HR .

3.2 $\dot{V}O_2$ kinematics

$\dot{V}O_2$ can be defined as the volume of oxygen we breath in (uptake) per unit time. $\dot{V}O_{2max}$ is the maximal limit of the ability to increase $\dot{V}O_2$, further increase in intensity do not yield a larger $\dot{V}O_2$. This is referred to as the aerobic capacity, maximal oxygen uptake or $\dot{V}O_{2max}$. Below the lactate threshold, LT (see section see section 3.3.1 for a definition and discussion of the lactate threshold) the rate of increase in $\dot{V}O_2$ uptake and demand is an approximately linear function of exercise intensity, whilst above the lactate threshold the function is nonlinear.

The $\dot{V}O_2(t)$ can be measured as a time series using breath by breath analysis equipment. Such recording are often taken in a lab whilst the athlete runs on a treadmill at various speeds however portable devices are also now available. It is also common to measure the volume of other gases other than the O_2 in each intake our outtake of breath. In this way it is possible to measure the chemical content of the gases we inspire and expire.

Figure 3 shows the on transient kinetics of $\dot{V}O_2(t)$ for four different intensities. It can be seen that moderate intensity shows an exponential like rise and plateau. The same can be seen in the response to a heavy exercise intensity, though this time there is a delay in reaching the plateau or steady state $\dot{V}O_2$. For very heavy intensity exercise the plateau is the $\dot{V}O_{2max}$, there is also a delay in reaching this plateau. For the severe intensity exercise the $\dot{V}O_2(t)$ rises very steeply until it is limited by the $\dot{V}O_{2max}$, which then becomes the plateau for remaining time the exercise can be carried out at.

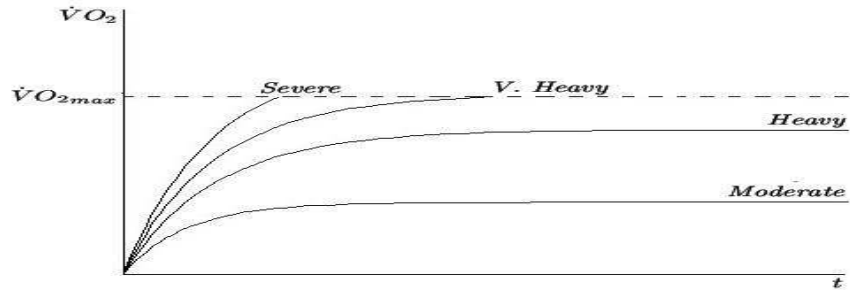


Figure 3: A Sketch of the on transient kinetics of $\dot{V}O_2(t)$ time series response to four different exercise intensities. Where moderate intensity exercise is below the LT , heavy is below the critical velocity v_{crit} (see section 3.4), very heavy is such that the oxygen demand is slightly greater than or equal to the $\dot{V}O_{2max}$ and severe is such that the oxygen demand is far greater than the $\dot{V}O_{2max}$.

Note if the exercise is to severe then the athlete may have to stop due to fatigue before they reach the $\dot{V}O_{2max}$. Figure 4 shows the off transient kinetics for the $\dot{V}O_2(t)$. In other words it shows how the $\dot{V}O_2(t)$ reduces as the body recovers to a new lower oxygen demand following exercise of different intensities.

3.2.1 Anaerobic capacity or oxygen deficit

The oxygen deficit is defined as the integral with respect to time of the difference between the oxygen demand for that particular exercise intensity and the oxygen uptake during the whole exercise bout (for a review see Saltin [56, 57], and Bangsbo [3, 4, 5]). It is used to quantify the anaerobic energy contribution (see section 3.3 for definition of the anaerobic energy contribution) to the work performed (see Saltin [56, 57], Bangsbo [3, 4, 5] and Medbo [42]). In short term non intense exercise below the lactate threshold, there is no problem, the problem arises during intense exhaustive exercise.

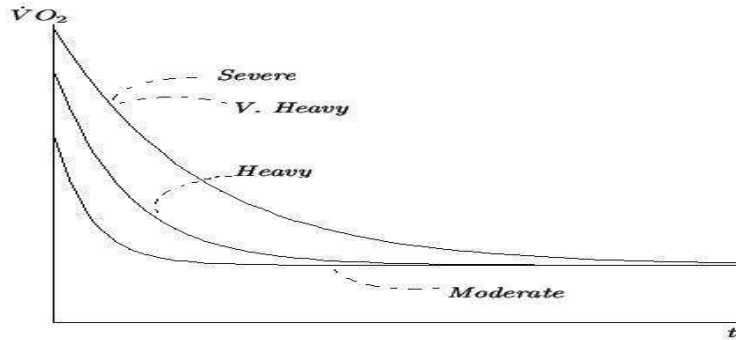


Figure 4: A Sketch of the off transient kinetics of $\dot{V}O_2(t)$ time series response to recovery (ie. to a lesser oxygen demand) from four different exercise intensities.

If the work exhausts the subject in a short duration it is likely that the maximum oxygen deficit is reached. Energy costs or oxygen demands must be accurately known to calculate the oxygen deficit. This is not difficult at sub maximal work loads below the lactate threshold where the steady state oxygen uptake (ie the plateau, see in the $\dot{V}O_2$ vs t curve) represents the oxygen demand. At exhaustive exercise, however the nature of the true cost is more complicated. The problem being that we cannot extrapolate from the sub maximal linear relationship as this would underestimate the oxygen deficit. We also cannot assume that the mechanical efficiency the subject remains constant with increasing intensity. It can be shown that the higher the intensity of the exercise the lower the mechanical efficiency of the athlete (see Saltin [56, 57], and Bangsbo [3]).

It can be shown that exercise involving more muscle mass results in a higher oxygen deficit. This can be seen in the fact that whole body exercise such as swimming or rowing have a maximal oxygen deficit more than 50% larger.

3.2.2 Three phase for pulmonary $\dot{V}O_2$

Hill and Lupton [31], recognized that the pulmonary $\dot{V}O_2$ rises approximately exponentially following the onset of moderate exercise. With more modern tools (breath by breath analysis), three phases can be observed in the rise in of $\dot{V}O_2$ (see Whipp [68]) and 'excessive' averaging. Phase 1 occurs in the first 15 to 25 seconds. The rise in $\dot{V}O_2$ is believed to be due primary to increased cardiac output and secondarily to changes in mixed venous O_2 content and lung gas stores. Phase 2 follows this, and in this phase there is an exponential rise to a steady state level of $\dot{V}O_2$ for moderate exercise below the lactate threshold, LT . Phase 3 represents the new steady state level of $\dot{V}O_2$, when it is achieved (ie. below LT). For exercise intensities above that corresponding to the lactate threshold however the $\dot{V}O_2$ kinetics is more complicated than the mono exponential model described above for moderate (below LT) intensities of exercise. Instead we have an additional slow component.

Hill and Stevens [33] conclude that “ at the onset of exercise in the severe intensity domain, $\dot{V}O_2$ is initially driven toward the O_2 demand, and then is limited by the achievable $\dot{V}O_2$ $\dot{V}O_{2_{peak}}$ ” Where $\dot{V}O_{2_{peak}} = \dot{V}O_{2_{max}}$ unless we have premature fatigue (see also the curve labelled severe in figure 3).

3.2.3 $\dot{V}O_2$ slow component

Below the LT , $\dot{V}O_2$ reaches a steady state after 3 minutes of constant work rate exercise (Davies, di Pampero, and Cerretelli, [23], Linnarsson [39], Whipp and Wassermann [67]) because the rate of ATP (see section 3.3 for more on ATP) utilization and aerobic regeneration of ATP becomes equal. The $\dot{V}O_2$ response at higher rates of work does not follow the steady state response (ie. a plateau). Instead at power outputs above the lactate threshold the oxygen consumption continues to increase either to the $\dot{V}O_{2_{max}}$ or to a delayed steady state $\dot{V}O_2$, i.e. $\dot{V}O_{2_{ss}}$. This slow drift upwards in $\dot{V}O_2$ during constant work rate exercise has been called the slow component of oxygen uptake kinetics. It has been speculated that this slow component is probably associated with ventilation, shifting of metabolic substrate from fat to carbohydrate, and or and increase in body temperature. While others believe the most likely mechanism however is an alteration in muscle fiber recruitment patterns, with the recruitment of more fast muscle fibers which are less efficient (see Gaesser and Poole [27] for a review).

3.2.4 $\dot{V}O_2$ drift

This is a similar but likely unrelated phenomena to that of the $\dot{V}O_2$ slow component. The phenomena is defined as a slow increase in $\dot{V}O_2$ during prolonged, sub maximal, constant power output exercise. Unlike the slow component $\dot{V}O_2$ drift is observed at power outputs well below lactate threshold and the magnitude of the increase in $\dot{V}O_2$ is much less. It is believed, to be due to an increase in ventilation, an increase in levels of circulating catecholamines and thermo regulatory constraints, see Wilmore and Costill [70].

3.2.5 Characterization of $\dot{V}O_2$ kinematics as a function of exercise intensity, Barstow [6]

During moderate exercise $\dot{V}O_2$ reaches a new steady state within 180 seconds in normal subjects, with little or non rise in blood lactate. The steady state $\dot{V}O_2$ increases linearly with work-rate. The time constant in phase 2 (after the first 15 – 20 seconds) is constant across work intensities and appears to reflect muscle oxygen utilization kinetics. However when heavier exercise is performed, which elevates blood lactate through the exercise, the $\dot{V}O_2$ response becomes more complex. The predominant phase 2 response continues to rise exponentially with about the same time constant as for moderate exercise, and the amplitude continues to be linearly related to work rate. However an additional slowly developing rise in $\dot{V}O_2$ is also usually observed, beginning 100 – 200 seconds into exercise. This additional $\dot{V}O_2$ delays attainment of a steady state, increasing the overall O_2 “cost” of the exercise and is statistically associated with the rate and magnitude of increase in blood lactate. Interestingly in children (and in some elite athletes see Billat [14]), neither the slow component nor the blood lactate rise as much during heavy exercise.

$\dot{V}O_2$ has been modelled in Barstow [6] using the following triple exponential form (see also figure 5).

$$\begin{aligned} \dot{V}O_2 = & \dot{V}O_2(0) + A_0^{on}(1 - e^{-\frac{t}{\tau_0^{on}}}) \quad \textit{phase1} \\ & + A_1^{on}(1 - e^{-\frac{(t-TD_1^{on})}{\tau_1^{on}}}) \quad \textit{phase2} \\ & + A_2^{on}(1 - e^{-\frac{(t-TD_2^{on})}{\tau_2^{on}}}) \quad \textit{slow component} \end{aligned} \tag{5}$$

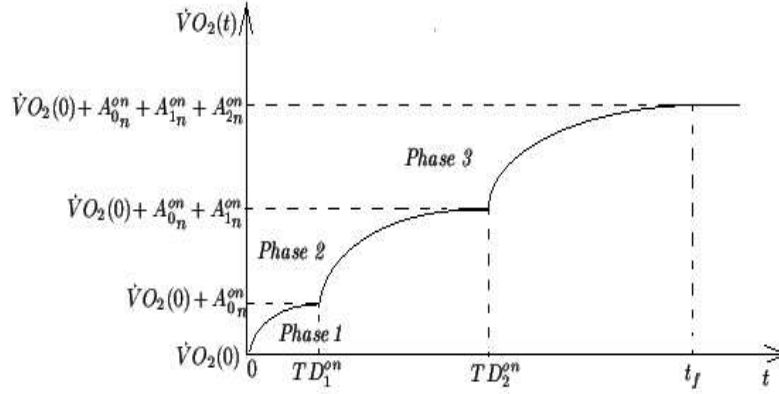


Figure 5: A sketch showing the 3 phases used to model oxygen uptake kinetics.

where $\dot{V}O_2(0)$ is the initial value of the $\dot{V}O_2$ at time $t = 0$. A_0^{on} , A_1^{on} and A_2^{on} are the asymptotic values, τ_0^{on} , τ_1^{on} and τ_2^{on} are the time constants, and TD_1^{on} and TD_2^{on} are time delays for each exponential term. We define $A_{0n}^{on} = A_0^{on}(1 - e^{-\frac{TD_1^{on}}{\tau_0^{on}}})$, $A_{1n}^{on} = A_1^{on}(1 - e^{-\frac{-(TD_2^{on}-TD_1^{on})}{\tau_1^{on}}})$ and $A_{2n}^{on} = A_2^{on}(1 - e^{-\frac{-(t_f-TD_2^{on})}{\tau_2^{on}}})$. The 3 phase model is such that after $t = TD_1^{on}$ we terminate phase 1 and put $\dot{V}O_2(TD_1^{on}) = \dot{V}O_2(0) + A_{0n}^{on}$, the second phase then starts and is added to $\dot{V}O_2(TD_1^{on})$. Phase 2 is terminated after $t = TD_2^{on}$, giving $\dot{V}O_2(TD_2^{on}) = \dot{V}O_2(0) + A_{0n}^{on} + A_{1n}^{on}$, the third phase then starts and is added to $\dot{V}O_2(TD_2^{on})$. Phase 3 is terminated after $t = t_f$ giving $\dot{V}O_2(t_f) = \dot{V}O_2(0) + A_{0n}^{on} + A_{1n}^{on} + A_{2n}^{on}$. Note that below the LT there is no slow component or phase 3 and hence the model will have only 2 phases. Note also when phase 1 is active phase 2 and the slow component are inactive due to the time delays TD_1^{on} and TD_2^{on} respectively. Phase 2 becomes active after $t = TD_1^{on}$, which is approximately 10 seconds, followed by the slow component after $t = TD_2^{on}$, which is approximately 180 seconds.

It should also be noted that many models have $A_0^{on} = 0$ as this initial cardiodynamic first phase does not directly represent muscle O_2 utilization, (Krogh and Lindhard [37]), instead they start the modelling after TD_1^{on} .

Also $TD_1^{on} = TD_2^{on}$ as its not necessarily proven that the slow component should really start after a different time delay than phase 2. In Engelen et al [26] and Ozyener etal [49] they model the off exercise kinematics as well, in the following form

$$\dot{V}O_2 = EE\dot{V}O_2 - A_0(1 - e^{-\frac{t}{\tau_0}}) - A_1(1 - e^{-\frac{(t-TD)}{\tau_1}}) - A_2(1 - e^{-\frac{(t-TD)}{\tau_2}}) \quad (6)$$

where the parameters are as before apart from $EE\dot{V}O_2$ which is the end exercise level of $\dot{V}O_2$ and the time delay $TD_1 = TD_2 = TD$ as the phase 2 dynamics and the slow component begin at different times during the intense exercise, but in the off phase they exist together at the time the off phase begins. Phase 1 is believed to be present in both the on and off responses to exercise.

3.2.6 Oxygen deficit solutions

In Bearden and Moffatt [7] they define the oxygen deficit in two ways, the traditional way $O_{2_{trad}}^{defi}$ and their modified way $O_{2_{b\&m}}^{defi}$, from equations 5.

$$\begin{aligned} O_{2_{trad}}^{defi} &= (\dot{V}O_2(0) + A_{0_n}^{on} + A_{1_n}^{on} + A_{2_n}^{on}).(t_f - t_0) - \int_{t_0=0}^{t_f} \dot{V}O_2 dt \\ O_{2_{b\&m}}^{defi} &= (\dot{V}O_2(0) + A_{0_n}^{on} + A_{1_n}^{on} + A_{2_n}^{on}).(t_f - t_0) - (A_{2_n}^{on}.TD_2^{on}) - \int_{t_0=0}^{t_f} \dot{V}O_2 dt \\ \int_{t_0=0}^{t_f} \dot{V}O_2 dt &= \dot{V}O_2(0).t_f + A_{0_n}^{on}(t_f - TD_1^{on}) + A_{1_n}^{on}(t_f - TD_2^{on}) + \\ &+ \int_{t_0=0}^{TD_1^{on}} A_0^{on}(1 - e^{-\frac{t}{\tau_0^{on}}}) dt + \int_{TD_1^{on}}^{TD_2^{on}} A_1^{on}(1 - e^{-\frac{(t-TD_1^{on})}{\tau_1^{on}}}) dt + \\ &+ \int_{TD_2^{on}}^{t_f} A_2^{on}(1 - e^{-\frac{(t-TD_2^{on})}{\tau_2^{on}}}) dt. \\ O_{2_{trad}}^{defi} &= -A_0^{on}.TD_1^{on}.e^{-\frac{TD_1^{on}}{\tau_0^{on}}} + A_1^{on} \left(TD_1^{on} - TD_2^{on}.e^{-\frac{TD_2^{on}-TD_1^{on}}{\tau_1^{on}}} \right) + \\ &+ A_2^{on} \left(TD_2^{on} - t_f.e^{-\frac{t_f-TD_2^{on}}{\tau_2^{on}}} \right) - A_0^{on}.\tau_0^{on} \left(e^{-\frac{TD_1^{on}}{\tau_0^{on}}} - 1 \right) - \\ &- A_1^{on}.\tau_1^{on} \left(e^{-\frac{TD_2^{on}-TD_1^{on}}{\tau_1^{on}}} - 1 \right) - A_2^{on}.\tau_2^{on} \left(e^{-\frac{t_f-TD_2^{on}}{\tau_2^{on}}} - 1 \right) \end{aligned} \quad (7)$$

$$\begin{aligned}
O_{2b\& m}^{defi} = & -A_0^{on} \cdot TD_1^{on} e^{-\frac{TD_1^{on}}{\tau_0^{on}}} + A_1^{on} \left(TD_1^{on} - TD_2^{on} \cdot e^{-\frac{TD_2^{on} - TD_1^{on}}{\tau_1^{on}}} \right) + \\
& + A_2^{on} (TD_2^{on} - t_f) e^{-\frac{t_f - TD_2^{on}}{\tau_2^{on}}} - A_0^{on} \cdot \tau_0^{on} \left(e^{-\frac{TD_1^{on}}{\tau_0^{on}}} - 1 \right) - \\
& - A_1^{on} \cdot \tau_1^{on} \left(e^{-\frac{TD_2^{on} - TD_1^{on}}{\tau_1^{on}}} - 1 \right) - A_2^{on} \cdot \tau_2^{on} \left(e^{-\frac{t_f - TD_2^{on}}{\tau_2^{on}}} - 1 \right)
\end{aligned} \tag{8}$$

If we look at solutions where we assume we have neither the first or third phase then we have $A_0 = A_2 = TD_1 = TD_2 = 0$ which gives $O_2^{defi} = A_1 \cdot \tau_1 \left(1 - e^{-\frac{t}{\tau_1}} \right)$ which when taken in the limit that t approaches infinity gives $O_2^{defi} = A_1 \cdot \tau_1$. Such solutions where there is no slow component work for intensities below the lactate threshold and for very intense exercise where the oxygen demand is in excess of the $\dot{V}O_{2max}$, see Whipp [68].

3.2.7 Solutions for the time spent at $\dot{V}O_{2max}$

Billat et al [12] used the constant velocity mono exponential version to calculate the time spent at $\dot{V}O_{2max}$, (i.e. $A_0 = A_2 = 0$) solving equation 5 for t gives

$$t = -\tau \ln \left[1 - \frac{\dot{V}O_2(t) - \dot{V}O_2(0)}{A_1} \right] \tag{9}$$

which is the time taken to arrive at $\dot{V}O_2(t)$. For $\dot{V}O_2(t) = \dot{V}O_{2max} - \delta$ the is term t is called the time to arrive within δ of $\dot{V}O_{2max}$, $TA_{\dot{V}O_{2max} - \delta}$. We now use this and the total time spent exercising, $tlim$ to calculate the total time above $\dot{V}O_{2max} - \delta$, which we term the $tlim_{\dot{V}O_{2max} - \delta}$

$$tlim_{\dot{V}O_{2max} - \delta} = tlim - TA_{\dot{V}O_{2max} - \delta}. \tag{10}$$

In this manner we can compare the time spent above $\dot{V}O_{2max} - \delta$ when exercising at other exercise intensities, if it is believed they can be modelled sufficiently well with the mono exponential function. For the full three phase work, we need to account for the time delay, $TD2$ before the start of the slow component. We look at the solution for an exercise duration greater than $TD2$

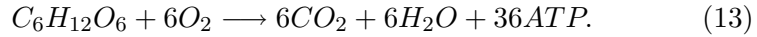
$$\dot{V}O_2(t) = A_1 + A_2(1 - e^{-\frac{(t-TD_2)}{\tau_2}}) \quad (11)$$

$$t = TD_2 - \tau_2 \ln \left[1 - \frac{\dot{V}O_2(t) - A_1}{A_2} \right] \quad (12)$$

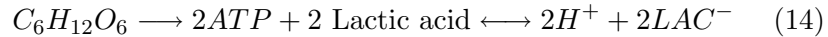
Where as before we let $\dot{V}O_2(t) = \dot{V}O_{2max} - \delta$ hence t becomes $TA_{\dot{V}O_{2max} - \delta}$. The time spent above $\dot{V}O_{2max} - \delta$ can then be found as before by using equation 10. This shows the limitations in the current model (i.e. equation 5) as we have to be able to find TD_2 before we can predict the $lim_{\dot{V}O_{2max} - \delta}$ and to do this requires a time series for that intensity. It would be better if we could predict such quantities for other velocities other than those we have data for, the same goes for all the other parameters. This point has been addressed in Stirling et al [60] along with other issues regarding the smoothness of the model.

3.3 Blood lactate $BLa(t)$ kinematics

To understand what blood lactate is we need first to understand the chemical equations for aerobic and anaerobic metabolism in the production of energy. The energy released from complete cellular breakdown of fuels is stored in the form of adenosine triphosphate (ATP). Below we present the aerobic breakdown (i.e. in the presence of oxygen) of a carbohydrate such as glucose is given by



This is a highly efficient process which results in enormous amounts of energy for work (ie. $36ATP$). Where as the anaerobic breakdown (i.e. in the absence of oxygen) of glucose



which is far less efficient (ie. only $2ATP$ as apposed to 36 is produced). Under physiological conditions the lactic acid produced dissociates almost immediately and completely lactate ions (Lac^-) and hydrogen ions (H^+). This eventually inhibits the enzymatic breakdown sequence as the enzymes involved in fuel breakdown operate within a narrow range of acidity.

The accumulation of H^+ ions causes much discomfort leading to an eventual reduction in the intensity of the exercise. Anaerobic metabolism is the main source of energy for very intense short duration exercises, ie. a 400 meter race, races over 1500 meters are have approximately a 50 : 50 balance between the two forms of metabolism and as the duration of the race increases then so does the percentage of the aerobic contribution. Due to the difference in the energy production in aerobic and anaerobic metabolism the more the energy demand can be met by aerobic metabolism the more efficient the process. This is one of the effects of increased fitness.

The concentration of blood lactate during incremental exercise reflects the difference between the release of lactate from the muscle (muscle efflux) and the uptake of blood lactate by muscle and other tissue. This means that the concentration of blood lactate doesn't necessarily reflect muscle lactate production. As a result it should be noted that the accumulation of blood lactate per se does not play an underlying role in the alteration of metabolic processes during exercise, (for a review see Brooks [17, 18]). However it is still believed that blood lactate concentration is a useful tool for predicting endurance performance and intensity of exercise training programs. One very common training session for example is 1 or 2 repetitions of 15 to 20 minutes at a velocity corresponding to the lactate threshold LT with a warm up and warm down and 5 minutes recovery easy running between the repetitions. The blood lactate is commonly measured at discrete intervals in the time series in exercise physiology labs along with the heart rate, $\dot{V}O_2$ and velocity while doing physiological testing.

3.3.1 *BLa* terminology: Lactate threshold and maximum lactate steady state

There are two obvious features of the blood lactate kinematics which are of interest from both a practical and mathematical point of view. One is the lactate threshold LT and the other is the individual anaerobic threshold, IAT . The lactate threshold, LT is not really a threshold in mathematical sense of the word as there is no abrupt change, however in exercise physiology it is defined as the point at which blood lactate begins to accumulate, above resting levels during exercise of increasing intensity. This can be seen as the point at which the gradient of the *BLa* vs V curve begins to rapidly increase its gradient, see figure 6. Another way of defining it is the highest $\dot{V}O_2$ or HR that can be attained during incremental exercise before an elevation in blood lactate is observed. This is also called the lactate break point,

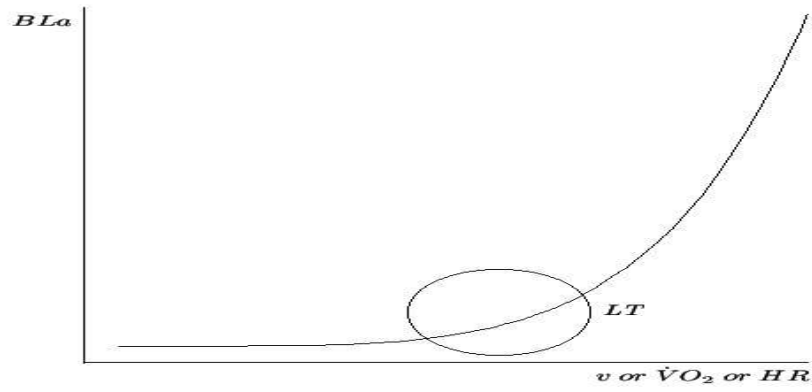


Figure 6: A sketch of the BLa vs v or HR or $\dot{V}O_2$, highlighting the lactate threshold, LT . Either of v , $\dot{V}O_2$ or HR are commonly used as the x axis variable.

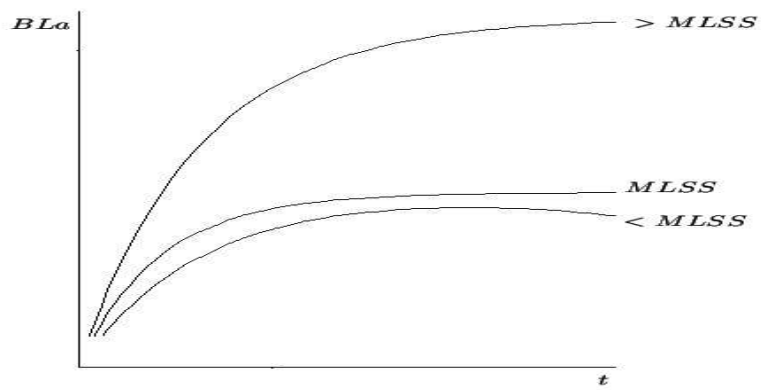


Figure 7: A sketch of the BLa vs t highlighting the maximum lactate steady state, $mlss$.

the onset of plasma lactate accumulation, the anaerobic threshold and the aerobic threshold. The individual anaerobic threshold, *IAT* is defined as the highest $\dot{V}O_2$ or *HR* that can be maintained over time (15 to 20 minutes) without a continual increase in the blood lactate, see figure 7. The term maximal lactate steady state, *MLSS* is also used. The definition of this term is far more rigorous from a mathematical point of view than that of the *LT*, however both terms are commonly used.

With training the lactate threshold occurs at a higher speed allowing the runner to run faster without accumulating lactate in the blood. This results from a greater ability to clear lactate from the muscles and also less lactate begin produced for the same work rate. The ability to exercise at a high intensity without accumulating lactate is beneficial to the athlete because lactate formulation contributes to fatigue.

3.3.2 The relationship between the ventilatory threshold and lactate threshold.

The so called thresholds for both the ventilation and lactate accumulation have been shown to occur together with a high correlation for metabolically normal people. (examples and references are given in Martin and Coe [41]). There is confusion regarding the terminology as there are two thresholds, and many names to describe them. The first threshold is observed during mild work (accompanied by breathing changes and a small rise in blood lactate concentration) the second threshold however occurs during more intense exercise (accompanied by breathing changes and and a steady accumulation of lactate).

The second threshold is the one often sensed by runners as a change in the breathing intensity such that the conversation stops. At the intensity of work which results in rapidly accumulating lactate levels elevated ventilatory removal of CO_2 can no longer maintain blood acidity within reasonable limits. As blood lactate levels rapidly rise the Ph of the blood begins to fall and the rising H^+ concentration provides a powerful ventilatory stimulus.

Gaesser and Poole [27] stated that “although the blood lactate profile is well correlated with the time course and magnitude of the $\dot{V}O_2$ slow component, the relationship is coincidental rather than causal. The most likely mechanism accounting for the slow component is muscle fibre recruitment”. There also exists work however which dispute the coincidental relationship between a blood lactate threshold and the ventilatory threshold. Examples and references of work in which it was found that the anaerobic and lactate

threshold do not coincide are show in the book of Weltman [66]. Weltman concludes that “recent studies have examined the anaerobic, ventilatory and lactate thresholds The results indicate that the three thresholds are determined by different underlying mechanisms and reflect different phenomena.”

3.3.3 *BLa vs t*

It can be shown that for low values of exercise intensity there is a minimal increase, if any, in blood lactate concentration with time. There are even some cases where the concentration decreases during low-intensity exercise as blood lactate is used as a substrate. For higher intensity exercise a velocity or power output is reached above which the blood lactate concentration is a nonlinear increasing function of time.

3.3.4 *BLa vs velocity*

It can be shown that blood lactate is an exponential function of intensity or velocity V . If incremental exercise is carried out it can be seen that for low intensities there is no increase from the base or resting levels of blood lactate concentration. This is so up until the lactate threshold, after which for increasing intensities of exercise we get an exponentially increasing value of the blood lactate concentration.

In all anaerobic training it is of value to have active rest, ie. low intensity exercise not exceeding a work intensity demanding above 60% of $\dot{V}O_{2max}$. lactate is then transferred quicker from the fatigued muscles because the perfusion of the muscle is maintained at a higher level than at rest. A prerequisite is that the rest exercise intensity is low enough not to cause further lactate formation (ie. must be below the lactate threshold). Note that even faster returns to pre-exercise blood lactate response could be noticed at relative work loads up to 80% of maximal oxygen uptake, $\dot{V}O_{2max}$ in endurance trained individuals, this is probably below there lactate threshold. Note the difference in the shape of the curves for different recovery intensities, (see figure 8), with 0% being approximately linear and 60% to 80% being exponential, with 80% having the steepest drop in BLa, see Saltin [57].

3.3.5 *BLa and heart rate or $\dot{V}O_2$ relationship*

The relationship between the blood lactate concentration and the $\dot{V}O_2$ has been subject to debate (see Myers etal [45]). Essentially the arguments

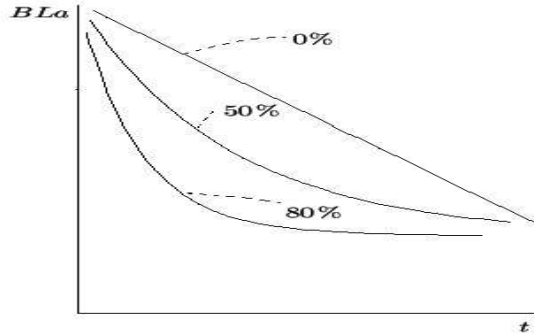


Figure 8: A sketch of the blood lactate time series for recovery at 3 different exercise intensities (given as a $\% \dot{V}O_{2max}$) from and elevated blood lactate level following hard exercise

are related to whether a continuous function can describe the blood lactate kinematics for tests of increasing intensity. Beaver et al [8] used a regression of the log of the lactate vs the log of the $\dot{V}O_2$ to try to highlight the lactate threshold. The lack of a threshold however was reported in Yeh et al [71]. It was found in Hughson et al [35] that the difference in fit between the two models is minor and it was believed the continuous model was a more appropriate model. This continuous model also described in Myers et al [45] is an exponential function in the following form,

$$BLa = a + b.e^{c.\dot{V}O_2} \quad (15)$$

where a, b, c are parameters. This shows that the blood lactate can be modelled as an exponential function of $\dot{V}O_2$ (and hence HR), for ramp exercise tests. It should be noted that this function appears similar though not identical to the dependency of the blood lactate on the exercise intensity of velocity, V . In fact many figures interchange the velocity, with the $\dot{V}O_2$ as the x axis measure of intensity.

Note that Billat [10] provides a method of determining the maximum lactate steady state, based on an interpolation between a value above and

one below this state, see Billat [10] for further details.

3.4 Speed: v_{mara} , v_{mlss} , v_{crit} and the minimum velocity to achieve $v\dot{V}O_2^{max}$ in an incremental step test.

Speed is another variable which is recorded when carrying out physiological test. The speed at which certain physiological phenomena such as the lactate threshold LT , maximum lactate steady state or $\dot{V}O_{2max}$ occur is often calculated in such tests and then used to set training sessions and record fitness levels. Billat and Koralsztein [9], and Billat [14] provide a detailed review of the subject, including its history, uses, definitions and protocols for determining it.

Daniels et al. [21] introduced the term velocity at $\dot{V}O_2^{max}$, or $v\dot{V}O_{2max}$ which was found as the first velocity to achieve $\dot{V}O_2^{max}$ in an incremental step test with 3 minute increments. The velocity was found to be close to that in a 3000 m race (with an approximate duration of 9 minutes) for elite female runners. They reported it to be a very useful term which combined $\dot{V}O_2^{max}$ and economy into a single variable, which could be used to identify differences between various runners.

There is a range of velocities for which there is a sustained increase in blood lactate and a decrease in arterial Ph with time. $\dot{V}O_2$ increases in a mono exponential way and stabilizes at approximately 80% for at least 1.5 hours of steady exercise (in top level marathon runners). After this time period we can get an increase in $\dot{V}O_2$ due to the so called $\dot{V}O_2$ drift which is due to thermoregulatory constraints. Such effects happen at a velocity, v_{mara} which is the best velocity at which to race a marathon it is approximately 80% of $v\dot{V}O_{2max}$. If we increase the velocity to v_{mlss} which is equal to approximately 85% of $v\dot{V}O_{2max}$ the maximal lactate steady state occurs, when the lactate levels are stable as the production of lactate is equal to the consumption. The time limit at this velocity is approximately an hour.

Hill and Lupton [31] said that when using Hill as the subject, “the rate of oxygen intake due to exercise increases as speed increases, reaching a maximum for the speeds beyond about 256m/min. At this particular speed, for which no further increase in O_2 intake can occur, the heart, lungs, circulation, and the diffusion of oxygen to the active muscle-fibres have attained there maximum activity. At higher speeds the requirement of the body for oxygen is far higher but cannot be satisfied, and the oxygen debt continually increases.” They then go on to say, “Considering the case of running,

there is clearly a critical speed for each individual at which there is a genuine dynamic equilibrium, breakdown being balanced by restoration, above which, however, the maximum oxygen intake is inadequate, lactic acid accumulating, a continuously increased oxygen debt being occurred, fatigue and exhaustion setting in.”

This critical velocity, the minimum velocity to elicit maximum oxygen uptake, or $\dot{V}O_2^{max}$, was defined by Moritani et al. [44] as close to the lactate threshold velocity. Volkov et al. [64] used this minimum velocity, or critical speed to measure the maximal aerobic capacity, the total oxygen consumption at $\dot{V}O_2^{max}$ when the subject is asked to run at the critical speed until exhaustion. Billat [14] defines the critical velocity to be the highest velocity for which the $\dot{V}O_2$ can reach a, delayed, sub maximal steady state, this is true also for the blood lactate Poole et al [50, 51]. Like wise Hill and Ferguson [32] demonstrated that “the v_{crit} is the threshold intensity above which exercise of sufficient duration will lead to the attainment of $\dot{V}O_2^{max}$ ”. Hill et al. [34] use this in the following definition of the severe intensity domain “the severe exercise intensity domain may be defined as that range of work rates over which $\dot{V}O_{2_{max}}$ can be elicited during constant-load exercise...This upper boundary is the highest work rate for which exercise duration is prolonged sufficiently (in this study, $136 \pm 17s$) to allow $\dot{V}O_2$ to rise to its maximal value. The lower boundary for severe exercise is just above v_{crit} , which is the highest work rate that is sustainable for a prolonged duration and that will not elicit $\dot{V}O_{2_{max}}$ ”. v_{crit} is at approximately 90% of $v\dot{V}O_{2_{max}}$ for well trained distance runners. At this velocity Billat [14], claims there is a slow component but it doesn’t cause the $\dot{V}O_2$ to reach $\dot{V}O_2^{max}$, instead the $\dot{V}O_2$ stabilizes at a sub maximal steady state value, (i.e. for an athlete running at 90% of $v\dot{V}O_{2_{max}}$ the $\dot{V}O_2$ could stabilize at 95% $\dot{V}O_2^{max}$. As we are above the MLSS the concentration of blood lactate increases as we accumulate more lactate due to there being more produced than consumed. The time limit at this velocity is reduced to less than 30 minutes due to rapid glycogen depletion.

Above the critical velocity (i.e. during high intensity exercise) neither the blood lactate or $\dot{V}O_2$ are stabilized and hence the $\dot{V}O_2$ continues to rise till it reaches the $\dot{V}O_{2_{max}}$ unless fatigue sets in before $\dot{V}O_{2_{max}}$ can be achieved. As shown in Billat and Koralsztein [9] there are a range of velocities which will achieve $\dot{V}O_{2_{max}}$ and there are many different ways to estimate $v\dot{V}O_{2_{max}}$. What can also be seen is that they don’t get the minimum velocity to achieve $\dot{V}O_2^{max}$ as it is shown that at 90% of the $v\dot{V}O_{2_{max}}$ some subjects reach $\dot{V}O_2^{max}$ in a run to exhaustion. The reason for this is

in the way the $v\dot{V}O_{2max}$ is determined by step test where the velocity is incremented every few minutes where as the time to exhaustion at 90% of the $v\dot{V}O_{2max}$ is of the order of 10 times greater than the step increment.

What is also of much interest is the time limit t_{lim} at $v\dot{V}O_{2max}$, (this was calculated using the three phase model in section 3.2.7) or the time to exhaustion when running at $v\dot{V}O_{2max}$. According to Billat and Koralsztein [9] the average t_{lim} is 6 minutes $\pm 25\%$. This t_{lim} is highly variable for groups of individuals with the same $v\dot{V}O_{2max}$, but for an individual the t_{lim} is reproducible (for a particular test protocol). It was shown that the t_{lim} at $\dot{V}O_2^{max}$ was correlated with the velocity at the lactate threshold (and maximal lactate steady state) expressed as a percentage of the $v\dot{V}O_{2max}$. This would explain the inter individual differences of the t_{lim} and suggest that the role of anaerobic contribution should be taken into account. Billat and Koralsztein [9] also showed that the t_{lim} at $\dot{V}O_2^{max}$ was correlated negatively with the $v\dot{V}O_{2max}$. Such that the runners who attained the highest $v\dot{V}O_{2max}$ spent the least time t_{lim} at $\dot{V}O_2^{max}$ before exhaustion.

4 Conclusions

The work presented here introduced some areas of biomechanics but focused mainly on models of the physiological response to exercise. Though the models presented here are not sophisticated from the point of view of modern mathematics, they are what is currently used. It can be seen that there are many open problems in the field of exercise physiology (and also biomechanics) and as a result the application of more modern and sophisticated techniques of analysis and modelling could provide very interesting and far reaching results. This is particularly the case with the use of tools from time series analysis (see Kants and Schrieber [36]) and non linear dynamics (see Guckenheimer and Holmes [29]) as most of the physiological variables have a time series which is a nonlinear functions of time and exercise intensity.

The use of more modern methods of analysis would be of much benefit for understanding the correlations and interactions between variables. These tools are also of much value in improving the identification of certain features of the time series of physiological variables, such as the lactate threshold, LT and identifying better their relationship with fitness and performance levels.

The use of modern mathematical methods for analysis and modelling of the processes we have described could not only have a large impact in the

development and understanding of training methodology and the testing of athletes involved in sport but also in medicine. Exercise physiology and biomechanics are fundamental areas of medicine involving not only the general health of the population but also such things as recover and recuperation following illnesses and injuries etc. The tools described here are also used in the diagnosis of many different disorders causing exercise intolerance, see Wasserman et al [65].

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References

- [1] Astrand PO., Rodahl K., Dahl HA., Stromme SB., 2003 "Textbook of Work Physiology: Physiological of Bases of Exercise", Human Kinetics.
- [2] Astrand, P.-O. and Saltin, B., 1961, "Oxygen uptake during the first minutes of heavy muscular exercise", Journal of Applied Physiology, **16**, 6, 971-976.
- [3] Bangsbo, J., 1996, "Physiological factors associated with efficiency in high intensity exercise," Sports Medicine, **22**, 5, 299-305.
- [4] Bangsbo, J., 1996, "Oxygen deficit: a measure of the anaerobic energy production during intense exercise?," Canadian Journal of Applied physiology, **21**, 5, 350-363.
- [5] Bangsbo, J., 1998, "Quantification of anaerobic energy production during intense exercise," Medicine and Science in Sports and Exercise, **30**,1, 47-52.
- [6] Barstow, T.J., 1994, "Characterization of $\dot{V}O_2$ kinetics during heavy exercise," Medicine and Science in Sport and Exercise, **26**, 11, 1327-1334.
- [7] Bearden SE., and Moffatt RJ., 2000, " $\dot{V}O_2$ kinetics and the O_2 deficit in heavy exercise, Journal of applied physiology **88** 1407-1412.

- [8] Beaver, WL., Wassermann, K., Whipp, BJ., 1985, "Improved detection of the lactate threshold during exercise using a log-log transformation," *Journal of applied physiology* **59** 11936-1940.
- [9] Billat, V.L., Koralsztein, J., 1996, "Significance of the Velocity at $\dot{V}O_{2_{max}}$ and the time to exhaustion at this velocity," *Sports Med.* **22** (2) 90-108.
- [10] Billat, V.L., 1996, "Use of blood lactate measurements for the prediction of exercise performance and control of training, recommendations for long distance running," *Sports Med.* **22** (3) 157-175.
- [11] Billat, V.L., Koralsztein, J., Morton HR., 1999, "Time in Human endurance models, from emperical models to physiological models," *Sports Med.* **27** (6) 359-379.
- [12] Billat, V.L., Bocquet, V., Slawinski, J., Laffitte, L., Demarle, A., Chassaing, and Koralsztein, J., 2000, "Intermittent runs at the velocity associated with maximal oxygen uptake enables subjects to remain at maximal oxygen uptake for a longer time than intense but sub maximal runs," *European Journal of Applied Physiology*, **81**, 188-196.
- [13] Billat, V.L., Bocquet, V., Slawinski, J., Laffitte, L., Demarle, A., Chassaing, P. and Koralsztein, J. P., 2000, "Effect of prior intimittent runs at $vVO_{2_{max}}$ on Oxygen kinetics during an all-out severe run in humans" *J. Sports Med., phys. fitness*, **40**, 185-194.
- [14] Billat, V.L., 2000, " $\dot{V}O_2$ slow component and performance in endurance sports," *British Journal of sports medicine*, **34** (2), 83-85.
- [15] Billat, V.L., 2001, "Interval training for performance: a scientific and emperical practice, part I, Aerobic interval training," *Sports Med.* **31** (1) 13-31.
- [16] Billat, V.L., 2001, "Interval training for performance: a scientific and emperical practice, part II, Anaerobic interval training," *Sports Med.* **31** (2) 75-90.

- [17] Brooks GA., 1985, "Anaerobic threshold: a review of the concept and directions for future research," *Med. Sci. in sports and exc.* **17**, 1, 22-31.
- [18] Brooks GA., 2002, "Lactate shuttles in nature," *Biochemical soc. trans.* **30**, 2, 258-264.
- [19] Burke, E.R. (Ed), 1998, "Precision heart rate training," Champaign IL, Human Kinetics.
- [20] Cavanagh PR., (Ed), 1990, "Biomechanics of distance running," Champaign IL, Human Kinetics.
- [21] Daniels, J., Scardina, N., Hayes, J., et al., 1984, "Elite and subelite female middle and long distance runners," in: Landers DM., editor. *Sport and Elite Performers*, Vol. 3, Proceedings of the 1984 Olympic Scientific Congress:1984 Jul 19-23, Oregon. Champaign (IL), Human Kinetics, 57-72.
- [22] Daniels, J., 1998, "Daniels' running formula," Champaign IL, Human Kinetics.
- [23] Davies, CT., Di Prampero, PE., Cerretelli, P., 1972, "Kinetics of the cardiac output and respiratory gas exchange during exercise and recovery," *Journal of applied physiology* **32**, 618-625.
- [24] De Wit B., De Clercq D., Aerts P., 2000, "Biomechanical analysis of the stance phase during barefoot and shod running," *Journal of Biomechanics*, **33**, 269-278.
- [25] De Clercq D., Aerts P., Kunnen M., 1994, "The mechanical characteristics of the human heel pad during foot strike in running: a in vivo cineradiographic study," *Journal of Biomechanics*, **27**, 1213-1222.
- [26] Engelen, M., Porszasz, J., Riley, M., Wasserman, K., Maehara, K., and Barstow TJ., 1996, "Effects of hypoxic hypoxia on O_2 uptake and heart rate kinetics during server exercise," *J. Appl. Physiol.* **81**(6), 2500-2508.
- [27] Gaesser and Poole, 1996, "The slow component of oxygen uptake in humans," *Exercise and sport science reviews*, **24**, 35-70.

- [28] Gajewski, J., Wojczuk, J., 1989. Changes in tremor following high intensity exercise. *Biology of Sport* **6** (3), 225-232.
- [29] Guckenheimer J. and Holmes P., 1983, "Nonlinear oscillations, dynamical systems and bifurcations of vector fields," Springer-Verlag.
- [30] Hennig, EM., Valiant GA., Liu Q., 1996, "Biomechanical variables and the perception of cushioning for running in various types of footwear," *Journal of Biomechanics*, **12**, 143-150.
- [31] Hill, A.V., Lupton, L., 1923, "Muscular exercise, lactic acid and the supply and utilization of oxygen," *Q. J. Med.*, **16** 135-171.
- [32] Hill, D.W., Ferguson, C.S., 1999, "A physiological description of critical velocity," *Eur. J. Physiol. Occup. Physiol.*, **79** (3), 290-293.
- [33] Hill, D.W., Stevens E.C., 2001, "The $\dot{V}O_2$ response to the onset of severe intensity exercise," *Can. J. Appl. Physiol.* **26** (4), 350-355.
- [34] Hill, D.W., Poole D.C., Smith J.C., 2002, "The relationship between power and time to achieve $\dot{V}O_{2max}$," **34** (4), 709-714.
- [35] Hughson, RL., Weisiger, KH., Swanson GD., 1987, "Blood lactate concentration increases as a continuous function in progressive exercise," *Journal of applied physiology* **62** 1975-1981.
- [36] Kantz H. and Schreiber T., 1999, "Nonlinear Time Series Analysis," Cambridge University Press.
- [37] Krogh, A., Lindhard, J., 1913, "The regulation of respiration and circulation during the initial stages of muscular work," *Journal of Physiology*, textbf47, 112-136.
- [38] Lamarra, I., Whipp, BJ., Ward, SA., and Wasserman, K., 1987, "Effect of interbreath fluctuations on characterizing exercise gas exchange kinetics" *J. Appl. Physiol.*, **62**, 2003-2012.
- [39] Linnarsson, D., 1974, "Dynamics of pulmonary gas exchange and heart rate changes at start and end of exercise," *Acta Physiol. Scand. Suppl.* **415** 1-68.

- [40] Margaria, R., Cerretelli, P., Hghenso, P., Sassi, G., 1963, "Energy cost of running," *Journal of applied Physiology*, **18**, 2, 367-370.
- [41] Martin, D.E. and Coe, P.N. , 1997, "Better training for distance runners," Champaign IL: Human Kinetics.
- [42] Medbo, J.I., Mohn, A.C., Tabata, I., 1988, Anaerobic capacity determined by maximal accumulated O_2 deficit," *J. Appl. Physiol.*, **64**, 50-60.
- [43] Mille-Hamard, L., Laffite, L., Demarle, A., Koralsztein, J. P. and Billat, V.L., 2000, "Oxygen-pulser steady state allows estimation of $\dot{V}O_2$, by heart rate monitoring in a sever submaximal run," *Biology of sport*, **17** 3, 389-399.
- [44] Moritani, T., Nagata, A., De Vries, H.A., et al., 1981, "Critical power as a measure of physical working capacityt and anaerobic threshold," *Ergonomics*, **24**, 339-350.
- [45] Myers, J., Walsh, D., Buchanan N., McAuley P., Bowes E., Froelicher V., 1994, "Increase in blood lactate during ramp exercise: comparison of continuous and threshold models," *Med. Sci. Sports Exerc.*, **26**, 11, 1413-1419.
- [46] Ng-Thow-Hing, V., Faloutsos, P., 2000, "Dynamic Animation and Control Environment (Dance), Software, version 1.0," University of Toronto.
- [47] Nigg BM., Herzog W., 1999, "Biomechanics of the musculo-skeletal system, second edition", John Wiley & Sons.
- [48] Noakes, T., 1991, "Lore of running. Discover the science and spirit of running," Champaign IL, Human Kinetics.
- [49] Ozyener, F., Rossiter, HB., Ward, SA., and Whipp, BJ., 2001, "Influence of exercise itnensity on the on- and off- transient kinetics of pulmonary oxygen uptake in humans," *Journal of Physiology*, **53.3**, 891-902.
- [50] Poole, D.C., Ward, S.A., Gardner G.W., Whipp, B.J., 1988, "Metabolic and respiratory profile of the upper limit for prolonged exercise in man," *Ergonomics*, **31**, 1265-1279.

- [51] Poole, D.C., Ward, S.A., Whipp, B.J., 1990, "The effects of training on the metabolic and respiratory profile of high intensity cycle ergometer exercise," *Eur. J. Appl. Physiol.* **59**, 421-429.
- [52] di Prampero, P.E., 1986, "The energy cost of human locomotion on land and in water," *Int. J. Sports Med.*, **7**, 55-72.
- [53] Robbins SE., Hanna AM., 1987, "Running-related injury prevention through barefoot adaptations," *Medicine and Science in Sports and Exercise*, **19**, 148-156.
- [54] Robbins SE., Gouw GJ., Hanna AM., 1991, "Running-related injury prevention through innate impact-moderating behaviour," *Medicine and Science in Sports and Exercise*, **21**, 130-139.
- [55] Robbins SE., Gouw GJ., 1991, "Athletic footwear: unsafe due to perceptual illusions," *Medicine and Science in Sports and Exercise*, **23**, 217-224.
- [56] Saltin, B., 1990, "Anaerobic Capacity: Past, present and Prospective," In Taylor, A., and Gollmack, P.D., (Eds), *Biochemistry of exercise VII, Human Kinetics: Champaign IL*, 387-412.
- [57] Saltin, B., 1987, "The physiological and biochemical basis of aerobic and anaerobic capacities in man; effect of training and range of adaptation," In Maehlum, S., Nilsson, S., Renstrom, P., eds. *An update in sports medicine: Proceedings from the second Scandinavian conference in sports medicine.* 16-59.
- [58] Saltin, B., Larsen, H., Terrados, W., Bangsbo, J., Bak, T., Kim, C.K., Svendenhag, J. and Rolf, C.J., 1995, "Aerobic exercise capacity at sea level and at altitude in Kenyan boys, junior and senior runners compared with Scandinavian runners," *Scandinavian Journal of Medicine in Sport*, **5**, 209-221.
- [59] Saltin, B., Kim, C.K., Terrados, N., Larsen, H., Svendenhag, J. and Rolf, C.J., 1995, "Morphology, enzyme activities and buffer capacity in leg muscles of Kenyan and Scandinavian runners," *Scandinavian Journal of Medicine in Sport*, **5**, 222-230.

- [60] Stirling, JR., Zakyntthinaki MS., Saltin B., 2003, "A model of oxygen up take kinetics: Including a method for caculating oxygen demand, deficit and debt," CRM preprint.
- [61] Stirling, JR., Zakyntthinaki MS., 2003, "Stability in sport," CRM preprint.
- [62] Swain, D.P. and Leutholtz B.C. , 1997, "Heart rate reserve is equivalent to $\% \dot{V}O_2$ reserve, not to $\% \dot{V}O_{2max}$," Medicine and Science in Sport and Exercise, **29**, 410-414.
- [63] Swain, D.P., Leutholtz, B.C. King, M.E., Haas, L.A. and Brauch, J.D., 1998, "Relationship between $\%$ heart rate reserve and $\% \dot{V}O_2$ reserve in treadmill exercise," Medicine and Science in Sport and Exercise, **30**, 2, 318-321.
- [64] Volkov, N.L., Shirkovets, E.A., Borilkevich, V.E., 1975, "Assessment of aerobic and anerobic capacity of athelets in treadmill running tests,""Eur. J. Appl. Physiol, **34**, 121-130.
- [65] WassermanK., Hansen JE., Sue DY., Casaburi R., Whipp BJ., 1999, "Principles of exercise testing and interpretation, including pathophysiology and clinical applications, third edition," Lippincott Williams and Wilkins.
- [66] Weltman A., 1995, "The Blood Lactate Response to Exercise (Current Issues in Exercise Science)" Human Kinetics.
- [67] Whipp BJ., Wasserman, K., 1972, "Oxygen uptake kinetics for various intensities of constant load work," Journal of applied physiology **33** 351-356.
- [68] Whipp, B.J., 1994, "The slow component of O_2 uptake kinetics during heavy exercise," Medicine and Science in Sport and Exercise, **26**, 11, 1319-1326.
- [69] Whipp, B.J., Rossiter, H.B. and Ward, S.A., 2002, "Exertional oxygen uptake kinetics: a stamen or stamina?," Biochemical Society transactions, 237-247.
- [70] Wilmore, J.H. and Costill, D.L. , 1999, "Physiology in Sport and Exercise," Champagne, IL: Human Kinetics.

- [71] Yeh MP., Gardener RM., Adams TD., Yanowitz, FG., Crapo RO., 1983, "Anaerobic threshold, problems of determination and validation," *Journal of applied physiology* **55** 1178-1186.
- [72] Zakynthinaki MS., Stirling JR., 2003, "Appearance of high frequency signals combined with fatigue-induced increases in tremor as a means for the anticipation and detection of muscular failure", submitted, *Journal of Biomechanics*.