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$\dot{V}O_2$ KINETICS IN SEVERE INTENSITY RUNNING

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This thesis has been submitted as a requirement for
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ABSTRACT

SCHOOL OF SPORT, HEALTH AND EXERCISE SCIENCES
Doctor of Philosophy

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This thesis aimed to investigate and model the $\dot{V}O_2$ response to exhaustive constant intensity running of a short duration.

Study 1 was a Douglas bag based study that compared the $\dot{V}O_2$ response of physically active subjects to 2, 5 and 8 minute bouts of constant intensity exhaustive exercise performed in both cycling and running. Nine male subjects took part and each completed a ramp test in addition to the three constant intensity tests in both modes of exercise. In the 5 and 8minute tests the subjects achieved 97.0 ± 4.2 and 97.5 ± 2.0 % for cycling, and 98.5 ± 1.8 and 99.2 ± 2.3% for running, of the ramp test $\dot{V}O_2$peak. In the 2 minute test, a significantly lower percentage was attained (89.9 ± 5.5% and 91.8 ± 2.5% for cycling and running respectively). In cycling $\dot{V}O_2$ was still increasing over the final minute of the test, whereas in running there was no difference between the last two 30 second samples (P=0.98). It was concluded that in severe intensity exercise of a short duration $\dot{V}O_2$ may not achieve its maximum and that in running it may plateau at this sub-maximal rate.

Study 2 validated the QP9000 for the measurement of $\dot{V}O_2$ during running on a breath-by-breath basis. Six male subjects performed a ramp test and tests at rest and at moderate and severe intensities. Each test was performed twice, once using the QP9000 and once using a Douglas bag system. No difference was found for $\dot{V}O_2$ between the two systems (P=0.358). The SD of the differences between the systems across exercise intensities was 97 ± 57 $ml.min^{-1}$. It was concluded that the QP9000 provides a valid measure of $\dot{V}O_2$ at all exercises intensities.

Study 3 investigated the $\dot{V}O_2$ response of trained runners to 800 m pace running, following a track based time trial. Eight male subjects ($\dot{V}O_2$max 68.8 ± 5.6 $ml.kg^{-1}min^{-1}$) took part in the study. That $\dot{V}O_2$ reached a plateau below $\dot{V}O_2$ was confirmed by a gradient of -29 ± 275 $ml.min^{-2}$ during the final 30 seconds of exercise. The asymptotic $\dot{V}O_2$ was only 85.3 ± 6.6 % of the $\dot{V}O_2$peak from a ramp test and the response was shown to be extremely fast (time constant (τ) of 10.7 ± 3.4 seconds). These breath-by-breath data confirmed the response indicated in Study 1.

Study 4 explored the single exponential model used to describe the $\dot{V}O_2$ response and the nature and level of breath-by-breath noise in severe intensity running. Five male subjects performed a ramp test to determine the speed at anaerobic threshold (AT). Each subject then performed five 8-minute runs at a speed corresponding to 90% of the AT and five exhaustive runs at a speed that would elicit exhaustion in about 2-minutes. Analysis of the noise to signal ratio of the severe intensity data showed that the noise was Gaussian and that averaging data over repeated transitions reduced this ratio. Computer simulations of noise equivalent to the noisiest subject's data demonstrated that the use of two repeated severe intensity transitions would give 95% confidence limits of < ± 1.2 seconds for τ.

Study 5 examined the effect of prior supra AT exercise on the $\dot{V}O_2$ response to exhaustive severe intensity running. Ten middle and long distance runners each completed a ramp test to determine AT and $\dot{V}O_2$peak. Subjects then ran exhaustive transitions, lasting approximately 2-minutes, that were preceded by moderate (90% AT) or heavy (50% of the difference between AT and $\dot{V}O_2$peak) intensity running. Each transition was repeated. Increased metabolic acidosis (from prior heavy intensity exercise) did not increase the asymptotic $\dot{V}O_2$ (P = 0.226), and this figure represented only about 90% of $\dot{V}O_2$peak from the ramp test. The mean response time (MRT) (time to reach 63% of the overall response) was faster following heavy exercise (20.9 ± 1.9 s vs. 18.9 ± 1.0 s, P<0.05). This was however due to a reduction in the duration of the initial (cardiodynamic) phase of the response rather than a speeding of the primary (phase 2) kinetics.

Study 6 analysed the differences in this response between sprint and endurance runners. Six male athletes were recruited for each group based on best times for 100 m and 10 000 m. Subjects performed repeated transitions at a speed that would elicit exhaustion in approximately 2-minutes. No difference was found in the model of the $\dot{V}O_2$ response between groups. When all subjects were analysed however a strong negative relationship was demonstrated between $\dot{V}O_2$peak (from a ramp test) and the percentage of this $\dot{V}O_2$peak that was reached in the constant speed test (r = -0.811, P = 0.001). It was concluded that the $\dot{V}O_2$ response was dependent on the aerobic capabilities of the individual.

In conclusion the thesis demonstrated a $\dot{V}O_2$ response in trained subjects during exhaustive severe intensity running that was different to that which is suggested by the majority of the literature. The $\dot{V}O_2$ response tends neither to $\dot{V}O_2$ required nor to $\dot{V}O_2$max, but rather tends to a plateau that is sub-maximal. This thesis was unable to identify the mechanisms that might result in such a shortfall in the response. However there appears to be a close link with the aerobic capabilities of the individual.

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CHAPTER 1

INTRODUCTION

Models of the determinants of running performance can be traced back to the groundbreaking work of A.V. Hill in the 1920s. Hill et al. (1924) suggested that performance was dependent on three factors. These were the energy demand ($\dot{V}O_2$ required), the maximum rate of oxygen uptake ($\dot{V}O_2max$) and the anaerobic capacity. Whilst the methods used by Hill et al. (1924) were primitive by modern standards and the measurement of anaerobic energy release grossly over estimated anaerobic capacity, the energy supply and demand model described by these three factors is the foundation on which current models of performance are built.

Hill et al. (1924) realised that there was inertia in the oxidative systems to meet the $\dot{V}O_2$ required and that consequently an oxygen deficit (O_2 deficit) would be incurred as anaerobic systems assisted to meet the energy demand at the onset of exercise. Furthermore Hill et al. showed that oxygen uptake response ($\dot{V}O_2$ kinetics) tended to the $\dot{V}O_2$ required in an exponential fashion, taking approximately 2.5 minutes to reach a steady state. Whilst this delay in $\dot{V}O_2$ achieving the $\dot{V}O_2$ required was not incorporated into Hill's model, it is apparent that he was aware that this was a weakness of that model. Hill was also aware that $\dot{V}O_2$ kinetics would differ at high exercise intensities. Indeed he noted that at high levels of lactate production a steady state in $\dot{V}O_2$ would not be achievable (Hill and Lupton 1923).

Both the rate and the magnitude of $\dot{V}O_2$ kinetics will have an impact on the relative aerobic and anaerobic energy contributions to exercise (Whipp 1994a). Specifically the rate of increase in $\dot{V}O_2$ will determine the magnitude of the O_2 deficit (reflecting anaerobic energy production) at the onset of exercise. The relative aerobic and anaerobic contributions to exhaustive exercise have traditionally been evaluated using the maximal accumulated oxygen deficit (MAOD) (Medbo et al. 1988). The majority of this research has used the Douglas bag method of gas collection. Whilst such systems are adequate for calculating total oxygen consumption, potentially useful information regarding the time

course of the $\dot{V}O_2$ response is not available. With the advent of reliable systems for the measurement of $\dot{V}O_2$ on a breath-by-breath basis, the kinetics of the $\dot{V}O_2$ response may be plotted and modelled (Lamarra and Whipp 1995).

At intensities where the $\dot{V}O_2$ required falls below the anaerobic threshold (AT)[1] the $\dot{V}O_2$ response has been shown to be similar to that described by Hill et al. (1924). After an initial delay phase (due to muscle to lung transit time) $\dot{V}O_2$ tends in an exponential fashion towards $\dot{V}O_2$ required, and reaches this asymptote within about 3 minutes (Whipp and Wasserman 1986). Intensities that have a $\dot{V}O_2$ required greater than the AT demonstrate a more complex response however (Barstow and Mole 1991). During these exercise intensities a delayed phase (termed slow component) becomes manifest after approximately 3 minutes that takes $\dot{V}O_2$ to a steady state that is greater than the predicted $\dot{V}O_2$ required (as predicted from a sub AT $\dot{V}O_2$-exercise intensity relationship) (Whipp and Ward 1990). At higher exercise intensities where blood lactate cannot stabilise but continues to rise during the exercise, this $\dot{V}O_2$ slow component does not reach a steady state but continues to rise until $\dot{V}O_2max$ is reached (Poole et al. 1988, 1990).

In short duration exhaustive exercise the $\dot{V}O_2$ required may even exceed $\dot{V}O_2max$ (Medbo et al. 1988). During such exercise (when the $\dot{V}O_2$ required exceeds $\dot{V}O_2max$), Whipp (1994a) argues that the $\dot{V}O_2$ response will tend exponentially to $\dot{V}O_2$ required but will be 'cut short' at $\dot{V}O_2max$. The presumption is then that all exercise where the $\dot{V}O_2$ required is close to or greater than $\dot{V}O_2max$, will result in the achievement of $\dot{V}O_2max$ (Hill & Ferguson 1999), unless the exercise is of such a short duration that there is insufficient time to reach $\dot{V}O_2max$ (Whipp 1994a). This assumption is widely accepted (Gaesser and Poole 1996) and has become fundamental to current understanding of $\dot{V}O_2$ kinetics and exercise intensity (Poole 1998; Ward 1999).

Mathematical models of exercise performance aim to provide sound physiological descriptions of the bio-energetic systems and their observed performance ability (Billat et

[1] The term AT is used in this thesis to describe a 'threshold' determined from either ventilatory or blood lactate measures. It is recognised that there are many conceptual difficulties with such a threshold and the use of the word anaerobic is not intended to imply that such a threshold is the result of insufficient O_2 supply.

al. 1999a). It was quickly recognised by those modelling the determinants of running performance that the $\dot{V}O_2$ response, missing from the model of Hill et al. (1924), needed inclusion (Sargent 1926). Current models, having the advantage of the large body of research into $\dot{V}O_2$ kinetics, have incorporated exponential functions to describe the $\dot{V}O_2$ response at the onset of exercise (Di Prampero 1986; Cappeli 1999). A similar exponential function is used in models of both running (Ward-Smith 1985) and cycling (Olds et al. 1983) performance. Based on current thinking about $\dot{V}O_2$ kinetics at very high intensities, these models assume that exhaustive exercise of a short duration will always tend to, and provided the duration is sufficient will result in the achievement of, $\dot{V}O_2max$. The definition of this duration differs between models (Di Prampero 1993; Peronnet and Thibault 1989), however all current models agree that exhaustive exercise of duration of less than 7 minutes will result in $\dot{V}O_2max$. The lower limit of this range (i.e. intensities where there is insufficient time for the $\dot{V}O_2$ response to reach $\dot{V}O_2max$) varies from about 50 to 150 seconds between models.

Current understanding of $\dot{V}O_2$ kinetics in short duration exhaustive exercise and models of performance in such events such as 800 and 1500 m running are in agreement in relation to the $\dot{V}O_2$ response. Both suggest that $\dot{V}O_2$ will rise in an exponential manner (tending to either $\dot{V}O_2max$ or $\dot{V}O_2$ required) and $\dot{V}O_2max$ will be attained. However, whilst research into exercise at such high intensities is far from extensive, there are data that question this assumption.

There is evidence to suggest that at very high intensities $\dot{V}O_2$ will tend neither to $\dot{V}O_2max$ nor to the $\dot{V}O_2$ required, but instead reach a plateau below its maximum rate. Astrand and Saltin (1967) examined the $\dot{V}O_2$ response at the onset of high intensity cycling exercise. The data from the shortest duration tests (which lasted ~ 2 minutes) showed that some subjects achieved a plateau in $\dot{V}O_2$ that was lower than that achieved in the longer tests, although this was not commented on by the experimenters. This phenomenon has been more clearly demonstrated in middle distance runners, running at 800 m race pace on a motorised treadmill (Spencer et al. 1996; Spencer and Gastin 2001). These aerobically trained individuals sustained a plateau in $\dot{V}O_2$ that was below $\dot{V}O_2max$ for approximately half of the run, despite the $\dot{V}O_2$ required being well in excess of $\dot{V}O_2max$. These data

suggested that $\dot{V}O_2$ might not tend to either $\dot{V}O_2max$ or $\dot{V}O_2$ required, and furthermore that this shortfall was not due to an insufficient duration for the response to fully develop. However, since this plateau below $\dot{V}O_2max$ was not apparent in all Astrand and Saltin's (1967) subjects, and as Spencer et al. did not use genuine constant intensity exercise, this phenomenon remains to be properly established. Furthermore, whether the clearer demonstration of this (plateau) response in the studies of Spencer et al. and Spencer and Gastin was due to the mode of exercise (running), or due to the superior level of aerobic training in those subjects compared to those of Astrand and Saltin is unknown.

The majority of research into $\dot{V}O_2$ kinetics has used cycling (Whipp & Wasserman 1972; Poole et al. 1988; Barstow & Mole 1991; Patterson & Whipp 1991; Barstow et al. 1996; Gerbino et al. 1996; McDonald et al. 1997; Zoladz et al. 1998), and the models we have of the $\dot{V}O_2$ response to different exercise intensities are based on this mode of exercise. Recent breath-by-breath studies of the $\dot{V}O_2$ response to treadmill running have shown differences in the $\dot{V}O_2$ response between the two exercise modes, during supra AT exercise (Billat 1998a; Jones et al. 1998).

Furthermore most studies have used untrained subjects. Elite distance runners have been shown to have different $\dot{V}O_2$ kinetics to untrained individuals (Billat 1997; Lucia et al. 2000). However whether this is due to training status (Womack et al. 1995; Phillips et al. 1995), mode of exercise (Billat et al. 1998a; Jones et al. 1998), or differences in muscle fibre type recruitment and distribution (Barstow 1996), all of which have been shown to affect $\dot{V}O_2$ kinetics, is unclear.

As mentioned previously, the $\dot{V}O_2$ response to exercise has been shown to be different and more complex in exercise intensities above AT, (Barstow & Mole 1991). Subjects exercising at these intensities demonstrate a delayed phase in $\dot{V}O_2$, occurring after about three minutes, that results in either an elevated steady state or a continual rise to exhaustion (Gaesser & Poole 1996), dependent on the severity of the exercise. This phenomenon has been termed the $\dot{V}O_2$ slow component and debate exists over the underlying physiological mechanisms. Research into $\dot{V}O_2$ kinetics during exercise at these intensities has therefore been extensive (Diamond et al.1977; Hagberg et al. 1978;

Barstow 1994; Billat et al. 1998). Conversely few studies have examined the $\dot{V}O_2$ kinetics of exercise at intensities that would result in exhaustion in a short duration (3 minutes or less). The nature of the response to the latter type of exercise is therefore unknown.

This thesis aimed to address the issues surrounding the $\dot{V}O_2$ response to exhaustive running of a short duration. There were a number of questions that could not be resolved with reference to the current body of research and so the thesis had several aims.

- First it was necessary to examine the effect of mode of exercise on the $\dot{V}O_2$ response to exhaustive exercise of a short duration in aerobically fit subjects. It is unclear, at present, whether the response shown for middle distance runners (Spencer et al. 1996), was due to the mode of exercise or the aerobically trained subject group.
- Second to investigate the incidence of a sub maximal plateau in $\dot{V}O_2$ during exhaustive running of a short duration using both off- and on-line analyses. Previous research demonstrating a sub maximal plateau has used only Douglas bags for cycling (Astrand and Saltin 1961) and a mixing chamber (Spencer et al. 1996) for running.
- Third to model the $\dot{V}O_2$ response to exhaustive running of a short duration, using breath-by-breath data.
- Fourth to test the assumptions underlying the model used, to calculate confidence limits for the resulting parameter estimates and determine how many repeated transitions data should be averaged over to give desired confidence limits for these parameter estimates.
- Fifth to investigate the effect of prior supra AT exercise on the $\dot{V}O_2$ response to exhaustive running of a short duration. Prior supra AT exercise has been shown to effect exercise of a lesser intensity (Gerbino et al. 1996).
- Finally to investigate the effect of aerobic capability on the $\dot{V}O_2$ response to exhaustive running of a short duration. The sub maximal plateau seen in $\dot{V}O_2$ for short duration exhaustive exercise has only been clearly demonstrated in the aerobically trained (Spencer et al. 1996).

Answers to these questions, it was hoped, would help to determine the physiological mechanisms that determine the $\dot{V}O_2$ response to short duration exhaustive running.

CHAPTER 2

REVIEW OF LITERATURE

2.1 The challenge of high intensity running

2.1.1 Middle distance running

The term middle distance has been applied to all track-running events between the distances of 800 and 3000 m (Brandon 1995), taking between approximately 2 and 10 minutes to complete (Camus 1992). Whilst research into the determinants of, and physiological mechanisms underlying, success in distance running events has been extensive (Costill 1967, 1971; Costill et al. 1973; Daniels 1974; Fink et al. 1977; Conley and Krahenbuhl, 1980; Pollock et al. 1980; Noakes et al. 1990), middle distance events have received little research attention (Padilla et al. 1992). The middle distance events lie between the sprint events where the energy requirement will be largely met by anaerobic sources and the largely aerobic distance events. Middle distance running requires a significant contribution from both aerobic and anaerobic energy pathways (Hill 1999), and the interplay between the two systems is thought to be an important aspect of performance (Daniels 1985).

The shorter of the middle distance events, the 800 and 1500 m, have a $\dot{V}O_2$ requirement that is close to or above $\dot{V}O_2max$ (Spencer et al. 1996). The relative contribution from aerobic and anaerobic energy sources has been the cause of considerable debate and difference of opinion amongst the scientific and coaching communities (Lacour et al.1990; Gamboa et al. 1996; Hill 1999; Gastin 2001).

2.1.2 Assessment of the anaerobic contribution and anaerobic capacity

Laboratory assessment of the anaerobic energy contribution to exercise and the evaluation of the total capacity for anaerobic energy provision have proved difficult. All of the methods available for whole-body exercise involve a number of assumptions and problems

that should be understood and identified if they are to be accepted as valid and reliable research tools.

The level of post exercise lactate concentration has been used to estimate the anaerobic energy yield (Margaria et al. 1963a). Problems exist in the assumption that blood lactate concentration is equivalent to muscle glycolytic rate, and even if it is a reasonable guide to glycolytic energy production it does not represent total anaerobic energy yield from all systems (Saltin 1989).

It has long been thought that the deficit in aerobic energy production, i.e. the anaerobic contribution to the exercise metabolism, is 'paid off' as excess $\dot{V}O_2$ during recovery and was termed oxygen debt (O_2 debt) (Hill and Lupton 1923). This O_2 debt was considered a measure of the anaerobic contribution to exercise, based on the assumption that the O_2 debt following exercise equalled the O_2 deficit that was incurred due to the inertia in aerobic energy production at the onset. The fast and slow phases in the recovery of $\dot{V}O_2$ were later divided into the alactic (ATP and PCr) and lactic (glycolitic) O_2 debts representing the recovery of those systems (Margaria et al. 1933). These assumptions were flawed (Gastin 1994), as shown by the dissociation between O_2 deficit and O_2 debt during any exercise that resulted in elevated blood lactate concentrations (Patterson and Whipp 1991). The majority of lactate produced during exercise is used as a substrate for oxidation and not for glycogen resynthesis (Gaesser and Brooks 1984) and more energy is required to oxidise this lactate than is liberated when lactate is produced (Saltin 1986). Other factors (not directly related to the anaerobic energy production) such as temperature and catecholamines may also influence the O_2 debt. Consequently the O_2 debt is likely to grossly overestimate anaerobic energy production during exercise (Green and Dawson 1993) and is more variable than the other available measures (Graham and Andrew 1973).

The O_2 deficit itself is widely used as a measurement of total anaerobic capacity (Medbo et al. 1988; Gastin and Lawson 1994) in exhaustive exercise and to calculate the anaerobic contribution to exercise of a given duration (Van Ingen Schenau 1992; Craig et al. 1993; Craig et al. 1995; Spencer et al. 1996). The concept of an oxygen deficit has been in existence for some time, being first introduced by Krogh and Lindhard in 1920, after their initial observations of the delay in oxidative metabolism at the onset of exercise (Krogh

and Lindhard 1913). The concept was reintroduced in 1969 by Hermansen, but calculation of O_2 deficit in exhaustive short duration exercise as a measure of total (and maximal) anaerobic capacity was not formally suggested until 1988 (Medbo et al. 1988). There are few arguments against the use of oxygen deficit to assess the anaerobic contribution to sub AT exercise (Saltin 1986). However, in supra AT exercise the $\dot{V}O_2$– work rate (WR) relationship is both non-linear and time dependent, therefore calculation of the $\dot{V}O_2$ required (and O_2 deficit) becomes problematic (Ward 1999). Two major assumptions underlie the calculation of the O_2 deficit: firstly that the $\dot{V}O_2$ requirement for all intensities, including those above that eliciting $\dot{V}O_2max$, can be calculated by extrapolation of a linear $\dot{V}O_2$-WR relationship; secondly that this energy demand will remain constant throughout exhaustive exercise (Bangsbo 1996a). It has now been established that the $\dot{V}O_2$-WR relationship is not linear above the AT (Bangsbo et al. 1993; Zoladz et al. 1995, 1998; Bearden and Moffatt 2001a). Oxygen uptake has been shown to increase in a non-linear manner above the AT (Hansen et al. 1988), with values exceeding those predicted from the sub AT $\dot{V}O_2$-WR relationship. This additional $\dot{V}O_2$ is delayed in onset (Whipp and Wasserman 1972) and therefore the nature of the $\dot{V}O_2$-WR relationship is dependent on the test protocol and the timing of the expired gas samples. Recent studies using a one legged exercise model suggest that the second assumption may also be flawed and the energy demand may vary during the course of a constant load bout of exercise (Bangsbo 1996b).

A further conceptual problem exists, in deciding what physiological systems contributing to the O_2 deficit are representative of the anaerobic energy yield or anaerobic capacity. Anaerobic energy provision is largely from the glycolytic system and some is via the ATP and PCr systems (Saltin 1989). However, also incorporated into the O_2 deficit is a certain amount of aerobic energy production. Oxygen bound to haemoglobin and myoglobin is reduced at the onset of exercise (Saltin 1986), and the O_2 deficit measured via pulmonary ventilation may not therefore represent that of the working musculature (Spriet et al. 1992; Graham 1996). Oxygen bound to myoglobin, although representing aerobic energy yield, is present within the muscle at the onset of exercise and blood stores of O_2, largely bound to haemoglobin, are provided by pulmonary gas exchange, albeit prior to the exercise. The question then arises of whether a measure of anaerobic capacity should represent purely

anaerobic energy yield, or all energy not directly gained via pulmonary ventilation during that exercise. The O_2 deficit and anaerobic capacity have never been clearly defined (Green 1994), and the investigator needs to consider the various energy producing elements that make up the total O_2 deficit.

The debate about the validity of the O_2 deficit has been intense (Bangsbo et al. 1990; Bangsbo 1996a, Medbo 1996). However, it is still widely used and considered to be a promising if not perfect measure of anaerobic metabolism (Gastin 1994). Oxygen deficit does also provide a useful index of $\dot{V}O_2$ kinetics at the onset of exercise, since any speeding of $\dot{V}O_2$ kinetics will result in a smaller O_2 deficit for a given $\dot{V}O_2$ required (Walsh 1992; Grassi et al. 2000). Furthermore it should be recognised that, consistent with research showing the O_2 deficit to be unchanged across all exhaustive exercise lasting longer than 2 minutes (Medbo et al. 1988), current mathematical models of running have incorporated a fixed anaerobic capacity that will be exhausted in all middle distance events.

2.2 Models of athletic performance

2.3.1 Early models

Throughout the 20th century, scientists have sought to model the limits of human capability, particularly in athletics and especially for the running events (Billat et al. 1999a). In the early part of the century models were purely empirical and concentrated on defining the speed-time relationship (Kennelly 1906; Meade 1916) based on world best times of the day. However, later models have attempted to explain performance based on the physiological processes underlying it (Morton and Hodgson, 1996).

2.3.2 The contribution of A.V. Hill

A.V. Hill (1925) praised the work of Kennelly. However, he recognised the need to identify the physiological determinants of fatigue to properly understand the speed-distance relationship. Hill et al. (1924), acting as their own subjects, ran around a 90 m grass running track, while collecting expired gases into Douglas bags, with a colleague calling out lap times to ensure an even paced run. In this way they investigated both the $\dot{V}O_2$ response and the $\dot{V}O_2$ required at various running speeds. This work was important for two reasons: firstly it was the first attempt to produce a model of performance based on the underlying physiology; and secondly, the suggested determinants remain central to contemporary models of performance.

Hill et al. (1924) suggested that the average running speed that could be maintained for a given distance was dependent on three factors. These were (first) the overall energy demand, (second) $\dot{V}O_2max$ and (third) the anaerobic capacity of the runner. It is testament to Hill that contemporary models of performance are still based on these three aspects of energy supply and demand.

Hill's subjects had to carry Douglas bags and respiratory tubing weighing approximately 10 pounds (4.5 kg), while running around a small circular track that made running at high speeds problematic. Despite these technical limitations the conclusions drawn from this work were impressive. It was observed that $\dot{V}O_2$ would rise in an exponential fashion towards a steady state that could be achieved in about 2.5 minutes at low exercise intensities. Hill and Lupton (1923) also realised that at higher exercise intensities a steady state in $\dot{V}O_2$ might not be achieved and linked this to the rate of lactate production and removal. This they argued meant not only that it was problematic to evaluate $\dot{V}O_2$ required at high intensities, if a steady state could not be achieved, but also that the exercise could not be maintained for a prolonged period of time due to fatigue.

2.3.3 *Refinement of Hill's model*

Whilst Hill et al. (1924) had realised that $\dot{V}O_2$ would take time to achieve a steady state and an oxygen deficit (referred to as oxygen debt) would be incurred, this was not included in the resulting performance model. However, Sargent (1926) developed the model, and took into account that $\dot{V}O_2$ would take time to reach its peak, addressing an assumption that Hill knew to be flawed in his proposed model. He assessed the oxygen demand at a variety of speeds in a single trained individual. Sargent's subject held his breath during the final 120 yards of each run and gas was collected on finishing until $\dot{V}O_2$ had reached resting values; both $\dot{V}O_2$ and O_2 debt were calculated from post exercise $\dot{V}O_2$. Theoretical times were calculated over a range of distances for this runner that agreed closely with actual times recorded. Whilst Sargent wrote of the striking agreement between predicted and actual times it should be remembered that only a single subject was used.

It was not until Henry in 1954 that the exponential increase of $\dot{V}O_2$ at the onset of exercise (suggested by Hill et al. (1924)) was incorporated into a model of performance. Henry's paper used the world record times of 1952 and was based on the findings of Sargent (1926). Unfortunately, Sargent (and Hill before him) had used the O_2 debt to calculate anaerobic capacity, and therefore a grossly overestimated figure of 18 litres was used. A typical figure for anaerobic capacity would be 5.5 litres, and even in athletes with a large anaerobic capacity, 18 litres represents more than double the figure that might be expected (Medbo et al. 1988; Medbo and Burgers 1990).

Lloyd (1966) (as had his predecessors) saw world records as important scientific data since (as in the acquisition of scientific data) a high degree of care and accuracy was required in the measurement, and similarly based his findings on best times of the day. Using the energy demand and O_2 debt calculations of Margaria el al. (1963b), Lloyd (1966, 1967) sought to test the model proposed by Hill. Margaria had realised that previous models of performance had overestimated the anaerobic contribution and concluded that the $\dot{V}O_2$-speed relationship was linear in treadmill running (acknowledging that air resistance would alter this relationship in outdoor running). Lloyd (1966) produced a comprehensive model that incorporated the exponential rise in $\dot{V}O_2$ used by Henry (1954). In addition, Lloyd described the anaerobic energy reserve decreasing as an exponential function of duration,

i.e. he proposed that the anaerobic capacity would not be fully exhausted in events of a very short duration. The model laid the way for contemporary models that describe this relationship between anaerobic and aerobic energy release at the onset of strenuous exercise in a similar fashion (Capelli 1999). Whilst contemporary models base this relationship on published results measured using breath-by-breath gas collection, Lloyd (1967) was tentative in his use of an exponential function.

2.3.4 $\dot{V}O_2$ kinetics and models of performance

During the 1970s, the advent of systems capable of calculating $\dot{V}O_2$ on a breath-by-breath basis resulted in considerable research into the kinetics of $\dot{V}O_2$. Consequently those attempting to model performance at the end of this decade were more confident to describe the inertia between rest and steady state $\dot{V}O_2$ using an exponential term. The term;

$$E\,\tau\,(1-\exp(-t/\tau))/\tau \quad \text{.....(2.1)}$$

was used to describe the rise in aerobic metabolism during constant intensity cycling (Wilkie 1980). Where E represented the steady state for $\dot{V}O_2$, t was time and τ was a constant. However, Wilkie was surprised that this model (when applied to data from trained cyclists), estimated a time constant (τ) of just 10 seconds, when a value of 30-40 seconds was expected. He explained this discrepancy as being the difference between working muscle $\dot{V}O_2$ and $\dot{V}O_2$ measured at the mouth. Recent research indicates that a difference does not exist between muscle $\dot{V}O_2$ kinetics and that measured at the mouth, however (Whipp et al. 1999).

The use of this exponential function to describe the aerobic and anaerobic energy cost at the onset of exercise has changed little, but has been refined. Ward-Smith (1985) praised Lloyd (1966) for including factors to describe anaerobic and aerobic energy transfer. Ward-Smith sought to improve on the model suggested by Lloyd based on principles of energy balance rather than mechanical efficiency. Aerobic energy production was modelled to increase exponentially towards an asymptote at $\dot{V}O_2$ required for all intensities

below $\dot{V}O_2max$, or at $\dot{V}O_2max$ for all intensities where $\dot{V}O_2$ required exceeded $\dot{V}O_2max$. This increase in aerobic energy production was in tandem with an exponential decrease in anaerobic energy production, and Ward-Smith linked $\dot{V}O_2$ to the rate of phosphagen degradation.

2.3.5 Current models of running performance

The function describing the interaction between aerobic and anaerobic parameters has altered little from that proposed by Wilkie (1980). Di Prampero (1986), who had worked with Margaria et al. (1963b), produced mathematical equations to predict performance in running, skating and swimming, as well as commenting on other forms of human locomotion. The models were comprehensive and examined many mechanical factors (air density, drag, terrain etc.). However, in terms of metabolic energy production the function was that used by Wilkie (1980). For running, the model was assessed against world record times for distances between 400 m and 10 000 m and with the exception of 400m a reasonable agreement was found. Di Prampero assumed a time constant of aerobic metabolism (τ) of 10 seconds (as had Wilkie 1980) and that the anaerobic energy reserve would be completely exhausted in all events. This work differed from the previous models in the conclusion that $\dot{V}O_2$ was not sustainable at its maximum for long duration events (>14 minutes). The model was further scrutinised specifically for middle distance running by calculating theoretical best times for a group of subjects, for distances of 800 to 5000 m (Di Prampero et al. 1993). The calculated times were close to actual times except in the 800 and 1000 m where the model overestimated the speed a subject could maintain. This underestimation was explained as an inability to exhaust the anaerobic capacity in short duration exercise, although this is contrary to previous research that has suggested that the anaerobic capacity can be exhausted within 2 minutes (Medbo et al.1988, Medbo and Tabata 1993).

The model proposed by Peronnet and Thibault (1989) does differ from that of Di Prampero in two key aspects. Firstly, whilst Di Prampero (1993) assumed a constant value for energy production derived from anaerobic processes, Peronnet and Thibault suggested that this value would decline with increasing running distance. Peronnet and Thibault based

this assumption on the work of Gollnick and Hermansen (1973). The second major difference is that Peronnet and Thibault introduced a factor that takes account of the decline in aerobic power with increasing duration. A factor (T_{MAP}) was identified that represents the maximum duration where $\dot{V}O_2$peak would equal $\dot{V}O_2$max, and this was estimated to be at about 420 seconds. This factor has been supported and used by Capelli (1999) and Ward-Smith (1999). Whilst a higher value has been suggested (Di Prampero 1993), the assumption exists in all current models that $\dot{V}O_2$ will attain $\dot{V}O_2$max if the duration of the exhaustive exercise is less than 420 seconds.

The other discrepancy among the current models is the value assigned to describe the speed of the $\dot{V}O_2$ response (τ). The differences appear to be a function of whether the assigned value was based on previous $\dot{V}O_2$ kinetics research, or whether it was based on the best fit of the model to the data. Peronnet and Thibault assigned a value of 30 seconds for τ, in line with typical values demonstrated in the research (Linnarson 1974). Capelli used a τ of 24 seconds based on the work of Binzoni et al. (1992), who calculated a similar τ using ^{31}P nuclear magnetic resonance spectroscopy. The τ of 10 seconds used by Wilkie (1980) and Di Prampero (1986) was calculated as the best fit for the model, and is much faster has been previously reported in previous research into $\dot{V}O_2$ kinetics (Linnarson 1974). As most models assume that 420 seconds is the maximum duration where $\dot{V}O_2$max will be achieved. It is also apparent that if the exercise is very short then $\dot{V}O_2$max will not be reached because the response has not had time to reach it. A τ of 10 or 30 seconds would equate a minimum duration to achieve ~99% $\dot{V}O_2$max, of approximately 50 or 150 seconds respectively.

2.3.6 Models of cycling performance

The models addressed so far are models that are specifically for running or examine several forms of human locomotion. Mathematical models specific to cycling performance have also been produced. These models obviously differ from those for running in that they include factors specific to cycling such as the mass of the bicycle, drag coefficients of

bike and rider, and rolling resistance. They are however, similar energy supply and demand based models and use the same exponential function to describe the interplay between aerobic and anaerobic energy production at the onset of exercise (Olds et al. 1993; Olds et al. 1995; Olds 2001). These models seem to differ from their running counterparts only in their readiness to accept oxygen deficit as a reliable measure of anaerobic capacity (Olds et al. 1993; Craig et al.1993; Olds et al. 1995; Olds 2001). Olds et al. make the assumption that $\dot{V}O_2$ would tend to and would be maintained at $\dot{V}O_2max$ for all events of 10 minutes and under. The time constant of $\dot{V}O_2$ (τ) was calculated to be 10 seconds by iteration of the model parameters, rather than by reference to research that suggests a slower $\dot{V}O_2$ response (Barstow et al. 1996).

Models of performance have changed little from the view of Hill et al. (1924) that performance was dependent on energy demand, $\dot{V}O_2max$ and anaerobic capacity. All current models (irrespective of mode of exercise) are based on this supply and demand principle and assume that for any event under 7 minutes duration $\dot{V}O_2$ will tend in an exponential manner to $\dot{V}O_2max$. The models differ only in terms of whether the anaerobic contribution differs across duration (Peronnet and Thibault 1989) and the speed of the $\dot{V}O_2$ response (Di Prampero 1993; Capelli 1999).

2.3 Domains of exercise intensity and $\dot{V}O_2$ kinetics

2.3.1 Overview

Differences have been demonstrated in $\dot{V}O_2$ kinetics, dependent on the intensity of the exercise performed (Whipp et al. 1980). Prior to discussing the $\dot{V}O_2$ response to various intensities of exercise, it is important to summarise current thinking about exercise intensity domains and what physiological parameters determine the upper and lower boundaries of each. In order to compare between research it is also important to identify the discrepancies that exist, between authors, in this terminology.

Categorisation of the domains of exercise intensity is often problematic since the definition of the upper and the lower limits of each domain is uncertain and different laboratory measures are used to calculate them. Also when exercise intensity is referenced as a percentage of $\dot{V}O_2max$, subjects may differ in terms of the exercise intensity domain they are performing in (Wetter et al. 1999). As a result, in addition to the terms discussed in this review, less well defined terms such as sub maximal exercise (Hughson and Smyth 1983), intense exercise (Houmard et al. 1991), and high intensity exercise (Bernard et al. 1998; Gerbino et al. 1996), also appear in the literature.

Within this thesis the terms *moderate intensity* exercise and *heavy intensity exercise* are used to describe intensities that fall below and above the AT respectively. All exercise intensities where the $\dot{V}O_2$ required is above the maximum intensity where a steady state may be achieved in blood lactate concentrations, i.e. maximum lactate steady state (MLSS), are referred to as *severe.* Identification of whether a given intensity is heavy or severe is problematic (see Section 2.3.2). However, since the focus of this thesis was exhaustive exercise and the longest exercise bout studied lasted 8 minutes (most were 2 minutes), it is safe to assume that all the exercise bouts studied must have been performed above MLSS and may therefore be termed severe intensity exercise.

2.3.2 Demarcation between the domains of exercise intensity

The term *moderate intensity* has been used to describe those exercise intensities that fall below the AT (Gaesser and Poole 1996; Xu and Rhodes 1999). Moderate intensities are consequently described as those that do not result in an increased (above resting levels) metabolic acidemia (Whipp 1994a). Some researchers have disputed the threshold model of lactate production and removal and therefore the existence of such a domain (Hughson and Green 1982; Hughson et al. 1987; Brooks et al. 1991). Controversy also surrounds the calculation of the AT, as there are various suggested methods, both from measurement of blood lactate and from respiratory data (Beaver et al. 1985; Beaver et al. 1986; Aunolo and Rosko 1992; Tokmakidis and Leger 1992). However, the majority of research on $\dot{V}O_2$ kinetics has used respiratory methods to calculate the AT. Despite the controversy surrounding the detection (or indeed the existence) of the AT (Hughson and Green 1982;

Yeh et al. 1983), this point has been taken to represent the upper limit of the domain of moderate intensity exercise. Many studies have used the AT to demarcate between the moderate and heavy exercise intensity domains (Hughson and Morrisey 1983; Poole et al. 1988; Barstow et al. 1994; McCreary et al. 1996; Koga et al. 1997; Jones et al. 1999) although each of these studies used a different method to calculate the AT. It is also apparent that authors are prepared to use the AT to distinguish exercise intensity domain despite arguing against its conceptual validity (Hughson et al.1987, 1993).

The AT has functional significance not only as the upper limit of the domain of moderate intensity exercise, but also as the lower limit of the next domain which is *heavy intensity exercise* (Patterson and Whipp 1991; Gaesser and Poole 1996). Consequently moderate and heavy intensity exercise is sometimes referred to as sub threshold and supra threshold exercise (Whipp and Ward 1990). The kinetics of $\dot{V}O_2$ have been demonstrated to respond differently in the domain of heavy intensity exercise (Linnarsson 1974), compared with moderate intensity exercise. Whilst the lower limit of this domain is reasonably well established (though the method for its calculation is disputed), the upper limit is perhaps less consistent in the literature. The upper limit of the domain of heavy intensity exercise is the highest work-rate that will result in a metabolic steady state. That is the highest work-rate at which steady state is achieved in both $\dot{V}O_2$ and lactate production and removal (MLSS) (Gaesser and Poole, 1996). Whilst some consensus exists on this point, there is disagreement about what physiological measure or parameter represents this upper limit. It has been suggested that the upper limit of the heavy intensity domain is the asymptote of the hyperbolic work rate-time to exhaustion relationship (Poole et al. 1988; Poole et al. 1990). This asymptotic value represents the theoretical maximum speed or power that can be maintained without fatigue (Moritani et al. 1981) , namely critical speed[2] (Hughson et al. 1984) or critical power (Monod and Scherrer 1965). There are conceptual limitations to this approach and it has been suggested that this asymptote will overestimate the MLSS (McLellan and Cheung 1992).

[2] The term critical speed rarely appears in current literature and critical velocity is generally used (Pepper et al. 1992; Billat et al. 1998a; Hill and Ferguson 1999). However since the focus of this thesis, and indeed the majority of such research, was treadmill running, which involves no displacement of the subject, the term critical speed was used in preference to critical velocity.

Exercise intensities that occur above those that are termed heavy, that is intensities that are above MLSS, are termed *severe* (Billat et al. 1998b; Gaesser and Poole 1996). This exercise intensity domain has also been termed *very heavy intensity exercise* (Wasserman et al. 1967; Stringer et al. 1995). The precise nature of the $\dot{V}O_2$ response in this domain is discussed in Section 2.3.5; it is however assumed that a steady state cannot be achieved and that exhaustive exercise in this domain will result in $\dot{V}O_2max$ provided it is of sufficient duration (Whipp 1994a). This means there are a range of intensities that will all result in $\dot{V}O_2max$. Therefore it is unwise to reference work-rates as $\%\dot{V}O_2max$ within the severe or indeed any other domain, since the theoretical work rate at which $\dot{V}O_2max$ is first achieved must be protocol dependent. Nevertheless, many studies have done so (Hagberg et al. 1978; Hagberg et al. 1980; Sady et al 1983; Hebestreit et al. 1998).

Due to the conceptual problems with referencing exercise intensity as a $\%\dot{V}O_2max$ alone, exercise intensity is often quantified as a percentage of the difference between $\dot{V}O_2$ at the AT and $\dot{V}O_2max$ (Koga et al. 1997; Bearden and Moffatt 2000). This difference is normally referred to as delta (Δ) (Armon et al. 1991; Billat et al 1999b). For example, for an individual with a $\dot{V}O_2max$ of 4 $L.min^{-1}$ and a $\dot{V}O_2$ at the AT of 2 $L.min^{-1}$, 50%Δ would be the intensity associated with a $\dot{V}O_2$ of 3 $L.min^{-1}$.

Whilst the term severe intensity exercise has been used to describe all work rates above the MLSS, those intensities where, in addition, the $\dot{V}O_2$ required is greater than $\dot{V}O_2max$ have been termed *supra maximal* by many authors (Katch 1973; Hughson, 1978; Yamaji and Shephard 1987; Graham and McLellan 1989; Itoh and Ohkuwa 1990; Rowland 1993; Norton et al. 1995; Laforgia et al. 1997). The term supra maximal is not used in this thesis since conceptually it is impossible for exercise intensity to be greater than maximal. In addition, as mentioned above, it is impossible to identify a single intensity that will result in the achievement of $\dot{V}O_2max$ (rather there is a range of such intensities). Consequently the upper limit to the severe intensity domain has not been established (Hill and Stevens 2001).

In practice few researchers actually identify the demarcation point between heavy and severe intensity exercise to ensure that exercise is performed in one domain or the other.

Critical speed / power is a controversial measure, and MLSS requires several long exercise tests to establish it, making it an impractical measure. Rather researchers have used exercise intensities with $\dot{V}O_2$ requirements that represent a high percentage of $\dot{V}O_2max$ (Williams et al. 1988; Billat et al.1988b) or exhaustive exercise that results in fatigue in a set (and short) duration (Hill and Stevens 2001).

2.3.3 $\dot{V}O_2$ kinetics in moderate intensity exercise

At moderate exercise intensities, after an initial delay, the $\dot{V}O_2$ response is considered to be exponential (Gaesser & Poole, 1996). The exponential behaviour of $\dot{V}O_2$ kinetics at the onset of constant intensity exercise was recognised as early as 1923 (Hill and Lupton). However, with the advent of sophisticated gas analysis systems that allow the calculation of $\dot{V}O_2$ on a breath-by-breath basis, it has been possible to examine the response in far greater detail. The whole response has been described as a single exponential function of the form:

$$\dot{V}O_2(t) = \text{Baseline} + \text{GAIN}\,(1-e^{-(t-\delta)/\tau}) \quad (2.2)$$

where $\dot{V}O_2(t)$ is $\dot{V}O_2$ at time t, Baseline is resting $\dot{V}O_2$, GAIN is the asymptotic value of $\dot{V}O_2$ (above Baseline) at steady state, δ is the time delay, and τ is the time constant.

In the moderate intensity exercise domain a steady state in $\dot{V}O_2$ is normally achieved within 3 minutes in healthy subjects (Gilbert et al. 1967; Xu and Rhodes), giving a time constant (τ) of about 30 seconds (Whipp and Wasserman 1986). The $\dot{V}O_2$ response to moderate intensity exercise has three distinct phases: a delay phase, the primary response, and the steady state. The first phase is essentially a delay, due to muscle to lung venous return (Whipp, 1994a). However, $\dot{V}O_2$ does increase during this period, principally due to an increase in cardiac output and therefore pulmonary blood flow (Wasserman et al. 1974). The exact nature of this first phase is not fully understood however. Blood extracted from a pulmonary arterial catheter showed a drop in O_2 saturation and an increase in the partial pressure of CO_2 during this initial phase (Casaburi et al. 1989a). Such changes suggest

mechanisms other than just an increase in cardiac output, may influence phase-1 $\dot{V}O_2$ kinetics.

The exponential rise to steady state therefore begins as venous blood from the exercising muscle reaches the lung, and represents muscle oxygen uptake and further increases in pulmonary blood flow (Whipp, 1994a). The time constant for this rise in $\dot{V}O_2$ (τ) is thought to be relatively unchanged across the range of moderate intensity work rates (Whipp & Ward, 1990), although recent evidence suggests it may increase with exercise intensity even in this domain (Brittain et al. 2001). For moderate intensity exercise, a steady state in both $\dot{V}O_2$ and lactate will follow the second phase, and the $\dot{V}O_2$-WR relationship is considered to be linear (Gaesser and Poole 1996).

Researchers have tended to model this response as a single exponential function. The exclusion of phase-1 from the modelling process has been justified because of the small number of data points within this phase (Grassi et al. 1996): phase-1 will only last approximately 15 seconds and breathing frequency will be low early in the transition to exercise. The modelling of $\dot{V}O_2$ kinetics in this exercise intensity domain has been achieved in a number of ways. Some investigators have used a single exponential function, modelled from all data points and including no time delay (δ) (Sady et al. 1983; Sietsma et al.1989). Other groups, recognising that the increase in $\dot{V}O_2$ during phase-1 will influence the modelling of the primary (phase-2) response, have treated phase-1 as a 'pure' delay. This involves including δ in the exponential and removing the phase-1 data points from the analysis (Whipp et al. 1980; Whipp et al. 1982a; Barstow et al 1994). Another approach has been to use the same model (exponential and delay), but simply remove the initial 25 seconds of data (Lamarra et al. 1987; Gerbino et al. 1996). More recently a two-component model has been used (Carter et al. 2002), that models the phase-1 response using the same exponential function as used to model the phase-2 response. This function does not contain a time delay (δ) for phase-1, but contains an independent GAIN (GAIN0) and time constant (τ0) for this phase. This gives the equation:

$$\dot{V}O_2(t) = \text{Baseline} + \text{GAIN0}\,(1-e^{-t/\tau 0}) + \text{GAIN1}\,(1-e^{-(t-\delta 1)/\tau 1}) \quad \text{.....(2.3)}$$

where GAIN1 τ1 and δ1 describe the phase-2 response. It should be recognised that the phase-1 response has never been demonstrated to be exponential in nature; the same exponential function is used for convenience rather than for a sound physiological reason (Whipp and Ozyener 1998). Even for phase-2 the use of an exponential function has occasionally been questioned (Taylor et al. 1999), and other mathematical functions (polynomials) have been used, albeit rarely.

2.3.4 $\dot{V}O_2$ kinetics in heavy intensity exercise and slow component

At exercise intensities that are above an individual's AT, the $\dot{V}O_2$ response has been demonstrated to be more complicated and is not adequately described by a simple mono-exponential function (Barstow and Mole,1991). The kinetics of the $\dot{V}O_2$ response in the domain of heavy intensity exercise have been described as a distortion of the basic kinetics seen in moderate intensity exercise (Whipp, 1994a). The $\dot{V}O_2$ response has been considered thus since phases-1 and -2 occur in a similar manner to those demonstrated in moderate intensity exercise, but instead of $\dot{V}O_2$ achieving a steady state in phase-2, a third phase of delayed onset emerges (Barstow and Mole 1991).

The delayed onset of this third phase has led to it being termed the *slow component* of $\dot{V}O_2$ (Poole 1994; Sloniger et al. 1996; Billat et al. 1998b; Obert et al. 1999). This slow component, whilst apparent in earlier studies (Astrand and Saltin 1961; Margaria et al. 1965), was not formally identified until 1972 (Whipp and Wasserman 1972). The slow component of $\dot{V}O_2$ occurs approximately 80-100 seconds after the onset of exercise in healthy subjects (Poole et al. 1994a). This third phase represents an excess $\dot{V}O_2$ that is over and above that which would be predicted from a linear $\dot{V}O_2$-WR regression calculated using sub AT work rates (Whipp and Ward, 1990). The $\dot{V}O_2$ slow component should not be confused with the *O_2 drift* that may be seen in moderate intensity exercise of a long duration (Kalis et al. 1988). The slow component is of greater magnitude and occurs only in exercise intensities that are above AT (Whipp and Mahler, 1980). The slow component can make a considerable contribution to the total $\dot{V}O_2$, sometimes as high as 1.5 $L.min^{-1}$, and also seems to have a different origin to O_2 drift. Possible physiological mechanisms that may underpin the slow component will be discussed later in this review.

The existence of a slow component challenges the widely held belief that the $\dot{V}O_2$-WR relationship is a linear one (Astrand and Rodahl 1986; Wilmore and Costill 1994). At moderate intensity exercise intensities the $\dot{V}O_2$-WR relationship is indeed linear (Gaesser and Poole 1996). However, for both cycling (Hansen et al. 1988; Zoladz et al. 1995; Zoladz et al. 1998) and treadmill running (Jones et al.1999), in the domain of heavy intensity exercise, the $\dot{V}O_2$-WR relationship has been demonstrated to be non-linear. Non-linearity of this relationship has obvious implications for calculating the O_2 deficit in the traditional manner, which relies on the accurate calculation of the $\dot{V}O_2$ required, and assumes this requirement to be constant (Bangsbo 1996a); see also Section 2.1.2. Barstow et al. (1993) found the phase-2 response to be linear even at high work rates, but the third slow component of delayed onset resulted in a deviation from linearity at intensities above AT. This non-linearity is often not recognised in incremental tests, as typically a fast ramp rate is used to bring an individual to exhaustion within approximately 10 minutes (Buchfuhrer et al. 1983). This does not allow sufficient time for the slow component to become manifest (Davies et al. 1982). A non-linear relationship has been shown when a slow rate of incrementation (e.g. 15 $W.min^{-1}$ in cycling), that allows a slow component to develop, is used (Whipp and Mahler, 1980; Hansen et al. 1988), however. The $\dot{V}O_2$-WR relationship is then not only non-linear but also time dependent. An incremental test with 1 minute stages is likely to produce a very different (and more linear) relationship to one with 5 minute stages where a slow component would be manifest.

In heavy intensity exercise, the basic kinetics of $\dot{V}O_2$ (as previously described for moderate intensity exercise) are distorted and an additional slow component is superimposed on the model (Whipp and Wasserman, 1972). Typically a slowing of kinetics is seen. This is may be a reflection of a greater τ in the second component, or an 'excess' $\dot{V}O_2$ that will reach a delayed steady state at a level above that predicted from a sub lactate threshold $\dot{V}O_2$-WR regression, or both (Patterson and Whipp 1991).

In the domain of heavy intensity exercise a second exponential term is used to describe the slow component. This term, which tends to a higher asymptote (GAIN2), has a separate time delay ($\delta 2$) and time constant ($\tau 2$) to describe the delayed onset;

$$\dot{V}O_2(t) = \text{Baseline} + \text{GAIN1}\,(1-e^{-(t-\delta 1)/\tau 1}) + \text{GAIN2}\,(1-e^{-(t-\delta 2)/\tau 2}) \quad (2.4)$$

An exponential function has been widely used to describe the slow component (Linnarson 1974; Camus et al. 1988; Barstow et al. 1996; Hebestreit et al. 1998; Bearden and Moffatt 2000). However, there is some controversy over whether the slow component is an exponential or a linear function (Barstow and Mole, 1991; Patterson and Whipp 1991; Gaesser and Poole 1996), particularly at the higher (severe) intensities where the theoretical asymptote may be above $\dot{V}O_2$max. Some studies have modelled the slow component rise as a linear function (Armon et al. 1991), whilst others have simply expressed the magnitude of the slow component as the difference between final $\dot{V}O_2$ and $\dot{V}O_2$ at 3 minutes (Billat et al. 1999b; Jones and McConnel 1999). This simple approach has been criticised however for underestimating the magnitude of the slow component (Bearden and Moffatt 2001b).

Several researchers have also attempted to incorporate the phase-1 response into the model (Barstow et al. 1996; Billat 2000) rather than regard it simply as a delay, since $\dot{V}O_2$ does increase during this phase. Again an exponential function has been used to describe this phase of the response, although as phase-1 is considered to begin at the onset of exercise this function does not contain a time delay (δ). This gives the equation:

$$\dot{V}O_2(t) = \text{Baseline} + \text{GAIN0}\,(1-e^{-(t)/\tau 0}) + \text{GAIN1}\,(1-e^{-(t-\delta 1)/\tau 1}) + \text{GAIN2}\,(1-e^{-(t-\delta 2)/\tau 2}) \quad (2.5)$$

Given the diversity of approach to modelling in this intensity domain, the willingness of authors to compare the various parameters between studies is surprising.

Research is also equivocal as to whether phase-2 $\dot{V}O_2$ kinetics are slowed (display a longer τ), in heavy compared with moderate intensity exercise. This debate primarily centres on two important studies from the same year. Patterson and Whipp (1991) found that the time constant increased with increasing exercise intensity whereas Barstow and Mole (1991) report no change. The two studies used slightly different mathematical models but both used a two-component model, with phase-1 regarded as a time delay. Whilst Barstow and

Mole did not demonstrate the slowing of kinetics shown by Patterson and Whipp, it is clear from their data that the highest work-rate resulted in the longest time constant. It may be that a small subject group (n = 4) and a large variability in this parameter prevented them from statistically demonstrating the same effect. Recently Carter et al. (2002), using a three-component model, showed that the phase-2 τ was increased in the heavy compared to the moderate intensity domain but unchanged across all supra AT work rates.

2.3.5 $\dot{V}O_2$ kinetics in the severe intensity domain

A key assumption exists surrounding the $\dot{V}O_2$ response to exercise in the severe intensity domain. Review papers are consistent that for severe intensity exercise, at a constant work rate, a slow component will be present and will be of a greater magnitude than is seen for heavy intensity exercise (Xu and Rhodes 1999). The response differs from that of heavy intensity exercise because $\dot{V}O_2$ is unable to reach a steady state but instead continues to rise until $\dot{V}O_2$max or exhaustion (Whipp and Ward 1990, Whipp 1994a, Whipp 1994b, Gaesser and Poole 1996, Xu and Rhodes 1999, Ward 1999). It is apparent, given this assumption, that a maximum steady state, in both blood lactate and $\dot{V}O_2$, is the logical lower limit for this exercise intensity domain. Research suggests that the highest work-rate at which a steady state in $\dot{V}O_2$ can be achieved coincides with the MLSS (Poole at al. 1988). However, the terms fatigue threshold and critical speed or power are also used to describe this same division of exercise intensity domains (Poole et al. 1988, Billat 2000).

When work rate is plotted against time to exhaustion the relationship is a hyperbolic one (Kennelly 1906; Hill 1925; Wilkie 1980; Billat 2000). When this relationship is plotted for an individual the asymptote represents the critical speed or power (Moritani et al. 1981). This asymptote is also often termed the fatigue threshold (Gaesser and Poole 1996; Billat 1999a; Ward 1999). The idea of a critical speed that represents the highest work rate that might be sustained for prolonged exercise is not a new concept (Hill 1924). In 1943 Francis (p. 315) described this asymptote (based on world running records) as the "dog trot" speed for a theoretical "perfect runner" that represented "the speed that he could maintain indefinitely without tiring (if it were not for lack of sleep, nourishment, etc.)". This early reference to critical speed highlights the fundamental flaw in the concept.

Francis (1943) believed that this intensity could be maintained indefinitely if substrates were available, whereas in reality time to exhaustion at such intensity is likely to be between 30 and 60 minutes (Housh et al. 1989; Scarborough et al. 1991).

Monod and Scherrer (1965) first identified the concept of a critical power for a given muscle. Moritani et al. (1981) then applied this critical power concept to whole body exercise on a cycle ergometer. Moritani et al. found no difference between critical power and power at the AT.

The calculation of critical power may be problematic due to the need to complete multiple tests at very high intensities and the subjective nature of fatigue (De Vries et al. 1982; Hill 1993; Bishop et al. 1997). Furthermore both critical power (De Vries et al. 1982, 1987; Jenkins and Quigley 1990) and critical speed (Highson et al. 1984) have been shown to be unreliable in calculating the highest work rate that may be sustained for a prolonged period of time.

With much controversy surrounding critical speed and power, it is surprising that it is widely accepted as being the demarcation point between the heavy and severe intensity domains (Poole et al. 1998; Hill and Smith, 1999). It is assumed to coincide with a maximum steady state in both blood lactate and $\dot{V}O_2$ (Ward 1999), although there is evidence to the contrary (McLellan and Cheung, 1992; Billat 1998a).

The physiological significance of the critical speed / power or fatigue threshold has been investigated, with particular reference to the behaviour of $\dot{V}O_2$. Poole et al. (1988, 1990) proposed that for any exercise intensity above critical power, the slow component of $\dot{V}O_2$ will drive $\dot{V}O_2$ to $\dot{V}O_2max$ instead of to a delayed steady state as would be the case in exercise intensities that fall below this threshold. The hyperbolic intensity-time to exhaustion relationship has critical power (CP) as its asymptote. The relationship between intensity and time to reach $\dot{V}O_2max$ has also been shown to be hyperbolic, yielding an asymptote that has been termed (CP') (Rowell et al. 1996). This second parameter (CP') theoretically represents the highest work rate that will not result in $\dot{V}O_2max$ being attained, and therefore the highest work rate where a steady state may be achieved. It has been demonstrated that CP and CP' are equal in both cycling (Hill and Smith 1999) and

running (Hill and Ferguson 1999). This has been interpreted as strong evidence that critical power is a good measure of the threshold between heavy and severe intensity exercise, and that all exhaustive exercise performed above this threshold will result in the attainment of $\dot{V}O_2$max (Hill and Smith 1999).

Billat et al. (1998a) demonstrated that it is possible for highly trained runners to reach a steady state in $\dot{V}O_2$ above their critical speed. Highly trained runners have also been shown to incur no $\dot{V}O_2$ slow component above critical speed (Billat et al. 1998b) when the slow component was calculated as the difference between the final $\dot{V}O_2$ and that at 3 minutes. However, Billat et al. have more recently used a three-component model to describe $\dot{V}O_2$ kinetics in the severe intensity domain and demonstrated a slow component of a small magnitude (Billat et al. 2000). There does though appear to be some question over the assumption that $\dot{V}O_2$ will reach $\dot{V}O_2$max for all exercise intensities above critical speed, particularly in highly trained populations.

The point that represents the change from heavy to severe intensity exercise is difficult to identify. This thesis was concerned with exhaustive exercise of a short duration however. The longest exercise transitions produced fatigue in approximately 8 minutes. Therefore the term severe was used to describe such transitions, since they must have been performed above MLSS in order for fatigue to occur in such a short duration.

2.3.6 $\dot{V}O_2$ kinetics in short duration exhaustive exercise

Surprisingly few investigators have focused on the $\dot{V}O_2$ response to exhaustive square wave exercise of a short duration. Much of the research has focused on very short duration, all out exercise (Katch 1973; Stevens and Wilson 1986; Kavanagh and Jacobs 1988), rather than the more constant intensity efforts that categorise athletic events such as middle distance running (Brandon 1995). In this exercise $\dot{V}O_2$ required might exceed $\dot{V}O_2$max and the third phase in $\dot{V}O_2$ kinetics (slow component) may not have sufficient time to become manifest (Whipp 1994a).

It has been established that $\dot{V}O_2$ kinetics are intensity dependent (Whipp, 1994a, 1994b). They are generally expressed as a mono-exponential function in such high intensity exercise, since no slow component will be present (Billat et al. 2000). However, when the $\dot{V}O_2$ required is greater than $\dot{V}O_2max$ modelling the response is problematic. In moderate and heavy intensity exercise $\dot{V}O_2$ will attain a steady state, providing the asymptote for the exponential function. In exhaustive exercise of a short duration this cannot occur, and the experimenter must decide whether this exponential should be expressed as relative to $\dot{V}O_2$ required (Margaria et al 1965), $\dot{V}O_2max$ (Hughson, 1978), or some other asymptotic value. During the modelling process the asymptote can be constrained to a given value (i.e. $\dot{V}O_2$ required or $\dot{V}O_2max$) or simply allowed to establish its own value from the data set. This is an important consideration, not only for the establishment of the asymptote, but also for the evaluation of the speed of the $\dot{V}O_2$ response. If the data set is modelled with the asymptote constrained to a $\dot{V}O_2$ requirement in excess of $\dot{V}O_2max$, it will display a greater τ than if unconstrained. Therefore caution should be exercised if such a constraint has been used, and conclusions have been drawn regarding the speed of the $\dot{V}O_2$ response. In any case such a constraint is problematic since it is impossible to accurately calculate the $\dot{V}O_2$ requirement by extrapolation of the $\dot{V}O_2$-WR regression (Ward 1999).

The first attempt to examine the $\dot{V}O_2$ response to constant intensity exercise of a short duration was by Astrand and Saltin (1961). Oxygen uptake was measured during exhaustive cycling of between ~ 2- and 8 minutes duration in five subjects. Expirate was collected into Douglas bags over periods of between 20 and 60 seconds. Astrand and Saltin (1961) reported that in a motivated subject 2 minutes exhaustive constant intensity cycling would give a $\dot{V}O_2$ that would be close to $\dot{V}O_2max$. However, it is apparent that in some of their subjects $\dot{V}O_2$ reached a plateau below its maximum in the shortest duration tests (2 - 3 minutes).

The $\dot{V}O_2$ kinetics in short duration exhaustive exercise (30-180 seconds) were further investigated by Margaria et al. (1965). Here a mixing chamber system was used in order to provide more data points for such short tests. Data were averaged every 10 - 15 seconds and the response was modelled. A semi-logarithmic function with $\dot{V}O_2$ required (above $\dot{V}O_2max$) as its asymptote was used. The results of this modelling suggested that $\dot{V}O_2$

kinetics were unchanged across all intensities with a half time of approximately 30 seconds. This would equate to a τ of about 43 seconds. There appear to be inconsistencies in this research however, in that Margaria et al. reported $\dot{V}O_2$ values that were higher (18%) than the reported $\dot{V}O_2max$ in the most severe intensity exercise (Hughson et al. 2000).

A similar approach to quantifying the $\dot{V}O_2$ response to short duration exhaustive exercise was used by Katch (1973), although in this case the exercise was a 1 minute 'all out' effort rather than a constant intensity exercise bout. Unlike Margaria et al., Katch (1973) considered that $\dot{V}O_2max$ necessarily represented the asymptote. Katch demonstrated a faster average half time for $\dot{V}O_2$ (18.4 seconds) than reported by Margaria et al. (1965). This equates to a τ of 26.6 seconds and would result in achievement of $\dot{V}O_2max$ in about 2 minutes. It should be remembered that this work rate would not be sustainable for 2 minutes, however. In comparing these findings to those of Margaria et al. (1965), it should be remembered that different mathematical models were used, and the faster time constant demonstrated by Katch (1973) will be the result of referencing the $\dot{V}O_2$ response to $\dot{V}O_2max$ rather than to the $\dot{V}O_2$ required. Gas samples were only collected every 20 seconds however and so whilst Katch (p. 200) described the model fit as "convincing", it should be realised that the model was fitted to just 3 data points and that the first of these would have been largely representative of the Phase-1 response.

The problem of whether $\dot{V}O_2$ kinetics should be referenced to $\dot{V}O_2max$ or the $\dot{V}O_2$ required was recognised by Hughson (1978). In that study subjects cycled to exhaustion at a power equivalent to 110 - 120% of $\dot{V}O_2max$ and the data were fitted with two models that differed in the asymptotic value ($\dot{V}O_2max$ v $\dot{V}O_2$ required). After 3 minutes of cycling the average $\dot{V}O_2$ for the seven subjects had reached only 79.5% of the $\dot{V}O_2$ required (approximately 90% $\dot{V}O_2max$). Hughson (1978) calculated the halftime for the $\dot{V}O_2$ response to be 36.6 seconds if referenced to $\dot{V}O_2max$, and 53.4 seconds if referenced to $\dot{V}O_2$ required (τ of 52.8 and 77.1 seconds respectively). When $\dot{V}O_2$ kinetics were referenced to $\dot{V}O_2max$ Hughson (1978) concluded, as had Katch (1973), that the response was well described as a single exponential term. However, Hughson considered that a single exponential model did not fit the data if referenced to $\dot{V}O_2$ required.

Hughson et al. further considered the issue of how to model data at such high intensities in a later study in which subjects completed exhaustive bouts of cycling at 57, 96 and 125 % $\dot{V}O_2$max (Hughson et al. 2000). Data from the 57 % $\dot{V}O_2$max tests (assumed to be moderate intensity exercise) were modelled using a two-component exponential model. Data from the two severe intensities were modelled in two ways: first as a three-component exponential model; and second as a semi-logarithmic model with $\dot{V}O_2$ required as its asymptote. When the data were modelled using the exponential functions the $\dot{V}O_2$ kinetics of the primary phase (phase-2) were shown to be faster at the severe intensities. Williams et al. (1988) had drawn a similar conclusion. However, since a three-component model was used in the severe intensity tests, τ was referenced to an asymptote that was below $\dot{V}O_2$max. This resulted in the apparent speeding of kinetics. Arguing that such an asymptote must be as a result of inadequate O_2 delivery and did not reflect where the response was originally tending to, Hughson et al. suggested that $\dot{V}O_2$ kinetics should not be modelled in this way. Therefore Hughson et al. applied a semi-logarithmic model (as previously applied by Margria et al. 1965) that was referenced to $\dot{V}O_2$ required. Using this model Hughson et al. (2000) demonstrated a slowing of $\dot{V}O_2$ kinetics at intensities close to or above $\dot{V}O_2$max. The model used, and more specifically whether the asymptote is constrained to the $\dot{V}O_2$ required, will have a considerable impact on the description of the response. It should also be noted that the semi-logarithmic model requires the accurate prediction of the $\dot{V}O_2$ required; and, as previously discussed, this is not possible (Bangsbo 1996a).

Hill and Stevens (2001) also attempted to answer the question of whether $\dot{V}O_2$ kinetics should be referenced to the $\dot{V}O_2$ required in the severe intensity domain. In this study data were modelled as an exponential function firstly using the complete data set and secondly using just the initial 45 seconds of data. Whilst the model using all data points reached an asymptote that was no different from $\dot{V}O_2$max, the model of the first 45 seconds produced an asymptote that was no different from the predicted $\dot{V}O_2$ required (calculated from sub AT exercise). This study gives strong support to the argument that $\dot{V}O_2$ kinetics should be referenced to $\dot{V}O_2$ required. The authors concluded that $\dot{V}O_2$max prevents the full

response from occurring and should not be considered the asymptote of the exponential response.

There were several limitations to the study of Hill and Stevens (2001) however. The phase-1 data were removed after visual inspection of the $\dot{V}O_2$ data and breath-by-breath data were modelled. The (mean ± SD) phase-1 duration was 17 ± 4 seconds. It appears that the initial 45 seconds of data may have been modelled on fewer than 30 seconds of breath-by breath data points on average. Furthermore, the exercise bouts were only performed once (rather than averaged over several repeated transitions). It is likely that the level of noise would be high (Lamarra et al. 1987) although the nature and magnitude of breath-by-breath noise has never been investigated in high intensity exercise. The SD of the asymptote for the first 45 seconds of data was also large (64% of the magnitude of the $\dot{V}O_2$ response itself). These factors would make it very unlikely for a difference to be shown between the asymptote and the predicted $\dot{V}O_2$ required. Finally, whilst Hill and Stevens claimed that all subjects were involved in physical fitness programmes, the aerobic ability of the subjects was limited ($\dot{V}O_2$max of 37 ± 11 ml.kg^{-1}min^{-1}).

In a review of $\dot{V}O_2$ kinetics across the range of exercise intensity domains, Whipp (1994a) argued that $\dot{V}O_2$ will always tend towards the $\dot{V}O_2$ required when this is above $\dot{V}O_2$max. Whipp stated that the mono-exponential response would be 'cut off' when $\dot{V}O_2$ reached its maximum rate, or in very short duration exhaustive exercise fatigue may occur before $\dot{V}O_2$max is reached

That short duration exhaustive exercise would always result in the achievement of $\dot{V}O_2$max was supported by Williams et al. (1988), who exercised subjects at 95, 100, 105 and 110% of the speed at $\dot{V}O_2$max on the treadmill. The mean time to exhaustion of these tests ranged from 126 seconds for the highest intensity to 301 seconds for the lowest. Peak $\dot{V}O_2$ from these exhaustive constant intensity tests was not different to $\dot{V}O_2$max, as calculated from an incremental test, supporting the view of Whipp (1994a, b) that all such intensities would result in the achievement of $\dot{V}O_2$max. However, contrary to the findings of Margaria et al (1965), Williams et al. (1988) found $\dot{V}O_2$ kinetics to be affected by exercise intensity. The highest exercise intensity resulted in a faster time constant (τ).

Williams et al. used a single exponential model that was not constrained to the $\dot{V}O_2$ required however. The view that all severe intensity exercise will result in $\dot{V}O_2max$ was reinforced by a similar study by the same group of researchers (Hill and Ferguson 1999). Again no difference in $\dot{V}O_2peak$ was found between exhaustive constant intensity exercise at intensities between 95 and 110% of the speed at $\dot{V}O_2max$ and that measured during an incremental test (Hill and Ferguson 1999). A similar result was demonstrated in children with no difference found in $\dot{V}O_2peak$ between an exhaustive incremental treadmill test and three severe intensity efforts (Rowland 1993).

The idea that exhaustive exercise of a very short duration results in $\dot{V}O_2$ rising in an exponential fashion towards the $\dot{V}O_2$ required but being 'cut off' at $\dot{V}O_2max$ (Whipp 1994a, b) has much support (Margaria et al. 1965; Williams et al. 1988; Hill and Ferguson 1999). However, it should be recognised that only Margaria et al. (1965) and Hughson et al. (2000) have actually modelled this response referenced to the $\dot{V}O_2$ required. Furthermore, the study of Margaria et al. has received criticism for possible technical limitations in the data acquisition (Hughson et al. 2000). The data of Margaria et al. (1965) were certainly modelled based on very few data points since sampling was at 10-15 second intervals and the majority of the tests lasted 1 minute or less. However there are also other published data that question the assumptions made (Whipp 1994a) about $\dot{V}O_2$ kinetics in severe intensity exercise.

The data of Astrand and Saltin (1961) that have been discussed previously do not wholly support these assumptions. It is apparent from this study that some of the subjects, during 2-3 minutes of exhaustive cycling, demonstrated a plateau in $\dot{V}O_2$ that was below $\dot{V}O_2max$. Subjects performing exhaustive cycling at 110-120% $\dot{V}O_2max$ have also been demonstrated to reach only 90% of $\dot{V}O_2max$ after 3 minutes of exercise (Hughson 1978). If these data cast some doubt over the assumption that $\dot{V}O_2$ is tending to the $\dot{V}O_2$ required, data published by Spencer et al. (1996) and Spencer and Gastin (2001) clearly demonstrate a very different response to that suggested by Whipp (1994a).

Spencer et al. (1996) published a study on trained sprinters and middle distance runners in which they evaluated the relative contributions of the aerobic and anaerobic systems, using

the accumulated oxygen deficit method, in the 400, 800 and 1500 m events. Oxygen uptake was monitored on a breath by breath basis as subjects ran on a motorised treadmill at speeds equivalent to their best track times. The sprint trained runners ran only the 400 m trial, whereas the middle distance runners were required to run both the 800 and the 1500 m trials. The results of the 800 and 1500m trials were striking. The mean $\dot{V}O_2$ required for the two trials was 112% $\dot{V}O_2$max (800 m) and 102% $\dot{V}O_2$max (1500 m), yet mean graphs of the $\dot{V}O_2$ response show a clear plateau of $\dot{V}O_2$ below $\dot{V}O_2$max in both trials. Peak oxygen uptake was lower in the 800 than the 1500 m trial (~91 v ~93 % $\dot{V}O_2$peak). In both trials a clear plateau was apparent.

Spencer et al. (1996) also highlighted the intensity dependence of $\dot{V}O_2$ kinetics. Comparing % $\dot{V}O_2$max reached after 30 seconds of exercise in the three trials, they assessed the speed of the aerobic systems to respond to increasing energy demand. Oxygen uptake was found to be at 94, 69 and 59% $\dot{V}O_2$max respectively for the 400, 800 and 1500 m trials. The sprint-trained group, unlike the middle distance runners, was able to get very close to $\dot{V}O_2$max in their 400 m trial, despite this being the shortest duration. It should be recognised that the sprinters were working at a much higher intensity, as expressed relative to $\dot{V}O_2$max, than the middle distance runners (170 vs. 112 and 102 %).

The work of Spencer et al. (1996) and Spencer and Gastin (2001), using a specifically trained population, questions two major assumptions regarding the kinetics of $\dot{V}O_2$ in high intensity exercise: first that $\dot{V}O_2$ will tend in an exponential fashion to the $\dot{V}O_2$ required (Whipp 1994a); and second that all intensities above critical speed will result in the achievement of $\dot{V}O_2$max. Spencer et al. did not comment on the significance of the $\dot{V}O_2$ response as the focus of the studies was quantification of the anaerobic contribution to the exercise. An important question remains then, as to whether Spencer et al. demonstrated a clearer plateau in $\dot{V}O_2$ (than had Astrand and Saltin (1961)), because of the difference in the mode of exercise or the difference in the level of aerobic training and capability of the subjects used.

In modelling the $\dot{V}O_2$ response to short duration exhaustive exercise, as with heavy and severe intensity exercise the method of mathematical modelling used varies considerably

between researchers, despite such a small amount of research being conducted in this area. As previously discussed controversy exists over whether the response should be modelled as tending to $\dot{V}O_2max$ (Williams et al. 1988) or the $\dot{V}O_2$ required (Hughson 2000). Curiously, even though the 'classic' view of $\dot{V}O_2$ kinetics in this intensity domain would describe an initial delay phase followed by the second primary phase that would take $\dot{V}O_2$ to $\dot{V}O_2max$ (Whipp 1994a), no researcher has used a two-component model to describe the response. The use of a two-component model has even been described as "inappropriate" (Hughson 1978 p. 43) based on the assumption that the $\dot{V}O_2$ response should be modelled as tending to the $\dot{V}O_2$ required.

Those modelling data in this domain (with the exception of Hughson et al. 2000) have chosen not to model the initial phase of $\dot{V}O_2$ kinetics. Rather they have chosen to incorporate this rise in $\dot{V}O_2$ into a single exponential describing the second phase. The same two phases are regularly modelled as two separate exponential parameters in moderate and heavy intensity exercise (Barstow and Mole 1991; Barstow et al. 1996; Jones and McConnell 1999), yet rarely is this first component incorporated into models of short duration exhaustive exercise. This anomaly is highlighted in a study where subjects exercised at 95, 100, 120 and 140% of the speed at which $\dot{V}O_2max$ was achieved in an incremental test (Billat 2000). In the 100, 120 and 140% trials the data were modelled using a single exponential (including no δ) to describe the whole response, whereas a three-component exponential (incorporating separate exponential functions to describe phase-1 and the slow component) was used to describe the 95% trial (Billat 2000). Such an approach, whilst mirroring what has been used in other studies, makes the comparison between the exercise intensities difficult. The intention here is not to suggest that the Phase-1 response should be included into the model; it is merely to stress the inconsistency of approach (even within a single study). The physiology surrounding this phase is poorly understood, there is a lack of data points early in the transition to exercise, and there is no reason to assume that the exponential model is appropriate for this purpose (Whipp and Ozyener 1998). Therefore, it is more appropriate to model phase-1 simply as a delay.

2.4 Physiological mechanisms underlying $\dot{V}O_2$ kinetics

2.4.1 Evidence for an oxygen delivery limitation

At the onset of exercise the oxidative systems are relatively slow to respond and anaerobic systems must meet the initial demand for ATP re-synthesis (Boutellier et al. 1984). There is no consensus on exactly which factors determine the $\dot{V}O_2$ response at the onset of exercise, with investigators arguing for both central (oxygen delivery) and peripheral (oxygen uptake at the working muscle) limitations to oxygen uptake (Roca et al. 1989; Wagner 1991, 1996; Poole and Richardson 1997; Richardson 1998; Di Prampero 1999; Grassi 2001; Hughson et al. 2001a). There is a large body of research that has attempted to identify the underlying physiological mechanisms that may limit and determine $\dot{V}O_2$ at the onset of exercise (Tschakovsky and Hughson 1999). Research has sought to investigate the factors responsible for both the primary (Phase-2) $\dot{V}O_2$ response (Walsh 1992) and the slow component of $\dot{V}O_2$ that is manifest in heavy and severe intensity exercise (Gaesser and Poole 1996). The Phase-2 response is representative of the full response in moderate intensity exercise and also potentially in very high intensity exercise where the sustainable duration does not allow for a third phase to be manifest.

The Phase-2 $\dot{V}O_2$ response is generally considered to be mono-exponential in nature and the asymptote of this response is a linear function of work rate (Walsh 1992). The deviation from linearity seen above the AT is caused by the increasing magnitude of the slow component (Zoladz et al. 1998). Furthermore, Phase-2 has been shown to be representative of muscle $\dot{V}O_2$ (Knight et al. 1992). This review will primarily examine the potential limitations to this phase.

The first phase of $\dot{V}O_2$ kinetics is regarded as essentially a delay phase that represents the venous transit time from the working muscle to the lung (Whipp 1994a). Despite this delay however $\dot{V}O_2$ and $\dot{V}CO_2$ (as measured at the mouth) have been shown to increase rapidly during this first phase. These changes are thought to be due to an increased cardiac output resulting in an increased pulmonary blood flow (Whipp and Ward 1990). The cardiodynamic dependence of $\dot{V}O_2$ at the onset of exercise was recognised early in

the 20th century (Krogh and Lindhard 1913) and has received support from recent studies. It has been shown (Cumin et al.1986) that there are proportional increases in pulmonary blood flow and $\dot{V}O_2$ during this initial phase, and that $\dot{V}O_2$ increases at the onset of exercise even when $\dot{V}_E$ is constrained to remain at resting levels (Weissman et al. 1982). It has also been shown that $\dot{V}O_2$ increases more slowly when stroke volume (and therefore presumably $\dot{Q}$) is above the normal resting level prior to exercise (Weiler-Ravell et al. 1983; Whipp et al. 1982a), further supporting this hypothesis. A recent study examining the $\dot{V}O_2$ kinetics of heart transplant patients demonstrated an abnormally slow Phase-1, due to the patients' inability to rapidly increase cardiac output at the onset of exercise (Mettauer et al. 2000).

Casaburi et al. (1989a) have presented evidence that questions the apparent consensus on the phase-1 $\dot{V}O_2$ kinetics. The study questioned the long standing assumption (Krogh and Lindhard 1913) that increases in $\dot{V}O_2$ (measured at the mouth) could not be due to a change in the circulating blood gas concentrations because of the transit delay from the working muscle. Casuburi et al. (1989a) showed a decrease in pulmonary-arterial oxygen concentration during this initial phase. The authors proposed that this increase in $\dot{V}O_2$ was due to washout from relatively underperfused areas. Further support for this hypothesis came from a study that showed no change in pulmonary-arterial blood gases during this phase when the exercise bout was preceded by light exercise rather than a period of rest (Casaburi et al 1989b).

Whilst the increase in $\dot{V}O_2$ that occurs during phase-1 does not represent oxygen consumption at the working muscle, it is important to recognise that $\dot{V}O_2$ does increase during this initial phase. Whether or not the initial phase is modelled may affect the calculated parameters for the second phase. Phase-1 is often constrained to an exponential fit (as is used for the following phases) (Barstow and Mole 1994; Barstow et al. 1996; Jones et al. 1999). However, this has never been demonstrated to be exponential (Whipp and Ward 1990) and has even been shown not to be exponential in some exercise transitions (Whipp and Ozyener 1998).

The principle phase of $\dot{V}O_2$ kinetics that represents oxygen consumption at the working muscle is Phase-2. Oxygen consumption measured at the mouth has been shown to correspond to muscle $\dot{V}O_2$ during this second phase (Knight et al. 1992; Poole et al. 1992). Unlike the Phase-1 response this second phase has been shown to follow first order exponential kinetics (Miyamoto et al. 1982; Gaesser and Poole 1996). This is perhaps surprising considering the number of complex reactions and steps in the oxygen pathway from atmosphere to mitochondria (Wagner 1992). That the primary $\dot{V}O_2$ response follows first order exponential kinetics and also has a linear relationship with work rate suggests that there may be a single rate limiting step in this process (Walsh 1992). Debate has long existed over whether this is an oxygen transport limitation (Hughson 1990) or a limitation in the ability of the muscle to use oxygen (Whipp and Mahler 1980).

The argument for a central limitation to $\dot{V}O_2$ kinetics has largely arisen from studies showing a change in time constant (τ) with a change in the inspired fraction of O_2 (F_IO_2) or following periods of training and detraining. A decreased F_IO_2 has been shown to result in a slowing of $\dot{V}O_2$ kinetics as demonstrated by an increased τ (Springer et al. 1991; Walsh and Banister 1995; Engelen et al. 1996). Conversely an increased F_IO_2 has resulted in a decreased τ (Pederson 1983; Walsh and Banister 1995; McDonald et al. 1997). However this effect has only been demonstrated in whole body exercise above the AT where differences in the overall response could be due to a change in the slow component. Indeed some studies have shown no difference in the $\dot{V}O_2$ response in either hypoxic or hyperoxic conditions (Linnarson et al. 1974). Gutierrez et al. (1989), using an isolated rabbit hind-limb preparation, showed that reduced oxygen transport resulted in decreased $\dot{V}O_2$ and an increase in anaerobic ATP resynthesis via the anaerobic glycolytic and PCr systems. However, changes in the response as a result of changes in the inspired O_2 fraction might also support the argument for a peripheral limitation, since arterial PO_2 will dictate the mitochondrial PO_2 required for adequate O_2 flux (Walsh 1992).

Patient populations suffering from disorders that result in an impaired capacity for oxygen delivery provide further evidence for a central limitation. Heart transplant (Mettauer et al. 2000) and peripheral arterial disease (Bauer et al. 1999) patients have a slow $\dot{V}O_2$

response. A peripheral compensation, resulting in an increased arterio-venous difference in O_2, has been shown in patients with chronic heart failure (Katz et al. 2000), however.

To examine whether oxygen transport might indeed be the limiting factor in $\dot{V}O_2$ kinetics, other investigators have sought to compare the various components of oxygen delivery to the overall $\dot{V}O_2$ kinetics. A close relationship has been shown between $\dot{V}O_2$ and cardiac output (Cummin et al. 1986), and the oral ingestion of a beta-blocker to slow heart rate during exercise is also associated with a slowed $\dot{V}O_2$ response (Hughson and Smyth 1983; Peterson et al. 1983). Ingestion of beta-blocker has also been shown to result in a compensatory arterial vasoconstriction to preserve blood pressure thus potentially limiting blood flow (Pawelczyk et al. 1992), but only in severe intensity exercise (Buckwalter et al. 1997, 1998). When heart rate and total cardiac output have been examined in healthy subjects, both have been shown to demonstrate a faster response than that of $\dot{V}O_2$ (Cerretelli et al. 1966; Davies et al. 1972; Linnarson 1974; Eriksen et al. 1990; De Cort et al. 1991). Furthermore, it has been shown that it is possible to reach $\dot{V}O_2max$ while cardiac output is still increasing (Faulkner et al. 1971). Phase-2 $\dot{V}O_2$ kinetics in children were shown to be no different to those of adults adults despite the children's slower heart rate kinetics (Cooper et al. 1985). Babcock et al. (1994) also demonstrated an increased τ as a function of age; but a similar relationship was not found with HR kinetics. Heart rate and cardiac output are therefore unlikely to dictate the speed of the primary $\dot{V}O_2$ response.

Further evidence to support a vascular limitation comes from the effect that a change in body position during exercise has on $\dot{V}O_2$ kinetics (Hughson et al. 1991). The onset of exercise has been shown to result in an initial fall in peripheral resistance and arterial pressure, although these parameters return to normal levels within 30 seconds (Sprangers et al. 1991). These vasodilatory changes occur in unison with abrupt increases in cardiac output (Loepky et al. 1981), and the combined effect is an increase in oxygen delivery at the onset of exercise. When exercise in the upright position is compared to supine exercise, both blood flow to the exercising legs and $\dot{V}O_2$ display slower kinetics in the supine position (McDonald et al. 1998), despite an increased cardiac output in this position (Hughson et al. 1993). However there was a smaller femoral arterial diameter in

the supine position, limiting the rate of blood flow at the onset of exercise; this diameter was still reduced after six minutes of exercise (McDonald et al. 1998).

It should be recognised that oxygen delivery to the muscle is dependent on the arterial concentration of O_2 which is itself dependent on other factors such as haemoglobin concentration (Kurdak et al. 1995), as well as cardiac output. Raynaud et al. (1973) have shown not only that muscle O_2 delivery has a faster $\dot{V}O_2$ response than that measured at the mouth but also that the O_2 concentration in venous blood returning to the lungs is highest during the first 40 seconds of exercise. Again this would suggest that O_2 delivery is not the controlling mechanism.

The muscle capillary bed where O_2 will be unloaded from the blood is another potential site for the regulation of $\dot{V}O_2$ kinetics (Roca et al. 1989; Walsh 1992). Blood flow measurements from electronically stimulated canine muscle suggest that blood flow to the muscle can respond faster than heart rate at the onset of exercise, making this an unlikely limitation (Honig et al. 1980). Muscle capillarity (Gollnick 1966), but not muscle blood flow (Holloszy 1973), is enhanced following a period of endurance training and $\dot{V}O_2$ kinetics are accelerated by such an intervention (Hickson et al. 1978; Powers et al. 1985). However, the O_2 extraction may be enhanced through the increased transit time the increased capillary density would allow, although Richardson et al. (1993) showed no transit time limitation in single leg exercise even with very high muscle blood flow (385 $ml.min^{-1}.100g^{-1}$).

2.4.2 Evidence for an O_2 uptake limitation to the primary $\dot{V}O_2$ response

Alternative hypotheses exist however arguing that the contractile activity of the muscle cell rather than O_2 availability determines the rate of aerobic ATP re-synthesis (Walsh et al. 1992; Richardson et al. 1999). Sahlin et al. (1988) found that the rate of change in a stepwise increase in exercise intensity (15 minutes gradual increase to achieve the exercise intensity or an immediate change) had no effect on the accumulated oxygen deficit incurred (and therefore presumably no effect on $\dot{V}O_2$ kinetics). They concluded that the rate of aerobic metabolism was governed by the metabolic status of the working

muscle, particularly increased ADP and P_i levels (as shown by biopsy), not by any delay in O_2 transportation since anaerobic energy production was unaffected by the rate of change in work rate. The potential peripheral regulators of this inertia in oxidative metabolism are of course numerous. There are many complex reactions underlying cellular respiration (Hultman et al. 1990; Greenhaff and Timmons, 1998) and it is beyond the scope of this review to identify them all. The ratios of ATP/ADP and NAD^+/NADH have both been suggested as potential regulators (Wilson 1994), and more recently the pyruvate dehydrogenase (PDH) complex reaction has been suggested as a possible limitation (Timmons et al. 1996). Connett and Honig (1989) failed to demonstrate a major regulatory role for ADP, however.

The peripheral mechanism that has received the most widespread support in the literature is the regulation of $\dot{V}O_2$ via the PCr shuttle (Sweeney 1994). The PCr system itself has an important role to play in anaerobic energy production at the onset of exercise (Maughan et al. 1997). The PCr shuttle hypothesis suggests that it may also provide a vital link between the site of ATP production (mitochondria) and the site of ATP usage (myofibrils) (Sweeney 1994). Reciprocal changes in the levels of PCr and creatine at both sites combine to regulate mitochondrial respiration. At the onset of exercise the decrease in PCr and increase in creatine at the myofibrils causes an increased flux of creatine to the mitochondria resulting in an increased ATP regeneration. This increased ATP generation will cause an increased O_2 uptake resulting in a decreased mitochondial PO_2, increasing the pressure gradient between blood and mitchondria, and therefore increasing O_2 flux into the muscle cell.

A relationship between the reduction in cellular PCr and ATP concentrations and the magnitude of the oxygen deficit has long been recognised (Knuttgen and Saltin, 1972). Oxygen uptake kinetics have also been shown to be slowed when PCr stores are reduced (Paganelli et al. 1989). If it is accepted that the PCr shuttle is the key regulator in phase-2 $\dot{V}O_2$ kinetics it should be expected that PCr itself should exhibit a similar time course to $\dot{V}O_2$. Furthermore, a linear relationship should exist between the level of PCr degradation and the rate of muscle oxygen consumption.

Advances in nuclear magnetic resonance (NMR) spectroscopy have enabled the investigator to examine the time course of PCr degradation in tandem with the measurement of $\dot{V}O_2$ (Whipp et al. 1999). Such research has revealed not only that PCr demonstrates first order exponential kinetics as does $\dot{V}O_2$ but also that the time course of the response is similar for PCr and $\dot{V}O_2$ for moderate intensity exercise (Mole et al. 1985; Meyer 1988; Marsh et al. 1993; McCreary et al 1996). That the relationship between the level of PCr degradation and the rate of O_2 consumption is a linear one has been demonstrated for *in vivo* animal muscle (Mahler 1985), *in situ* animal muscle (Meyer 1988), and exercising muscle in man (Linnarson et al. 1974). These studies suggest that O_2 flux will not occur without PCr degradation and that the rate of O_2 flux is proportional to the rate of PCr degradation (Walsh 1992).

There is compelling evidence therefore for both a central (blood flow) and a peripheral (PCr mediated) control of the primary kinetics of $\dot{V}O_2$. However, it should be recognised that the dominant factor in this control may depend on the intensity and the nature of the exercise (particularly the amount of muscle being exercised) (Di Prampero 1985).

2.4.3 Potential mechanisms of the $\dot{V}O_2$ slow component

Of the possible mechanisms responsible for the slow component, lactate has received probably the most attention, since the slow component itself has been shown to be closely tied to the lactate response (Whipp and Wasserman 1986; Barstow et al. 1993). Reductions in end exercise lactate concentrations following endurance training coincide with a reduction in the magnitude of the slow component (Casaburi et al. 1987) and Ryan et al. (1979) reported an increased exercising $\dot{V}O_2$ after L-(+)-lactate infusion.

An increase in $\dot{V}O_2$, it has been suggested, might be due to a lactate induced increase in gluco/glyconeogenesis, although the impact of this on metabolic rate is likely to be small (Gaesser 1994). A perhaps more plausible hypothesis is that the slow component is mediated by a lactic acidosis mediated Bohr effect, allowing a greater aerobic contribution to metabolism (Wasserman et al. 1991; Stringer et al. 1994). This possibility is supported by a high correlation between the magnitude of the slow component and the decrease in

haemoglobin saturation during cycle exercise (Belardinelli et al. 1995). Whilst such an hypothesis may explain the delayed onset of the slow component, it does not explain why there appears to be an increased $\dot{V}O_2$ required in heavy intensity exercise over and above that predicted from a sub AT $\dot{V}O_2$-WR relationship (Zoladz et al. 1995; Jones et al.1999; Morton and Billat 2000).

Steed et al. (1994) demonstrated a slow component in treadmill running without a significant rise in blood lactate concentration. Barstow (1994) demonstrated that femoral vein lactate levels were elevated in advance of the manifestation of a slow component suggesting no functional coupling of the two. Finally an increase in lactate concentration due to either L-(+)-lactate or adrenaline infusion was found not to elevate $\dot{V}O_2$ in canine muscle (Gaesser 1994; Gaesser et al. 1994; Poole et al. 1994b). This suggests that the relationship between the slow component and blood lactate is not one of cause and effect.

Adrenaline has also been suggested as a possible mediator of the slow component (Gaesser and Poole 1996). Whilst the threshold behaviour of adrenaline is similar to that of lactate (Turner et al. 1995; Dickhuth et al. 1999; McMorris et al. 2000) a causative link does not seem to exist with the slow component. Resting $\dot{V}O_2$ is elevated by adrenaline infusion (Staten et al. 1987), but exercise $\dot{V}O_2$ is unaffected despite a large increase in adrenaline levels (Gaesser et al. 1994).

It is apparent that there will be an oxygen cost for the work of the respiratory muscles during exercise and that the increasing ventilation required at higher exercise intensities will increase total $\dot{V}O_2$ (Bartlett et al. 1958). Since ventilation will increase during the period where a slow component is incurred there will be an increase in the oxygen cost of this respiratory work (Hagberg et al. 1978). It follows that there must be some contribution therefore to this excess $\dot{V}O_2$ from respiratory muscle work, since $\dot{V}_E$ typically increases by as much as 60 $L.min^{-1}$ between the third minute and the end of severe intensity exercise (Poole et al. 1991). However, when the additional O_2 cost of this respiratory work is examined (Aaron et al. 1992), it is clear that this will only account for approximately 18-23 % of the excess $\dot{V}O_2$ of the slow component (Womack et al. 1995; Gaesser and Poole 1996).

There is evidence that the source of the majority of the $\dot{V}O_2$ slow component is the working (skeletal) muscle (Mole and Coulson 1985; Vollestad et al. 1990; Poole 1994). Poole et al. (1991) made simultaneous measurements of both leg and pulmonary $\dot{V}O_2$ during heavy intensity cycling, and showed that 86% of the additional rise in $\dot{V}O_2$ beyond the third minute of exercise could be attributed to a rise in leg $\dot{V}O_2$. It is established that the majority of this excess $\dot{V}O_2$ originates in the working musculature and is peripheral in nature (Poole 1994). The exact mechanism responsible has not been established but the most widely accepted hypothesis involves the recruitment of less efficient type II muscle fibres (Borrani et al. 2001; Lucia et al 2002a). Such a hypothesis might explain both the excess $\dot{V}O_2$ and the delay in onset of the slow component (Whipp 1994a; Poole et al 1994a; Barstow et al. 1996; Billat 2000). The question of the mechanism responsible for such a shift in fibre recruitment pattern remains unresolved. However, it has been suggested that a decreasing pH, whilst having little effect on mitochondrial respiration, might affect the contraction of working muscles and result in the recruitment of less efficient units (Willis and Jackman, 1994).

2.5 Factors shown to influence the $\dot{V}O_2$ response to a given exercise intensity

2.5.1 *The effect of prior exercise on $\dot{V}O_2$ kinetics*

It has been demonstrated that $\dot{V}O_2$ kinetics are faster for heavy intensity exercise if the exercise is preceded by a bout of heavy intensity exercise (Gaushe et al. 1989; Gerbino et al. 1996). The use of a mono-exponential model (as used in these studies) has been criticised (Morton 1987; Burley et al. 2000) since it does not discern between the primary and Phase-3 responses. Prior moderate intensity exercise does not accelerate $\dot{V}O_2$ kinetics above that seen from rest (Gerbino et al. 1996), however. Furthermore, the intensity of prior exercise was found not to affect the response to moderate intensity exercise (McDonald et al. 1997). There are potential performance benefits of prior heavy intensity exercise, as it may reduce the overall O_2 cost of the exercise (Gerbino et al. 1996; McDonald et al. 1997), and perhaps therefore spare the anaerobic capacity.

However, it is unclear whether prior exercise improves running (Andzel 1978) or swimming (Houmard et al. 1991; Mitchell and Huston 1993) performance (as shown by changes in performance time or time to exhaustion).

Gerbino et al. (1996) and McDonald et al. (1997) hypothesise two possible mechanisms responsible for the effect of supra AT exercise on the $\dot{V}O_2$ kinetics for a subsequent bout of heavy intensity exercise. Since the speeding of $\dot{V}O_2$ kinetics is seen only if the prior exercise is performed above the AT, both proposed mechanisms involve the effects of metabolic acidosis. First, O_2 delivery may be decreased due to vasodilation and an already elevated blood flow; second, an acidemia induced Bohr effect (rightward shift of the oxy-haemoglobin dissociation curve) may result in an improved diffusion between capillary blood and mitochondria. The preferred explanation of these authors is that the effect was due to a blood flow limitation since the increased acidosis had no effect on a bout of moderate intensity exercise (Gerbino et al. 1996). However this does assume that a different mechanism determines the $\dot{V}O_2$ response in different intensity domains. That is it assumes that O_2 delivery is only limiting for supra AT exercise intensities.

Burnley et al. (2000) and Bearden and Moffatt (2001c) revisited the work of Gerbino et al. (1996) and McDonald et al. (1997). Both Burnley et al. and Bearden and Moffatt, challenged the conclusion that the primary $\dot{V}O_2$ response to heavy intensity exercise was faster following prior heavy intensity exercise (Gerbino et al. 1996). Burnley et al. (2000) replicated the study of Gerbino et al. (1996), but modelled the data using a three-component exponential model in order to separate the phase-2 from the phase-3 response. McDonald et al. (1997) had already modelled similar data in such a way, but had been criticised (by Burnley et al.) for analysing the data as an overall mean response time (MRT), which does not distinguish between the primary response and the phase-1 or slow component responses (Burnley et al. 2000). The MRT is the time taken to reach 63% of the overall response (effectively the sum of the delay(s) and the constant(s) for an exponential phase or the whole response). Modelling of each phase of the $\dot{V}O_2$ response separately, suggested that the apparent speeding of $\dot{V}O_2$ kinetics with prior heavy intensity exercise, was not due to acceleration of the phase-2 response. The apparent acceleration of the overall response was rather from a reduction in the magnitude of the slow component (Burnley et al. 2000; Bearden and Moffatt 2001c). This questioned the

assumption that the acidosis from prior heavy exercise (improved muscle blood flow and Bohr effect) alters the primary kinetics in the first few minutes of exercise (Burnley et al. 2000).

Simultaneous sampling of venous blood and measurement of blood flow using Doppler ultrasound have been used to further investigate the mechanisms underlying the changes in $\dot{V}O_2$ kinetics in a second bout of heavy intensity exercise (McDonald et al. 2000). Both muscle blood flow and $\dot{V}O_2$ were elevated in the first 30 seconds of a second bout of handgrip (not whole body) exercise. This study, in contrast to the findings of Burnley et al. (2000), clearly demonstrated a link between an improved muscle blood flow and accelerated $\dot{V}O_2$ kinetics. These contrasting results have lead to debate between these two research groups (Jones et al. 2001; Hughson et al. 2001b).

Oxygen uptake kinetics are faster during a step change in exercise intensity (i.e. from a low to a higher work rate), compared to exercise from rest (Diamond et al. 1977; Hughson and Morrissey 1982). These two studies drew contrasting conclusions about the mechanism of this effect. Hughson and Morrissey (1982) suggested it was due to improved O_2 transport, whilst Diamond et al. (1977) suggested an improved O_2 uptake. Yoshida et al. (1995) sought to answer this question by performing exercise-to-exercise transitions of single leg exercise, but performed the second bout of exercise with either the same leg or the other leg. Oxygen uptake kinetics were speeded in the pre-exercised leg but not in the other despite a similar increase in cardiac output. It was concluded that the speeding of kinetics was due to an improved O_2 uptake, due to local metabolic conditions, and not an improved O_2 transport (Yoshida et al. 1995).

Several authors have manipulated acid base status to investigate its effect on exercise. However an increased alkalosis, through sodium bicarbonate ingestion, was shown not to affect the $\dot{V}O_2$ response for heavy intensity exercise (Heck et al. 1998). Furthermore. time to exhaustion at 90 and 95% $\dot{V}O_2$max has been shown to be decreased when acidosis is increased, and increased when alkalosis is increased, through prior administration of drugs (Jones et al. 1977; Sutton et al. 1979). Oren et al. (1982) examined the effect of both acidosis and alkalosis on respiratory parameters, during moderate intensity cycling. Whilst acid base status was found to affect the kinetics of both $\dot{V}_E$ and $\dot{V}CO_2$, no effect

was demonstrated for $\dot{V}O_2$ kinetics. This supports the findings of Gerbino et al. (1996), who reported a warm up effect in heavy but not in moderate intensity exercise.

2.5.2 Fibre type and $\dot{V}O_2$ kinetics

Skeletal muscle is considered to comprise two major muscle fibre types, generally referred to as fast and slow twitch, or type I and type II fibres (Brooke and Kaiser 1970). Type II fibres are often subdivided into type IIa and type IIb, depending on the oxidative capacity of the fibres (Gollnick and Matoba 1984). It is established that type II muscle fibres have a smaller potential for aerobic energy production and consume more O_2 for the same power output when compared to type I fibres (Crow and Kushmerick 1982; Willis and Jackman 1994). Furthermore it has been shown that highly trained athletes with a high percentage of type I fibres exhibit good cycling economy, i.e. a low $\dot{V}O_2$ cost for a given power output (Coyle et al. 1982; Horowitz et al. 1994). This effect does however seem less apparent at very high work rates (Suzuki 1979). Endurance runners are known to have a higher percentage of type I fibres than sprint runners (Gollnick et al. 1973, Costill et al. 1976), and also have better running economy (Kaneko et al. 1983).

There are several reasons why type II fibres are likely to be less efficient than type I fibres. Type II fibres produce more heat for a given tension, have a greater calcium pump activity, greater actinomyosin turnover, and a lower mitochondrial P/O ratio (Crow and Kushmeric 1982; Barstow et al. 1996). This reduction in mitochondrial P/O ratio may be due to type II fibres having a greater reliance on the FAD linked α-glycerophosphate shuttle over the NAD linked malate-aspartate electron-transfer shuttle (Sanchz and Henriksson 1987). Indeed any greater input from this less efficient hydrogen transfer shuttle, for example due to saturation of the other, would result in an increased $\dot{V}O_2$ (Whipp 1994a).

It has been established that there is an increasingly greater recruitment of type II muscle fibres as work rate increases (Vollestad and Blom 1985). Type II fibres have been shown (in the mouse) to demonstrate slower $\dot{V}O_2$ kinetics than Type I (Crow and Kushmeric 1982), potentially leading to a delay in the attainment of a steady state. However, Barstow and Mole (1991) note that the different response times of fibre types cannot, per se,

explain the delayed onset of the slow component. Whatever the mechanism responsible for the slow component, it does not become manifest until later in the exercise. A study simultaneously measuring $\dot{V}O_2$ and integrated electromyogram (iEMG) (quadriceps) during cycling has demonstrated an increase in muscular recruitment in association with the slow component of $\dot{V}O_2$ (Shinohara and Moritani 1992). This suggests that the excess $\dot{V}O_2$ may be coupled to a change in recruitment pattern and perhaps an increase in the recruitment of less efficient type II muscle fibres.

This review has so far examined the effect of fibre type on the slow component of $\dot{V}O_2$. Yet it should be realised that the reduced efficiency of type II fibres, coupled with their slower oxygen uptake kinetics, will also affect the primary phase of the $\dot{V}O_2$ response (phase-2). As previously stated, endurance athletes have been shown to exhibit a relatively high percentage of type I muscle fibres (Gollnick et al. 1972; Costill et al. 1976). Endurance athletes also exhibit faster primary kinetics than sprint athletes (Edwards et al. 1999) as well as a smaller slow component (Billat 1998a). Barstow et al. (1996) found that fibre type significantly affected both fast and slow phases of $\dot{V}O_2$ kinetics for heavy intensity exercise. However, such a comparison has yet to be conducted at severe intensities where a two-phase response might be expected.

2.5.3 Effects of training status and aerobic fitness on $\dot{V}O_2$ kinetics

Similar to (and perhaps linked to) muscle fibre type, the training status of an individual has been shown to affect both primary and slow component phases of $\dot{V}O_2$ kinetics. Endurance training has been demonstrated to accelerate primary $\dot{V}O_2$ kinetics (Hagberg et al. 1980; Taylor et al. 1999), and individuals with a higher $\dot{V}O_2$max have been shown to exhibit faster kinetics (Whipp and Wasserman 1972; Powers et al 1985). A period of endurance training has been demonstrated to shorten the phase-2 τ in both sedentary individuals (Berry and Moritani 1985) and previously trained subjects (Norris and Peterson 1997). This training effect has been shown to speed $\dot{V}O_2$ kinetics both at the same absolute (Cerretelli et al. 1979) and at the same relative exercise intensity (Hickson et al.

1978). Further evidence of this training effect comes from the fact that $\dot{V}O_2$ kinetics are slowed following a period of bed rest (Convertino et al. 1984).

The physiological processes responsible for this training effect are difficult to identify, since the limiting factors underlying the primary $\dot{V}O_2$ kinetics have yet to be established. The influence of endurance training on $\dot{V}O_2$ kinetics can be interpreted as evidence for both a central and a peripheral limitation (Walsh 1992), since there is evidence of endurance training improving both O_2 delivery and O_2 consumption.

The heart, is known to be adaptable to increases and decreases in physical activity (Asrand and Rohdahl, 1986), and indeed Peronnet et al. (1981) reported an increased left ventricular and end-diastolic volume following a period of endurance training. In addition to improvements in cardiac output, O_2 delivery may be increased by an enhanced O_2 carrying capacity of the blood due to increased total haemoglobin (Ekblom et al. 1968). Endurance training may also result in improved O_2-haemoglobin dissociation from higher levels of 2,3-diphosphoglycerate (Klein et al. 1980) and an improved blood flow to the muscle (Terjung et al. 1990).

There are also many peripheral adaptations that might account for a speeding of $\dot{V}O_2$ kinetics following endurance training. It has long been established that in addition to the improvements in $\dot{V}O_2$max that may be seen following a period of endurance training a large increase will be seen in the levels of oxidative enzymes in skeletal muscle (Gollnick et al. 1973; Taylor and Bachman 1999). This increase is often in fact so much larger than the increase in $\dot{V}O_2$max itself that it is unlikely to be the major reason for such an increase (Gollnick and Saltin 1981). In addition, muscle in the trained state is likely have an increase in mitochondrial size and number (Saltin et al. 1976), increasing peripheral oxidative capacity and therefore reducing diffusion distance between blood and mitochondria. It has been suggested that as mitochondrial respiration competes with glycolysis for a common substrate (e.g. ADP), this increase in mitochondrial respiratory power would result in faster $\dot{V}O_2$ kinetics, reducing the flux through the glycolytic pathway (Walsh 1992). The strong relationship that has been demonstrated between τ and AT during endurance training supports such a hypothesis (Yoshida and Udo, 1991).

However, a more recent study showed that $\dot{V}O_2$ kinetics were speeded after only a few days of endurance training (Phillips et al. 1995), at a time when no changes in mitochondrial enzyme activity could be detected.

Whilst the physiological mechanisms responsible are perhaps not fully understood, endurance training has been demonstrated to affect phase-2 $\dot{V}O_2$ kinetics (Hickson 1978; Cerretelli et al. 1979; Hagberg 1980; Berry and Moritani 1985; Powers 1985). A significant reduction in end exercise $\dot{V}O_2$ and the magnitude of the $\dot{V}O_2$ slow component has also been shown after 6 weeks (Womack et al. 1995; Carter et al 2000b), 7 weeks (Poole et al. 1990) and 8 weeks (Casaburi et al 1987; Belman and Gaesser 1991) of endurance training. If the subject is exercised at the same absolute work rate during pre and post training tests (Casaburi et al. 1987; Womack et al. 1995), increases in both $\dot{V}O_2$max and AT will greatly impact on the relative work rate (Yoshida et al.1982; Poole et al 1991). When subjects are exercised at the same relative intensity the results are equivocal. Poole et al. (1990) found the reduction in slow component was considerably less clear, whilst Carter et al. (2000b) showed a significant reduction through endurance training. Studies that have examined highly trained endurance athletes do suggest that a different response is seen in the highly trained however, and have reported either a small or no slow component (Billat 1998a; Lucia et al. 2000; Lucia et al. 2002b).

The exact mechanism behind this training effect is difficult to pinpoint since the underling physiology of the slow component is itself elusive. Womack et al. (1995) found that, similar to changes in phase-2 kinetics, the effect on the slow component occurs early in the training programme. These training studies again highlighted the close link between blood lactate and the slow component (Casaburi et al. 1987), but no causative link was found with either lactate or adrenaline (Gaesser 1994; Womack et al.1995). Casaburi et al. (1987) suggested that a small percentage of the reduction in excess $\dot{V}O_2$, may be due to a training induced reduction in pulmonary ventilation. However, the most accepted explanation is that the slow component is caused by a change in fibre recruitment pattern (Whipp 1994b). Gaesser and Poole (1996) postulate that this training effect reflects an increase in the recruitment of more efficient type I fibres and a reduction in type-II fibre recruitment.

Identification of the exact physiological mechanisms responsible for endurance training induced alterations in $\dot{V}O_2$ kinetics has yet to be achieved. It has been clearly established though that the training status of an individual will have a profound effect on both phase-2 and slow component $\dot{V}O_2$ kinetics. It should also be recognised that relatively few investigations have used specifically trained populations, and much of the current understanding of $\dot{V}O_2$ kinetics has arisen from the study of sedentary or in some cases patient populations. Caution should be exercised in applying current understanding to trained athletes, since the highly trained have been shown to differ in their $\dot{V}O_2$ response and may not conform to existing models (Billat et al. 1998a).

2.5.4 Mode of exercise

The majority of the research into $\dot{V}O_2$ kinetics has used cycling as the mode of exercise (Astrand and Saltin 1961; Margaria et al 1965; Katch 1973; Whipp and Wasserman 1972; Hagberg et al 1980; Hansen et al.1988; Patterson and Whipp 1991; Barstow et al. 1996; Gerbino et al. 1996; McDonald et al.1997; Zoladz et al.1998). This has created difficulties for anyone studying $\dot{V}O_2$ kinetics during running or other forms of exercise since current ideas and models of $\dot{V}O_2$ kinetics have been developed using cycling. Such difficulties are not new however. Indeed in 1925 A.V. Hill (p. 484) wrote in the Lancet of research using the cycle ergometer: "nearly all the laboratory observations on man, in connection with muscular exercise have been made with that implement."

Cycling exercise has many advantages over running for the investigator. When compared with treadmill exercise, cycling offers a greater range of intensities within the moderate intensity domain. When running there is a relatively narrow range of speeds that fall below AT that would not result in the subject walking (particularly in sedentary populations). The economy of walking differs from that of running (Menier and Pugh 1968) making it difficult to produce a sub AT $\dot{V}O_2$-speed regression. Mechanical changes are also likely to occur as treadmill speed increases (Cavanagh and Kram, 1985), whereas the mechanics of cycling are relatively unchanged across intensities. Cycling may also offer a safer mode of exercise for exhaustive testing, as the subject's mass is supported.

Finally recent studies suggest that the slow component is larger for cycling than for running (Jones and McConnell 1999; Billat et al. 1999b), so the former perhaps lends itself better to investigations of the underlying physiology of the slow component.

Research into the $\dot{V}O_2$ response to running in the heavy and severe intensity domains has demonstrated differences in comparison with cycling. Controversy over the existence of a slow component in running has arisen. Billat et al. (1998a), in a study using highly trained endurance runners, exercised subjects at supra critical speed intensities and showed that these athletes maintained a steady state $\dot{V}O_2$ and did not demonstrate a slow component. Whether this absence of the slow component was due to the training status of the subject group or the mode of exercise is uncertain. Other studies have shown that a slow component does exist in running (Steed et al. 1994), but it seems that the magnitude of this response is less than is seen in cycling (Jones and McConnell 1999; Billat et al. 1999b; Billat et al.2000; Carter et al. 2000a). However, a recent study investigating world class cyclists reported a slow component of a very small magnitude (Lucia et al. 2000).

Comparisons have been made between the $\dot{V}O_2$ responses of cycling and running in the same subjects (Billat et al 1998b, Billat et al 1999a, Carter et al. 2000a) and all have confirmed that the slow component is smaller in running. Billat et al. (1998b, 1999a) used a subject group that was highly trained in both modes of exercise (triathletes) to ensure that any effect was not the result of training status in one of the exercise modes. Both Billat et al. and Carter et al. explain the discrepancy in terms of difference in the type and pattern of muscular contractions between the two modes of exercise, arguing for a higher intra-muscular tension in cycling and a greater eccentric component in running. If the hypothesis is accepted that the $\dot{V}O_2$ slow component is largely due to the recruitment of less efficient muscle fibres, such differences in muscular contraction and recruitment patterns may in part explain the different $\dot{V}O_2$ response.

It is clear then that applying current understanding of $\dot{V}O_2$ kinetics to treadmill exercise may be problematic. Differences exist between cycling and running in the $\dot{V}O_2$ response to heavy and severe intensity exercise. The data of Spencer et al. (1996), for trained runners, suggests a very different response to short duration exhaustive exercise than otherwise reported (Hill and Stevens 2001).

In the laboratory setting it is necessary to use motorised treadmills to investigate the aerobic parameters of running exercise. Improvements in portable gas analysis systems are beginning to make $\dot{V}O_2$ measurement on the track a possibility (King et al. 1999; McLaughlin et al. 2001), and recent research has used such technology (Billat et al. 2000). Future research is likely to make further use of this technology; however the need to control exercise intensity and the greater accuracy of laboratory based equipment means most running research has used and will use the motorised treadmill. It should be recognised that mechanical differences have been demonstrated between treadmill and track running (Elliot and Blanksby 1976). Researchers have also found it difficult to replicate track performance in the laboratory environment (Falk et al. 1996; Spencer et al.1996; Hill 1999). Moreover, it should be realised that constant intensity (or square wave) exercise testing is itself limited since this does not mirror genuine athletic performance. The initial higher intensity spurt, or fast start, often seen in athletic performance might have a performance benefit in speeding $\dot{V}O_2$ kinetics and reducing O_2 deficit (sparing anaerobic capacity) (Secher et al. 1982).

2.6 Summary

There are several key points from this review of literature that are of particular relevance to an investigation of $\dot{V}O_2$ kinetics in exhaustive running of a short duration. Firstly there are running events that take place at intensities where the $\dot{V}O_2$ required exceeds $\dot{V}O_2max$ yet the aerobic contribution to the exercise is considerable. The 800 m is perhaps the best example of such an event. The speed of adaptation of the aerobic system ($\dot{V}O_2$ kinetics) is an important determinant of performance since it will dictate the relative aerobic and anaerobic energy contributions during the event. Little investigation of $\dot{V}O_2$ kinetics has been carried out in running exercise and even less into the response to exhaustive running of a short duration.

Current thinking regarding the behaviour of $\dot{V}O_2$ during exercise in the severe intensity domain is that $\dot{V}O_2$ will tend, at the onset of exercise (in an exponential manner), towards the $\dot{V}O_2$ required. If the $\dot{V}O_2$ required is greater than MLSS, an additional slow component will take the response to $\dot{V}O_2max$. Where the $\dot{V}O_2$ required exceeds $\dot{V}O_2max$ the phase-2 response will end (prematurely) at $\dot{V}O_2max$. The assumption exists then that all running speeds above MLSS will result in the achievement of $\dot{V}O_2max$. There is research using aerobically fit subjects that raises questions regarding this assumption, however. Whether this different response is related to the mode of exercise or the subject's aerobic capability remains to be established.

Mathematical models that seek to predict exercise performance all make the assumption that short duration exhaustive exercise will always result in the achievement of $\dot{V}O_2max$. Current models assume that $\dot{V}O_2$ will rise as a single exponential response towards $\dot{V}O_2max$. In trained populations this may not be the case (Spencer and Gastin 2001). Any shortfall in this $\dot{V}O_2$ response will result in an over-estimation of the sustainable speed for a given event. Running events of a short duration have proved particularly problematic for these models, and the sustainable speed has been overestimated for the 800 m (Di Prampero et al. 1993).

If there is a shortfall in the $\dot{V}O_2$ response to short duration exhaustive exercise, the reason for such a shortfall is unclear. This shortfall in $\dot{V}O_2$ has only been seen in the aerobically trained, but such a response has never been clearly identified or described by modelling. Any model used would require close scrutiny however, since the nature of breath-by-breath noise has never been investigated in severe intensity exercise.

Identification of the cause of such a shortfall in $\dot{V}O_2$ is difficult, since the mechanisms underlying the response even at the lower exercise intensities have not been firmly established. The shortfall may be linked to the aerobic capability of the subject as this response has only been identified in aerobically trained subjects. If the limitation is one of O_2 delivery, there may be a performance benefit in increasing metabolic acidosis prior to exercise (this strategy has been shown to speed $\dot{V}O_2$ kinetics during heavy intensity exercise).

CHAPTER 3

GENERAL METHODS

3.1 Laboratory and procedures

All laboratory-based experiments were conducted in a BASES (British Association of Sport and Exercise Sciences) accredited laboratory at University College Chichester. The laboratory was air-conditioned (Toshiba, Japan) and temperature was controlled at 19 ± 1 °C. A window was left partially open during testing to ensure that the laboratory had sufficient ventilation to maintain constant inspired gas fractions. Barometric pressure was measured using a wall mounted mercury barometer (Griffin and George Ltd., London, UK) and ambient temperature was measured using an electronic temperature probe (Hannah Instruments, US).

3.2 Subjects

The subjects that participated in the present thesis were all volunteers. Each was required to give written informed consent for each of the studies and also completed a medical history questionnaire. The subjects were male and were recruited largely from the Athletics Club at University College Chichester and from the local athletics club (Chichester Runners and Athletics Club). Studies 1, 2, and 4, that required physically fit rather than specifically trained individuals, also recruited subjects from other Sports Clubs at University College Chichester. Studies 3, 5 and 6 required a more specifically trained subject group however, and also recruited from an elite training group of distance and middle distance runners from the Horsham area.

3.3 The motorised treadmill

Treadmill tests were performed on a Quinton Q65 motorised treadmill (Quinton Instrument Co., Seattle, US). The treadmill runway consisted of a continuous nylon belt running around two metal rollers and over a metal platform. A DC motor drove the front rollers. The flat metal bed surface of the runway was regularly lubricated with a silicon-based lubricant to prevent the nylon belt sticking on foot-strike.

The manufacturer's speed controls and display were situated on a front panel that was visible to the subject. A new controller was manufactured and fitted to the handrail, and a second display was fitted perpendicular to the first and so visible only to the experimenter. The original display was obscured from the subject during all tests.

Since the studies included in this thesis demanded flat running, there was no need to calibrate gradients on the treadmill, merely to ensure that the treadmill was at a 0° at the start of each test. The front of the treadmill was supported on lockable casters, and the rear had adjustable feet, so that the platform could be set to be horizontal. The use of a spirit level ensured, that the runway was completely horizontal when the treadmill was displaying 0°. A zero gradient was used since this was considered to be most appropriate for track runners. An increased gradient is often used to prevent a cadence limitation prior to the achievement of $\dot{V}O_2max$ in incremental tests. However the increased gradient increases the $\dot{V}O_2peak$ achieved and is unnecessary in an incremental test as middle- and long-distance runners are able to reach a plateau in $\dot{V}O_2$ at a zero gradient (Draper et al. 1998).

Prior to each study the belt speed of the treadmill was checked against the displayed speed (on the second display unit used by the experimenter). Belt speed was determined by marking the belt and bed of the runway and timing (using a hand held digital stopwatch (ATP, Leics., UK)) a given number of complete revolutions of the belt, having measured the length of the belt. Speeds between 6 and 24 $km.h^{-1}$ were assessed, and the results were averaged over 10 trials at each speed. In all cases the displayed speed was within 0.04 $km.h^{-1}$ of the speed calculated from the timing of the belt. A data set from such a

calibration is shown in Table 3.1. The accuracy of the display was independent of whether a subject was running on the belt.

Table 3.1: Agreement between the belt speed (as calculated from timing the belt revolutions) and the display speed

Display Speed ($km.h^{-1}$)	Belt Revolutions (revs)	Time (s)	Belt Speed ($km.h^{-1}$)
6.0	27	59.90	6.02
8.0	36	60.10	8.00
10.0	45	60.04	10.01
12.0	54	60.05	12.01
14.0	63	60.02	14.02
16.0	72	60.06	16.01
18.0	81	60.00	18.03
20.0	90	59.98	20.04
22.0	99	60.05	22.02
24.0	108	60.08	24.01

3.4 Cycle ergometry

The focus of this thesis was running and so the majority of the data presented were collected on a motorised treadmill. Study 1 (Chapter 4) however dealt with high intensity exercise in both cycling and running, and so a cycle ergometer was necessary for data collection. Cycle tests were carried out using a friction braked cycle ergometer (Monark 814E; Monark, Varberg, Sweden).

The ergometer had been adapted and fitted with an adjustable saddle (adjustable both horizontally and vertically) and drop handlebars. The horizontal and vertical saddle adjustments had numbered holes in the tubing for each possible position and care was taken to ensure that for a given subject the same position was used for all tests. The manufacturer's display was replaced with an electronic cycle computer (BC 1100; Sigma Sport, Poole, UK), which monitored cadence via a sensor located on the pedal crank. The ergometer is frictionally braked by a cord that encircles the flywheel and connects to the

weighted cradle at the front of the ergometer. The tension in this cord and the height of the weighted cradle affects the level of friction applied to the flywheel. The ergometer was calibrated according to the manufacturer's instructions on each day of testing. The individual weights were calibrated against a standard weight prior to testing.

The flywheel of a cycle ergometer exhibits a certain level of inertia at the onset of exercise if the test begins from a static start. Whilst this would have a negligible effect on the results of a ramp, in square wave exercise this would be problematic since a greater power output would be necessary to overcome this inertia. The result would in effect not be a constant intensity test, as a higher power output would be required to overcome the inertia of the flywheel. To counter this problem, prior to all cycle tests the subject performing unloaded cycling (for a few seconds) with the experimenter supporting the weighted cradle. The test began as the experimenter dropped the weighted cradle and the subject maintained the same cadence at the increased load.

3.5 Peak heart rate

Peak heart rate (HRpeak) was measured using short-range telemetry (Vantage NV; Polar Electro Oy, Kempele, Finland). This consisted of a chest-mounted transmitter on an elastic strap and a wrist mounted monitor. The wrist unit was always positioned on the subject's back via the elastic strap so that the display was not visible to the subject. The monitor recorded heart rate (HR) every 5 seconds and the highest value was taken to be HRpeak.

3.6 Blood lactate

Anaerobic threshold was determined using respiratory data so blood lactate was not measured either during or before any exercise test. A fingertip capillary blood sample was however drawn 1 minute post all exercise tests and assayed for blood lactate concentration (YSI 2300 StatPlus Analyser; Yellow Springs Industries, Ohio, US). The YSI 2300, which

uses the enzymatic method of blood lactate determination, was checked daily (prior to testing) against known standards ranging from 2.5 to 12.5 mmol.L^{-1}. The fingertip site was first wiped with a swab saturated with 70% isopropyl alcohol BP and punctured using an automated lancet (H & MS, Northampton, UK). Approximately 40 µl of blood was then drawn into a plain micro capillary tube and dispensed into a 0.5 ml Eppendorf tube. The sample was immediately assayed for lactate.

3.7 Off-line gas analysis

Whilst the majority of the experiments in this thesis used on-line analysis of expired gases, Study 1 (Chapter 4) was completed before an on-line gas analysis system was installed in the laboratory. Furthermore Study 2 (Chapter 5) used off-line measurement as a criterion measure against which the on-line analysis system was validated.

Subjects wore a nose clip and breathed through a low resistance one-way valve box (Jakeman and Davies, 1979). The inspired side of this valve box opened directly to the atmosphere and the expired side was connected via a 1.5 m length of Falconia tubing (3cm internal diameter) (Baxter, Woodhouse and Taylor; Maclesfield, UK) to the Douglas bags. The valve box was constructed from clear perspex so that the experimenter could observe the movement of the diaphragm on the inspired side to determine when the subject was inspiring and expiring.

Expired air was collected in 200 L polyethylene Douglas bags (Cranlea and Co., Birmingham, UK). The bags were arranged in racks of four, with each bag fitted with a two way valve that could vent expired gas either into the Douglas bag or out into the laboratory. The four bags were arranged in a square and connected by a length of plastic tubing (2 cm internal diameter) (Figure 3.1). Expired air had to pass all other two-way valves on-route to bag 1; this meant that 4 consecutive collections could be made without the loss of any expired gas because the opening of the next bag effectively closed the previous one. When it was necessary to change from rack to rack expired gases were lost into the laboratory for approximately 10 seconds. It took approximately 5 seconds to

change the rack but a further 5 seconds were used (once the new rack was connected) to ensure that the plastic tubing (that had a volume of approximately 0.5 L) was flushed with expired gases rather than ambient air. The racks were constructed from lightweight box tubing and each had four casters attached to the feet to enable them to be easily manoeuvred around the laboratory.

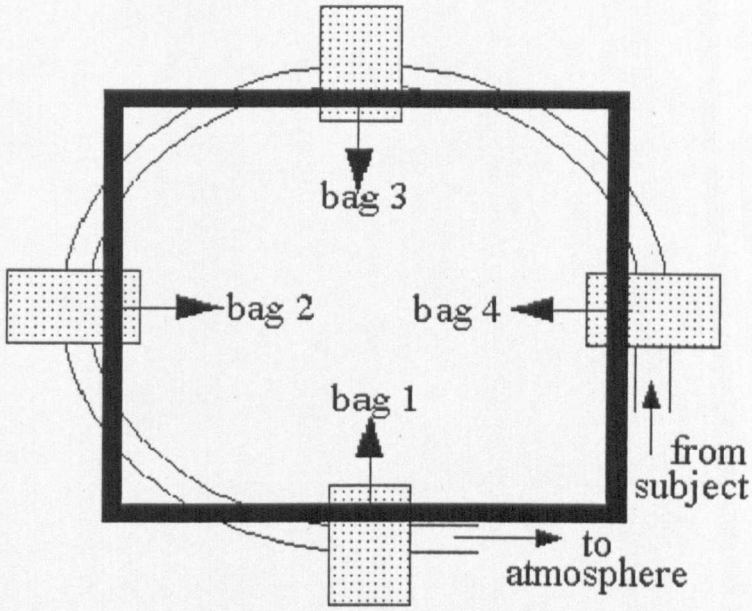

Figure 3.1: Arrangement of Douglas bags on rack (black square). The clear plastic tubing (not shaded) connects the bags together via four two-way valves (shaded).

All collections of expired gases were made from inspiration to inspiration to prevent the subject trying to breath through the system while a two-way valve was moving. This also insured that only whole breaths were collected. When subjects finished an exhaustive test, either by jumping from the treadmill or by stopping cycling, the experimenter always closed the final bag on the next inspiration, which would be slightly after the termination of the exercise. All collections were timed using a hand held digital stopwatch (ATP, Leics., UK), which was capable of storing the split-time for each individual bag.

The volume of expirate collected into the Douglas bag was determined using a Harvard digital dry gas meter (Harvard, Edenbridge, UK). The probe of a thermister was inserted into the inlet port of the dry gas meter and expirate temperature was measured individually for each Douglas bag. Two further corrections were made to this bag volume in the calculation of minute ventilation ($\dot{V}_E$), firstly to allow for the volume that was lost during sampling of expired gas fractions (F_EO_2 and F_ECO_2) and secondly to correct any measurement error of the dry gas meter.

When expired gases were sampled, the sample passed first through a flow meter (Platon Instrumentation, Hants, UK), which was adjusted to maintain a sample flow of 400 $ml.min^{-1}$. The gas-sampling period was 75 seconds, and therefore 0.5 L was added to each bag volume prior to the calculation of $\dot{V}_E$.

The dry gas meter was calibrated before each study. It is difficult to make fine adjustments to the dry gas meter and so a correction factor was used to calibrate the measured volumes. A 7 L precision syringe (Hans Rudolf Inc., Kansas, US) was used to put known volumes of air into the Douglas bags that were then measured using the dry gas meter. A mean value from two complete racks of Douglas bags (8 bags) was used for each volume. A linear regression was then used to derive the correction factor from these data. A typical data set and regression equation is shown in Figure 3.2. The resulting regression equation is in the form y = mx, where y is the actual or syringe volume, x is the measured volume and m is the correction factor (the regression was forced through the origin).

The volume of gas for each Douglas bag collection was calculated as:

Douglas bag vol. = (meter vol. x vol. calibration factor) + sample vol.....(3.1)

This volume (ATPS) was then adjusted based on the duration of the collection to give minute ventilation ($\dot{V}_{E(ATPS)}$). Volumes were then adjusted to STPD values to be used in the calculation of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ in the usual manner (McArdle et al. 1991 p.797).

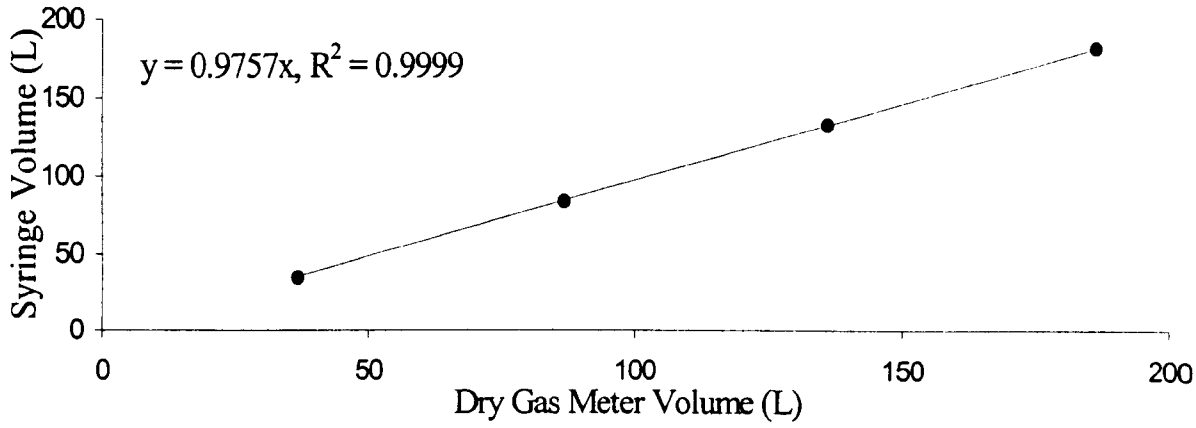

Figure 3.2: Typical data set and regression line used to calculate the gas volume correction factor.

The expired fractions of O_2 and CO_2 were determined using a paramagnetic oxygen analyser and an infrared carbon dioxide analyser (Servomex Series 1400; Servomex PLC., Crowborough, UK). Infrared analysers compare the oscillating signals of infrared radiation that is sent through the sample gas and a reference cell. Paramagnetic analysers make use of the paramagnetic properties of oxygen. The oxygen concentration is determined by the amount of electrical current needed to cancel the rotation of a nitrogen filled bell suspended in a magnetic field. Both infrared and paramagnetic analysers have been shown to be accurate in the measurement of expired gases (MacFarlane 2001). These analysers, since they make their measurements based on the partial pressure of a gas within a mixture, are sensitive to changes in barometric pressure and to the water vapour content of the gas mixture. It was important therefore that the analyser was calibrated regularly and that the water vapour content of both the sample and the calibration gases was standardised. The gas analysers were calibrated using a two-point calibration process for both O_2 and CO_2. The calibration process was performed immediately before the expirate from each test was analysed. Bottled nitrogen (Linde Gases, UK) was used to zero both analysers. An upper limit was then set for O_2 by passing outside air through the analyser and setting the span to 20.93%. Finally the span was set for CO_2 using a bottled gas mixture containing approximately 15.5 – 16.5 % O_2 and 5.0 – 6.5 % CO_2 in nitrogen (Linde Gases, UK). This process effectively provided a check on the calibration for O_2,

since any error in the calibration would be seen at this point with a concentration close to expirate.

As discussed above the sample was drawn through the gas analysers at a constant flow rate (400 ml.min^{-1}) and all measurements were timed for 75 seconds. All gases were passed through a 30 cm section of Nafion tubing (Omnifit Ltd, Cambridge, UK) and a Buhler PKE3 condensor (Patterson Insruments Ltd., Leighton Buzzard, UK) prior to the sample being presented to the gas analyser. The Nafion tubing was suspended in water, and since this tubing allows water to permeate its membrane but does not allow gas to permeate, all gases were saturated prior to reaching the condenser. The condenser then cooled the gases to 5° C to reduce the water vapour pressure (P_{H_2O}). Whilst this meant that gases were not presented as dry gases to the analyser, the system did ensure that the P_{H_2O} was both low (6.8 mmHg) and the same for all gases. Control of P_{H_2O} is an important consideration for analysers that measure the partial pressure of a gas within a mixture such as those used for off-line analysis in this thesis.

Prior to testing all Douglas bags were flushed with room air (minimum of 50 L), to ensure that the residual contents of each was room air. The expired fractions of O_2 and CO_2 could therefore be corrected for the effect of the dilution of the sample by the residual volume of room air, once the residual volume of each Douglas bags had been determined. The residual volume was calculated by first collecting at least 50 L of expirate in a Douglas bag. The O_2 and CO_2 concentrations of both this expirate and the room air were determined before the Douglas bag was evacuated. Seven litres of room air was then added to the bag from a precision syringe (Hans Rudolf Inc., Kansas, US). The O_2 and CO_2 concentrations of the contents of the Douglas bag were then determined for a second time. The residual volume could thus be calculated using either of the following equations:

Residual vol (L) = 6.9 * ($\%O_2$amb - $\%O_2$post) / ($\%O_2$post - $\%O_2$pre).....(3.2)

Residual vol (L) = 6.9 * ($\%CO_2$amb - $\%CO_2$post) / ($\%CO_2$post - $\%CO_2$pre).....(3.3)

where 6.9 is the approximate syringe volume in litres (ATPD) (amb is the ambient room concentration, pre is the expirate concentration before emptying the bag, and post is the concentration following the addition of the 7 litres of room air. Table 3.2 shows the data collected from eight Douglas bags and the calculated residual volumes. As a result of this analysis a value of 500 ml was used as the typical residual volume. Expired gas fractions (O_2 and CO_2) were therefore corrected using the equation:

Corrected % = measured % + (500 / bag volume (ml)) x (measured % - ambient %).....(3.4)

Values of 20.90 and 0.10 % were found to be typical concentrations of O_2 and CO_2 in the laboratory and were the assumed values for ambient air in this equation and in the calculation of $\dot{V}_{O_2}$.

Table 3.2: Residual volumes of eight Douglas bags as calculated from either O_2 or CO_2 concentrations

Douglas bag	Residual volume (O_2) (ml)	Residual volume (CO_2) (ml)
1	402	442
2	393	453
3	539	597
4	547	591
5	423	476
6	586	646
7	488	559
8	461	507
Mean ± SD	480 ± 72	534 ± 75

3.8 On-line gas analysis

The thesis used primarily breath by breath measurements of $\dot{V}_{O_2}$. The QP9000 (Morgan Medical, Rainham UK) was used for on-line gas analysis. The QP9000 is a mass spectrometer based system, combined with a turbine for the measurement of gas flow and volume. The paramagnetic and infrared devices previously described for off-line analysis calculate the concentration of a specific gas within a mixture by analysing the partial pressure exerted by that gas. The mass spectrometer determines the concentration of a gas within a gas mixture by categorising the molecules within the gas mixture according to mass.

The QP9000 employs a quadrupole type mass spectrometer. Gas molecules are converted to ions (molecules with either a positive or a negative electrical charge) so that they may be manipulated electronically without greatly altering the mass of the molecule. This is achieved in the QP9000 by a process of electron bombardment via heated filaments within the ioniser. The ion detector within the analyser head is able to determine the gas species emerging based on the electrical current from these ions. The term 'mass spectrometer' is therefore a misleading one since gas specie are not separated according to mass but rather according to the mass / charge ratio (Morgan Medical 1999).

In off-line analysis the effect of water vapour is controlled by measuring the temperature of the expirate and subtracting the P_{H_2O} that corresponds to that temperature from barometric pressure in the calculation of volume (making the assumption that the gas sample is 100% saturated). The QP9000 also assumes the expirate to be fully saturated and must also assume an expirate temperature since this is not measured. An expirate temperature of 32° C is therefore assumed in the calculation of $\dot{V}_E$. When the sample has a variable water vapour content as in on-line analysis it is also problematic to calculate gas concentrations by partial pressure. The mass spectrometer is reasonably insensitive to changes in pressure, due to the different operating principles. Water vapour is a problematic gas to measure, in terms of both the response time and the tendency for it to condense within the sample capillary and analyser. The QP9000, whilst recognising gas from the water vapour mass spectra, disregards water vapour from any analysis. Effectively then, the measured fractions are those of the dry volume.

Prior to each exercise test the mass spectrometer was calibrated using a bottled gas of known concentrations (Linde Gases, UK). Unlike the gas cylinder used in the calibration of the off-line equipment, this cylinder contained argon in addition to the other three gases (oxygen, carbon dioxide and nitrogen). A two point calibration as was used off-line is not possible using this system.

The calculation of gas volume was achieved on-line using a low resistance turbine device (0.65 $cmH_2O.L^{-1}sec^{-1}$ at 8.5 $L.sec^{-1}$). This compares very favourably with the off-line low resistance valve (3 $cmH_2O.L^{-1}sec^{-1}$ at 5 $L.sec^{-1}$) (Jakeman and Davies 1979). The turbine device had a spinning propeller at the centre of a clear perspex tube. Each revolution of this propeller was detected by a photocell in order to determine the gas volume. It is important to recognise that such a device, unlike a simple Douglas bag, is measuring gas flow not gas volume, whilst it may be reasonable to suppose that a single revolution is equal to a finite gas volume. Turbines have the advantage over other types of flow device in that they are fairly insensitive to changes in temperature, humidity and gas composition (MacFarlane 2001). Turbine devices have been shown to suffer from linearity problems however due to the friction and inertia properties of the propeller. This is seen at low flow rates when a 'lag-before-start' may occur and at high flow rates when a 'spin-after-stop' may occur (Yeh et al. 1987). Such errors will impact on the calculation of $\dot{V}O_2$. The turbine device (Interface Associates, Alifovieja US) used in conjunction with the QP9000 was designed to exhibit a minimum inertia to reduce this error. However the device cannot be calibrated accurately across a range of flow rates (to compensate for such effects) since it is not possible to accurately measure the flow rate applied.

The turbine itself was connected to the mouthpiece via a short plastic tube. The plastic tube also had connections to the gas sampling capillary and to a saliva trap. The capillary entered from the top of the tube and the length of the capillary was adjusted so that its aperture was central to the plastic tube. At the bottom of the plastic connecting tube was a slotted aperture where the saliva trap was connected. This plastic cylinder collected saliva that entered the mouthpiece and prevented it being sucked into the capillary. Therefore, whilst the turbine cartridge had a dead space of only 38 ml, the dead space of the complete set-up during testing was 114 ml. This is greater than the low resistance breathing valve used in the off-line experimentation that had a dead space of 80 ml. The length of the

mouthpiece and turbine once connected was 168 mm and the flow transducer and connecting cable were placed 122 mm from the mouthpiece aperture. It was therefore necessary for the equipment to be supported by headgear. The headgear was adjustable for each subject and had a supporting arm that connected to a metal ring that was positioned around the turbine cartridge. The arrangement of the capillary and turbine cartridge in relation to the mouthpiece is shown in Figure 3.3. A disadvantage of this set-up was that there was a distance of 70 mm between the flow measurement (turbine) and the gas concentration measurement (capillary). It is assumed in the calculation of $\dot{V}O_2$ and $\dot{V}CO_2$ that the same gas was analysed for both flow and concentration. It is not possible to sample the gas at the same point as flow with a turbine device. The distance between the two measuring points should however be minimised.

The turbine flow device was calibrated immediately prior to each test (this was particularly important as three turbines were used during the studies to allow for sterilisation and drying to take place after each test). Prior to this calibration the ambient temperature and relative humidity were entered so that they could be used in the calibration algorithm. A 3 L (Hans Rudolf Inc., Kansas, US) precision syringe was used to calibrate the equipment. Ten strokes of the syringe are used and the QP9000 calculates the mean volume. This process was repeated until the QP9000 returned a value that was within 0.01 of 3 L. This process does allow the syringe volume to be applied at varying flow rates. However since a mean value was taken from the ten syringe strokes this would not compensate for any non-linearity of the turbine response, and the accuracy of the turbine and transducer was potentially still a function of flow rate (Lamarra and Whipp 1995).

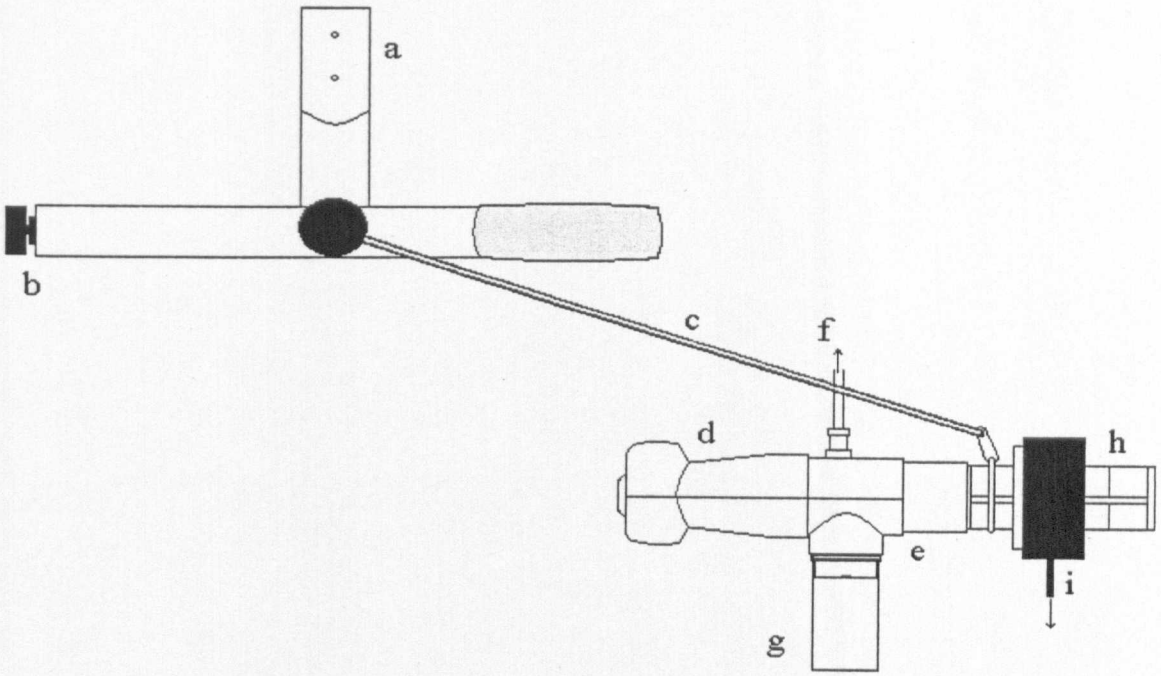

Figure 3.3: Schematic of the layout of the mouthpiece, turbine and supporting headgear, where a) is the headgear, b) the headgear adjustment, c) the supporting arms, d) the rubber mouthpiece, e) the plastic pipe, f) the gas sampling capillary, g) the saliva trap, h) the turbine cartridge and i) is the flow transducer.

The assumption that the expirate temperature is 32° C and remains constant throughout an exercise test is a potential source of error. During off-line analysis it is a relatively simple task to measure the temperature of the expirate; provided the equipment has been appropriately calibrated and that the timing of the gas collection was accurate, the measurement of $\dot{V}_E$ can be made with precision. During on-line analysis the assumption is made that the temperature of expirate remains constant throughout each breath and irrespective of respiratory frequency (f_R). Since both of these assumptions are to some extent flawed they will contribute in some manner to the measurement error. There may be a potential intensity effect on the measurement of $\dot{V}_E$ since f_R will increase with increasing exercise intensity.

Highlighting the potential error in the measurement of $\dot{V}_E$ is particularly pertinent, since similar to the off-line analysis the QP9000 uses the 'Haldane Transformation' to determine the inspired volume of O_2. The transformation, whilst generally attributed to Haldane, was actually outlined in 1888 by the German scientists Geppert and Zuntz (Poole and Whipp

1988). The principle assumption underlying the transformation is that nitrogen is metabolically inert and so inspired and expired volumes of nitrogen are equal. This assumption has been shown to be incorrect (Dudka et al. 1971), but the effect is negligible when values are averaged over a 60 second period (Wilmore and Costill 1974), as in the Douglas bag analysis. Owing to the difficulties with measuring the inspired volume (temperature and inequalities between the inspired and expired signals) the QP9000 calculates $\dot{V}O_2$ using the same principle. That this is an incorrect assumption within the context of a single breath (Wessel et al. 1979) adds both to the measurement error and the level of noise on the breath-by–breath data. Such analysis has however been shown to result in strong agreement with the Douglas bag method of analysis, when averaged over a reasonable number of breaths (Bassett et al. 2001). The QP9000 does have the ability to measure the inspired gas fraction. Whilst inspired volume is not measured, the bi-directional turbine device is important since the inspiration and expiration periods are determined by the change in direction of the turbine signal.

Most computerised breath-by-breath systems do not use inspired measurements and instead make their computations from expired measurements only (Bassett et al. 2001). Since equilibrium of inspired and expired nitrogen is not a safe assumption within a single breath (Wessel et al. 1979), some researchers have attempted to measure both inspired and expired gas and flow signals in an attempt to negate the need to use the Haldane transformation. Such an approach is however itself problematic and will result in a greater ratio of noise to signal on the measured response (Beaver et al. 1981). This noise is primarily the result of considerable fluctuations between breaths in the amount of O_2 or CO_2 that is stored in the lung. In order to reduce this high level of noise it is necessary to estimate the 'true' alveolar gas exchange and a variety of algorithms have been produced in order to make this estimation (Swanson 1980; Beaver et al. 1981; DiPrampero and Lafortuna 1989). There is however no generally accepted method of making this calculation (Allen et al. 1984).

The final calibration that was carried out prior to each test was the delay time. The electronic signal produced from the turbine sensor has a smaller transit delay than the gas signal at any given time point during exercise (Proctor and Beck 1996; Arieli and Van Liew 1981). Again it was essential that this calibration was performed routinely since

different capillaries were used during testing and each had a slightly different transit delay time. The delay was calculated by blowing expirate from the 3 L syringe into the mouthpiece. The delay was then calculated as the difference in time from the recognition of the turbine signal to the recognition of an increased CO_2 concentration. This process was repeated at least three times, to confirm that the result was consistent, before this figure was accepted as the transit delay. Typical delay times were 420 – 460 ms. Delay times longer than 460 ms indicated that there was a potential blockage within the capillary and it was then replaced and the system was re-calibrated prior to testing. In addition to this transit delay there will also be the minimal response time of the mass spectrometer itself (Noguchi et al. 1982). This is not corrected for by the QP9000. As the QP9000 has a response time of <30 ms the effect will be small however. Such an effect would be potentially much larger in systems that use a mixing chamber (Bassett et al.2001).

3.9 Protocols

The Ethics Committee of University College Chichester approved all protocols used in the studies contained within this thesis. Subjects were given details of the experimental procedures prior to giving their written consent to participate. The informed consent form is included as Appendix 2. Subjects were also required to complete a health history questionnaire (Appendix 3) prior to any exercise testing taking place. It was made clear to subjects that they could terminate exercise tests at any time should they experience any adverse reactions. They were shown how to lift themselves safely clear of the treadmill in order to terminate the test and were encouraged to practise this prior to the testing. Subjects were also familiarised with where the 'Stop' (stops treadmill belt quickly but gradually) and 'Emergency Stop' (stops belt immediately and belt cannot be restarted until the treadmill has been re-set) controls were located. A large crash mat was positioned to cover the wall to the rear of the treadmill during all tests.

All tests except for the cycle tests in Study 1 (Chapter 4) were treadmill based. Ramp tests to exhaustion were used to determine both $\dot{V}O_2$peak and the AT. The AT was determined from respiratory data (averaged for every complete 15 second period during the ramp test), primarily from $\dot{V}CO_2$ vs. $\dot{V}O_2$ (Beaver et al. 1986), but secondary plots of ventilatory

equivalents and end tidal gas concentrations were used to verify this calculation (Whipp et al. 1986). The $\dot{V}O_2$peak was taken to be the highest value for any individual collection during off-line testing and the highest value from a rolling 15 second average of the second-to-second data collected on-line.

For all tests subjects were, having finished a moderate intensity warm-up 5 minutes before, instructed to breath for 2 minutes through the gas analysis system (off- or on-line) prior to the test commencing. This was to ensure that the equipment was flushed with expirate and $\dot{V}O_2$ was at a genuine resting level prior to testing. (The latter was important to establish baseline values for modelling the $\dot{V}O_2$ response.) This period of 2 minutes rest was conducted with the subject either standing astride the treadmill belt (that was accelerated during this period to the required starting speed) or sitting on the cycle ergometer. Tests commenced with the subject jumping on the moving treadmill belt or with the experimenter dropping the weighted cradle once the subject reached the required cadence. The treadmill was fitted with two handrails, which enabled the subjects to lift themselves on to or clear of the treadmill. During constant intensity tests the treadmill was running at very high speeds so subjects remained in contact with these rails for a few seconds until they had reached the required leg speed. Each test was terminated when the subject lifted himself clear of the treadmill belt or became unable to maintain the required cadence on the cycle ergometer. During cycle tests subjects were warned if pedal cadence dropped more than 5 rpm below the required rate; if the subject was unable to respond, the experimenter terminated the test.

Treadmill speeds and cycle power outputs for square wave exercise tests were estimated from the ramp to exhaustion based on peak speed/power. Square wave tests were however designed to be of a certain duration rather than at a particular work-rate. Previous work from this laboratory (Draper, unpublished) had found a large variation in duration when subjects worked at a percentage of peak work rate or $\dot{V}O_2$peak. Some of the subjects had tested previously in this laboratory and so some of the necessary information was available. There were occasions when within a study, a subject's first square wave test duration, was unacceptably short or long. In this case the estimated speeds or powers were recalculated and the subject repeated the test on a separate day, before completing any further tests. In this way the counterbalanced design of the studies was not compromised.

3.10 Data analysis

Analysis of the off-line data was conducted using Excel for Windows (Version 8.0). The corrections outlined in this chapter (for the dry gas meter calibration factor, room air gas concentrations and residual volume of the Douglas bags) were added to the basic Haldane transformation and volume standardisation calculations. The on-line data were also exported to Excel for further analysis. The QP9000 returns a time point for each breath that is the end of expiration. However, since values for $\dot{V}O_2$ etc. represent mean values across the breath, these time points were first realigned to the middle of each breath. Using these corrected times the breath-by-breath $\dot{V}O_2$ data were then interpolated to second-to-second data. As well as reducing the level of noise present on the data this process (see Chapter 7) also enabled the data to be averaged across repeated transitions.

All data were checked for normality using the approach suggested by Vincent (1999). In all cases, data were found to be acceptably normal. That is, the figures for skewness and kurtosis were less than twice the respective standard errors.

Non-linear regression procedures were used to model the data. Data were modelled using the interpolated second-to-second data (in some cases averaged over multiple transitions). It was decided to model the response as a single exponential function that incorporated a delay (δ) rather than attempt to model phase-1. The first 15 seconds of data were removed from the modelling of severe intensity data and the first 25 seconds were removed from any modelling of moderate intensity data. This approach of only modelling the phase-2 data points has been widely used (Whipp et al. 1980; Patterson and Whipp 1991; Gerbino et al. 1996). That sufficient data had been removed was confirmed by applying the criteria of Mettauer and colleagues (2000) to determine the end of phase-1 (see Chapter 8 for details). This was important, as such an approach had never been applied to a trained subject group during exhaustive square wave exercise of a short duration.

The removal of the phase-1 data has a clear rationale and is not done merely for convenience. Firstly there are insufficient data points during the phase-1 response (Grassi et al. 1996), and secondly there exists no experimental evidence nor physiological rationale to suggest that this initial phase is exponential in nature (Whipp and Ozyener 1998). As

previously discussed most breath-by-breath systems (including the QP9000) use the Haldane transformation to determine $\dot{V}O_2$. However the assumptions underlying this transformation are flawed in the context of a single breath due to the changes in lung gas stores and their subsequent effect on alveolar gas exchange. This effect will be particularly great during this initial non-steady state period. It is therefore unwise for researchers using a Haldane based calculation of $\dot{V}O_2$ to model this phase. Finally the confidence limits of any parameter derived from the modelling process are dependent on both the level of noise on the data and the magnitude of the response (Lamarra et al. 1987). Therefore, even if the noise level is no higher than during the primary (phase-2) response, the confidence limits of the calculated phase-1 parameters will be wide. The magnitude of the phase-1 response is typically only 50 % of the phase-2 response even at moderate exercise intensities and is much lower than this (~30 %) at severe exercise intensities (Carter et al. 2002).

Non-linear regression, analysis of variance (ANOVA) tests, independent samples t-tests, correlations and plots of frequency distribution were all conducted using SPSS for Windows (Version 9.0). All other analyses used Excel. For each ANOVA, the degrees of freedom were corrected for any violation of the sphericity assumption. This correction was performed in line with the recommendation of Huynh and Feldt (1976). That is, the Huhnh-Feldt correction was used when an estimate of the true value for ε [the average of the Huynh-Feldt and the Greenhouse-Geisser ε values (Howell 1997)] was ≤ 0.75 and the Greenhouse-Geisser correction was used when this estimate was > 0.75.

CHAPTER 4

STUDY 1: THE INFLUENCE OF INTENSITY AND MODE OF EXERCISE ON THE $\dot{V}O_2$ RESPONSE TO EXHAUSTIVE SQUARE WAVE EXERCISE

4.1 Introduction

Research into the kinetics of $\dot{V}O_2$ has been considerable (Whipp et al. 1982a; Barstow and Mole 1991; Patterson and Whipp 1991; Poole et al. 1991; Barstow1994; Barstow et al. 1994). The focus of this research has however tended to be within the moderate and heavy intensity domains. The $\dot{V}O_2$ response to exhaustive exercise of a short duration, where the $\dot{V}O_2$ required is close to or above $\dot{V}O_2$max, is not well understood.

Within competitive sport there are events which demand the competitor to compete at such high intensities (middle distance running events and certain track cycling events). These events demand high levels of both speed and endurance and therefore demand a large energy contribution from both aerobic and anaerobic energy systems. The $\dot{V}O_2$ response is an important determinant of the aerobic contribution and thus potentially of performance but has received relatively little attention. Determining the $\dot{V}O_2$ response to exhaustive severe intensity exercise of an appropriate duration is therefore an important step in understanding the physiology underpinning such events.

All severe intensities are expected to result in a slow component that (unlike that seen in heavy exercise) will not attain a steady state but rather will continue to rise until $\dot{V}O_2$max is reached (Whipp and Ward 1990) or the exercise is terminated. The assumption is then that, provided the duration is sufficient, severe intensity exercise will always result in the achievement of $\dot{V}O_2$max. This assumption has received considerable support (Hill and Ferguson 1999; Hill and Stevens 2001) and is central to current conceptions of the physiological response to the various exercise intensities (Poole et al. 1988; Gaesser and Poole 1996). Furthermore current mathematical models of athletic performance use a simple mono-exponential function and an asymptote equal to $\dot{V}O_2$max to describe the $\dot{V}O_2$ response for all events of a short duration (<420 s) (Capelli 1999).

There is however research that suggests $\dot{V}O_2$ may plateau at a rate below $\dot{V}O_2$max during exhaustive exercise at intensities that elicit exhaustion in less than 5 minutes. Astrand and Saltin (1961) showed such a response in three out of five subjects in exhaustive cycling exercise of less than 3 minutes duration, although they did not comment on it. This same phenomenon was however clearly demonstrated in treadmill exercise (Spencer et al. 1996; Spencer and Gastin 2001). When trained middle distance runners ran at 800 and 1500 m race pace on the treadmill, $\dot{V}O_2$ reached a plateau below $\dot{V}O_2$max approximately halfway through each run.

There are two reasons why Spencer et al. (1996) and Spencer and Gastin (2001) may have shown a very different $\dot{V}O_2$ response to that shown by other researchers. Whereas other researchers have used cycling (Hughson et al. 2000; Hill and Stevens 2001) and moderately trained subjects (Hill and Ferguson 1999; Hughson et al. 2000; Hill and Stevens 2001), Spencer et al. (1996) and Spencer and Gastin (2001) used running exercise and a highly trained population.

Both mode of exercise (Billat et al. 1998b) and the level of aerobic training (Hagberg et al. 1980) have been shown to influence the $\dot{V}O_2$ response at lower exercise intensities. Since exhaustive severe intensity exercise has never been examined using aerobically trained subjects in cycle exercise, it is unclear whether the reported differences in the $\dot{V}O_2$ response were due to the mode of exercise or the trained nature of the subject population. Furthermore Spencer et al. (1996) and Spencer and Gastin (2001) were not directly investigating the $\dot{V}O_2$ response and did not use genuine square wave exercise transitions. It still remains therefore for a plateau in $\dot{V}O_2$ below $\dot{V}O_2$max during exhaustive square wave exercise, to be clearly identified and described in any mode of exercise.

The purpose of the present study was to investigate the effect of both exercise intensity and mode of exercise on the $\dot{V}O_2$ response to exhaustive square wave exercise of 2, 5 and 8 minutes duration. Two minutes was chosen as the shortest duration as this was similar to that which Spencer et al. (1996) had used for their 800 m trials. Eight minutes was chosen as a suitable duration to assess the presence of a $\dot{V}O_2$ slow component.

4.2 Method

4.2.1 Subjects

Nine male subjects (mean ± SD: age 25.4 ± 4.4 years, height 1.82 ± 0.07 m, mass 79.5 ± 8.6 kg) participated in the study. Subjects were trained, though not specifically for running or cycling.

4.2.2 Study Design

All tests were performed on separate days, and subjects were required to complete four running and four cycling tests. An initial ramp test to exhaustion was performed on the first visit to the laboratory, to determine $\dot{V}O_2$peak, peak speed, and an individual relationship between work rate and $\dot{V}O_2$. Exercise intensities that would give test durations of approximately 2, 5 and 8 minutes were firstly estimated from peak power or speed from the progressive ramp test.

Three bouts of exhaustive square wave exercise (at intensities selected to elicit exhaustion in approximately 8, 5, and 2 minutes) then followed. For each subject all four tests (including the ramp test) were completed in one mode of exercise before any were completed in the other. Four subjects completed the cycling transitions and five the running transitions first. The sequence for completion of the square wave tests was randomised.

4.2.3 Cycling Tests

For the ramp test, subjects cycled at either 80 or 90 rpm (whichever was the preferred cadence) and this preferred cadence was then used for the *square wave* tests that followed. The test load was increased by 0.1 kg every 20 seconds. This resulted in ramp rate of 22.0 or 24.7 $W.min^{-1}$, depending on cadence. The starting load was determined depending on the fitness of the subject to elicit exhaustion in approximately 10 minutes.

Prior to each test, subjects performed a 5 minute warm up at 50% of peak power (estimated in the case of the ramp test) from the ramp test. A 5 minute rest period then followed, during which subjects were encouraged to stretch.

4.2.4 Running Tests

For the ramp test speed was increased by 0.1 $km.h^{-1}$ every 5 seconds (a ramp rate of 1.2 $km.h^{-1}min^{-1}$). The starting speed was determined depending on the fitness of the subject to elicit exhaustion in approximately 10 minutes.

Prior to each test, subjects performed a 5 minute warm up at 50% of peak speed power (estimated in the case of the ramp test) from the ramp test. A 5 minute rest period then followed, during which subjects were encouraged to stretch.

4.2.5 Collection of expirate

Expired gases were collected, using the Douglas bag method, throughout each test. For the progressive tests, 60 second collections were taken. In the *square wave* tests, 30 second gas collections were taken throughout the first 4 minutes of the test and 60 second collections thereafter. For the final collection, in any test, the collection period needed to be at least 20 seconds to be included in the data analysis.

4.2.6 Data analysis

Similar to other research (Hughson et al. 2000) a slightly higher $\dot{V}O_2$ was recorded in the longer square wave tests than during the progressive ramp test in some subjects. Consequently $\dot{V}O_2max$ was taken to be the highest $\dot{V}O_2$ achieved in any of the four tests. The $\dot{V}O_2$ slow component was quantified as the difference between the $\dot{V}O_2$ at the sampling period between 2.5 and 3.0 minutes and the final $\dot{V}O_2$ for the 5 and 8 minute

tests. Whether a plateau had occurred in $\dot{V}O_2$ was determined by evaluating the difference between the last two collection periods ($\Delta \dot{V}O_2$).

The influence of exercise mode and intensity was investigated with a 2 x 3 (exercise mode x test duration) repeated measures ANOVA. Since no significant interactions were found (see section 4.3), main effects for test duration were investigated post hoc using Bonferonni corrected t-tests performed on combined (mean) data from both cycling and running tests. The P values given for this analysis are the corrected (Bonferonni) values. In instances where this corrected P value was greater than 1, this P value is not given. Differences between the cycling and running conditions from the ramp test, the magnitude of the slow component and the $\Delta \dot{V}O_2$ between the last two collection periods of the two minute tests were evaluated using paired t-tests. An alpha level of 0.05 was used for all tests. Group data are mean ± SD unless otherwise stated.

4.3 Results

The $\dot{V}O_2$max (defined as the highest $\dot{V}O_2$ achieved in any of the tests in each exercise mode) did not differ (P=0.15) between running and cycling (4.76 ± 0.51 and 4.64 ± 0.51 $L.min^{-1}$ or 60.0 ± 2.9 and 58.5 ± 3.3 $ml.kg^{-1}min^{-1}$). The intensity (calculated from the $\dot{V}O_2$-WR relationship from the ramp test), expressed as a percentage of $\dot{V}O_2$max, was 82.8 ± 6.0, 91.7 ± 7.2 and 106.7 ± 5.4 % respectively for the 2, 5 and 8 minute cycling tests. For the running, the intensity, expressed as a percentage of $\dot{V}O_2$max, was 90.3 ± 3.3, 97.7 ± 2.5 and 109.9 ± 4.3 % respectively for the 2, 5 and 8 tests.

Peak values from the square wave tests, together with test duration and the percentage of $\dot{V}O_2$max attained, are contained in Table 4.1 (cycling) and Table 4.2 (running). A degree of variability in test duration of the square wave tests was inevitable due to the exhaustive nature of the tests. However there was no interaction (mode x duration) found for test duration (P = 0.754) and no main effect for mode of exercise difference in test duration

(P=0.761). As expected a main effect was found for duration ($P < 0.001$) and all three test duration were different from each other ($P < 0.001$ in all cases).

Table 4.1: Peak values from, and test duration of, the square wave cycling tests

	Test Duration		
	8 minutes	5 minutes	2 minutes
$\dot{V}O_2$ (L.min^{-1})	4.53 ± 0.48	4.51 ± 0.58	4.19 ± 0.64
% $\dot{V}O_2$max	97.5 ± 2.0	96.9 ± 4.2	89.9 ± 5.5
HR (b.min^{-1})	183.1 ± 9.5	180.2 ± 8.3	174.7 ± 9.5
Lactate (mmol.L^{-1})	8.64 ± 1.62	7.96 ± 0.83	7.53 ± 1.03
RER	1.18 ± 0.07	1.25 ± 0.09	1.36 ± 0.11
Test Duration (min)	8.55 ± 1.88	4.65 ± 0.75	2.25 ± 0.43

Table 4.2: Peak values from, and test duration of, the square wave running tests

	Test Duration		
	8 minutes	5 minutes	2 minutes
$\dot{V}O_2$ (L.min^{-1})	4.73 ± 0.53	4.69 ± 0.50	4.37 ± 0.47
% $\dot{V}O_2$max	99.1 ± 2.2	98.4 ± 1.8	91.7 ± 2.5
Heart rate (b.min^{-1})	191.4 ± 13.7	186.3 ± 10.8	182.6 ± 9.5
Lactate (mmol.L^{-1})	7.27 ± 1.46	7.38 ± 1.12	7.12 ± 1.44
RER	1.13 ± 0.06	1.18 ± 0.05	1.30 ± 0.08
Test Duration (min)	8.43 ± 0.88	4.76 ± 0.72	2.01 ± 0.20

For $\dot{V}O_2$peak no interaction (mode x duration) was found (P = 0.978) and no main effect for mode (P = 0.222). A main effect was found for duration (P < 0.001), where $\dot{V}O_2$peak was found to be lower in the 2 minute test than either the 5 or 8 minute tests (P < 0.001 in both instances). No difference was found between the 5 and 8 minute tests.

For HRpeak no interaction (mode x duration) was found (P = 0.625). A main effect was found for mode of exercise (P = 0.006) with running producing the higher values. A main effect was also found for test duration (P = 0.002), with the 2 minute test producing higher values than both the 5 (P = 0.001) and 8 minute (P = 0.003) tests. There was no difference in HRpeak between the 5 and 8 minute tests (P = 0.208).

For post exercise lactate concentration no interaction (mode x duration) was found (P = 0.058). Furthermore no main effects were found for either mode of exercise (P = 0.135) or test duration (P = 0.254).

For peak RER no interaction (mode x duration) was found (P = 0.775). A main effect was found for mode of exercise (P = 0.025) with cycling producing the higher values. A main effect was also found for test duration (P < 0.001), with the 2 minute test producing higher values than both the 5 and 8 minute tests (P < 0.001 in both instances). Furthermore the 5 minute test produced higher values than the 8 minute tests (P = 0.008).

The $\dot{V}O_2$ responses of a typical subject, expressed as % $\dot{V}O_2$max, are contained in figures 4.1 (cycling) 4.2 (running). The $\dot{V}O_2$ response of this subject is consistent with the mean response shown in figures 4.3 (cycling) and 4.4 (running). It is clear that $\dot{V}O_2$peak is lowest in the 2 minute test for both modes of exercise. It is also apparent that for the 2 minute test a plateau in $\dot{V}O_2$ occurred in the running test, but not in the cycling test. To investigate this apparent difference in the response between the two modes of exercise, the $\Delta\dot{V}O_2$ was calculated between the last two collection periods in the 2 minute test. A clear difference was shown in the $\dot{V}O_2$ response. In the 2 minute cycling test $\dot{V}O_2$ increased across the final two collection periods (3.99 ± 0.63 vs. 4.17 ± 0.65 $L.min^{-1}$; P = 0.007). In the 2 minute running test no such increase was observed (4.33 ± 0.46 vs. 4.33 ± 0.45

L.min^{-1}; P = 0.983). This equated to a $\Delta \dot{V}O_2$ of 0.18 ± 0.15 L.min^{-1} in the cycling test and 0.00 ± 0.09 L.min^{-1} in the running test.

Investigation of the slow component in the 5 minute tests showed no difference between the exercise modes (P=0.178). The magnitude being 0.26 ± 0.15 L.min.$^{-1}$ for cycling and 0.17 ± 0.12 L.min.$^{-1}$ for running. For the 8 minute tests however, the slow component was higher in cycling than running (0.59 ± 0.16 vs. 0.42 ± 0.08 L.min.$^{-1}$; P=0.013).

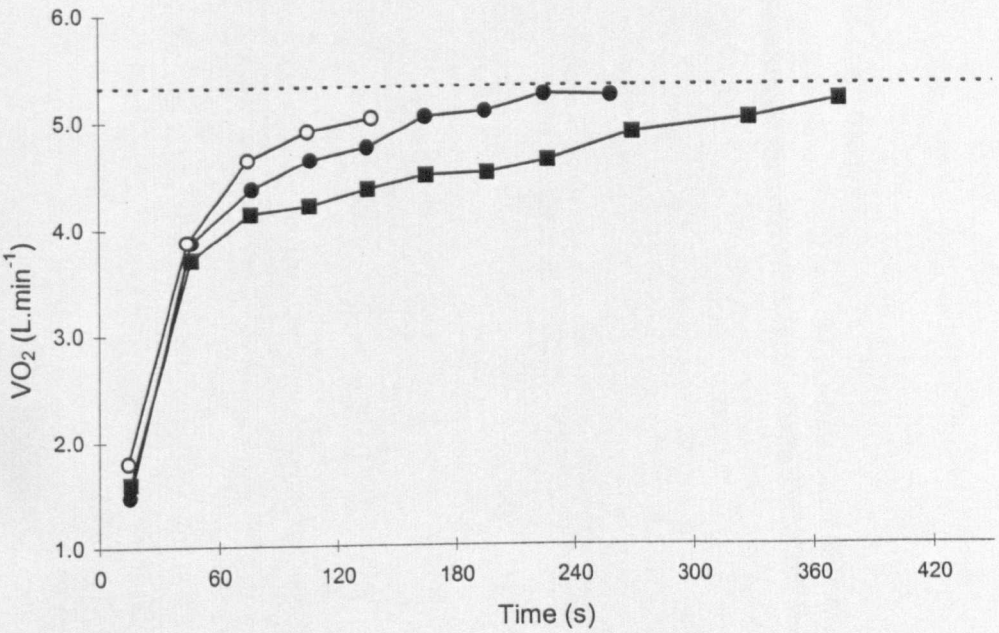

Figure 4.1: Oxygen uptake response for exhaustive square wave cycling lasting approximately 8 min (■), 5 min (●)and 2 min (○) for a representative subject. Broken line indicates $\dot{V}O_2$max.

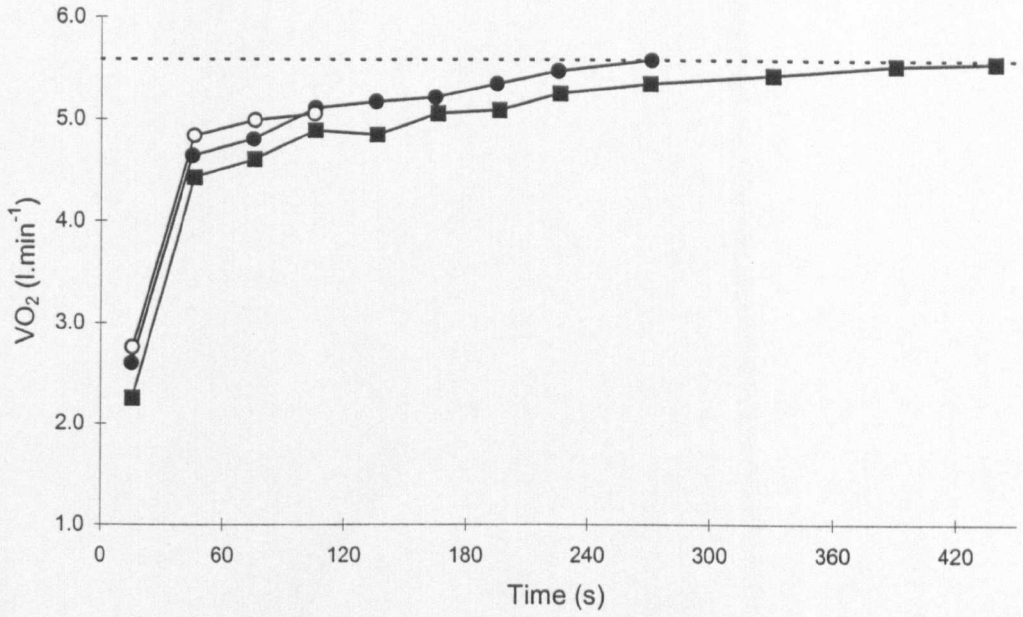

Figure 4.2: **Oxygen uptake response for exhaustive square wave running lasting approximately 8 min (■), 5 min (●)and 2 min (○) for the same representative subject. Broken line indicates $\dot{V}O_2max$.**

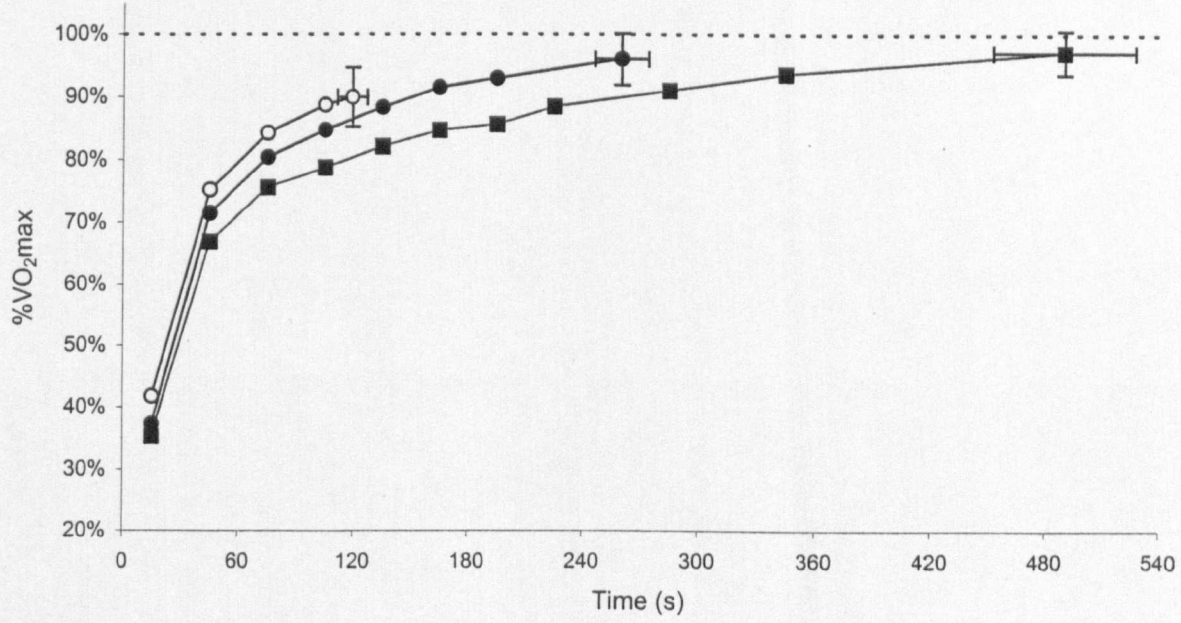

Figure 4.3: **Mean $\dot{V}O_2$ response (expressed as $\%\dot{V}O_2max$) for exhaustive square wave cycling lasting approximately 8 min (■), 5 min (●)and 2 min (○) for all subjects**

Note: Error represent the SEM. For clarity they are omitted from all but the final data points.

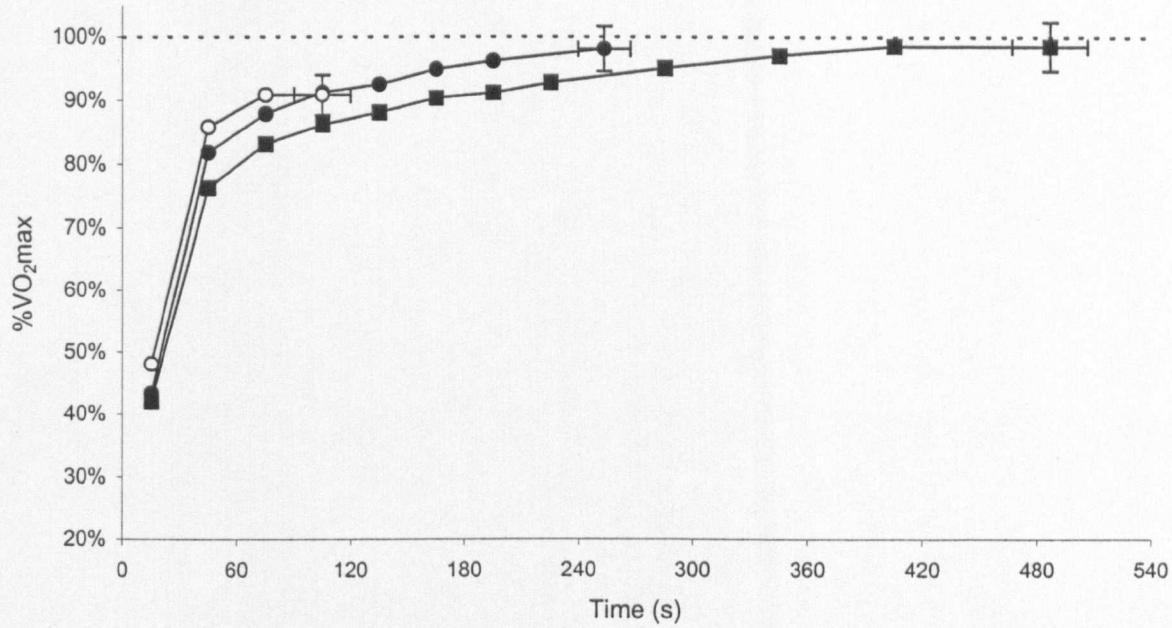

Figure 4.4: Mean $\dot{V}O_2$ response (expressed as $\%\dot{V}O_2max$) for exhaustive square wave running lasting approximately8 min (■), 5 min (●)and 2 min (○) for all subjects
Note: Error represent the SEM. For clarity they are omitted from all but the final data points.

4.4 Discussion

The present study demonstrated that the $\dot{V}O_2$ response is affected by exercise intensity for exhaustive square wave cycling and running in the severe intensity domain. The square wave exercise tests lasting approximately 2 minutes produced a significantly lower $\dot{V}O_2peak$ than either the 5 or the 8 minute tests. This was shown in both running and cycling using aerobically fit subjects. There was no difference in $\dot{V}O_2peak$ between the 5 and 8 minute tests, suggesting that $\dot{V}O_2$ is able to reach its maximum in exhaustive exercise of such duration.

Exercise intensities were used that would result in exhaustion in a specific duration, as opposed to workrates equating to a specific percentage of $\dot{V}O_2max$ (Hebestreit et al. 1998; Hughson et al. 2000), or a specific absolute workrate (Margaria et al 1965). The square

wave test of approximately 2 minutes duration was the only test for which the theoretical $\dot{V}O_2$ required was greater than $\dot{V}O_2max$. The variance in workrates when expressed as a percentage of $\dot{V}O_2max$ (see Section 4.3) highlights the problem of setting exhaustive workrates as a percentage of $\dot{V}O_2max$ in severe intensity exercise. This variability, perhaps due to differences in anaerobic capabilities, may explain contrasting results in the literature.

The results show that the assumption that $\dot{V}O_2$ will always attain $\dot{V}O_2max$ in severe intensity exhaustive exercise (Whipp 1994a) is incorrect. The response demonstrated in the 2 minute trials was similar to that reported by Spencer et al. (1996) and Spencer and Gastin (2001) in 800 m runners. Furthermore the $\dot{V}O_2$ response (Figures 4.1 to 4.4) appeared to be tending neither towards the $\dot{V}O_2$ required nor to $\dot{V}O_2max$, but rather to a plateau which was below $\dot{V}O_2max$. This plateau effect was especially pronounced in running exercise.

The shortest duration exhaustive square wave exercise then showed a response that, according to current understanding of $\dot{V}O_2$ kinetics (Gaesser and Poole 1996), was atypical. Examination of the data from the ramp test and the longer square wave tests however showed responses that would be considered typical. In the 5 and 8 minute tests a slow component was incurred and $\dot{V}O_2$ continued to rise until $\dot{V}O_2max$ was achieved. Furthermore, consistent with previous research, the slow component was shown to be greater in cycling than running (Jones and McConnell 1999; Billat et al. 1999b; Billat et al.2000; Carter et al. 2000a). That the subjects demonstrated such typical $\dot{V}O_2$ responses to the other exercise tests suggests that the shortfall in the $\dot{V}O_2$ response observed in the 2 minute test is a genuine phenomenon and not the result of an atypical subject group.

The $\dot{V}O_2$ response to severe intensity running shown by Spencer et al. (1996) differed from that reported in other research (Hill and Ferguson 1999; Hughson 2000; Hill and Stevens 2001). The present study sought to answer two important questions regarding the findings of Spencer et al. (1996). Firstly, could the apparent shortfall in the $\dot{V}O_2$ response be demonstrated in genuine square wave exercise? Secondly, if such a response was shown in square wave exercise, was it due to the mode of exercise or the training status of

the subjects? The response shown in the present study in the 2 minute tests supports the findings of Spencer et al. (1996) and suggests that the plateau seen in $\dot{V}O_2$, below $\dot{V}O_2max$, is a reproducible phenomenon in exhaustive running of this duration. The shortfall in the $\dot{V}O_2$ response was similar in cycling but a clear plateau was not shown.

It is well established that at all exercise intensities there are distinct phases of $\dot{V}O_2$ kinetics (Xu and Rhodes 1999). The first phase can be regarded as essentially a delay phase, representing an increased venous return. This is followed by a second phase representing the rate of oxygen consumption at the working muscle (Barstow et al 1990). The present study used a Douglas bag system and expired gases were collected continuously over 30 second periods during the initial stages of the tests. A lack of data points made it impossible to distinguish between phase-1 and the primary (phase-2) response and therefore to model the response in any meaningful way. However, the $\dot{V}O_2$ varied very little over the second minute of the 2 minute tests. If this apparent plateau in $\dot{V}O_2$ was the asymptote of an exponential response, the kinetics were very fast as represented by τ (time taken to reach 63% of the asymptote). A breath-by-breath analysis of the response would be necessary to investigate the nature of this response, however.

An important issue regarding the nature of the $\dot{V}O_2$ response to the 2 minute test is whether the primary $\dot{V}O_2$ response was tending towards the observed plateau throughout the exercise. Alternatively $\dot{V}O_2$ may have been tending towards a higher asymptote, possibly the $\dot{V}O_2$ required (Whipp 1994a; Hill and Stevens 2001), but this response may have been limited by some other mechanism. Whether the $\dot{V}O_2$ response to exercise of this intensity should be referenced to its apparent asymptote has been the focus of considerable debate (Margaria et al. 1965; Williams et al. 1988; Hughson et al. 2000; Hill and Stevens 2001). This debate has however really only questioned whether such a response would be tending to $\dot{V}O_2$ required or to $\dot{V}O_2max$. Where the response tends towards will greatly influence the τ, and therefore whether $\dot{V}O_2$ kinetics are seen to be faster or slower in short duration exhaustive exercise when compared to heavy and moderate exercise intensities.

Hughson et al. (2000) collected breath-by-breath data on subjects performing exhaustive cycle exercise at 96 and 125 % of $\dot{V}O_2$peak and attempted to model the response. In contrast to the present study $\dot{V}O_2$ achieved maximal or near maximal rates at both intensities (101.3 ± 7.4 and 96.1 ± 9.0 % $\dot{V}O_2$peak at 95 and 125 %% $\dot{V}O_2$peak respectively). However, these large standard deviations suggest a far from consistent response. The average duration for the most severe exercise was little more than 1 minute and a shortfall might therefore be expected in these shortest tests because of insufficient time for the response to fully develop. A phase-2 time constant of 12.5 seconds was reported however, suggesting that if the response was tending to $\dot{V}O_2$ required, there was sufficient time for the achievement of $\dot{V}O_2$peak. When modelling this response Hughson et al. (2000) used both a three-component exponential model and a semi-logarithmic model. The three component model showed an apparent speeding of the phase-2 $\dot{V}O_2$ kinetics with increasing exercise intensity. The semi-logarithmic method was constrained to have the $\dot{V}O_2$ required (i.e. 96 and 125% $\dot{V}O_2$peak) as its asymptote. This model showed phase-2 $\dot{V}O_2$ kinetics to be slowed in short duration exhaustive exercise relative to moderate intensity exercise. Hughson et al. (2000) concluded that both the asymptotic value of phase-2 (relative to $\dot{V}O_2$peak) and the apparent slowing of $\dot{V}O_2$ kinetics were due to inadequate O_2 delivery. They argued that $\dot{V}O_2$ kinetics tend initially to the $\dot{V}O_2$ required but are limited by O_2 delivery giving an apparent rather than actual asymptote, and that the response should therefore be constrained to the $\dot{V}O_2$ required. The data from the present study (figures 4.3 and 4.4) show no evidence of a third phase of $\dot{V}O_2$ kinetics (as shown by Hughson et al. 2000) during the 2 minute trials, although breath-by breath data would be needed to confirm that it was in fact a two-phase response.

It has previously been demonstrated that prior exercise in the heavy intensity domain (i.e. supra AT exercise) speeds the $\dot{V}O_2$ response (Gaushe et al. 1989; Gerbino et al. 1996; McDonald et al. 1997). Vasodilation and elevated muscle blood flow, coupled with an acidemia improved diffusion gradient, have been suggested as the probable mechanisms for the adaptation. It was ensured in the present study that all subjects performed a warm up of a moderate (not heavy) intensity exercise (50% peak speed) prior to testing.

Peak HR was lower in both 2 minute tests, compared with the 5 and 8 minute tests, in both exercise modes. Subjects were unable to reach a maximum HR, and potentially were unable to reach maximum cardiac output therefore, during exhaustive exercise of approximately 2 minutes duration. However, previous research suggests that HR and cardiac output kinetics are unlikely to limit $\dot{V}O_2$ at the onset of exercise (Cerretelli et al. 1966; Davies et al. 1972; Linnarson 1974; Eriksen et al. 1990; De Cort et al. 1991).

Whatever the underlying physiological mechanisms, the finding that $\dot{V}O_2$ may plateau below $\dot{V}O_2$max in aerobically fit individuals in running events of approximately 2 minutes, has implications for our understanding of performance in middle distance running, particularly the 800 m. The results of the present study challenge the validity of the existing models of running performance that assume a $\dot{V}O_2$ response that tends to $\dot{V}O_2$max (Di Prampero 1986; Peronnet and Thibault 1989; Di Prampero et al. 1993; Olds et al. 1993; Capelli 1999). That the nature of this response is poorly understood is perhaps an important factor in the lack of consensus among both sports scientists and coaches about the relative aerobic / anaerobic contributions to metabolism in middle distance running events (Gamboa et al. 1996; Hill 2000).

Considerable research has focused on the third slow phase of $\dot{V}O_2$ kinetics and the possible physiological mechanisms underlying it (Barstow et al. 1996; Patterson and Whipp 1991; Poole et al. 1988; Whipp and Wasserman 1972). More recently debate has arisen over whether the response is the same in cycling and running. Research has been equivocal, showing a similar response in both exercise modes (Bernard 1998), no significant third phase in running (Billat et al. 1998a), or a lesser but still significant third phase in running (Jones et al. 1999). Comparisons between these studies are difficult however due to the large variation in the aerobic capabilities of the subjects. The present study did demonstrate that a third phase was manifest in both exercise modes. No difference was found in the magnitude of the slow component in the 5 minute test, but in agreement with Jones et al. (1999) this third phase was shown to be of a significantly greater magnitude in cycling for the 8 minute test.

In summary, the present study showed that in exhaustive square wave exercise in the severe intensity domain, the $\dot{V}O_2$ response is affected by exercise intensity. It was

demonstrated for the first time that in genuine square wave exercise at intensities severe enough to result in fatigue in approximately 2 minues, $\dot{V}O_2$ reached a plateau below its maximum rate. This phenomenon was particularly clear in running although the response was similar for cycling. However, the cycling response was still rising during the last 30 seconds of exercise and could be interpreted as tending to $\dot{V}O_2max$ (albeit slowly). The present study showed that the plateau in $\dot{V}O_2$ was then, in part, dependent on the mode of exercise and the following studies therefore used running, where the plateau had been clearly demonstrated. That $\dot{V}O_2$ may plateau below $\dot{V}O_2max$ challenges current assumptions about $\dot{V}O_2$ kinetics and questions the validity of existing models of athletic performance. The Douglas bag analysis of expired gases did not allow modelling of the data however. A breath-by-breath analysis of the response was therefore warranted.

CHAPTER 5

STUDY 2: VALIDATION OF THE QP9000 FOR THE MEASUREMENT OF $\dot{V}O_2$, $\dot{V}CO_2$ AND $\dot{V}_E$ IN TREADMILL RUNNING

5.1 Introduction

The measurement of pulmonary gas exchange on a breath-by-breath basis has become routine and necessary for a variety of investigations (Lamarra and Whipp 1995). Breath-by-breath measurement saves time and offers sufficient data to evaluate non-steady state exercise (Veersteeg and Kippersluis 1989). Consequently there are many commercially available systems offering on line analysis (King et al. 1999).

On-line analysis has progressed since early semi-automated systems where expired gases went first to a mixing chamber and then to a gas meter, via three anaesthesia bags (Wilmore and Costill 1974). Fully automated systems were later developed that used a flow transducer and a mixing chamber (Wilmore et al. 1976). Such systems were further developed to align the ventilation signal to the measurement of mixed expired gases (Jones 1984). Mixing chamber based systems are ideal for the measurement of gas exchange variables as mean values over 15 or 30 second periods, but if assessment of non-steady state exercise is required they may be problematic as the response is likely to be slurred (Whipp and Lamarra 1995). Most modern systems assess pulmonary gas exchange on a breath-by-breath basis, where the sampling line from the gas analysers is connected directly to the mouth and a mixing chamber is not necessary (Holly 1993).

The QP9000 is a mass spectrometer based breath-by-breath system that provides almost continuous measurement (each gas channel is measured every 20 ms) of dry gas concentrations. The gas concentration signals are time aligned and coupled with the gas volume signal from a turbine flow meter.

Whilst the use of on-line analysis systems has increased, off-line systems, involving expirate collection using meteorological balloons or Douglas bags and analysis using electronic gas analysers, remain the gold standard against which other methods are compared (Davis 1995). Several researchers have used such off-line systems as criterion

measures against which to compare breath-by-breath equipment (Veersteeg and Kippersluis 1989; King et al. 1999; McLaughlin et al. 2001). Whilst the QP9000 has been used in several published clinical experiments (Corfield et al. 1999; Clar et al. 1999; Harty et al. 1999), a comprehensive validation of the QP9000, against a criterion measure, has not been reported.

Atkinson and Nevill (1997) emphasise the importance of assessing both validity and reliability when evaluating equipment. Validity (or accuracy) refers to how truthful a measure is, i.e. how well the measured value agrees with the true or criterion value. Reliability (or precision) refers to the consistency or repeatability of a measure (Morrow et al. 1995).

Methods used to compare two measures have been the subject of some controversy. Traditionally correlation coefficients have been used (Davis et al. 1976), and more recently the coefficient of variation (a measure of typical error) (Hopkins 2000) has also been suggested. Both these approaches have been criticised (Bland and Altman 1986, 1995; Atkinson and Neville 2000). Bland and Altman (1986) advised against the use of the Pearson Correlation Coefficient since it would not take account of systematic bias and is heavily dependent on sample heterogeneity. However, similar studies to the present one have used the correlation approach (Jones 1984; Rietjens et al. 2001). The coefficient of variation (calculated as SD / mean and expressed as a percentage) has also been criticised (Atkinson and Neville 2000), due to the flawed assumption that heteroscedastic error is always present (i.e. that the largest test-retest differences occur where the measured values are highest). The limits of agreement (LOA) approach, appears to be the preferred method in modern studies (King et al. 1999; Lamb et al. 1999; McLaughlin et al. 2001; Rietjens et al. 2001). Whatever approach is undertaken it is important that the comparison is made across an appropriate physiological range (Davis 1995) for the purpose that the instrument is designed for.

Limits of agreement is a technique that allows systematic and random errors to be considered separately. For example, in method comparison studies, the difference between the two methods is determined for each subject. The mean of these differences is representative of the systematic error (bias); the standard deviation (SD) of the differences

is representative of the random error (uncertainty). Typically the 95% 'Limits of agreement' would be obtained by adding and subtracting 1.96 × the SD to the mean difference (Atkinson and Nevill, 2000). The appeal of the method is that the calculations involved are straightforward and the limits can be presented visually using the so-called 'Bland and Altman plot' (Bland and Altman 1986).

The purpose of the present study was to evaluate the validity and reliability of the QP9000 in the assessment of $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$. The comparisons were made against a Douglas bag based system and across an appropriate range of exercise intensities.

5.2 Method

5.2.1 Subjects

Six males who were physically active, but not specifically running trained (mean ± SD: age 28.2 ± 3.7 years, height 1.83 ± 0.07 m, mass 81.1 ± 10.6 kg) participated in the study.

5.2.2 Study design

The study involved four separate protocols. Each was performed four times by each subject (twice each for on-line and off-line systems). This enabled comparisons within as well as between each system to be made. The turbine used in the on-line measurement is bi-directional and therefore, unlike the valve box used off-line, has only a single aperture for both inspirate and expirate. This meant that simultaneous measurement using the two systems was not possible.

The four protocols were rest (8 min), a ramp test (to exhaustion), a square wave test at a moderate intensity (8 min) and a square wave test at a severe intensity (to exhaustion). The resting conditions were performed 5 minutes prior to the ramp tests and the moderate intensity tests were performed 5 minutes prior to the severe intensity test, since the $\dot{V}O_2$ response for heavy intensity exercise has been shown to be unaffected by prior moderate intensity exercise (Gerbino et al. 1996). The resting - ramp combinations were performed

before the moderate – severe combination. This was done because the data from the ramp test were needed to calculate the moderate and severe intensity speeds. Each session of data collection was at least 24 hours apart. The order in which the systems were used was counterbalanced.

5.2.3 Test Protocols

All tests were conducted on the motorised treadmill and the resting data were collected with the subject standing on the treadmill belt. In the resting condition subjects stood on the treadmill belt and breathed through the appropriate apparatus for 2 minutes prior to data collection. The same 2 minute period of (standing) rest also preceded each of the other protocols. Expired gases were collected over two consecutive 4 minute periods, with data from the second of these 4 minute periods being retained for analysis. The resting condition was performed on the treadmill since later studies would require the calculation of a baseline $\dot{V}O_2$ representing the $\dot{V}O_2$ immediately prior to exercise.

The speed for the ramp test was increased by 0.1 $km.h^{-1}$ every 5 seconds (a ramp rate of 1.2 $km.h^{-1}min^{-1}$) throughout the ramp test. The starting speed was estimated depending on the fitness of the subject to elicit exhaustion in approximately 10 minutes. Whilst subjects were encouraged on each occasion to run for as long as possible, only complete 60 second periods were used in the data analysis. Data from the first two minutes of the test were excluded from the analysis.

The moderate intensity test was run at a speed equivalent to approximately 50% of the peak speed achieved during the ramp tests. Subjects exercised for 8 minutes, and the data from the final 60 second period were used in the analysis. The severe intensity test was run to exhaustion at an intensity equivalent to approximately 95% of the peak speed achieved during the ramp test. Data from the 4^{th} minute of exercise were used for the data analysis, since this was the last 60 second time period achieved by all subjects

5.2.4 Expirate collection and analysis

The off-line analysis was conducted using the Douglas bag (DB) method. Continuous 60 second gas collections were taken for all conditions except the resting condition, where two 4 minute collections were taken. Longer collections were used at rest to ensure a sufficient volume of expirate was collected, to reduce the risk of error in the measurement of F_EO_2 and F_EO_2 due to the residual volume of the Douglas bag. The absolute error in $\dot{V}_E$ is constant (Wood 1999), therefore the relative error in the determination of $\dot{V}O_2$ decreases as $\dot{V}_E$ increases (see section 3.7). The first two collections from the ramp test were excluded from the analysis for the same reason.

On-line (B x B) analysis was performed using the QP9000. Variables determined on a breath-by-breath basis were averaged to match the same 60 second or 4 minute collection periods that were used for the off-line gas analysis.

5.2.5 Data Analysis

The QP9000 was assessed for both validity and reliability for $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}_E$ across an appropriate range of exercise intensities. The focus was on these three variables as other important respiratory variables such as RER and ventilatory equivalents are simply ratios of these three primary variables.

To determine the statistical significance of any differences, relative to random error, between systems, two approaches were adopted. First a 2 x 3 (system × intensity) repeated measures analysis of variance (ANOVA) was used to evaluate data from the resting, moderate intensity and severe intensity tests. Second a paired t-test was used to compare the data from the final minute of the ramp test. Since each test was performed twice for each system, averaged data from the two tests were used in the analysis. Repeated paired t-tests (Bonferonni corrected) were used post hoc to highlight significant differences from the ANOVA.

To evaluate the reliability of the QP9000, in comparison to that of the DB system, data from the ramp tests were used. For each subject, and for each system, the difference

between test 1 and test 2 was determined for each minute of the test (excluding the first 2 minutes of the test). Similarly, the difference between test 1 for the DB system and test 1 for the B × B system was determined, for each subject and for each minute of the test. The same approach was adopted for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$.

The two-way ANOVA described above revealed significant interactions for both $\dot{V}CO_2$ and $\dot{V}_E$. This suggested that the difference between systems is a function of exercise intensity for these variables. Hence, for each subject the differences between test 1 for the DB system and test 1 for the B × B system were regressed on the mean of the two values for both $\dot{V}CO_2$ and $\dot{V}_E$. The slope of the regression equation was always less than 0.22 for $\dot{V}CO_2$ and 0.11 for $\dot{V}_E$, and was not significantly different from zero for $\dot{V}CO_2$ ($p = 0.092$) or $\dot{V}_E$ ($p = 0.326$) (1-sample t-test). However, since the slopes of these regressions were not all in the same direction the SD of the residual differences were used as an index of intra-individual variation, for each comparison and for each variable. For each variable, intra-individual variation was evaluated by performing a one-way repeated measures ANOVA on the intra-individual SDs across the three comparisons. The alpha level was set at 0.05% for all tests. Data are mean ± SD.

5.3 Results

Data collected at rest, from both square wave tests, and from the final minute of the ramp test are contained in Table 5.1. As expected, the two-way ANOVA revealed a main effect for exercise intensity in all three variables ($P<0.001$).

Table 5.1: Data from resting (rest) and square wave (moderate and severe) tests, together with data from the final minute of the ramp test (max) for on-line (BxB) and off-line (DB) data collection systems as well as the difference between systems (diff) (BxB – DB)

		BxB	DB	Diff
$\dot{V}O_2$	Rest	443 ± 116	408 ± 65	35 ± 65
($ml.min^{-1}$)	Moderate	2591 ± 392	2527 ± 359	64 ± 105
	Severe	4180 ± 629	4204 ± 616	-24 ± 89
	Max	4159 ± 725	4155 ± 713	4 ± 69
$\dot{V}CO_2$	Rest	378 ± 110	344 ± 56	34 ± 58
($ml.min^{-1}$)	Moderate	2258 ± 328	2294 ± 337	-9 ± 92
	Severe	4642 ± 714	4794 ± 708	-152 ± 114
	Max	4568 ± 775	4747 ± 830	-178 ± 115
$\dot{V}_E$	Rest	12.3 ± 3.3	12.0 ± 2.5	0.2 ± 2.2
($L.min^{-1}$)	Moderate	62.8 ± 8.0	65.6 ± 7.7	-2.9 ± 1.9
	Severe	139.8 ± 23.3	147.5 ± 23.0	-7.7 ± 4.6
	Max	136.6 ± 23.6	142.6 ± 25.7	-6.0 ± 4.0

Analysis of variance for $\dot{V}O_2$ revealed no interaction (P=0.210), and no main effect for system (P = 0.358). For $\dot{V}CO_2$ the ANOVA did reveal an interaction (P = 0.011), but there was no main effect between systems (P = 0.119). For $\dot{V}_E$ there was an interaction (P = 0.003) and a main effect for system (P = 0.010), with off-line analysis recording the higher values. Post hoc analysis demonstrated a significant difference for $\dot{V}_E$ at both moderate and severe exercise intensities (P=0.037 and P=0.029 respectively), whilst $\dot{V}CO_2$ showed a difference which was close to statistical significance only at the highest exercise intensity (P=0.067).

The paired t-test on data from the final period of the ramp test confirmed this intensity effect. No significant difference was found between the two systems for $\dot{V}O_2$ (P=0.893). Differences were shown however for both $\dot{V}CO_2$ (P=0.012) and $\dot{V}_E$ (P=0.014). One-way ANOVA revealed no differences between conditions for the intra-individual SD (P=0.146, 0.645 and 0.716, for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ respectively). Individual SDs and the group mean ± SD of the individual SDs are contained in Table 5.2.

Table 5.2: Intra-individual SDs for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$, in the comparisons between on-line test 1 and off-line test 1 (BxB1 v DB1), on-line test 1 and on-line test 2 (BxB1 v BxB2), and off-line test 1 and off-line test 2 (DB1 v DB 2).

	Subject	BxB 1 v DB 1	BxB 1 v BxB 2	DB 1 v DB 2
$\dot{V}O_2$ ($ml.min^{-1}$)	1	183	73	180
	2	52	40	99
	3	143	133	49
	4	46	39	71
	5	102	47	82
	6	55	43	108
	MEAN	**97**	**62**	**98**
	SD	**56**	**37**	**45**
$\dot{V}CO_2$ ($ml.min^{-1}$)	1	127	53	79
	2	52	39	92
	3	108	130	73
	4	51	213	81
	5	67	64	44
	6	64	48	56
	MEAN	**78**	**91**	**71**
	SD	**32**	**68**	**18**
$\dot{V}_E$ ($L.min^{-1}$)	1	6.5	6.5	2.1
	2	2.1	1.2	3.4
	3	4.0	4.5	2.6
	4	2.8	5.2	3.1
	5	2.7	2.2	2.6
	6	4.3	1.6	4.3
	MEAN	**3.7**	**3.5**	**3.0**
	SD	**1.6**	**2.1**	**0.8**

Note: $\dot{V}CO_2$ and $\dot{V}_E$ data are SD of residuals from regression (see Section 5.2.5)

5.4 Discussion

The principal finding from the present study was the agreement between systems for the measurement of $\dot{V}O_2$. The QP9000 was assessed for both systematic bias and random error across a range of metabolic rates from rest to maximal exercise.

The present study focused only on the measurement of $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$. Other variables such as RER and ventilatory equivalents were not examined in this analysis, although they are important in the assessment of respiratory function. However, since they represent ratios of the three primary variables ($\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$), they should be both reliable and valid provided the primary variables can be shown to be so. Previous validation studies have also concentrated solely on $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ (King et al.1999; McLaughlin et al. 2001).

There was no evidence of systematic bias for $\dot{V}O_2$ at any exercise intensity. Results from the two-way ANOVA suggested that systematic bias was present for both $\dot{V}CO_2$ and $\dot{V}_E$, however. The largest differences were at the highest exercise intensities for both variables. Results also showed that $\dot{V}_E$ was measured systematically lower using the QP9000 data collection system across the range of exercise intensities. The analysis of the final period of the ramp test provided further evidence of this intensity effect, with no difference in $\dot{V}O_2$ but differences in both $\dot{V}CO_2$ and $\dot{V}_E$.

Examination of the mean and SD of the differences (Table 5.1), showed that despite the interaction demonstrated for $\dot{V}CO_2$ and $\dot{V}_E$, the level of bias and of random error for all three variables examined was relatively small, across all exercise intensities. The calculation of the SD of the differences also allowed for comparisons to be drawn between this study and previous research which has used a LOA approach. Limits of agreement are calculated as ± 1.96 x the SD of the differences (Bland and Altman 1986). Such comparison shows that the QP9000 demonstrates a better level of validity than has been reported for other published studies on breath-by-breath systems (King et al.1999; McLaughlin et al. 2001). Caution should be exercised in applying such comparisons however. Limits of Agreement themselves have confidence limits, which will be dependent on the size of the sample (Bland and Altman 1999). As the present study used

only six subjects these confidence limits would be expected to be wide. The conclusions from this part of the analysis were that, whilst there existed some issues with the QP9000's measurement of $\dot{V}CO_2$ and $\dot{V}_E$, the bias was small and the LOA were narrow (0.19, 0.15 and 7.25 $L.min^{-1}$ for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ respectively) for all variables.

An examination of resting values was included in the analysis, due to the need to accurately measure a baseline $\dot{V}O_2$ in future studies. The measurement of a pre exercise $\dot{V}O_2$ is important if the $\dot{V}O_2$ response to exercise is to be modelled. It was for this reason that the resting data were collected with the subject standing on the treadmill belt as if pre-test. Agreement was demonstrated for all variables between the systems at rest.

The data from the ramp tests were used to compare the intra-individual variability between the systems. For $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$, the variability was no greater for the QP9000 than for the criterion Douglas bag system. Analysis of the ramp data therefore showed the QP9000 to be a reliable instrument in the assessment of pulmonary gas exchange.

In summary, the QP9000 proved to be both valid and reliable for measuring $\dot{V}O_2$ across the full range of exercise intensities. The interactions observed for $\dot{V}CO_2$ and $\dot{V}_E$ raised concerns about the validity of the system to measure these parameters at high exercise intensities. The purpose of the present thesis was to examine the $\dot{V}O_2$ response to severe intensity running and the QP9000 was demonstrated to be a valid and reliable tool for this purpose.

CHAPTER 6

STUDY 3: BREATH-BY-BREATH ANALYSIS OF THE $\dot{V}O_2$ RESPONSE TO 800 M RUNNING IN TRAINED RUNNERS

6.1 Introduction

Chapter 4 showed a $\dot{V}O_2$ response to exhaustive square wave running over a duration of approximately 2 minutes that resulted in a $\dot{V}O_2$ plateau below $\dot{V}O_2max$. This response is contrary to current understanding of $\dot{V}O_2$ kinetics in severe intensity exercise, where it is expected that $\dot{V}O_2$ will tend to $\dot{V}O_2$ required and will always attain $\dot{V}O_2max$ (provided there is sufficient time for the response to reach $\dot{V}O_2max$). There is however literature that has demonstrated a similar response to severe intensity running in the highly trained (Spencer et al. 1996; Spencer and Gastin 2001). The data included in Study 1 (Chapter 4) were collected using the Douglas bag method, and whilst a clear trend was apparent, it was based on a limited number of data points.

In athletics the events that are of a short duration and in the severe intensity domain are the middle distance events. The 800 m is the event that best matches the 2 minute exercise duration used in Chapter 4. Although the world record for men in the 800 m is 101.73 seconds, 2 minutes represents a reasonable time for a club level runner.

Replicating track performance in the laboratory is problematic. First there are issues with how to account for air resistance. Second many athletes struggle to run at high speeds on a motorised treadmill (Hill 1999). There have yet to be published any breath-by-breath data of genuine square wave running in a trained population. Consequently the nature of this response and how it may differ from that of other exercise intensities is not known.

Study into $\dot{V}O_2$ kinetics using breath-by-breath data is now commonplace and the fitting of mathematical models to describe the response is widespread. Breath-by-breath data encompass not only the underlying response but also fluctuations in breathing that will result in noise. The level of this noise differs widely between individuals (Lamarra et al. 1987). However very few researchers have attempted to evaluate the confidence limits of

the parameter estimates from a given model or assess the impact of this noise on the reliability of such models. Lamarra et al. (1987) and Potter et al. (1999) have attempted to assess the impact of the level of noise on the parameter estimates, namely the time constant (τ) and the delay (δ). However both studies were limited to moderate intensity exercise and Potter et al. investigated the responses of children. Lamarra et al. showed that the noise encountered with breath-by-breath data is Gaussian in nature, and that the confidence limits of τ and δ are to a large extent dependent on the level of this random noise. Potter et al. (1999) showed that in the majority of subjects noise was not Gaussian. However Potter et al. used a different method to Lamarra et al. to evaluate the distribution of the noise and a child population (this is method is investigated in Study 4 (Chapter 7)).

Lamarra et al. (1987) went on to describe this effect of noise on the calculation of τ and δ with the equation:

$$K_1 = L (SD / GAIN) \quad (6.1)$$

where $\pm K_1$ are the 95% confidence limits of the parameter estimate, L is a constant, SD is the standard deviation of the noise and GAIN is the magnitude of the response (the difference between the asymptotic value of $\dot{V}O_2$ and the baseline or resting $\dot{V}O_2$). It is apparent from the data presented by Lamarra et al. that the value of the constant L was approximately 50 for the conditions they investigated. However L is a function of both the time constant (τ) of the response and the amount of data available for the model fitting. The short duration exhaustive exercise (2 minutes) shown in Study 1 combined a small amount data with a seemingly fast $\dot{V}O_2$ response. It remains to be established what a typical value would be for L in exhaustive square wave exercise of a short duration (2 minutes).

Provided the noise is Gaussian, the SD of the response can be reduced with the use of multiple transitions where data are first interpolated to second-to-second values and then averaged across transitions. Lamarra et al. give a second equation that may be used to determine how many of these repeated transitions must be completed to give a desired confidence limit for τ and δ, once L has been established:

$$n = [(L \times SD)/ (K_n \times GAIN)]^2 \quad \text{.....(6.2)}$$

where n is the number of transitions required to produce the desired confidence interval ($\pm K_n$).

The use of multiple transitions is normal in studies that attempt to model the $\dot{V}O_2$ response to exercise (Whipp et al. 1982; Barstow and Mole 1991; Patterson and Whipp, 1991; Jones and McConnell 1999). However, such studies do not appear to base the number of transitions completed on the desire to achieve any given level of confidence in the parameter estimates. Since the number of transitions required to achieve a given confidence interval will be dependent on the magnitude of the ratio of the SD to the GAIN, fewer transitions should be required at high exercise intensities. The gain would be large at these high intensities, and noise is thought to be independent of exercise intensity (Lamarra et al. 1987), although only moderate intensities have been investigated. That fewer repetitions are required at the highest intensity is often assumed. Hughson et al. (2000) used data averaged over four transitions for moderate intensity exercise, whereas only two transitions were averaged in the severe intensity domain. Whilst this assumption is perhaps a logical step, it remains to be demonstrated that noise is independent of exercise intensity in the severe intensity domain.

It is generally considered that exhaustive exercise performed in the severe intensity domain will result in the attainment of $\dot{V}O_2max$ (Hill and Smith 1999). In exercise performed at intensities severe enough that the $\dot{V}O_2$ required is in excess of $\dot{V}O_2max$ modelling the response is problematic. Whether the primary (phase-2) response should be referenced as tending to $\dot{V}O_2max$ or to the $\dot{V}O_2$ required has proved controversial when modelling such exercise (Hughson 1978). However a recent study modelled severe intensity data separately using either breath-by-breath data from the whole test or data from just the initial 45 seconds of exercise (Hill and Stevens 2001). The study showed no difference between the projected asymptote from the first 45 seconds of data and the theoretical $\dot{V}O_2$ required (Hill and Stevens 2001). This was interpreted as evidence that $\dot{V}O_2$ should be referenced as tending to $\dot{V}O_2$ required at such high intensities. The work of Hill and Stevens (2001) provides support for the view that $\dot{V}O_2$ initially tends to the $\dot{V}O_2$ required but the response is cut off when $\dot{V}O_2$ is reached.

The present study set out to do four things. First, to confirm that $\dot{V}O_2$ would plateau below $\dot{V}O_2max$ during severe intensity running at 800 m race speed in trained middle- and long-distance runners (i.e. that the results from the Douglas bag system were genuine and not an artefact of that system). Second, to describe the $\dot{V}O_2$ response using a simple mathematical model and assess the confidence limits of the parameter estimates of this model. Third, to apply the equations of Lamarra et al. (1987), in particular to calculate a value for L, for data collected in the severe intensity domain. Finally, to investigate whether $\dot{V}O_2$ was tending to the (sub maximal) plateau value or to a higher asymptote ($\dot{V}O_2$ required or $\dot{V}O_2max$) by applying the method used by Hill and Stevens (2001).

6.2 Method

6.2.1 Subjects

Eight male subjects (mean ± SD: age 22.4 ± 4.5 years, height 1.80 ± 0.06 m, mass 68.8 ± 5.8 kg) volunteered for the study. All were trained competitive middle- and long-distance runners, with a (mean ± SD) $\dot{V}O_2peak$ from an exhaustive progressive test of 68.8 ± 5.6 $ml.kg^{-1}min^{-1}$. They were all familiar with both treadmill running and laboratory testing and were asked to arrive for each laboratory session in a rested state and to suspend their normal training for at least 24 hours prior to testing.

6.2.2 Study design

The subjects completed three test protocols on three separate days. These were an individual 800 m track time trial, a (treadmill) ramp test to exhaustion, and a square wave treadmill run at 800 m pace. All subjects performed the three tests in this order. The ramp test was used to determine $\dot{V}O_2peak$, HRpeak and the speed at the AT.

Subjects followed their usual warm up and stretching routine prior to the track time trial. During the laboratory testing they performed a 5 minute run at a moderate intensity 7 minutes prior to each test as a warm-up. The speed for this run was estimated for the warm

up prior to the ramp test but was calculated as a speed equivalent to 90% of the AT, determined from respiratory data from the ramp test (Beaver et al. 1986), for the warm up prior to the exhaustive run at 800 m pace. During the 7 minute period between the warm–up and test, subjects were encouraged to stretch for 5 minutes. A 2 minute period of breathing through the gas collection apparatus followed. Data from this period were used to determine a baseline $\dot{V}O_2$.

6.2.3 Test Protocols

The track time trials took place on a standard 400 m outdoor athletics track. All time trials were performed within an hour of each other and subjects all ran individually. The testing was performed in winter and air temperature was 9.4°C (determined from a hand held electronic thermometer (Hannah Instruments, US)). Wind speed was light (2.4 $km.h^{-1}$) as measured by a hand anemometer (Munro Ltd., London UK)). Times were recorded using a hand held digital stopwatch (ATP, Leicester UK).

The speed for the ramp test was increased by 0.1 $km.h^{-1}$ every 5 seconds (a ramp rate of 1.2 $km.h^{-1}min^{-1}$) throughout. The starting speed was estimated depending on the fitness of the subject to elicit exhaustion in approximately 12 minutes. A longer duration than was used in study 1 was desirable in the present study to secure sufficient data for the determination of the AT.

The speed for the square wave test was equivalent to the subject's average speed from the track time trial plus 1 km^{-1} (to make some allowance for air resistance encountered outside on the track). Pilot square wave tests (n = 8) were carried out prior to the study at average track speed (n = 4) and at average track speed plus a speed individually calculated to compensate for the extra $\dot{V}O_2$ cost of running at the average track speed (n = 4). This extra speed was calculated from the individual subject's $\dot{V}O_2$-running speed relationship and published equations to determine body surface area (Dubois and Dubois 1916) and air resistance (Pugh 1970). No subjects were able to maintain the air resistance corrected speed for a duration comparable to the track times (mean ± SD: 67 ± 12 s for the treadmill run v 119 ± 0 s for the track run), and in the trials at track speed several subjects were able

to continue for too long (mean ± SD: 158 ± 10 s for the treadmill run v 140 ± 4 s for the track run). (Pilot test data are given in Appendix 6 (Table A6.2).) Therefore track speed plus 1 $km.h^{-1}$ was adopted as a simple compromise, to give a test duration of approximately 2 minutes.

6.2.4 Expirate analysis

Expired gases were analysed on-line using the QP9000.

6.2.5 Data Analysis

Breath-by-breath data were first converted to second-to-second data using linear interpolation between breaths. Data from the ramp tests were used to determine $\dot{V}O_2$peak and AT. Rolling 15 second averages were calculated for $\dot{V}O_2$ for every complete 15 second period during the test, in order to determine $\dot{V}O_2$peak (the highest of these values), for both the ramp test and the square wave test. The speed at the AT was determined from the ramp test data using the V-slope method as described by Beaver et al. (1986).

The HRpeak was compared between all three test conditions using one way ANOVA. The $\dot{V}O_2$peak was compared between the two laboratory based tests using a paired t-test.

In modelling the $\dot{V}O_2$ response for each individual, it was decided to remove the data from the first phase of the response (see section 3.10) (Chapter 3). Previous researchers adopting this approach have excluded the initial 25 seconds of data (Whipp et al. 1982a; Lamarra et al. 1987; Gerbino et al. 1996). In the present study however, due to the short duration of the exercise, and after visual inspection of the data revealed that the first phase was completed much earlier than 25 seconds, only the first 15 seconds of data were excluded from the modelling of the response. The data were modelled using SPSS for Windows (version 9.0). Non-linear regression (least sum of squares by iteration) was used to estimate the gain (asymptotic value of $\dot{V}O_2$ above baseline), the time constant (τ) of the

response, and the time delay (δ) of the phase-2 response beginning. The single exponential equation:

$$\dot{V}O_2(t) = \text{baseline} + \text{GAIN} \times (1-e^{-(t-\delta)/\tau}) \quad (6.3)$$

was used for this purpose. The baseline ($\dot{V}O_2$) was calculated as the average $\dot{V}O_2$ for the sixty second period immediately preceding the square wave test. No parameters were constrained and the resulting parameter estimates were therefore simply those that produced the best fit model.

The approach described by Hill and Stevens (2001) was used to investigate the asymptote of the model. The data from the first 45 seconds of the exercise (30 seconds of data since 15 had already been removed) were re-modelled using equation 6.3. The calculated GAIN and τ were then compared to those calculated from the whole data set using paired t-tests. The theoretical O_2 demand was calculated from the test speed and the individual $\dot{V}O_2$-running speed regression derived from the ramp data.

The issue of whether $\dot{V}O_2$ had truly plateaued was addressed by examining the data from the final 30 seconds of the square wave tests. A gradient was calculated from these data for each subject and a mean gradient when the data for each second were averaged across the subjects. The gradients were compared to zero using a one sample t-test. Any outlying data points were removed prior to this analysis. Outliers were defined as being more than 3 SDs from the mean (Lamarra et al. 1987). It is normal for a small number of outliers to be present in breath-by-breath data (Lamarra et al. 1987; Potter et al. 1999).

The level of noise on the data was also evaluated, again using data from the final 30 seconds of the square wave tests. A one-sample t-test revealed that the gradient, when these data were regressed on time, was not different from zero ($P = 0.794$). However, since these small gradients were not all in the same direction, the SD of the residuals (around this regression line), rather than the SD of the differences from the mean, was used to quantify the random error. This was done for both second-to-second and breath-by-breath data.

Whilst SPSS returns 95% confidence limits along with the parameter estimates, such limits should not be used (Mutulsky and Ransnas 1987). Exact confidence limits cannot be calculated for non-linear functions; reported confidence limits are based on linearising assumptions and will underestimate actual confidence limits. Simulations described by Lamarra et al. (1987) were used to calculate confidence limits for the GAIN, τ and δ. It was decided to calculate confidence limits for GAIN in addition to those described by Lamarra et al. (τ and δ) since this was a primary focus of this study.

Simulations were calculated by first calculating the SD / GAIN ratio for each subject. The subjects who returned the highest and lowest ratios were used as the worst and best case scenarios respectively. Parameter estimates from these subjects were used to generate 500 data sets per subject based on the underlying response superimposed with random noise with a SD equal to the individual SD. Each data set was then modelled by the same iterative method as the test data, and confidence limits (1.96 x SD) for each parameter estimate were calculated from the values returned from these 500 model fits. These confidence limits were then used, in conjunction with the gain of the underlying response and the SD of the noise, to calculate a value for the constant L described by Lamarra et al. (1987), by manipulating Equation 6.1 to give:

$$L = [K_1 / (SD / GAIN)] \quad (6.4).$$

This calculated value for L was then used to calculate the number of transitions required to return given confidence limits parameter estimates of τ and δ in the severe exercise domain (Equation 6.2).

Relationships between $\dot{V}O_2peak$ (as an index of aerobic capability) and the parameter estimates were evaluated. This analysis used Pearson correlation coefficients. The alpha level was set at 0.05% for all tests. Data are mean ± SD unless otherwise stated.

6.3 Results

Results from the three tests, including peak values for $\dot{V}O_2$ and HR, are contained in Table 6.1. Peak $\dot{V}O_2$ from the treadmill run at 800 m pace was found to be significantly lower than that recorded from the ramp test ($P = 0.008$) despite the exhaustive nature of both tests. There was no difference in HRpeak between the three tests ($P = 0.243$). Speed and $\dot{V}O_2$ at the AT were calculated as 12.9 ± 1.6 km.h^{-1} and 3.11 ± 0.47 L.min^{-1} respectively.

Table 6.1: Results from the track time trial (800 m track), laboratory based square wave run at 800 m pace (square wave), and ramp test (ramp)

	800 m TRACK	SQUARE WAVE	RAMP
Duration (s)	118 ± 21	132 ± 11	737 ± 42
Average Speed (km.h^{-1})	21.9 ± 1.9	22.9 ± 1.9	N/A
$\dot{V}O_2$peak (L.min^{-1})	N/A	4.14 ± 0.32	4.74 ± 0.55
$\dot{V}O_2$peak (ml.kg^{-1}min^{-1})	N/A	60.2 ± 1.2	68.9 ± 5.6
HRpeak (b.min^{-1})	190 ± 13	186 ± 12	192 ± 4

The $\dot{V}O_2$ response to the exhaustive square wave test is shown, along with the residual errors, for a representative subject in Figure 6.1. The modelled response is only shown for phase –2 (i.e. from 16 seconds onwards). The mean response for all subjects, expressed as a percentage of $\dot{V}O_2$peak from the ramp test, is shown in Figure 6.2.

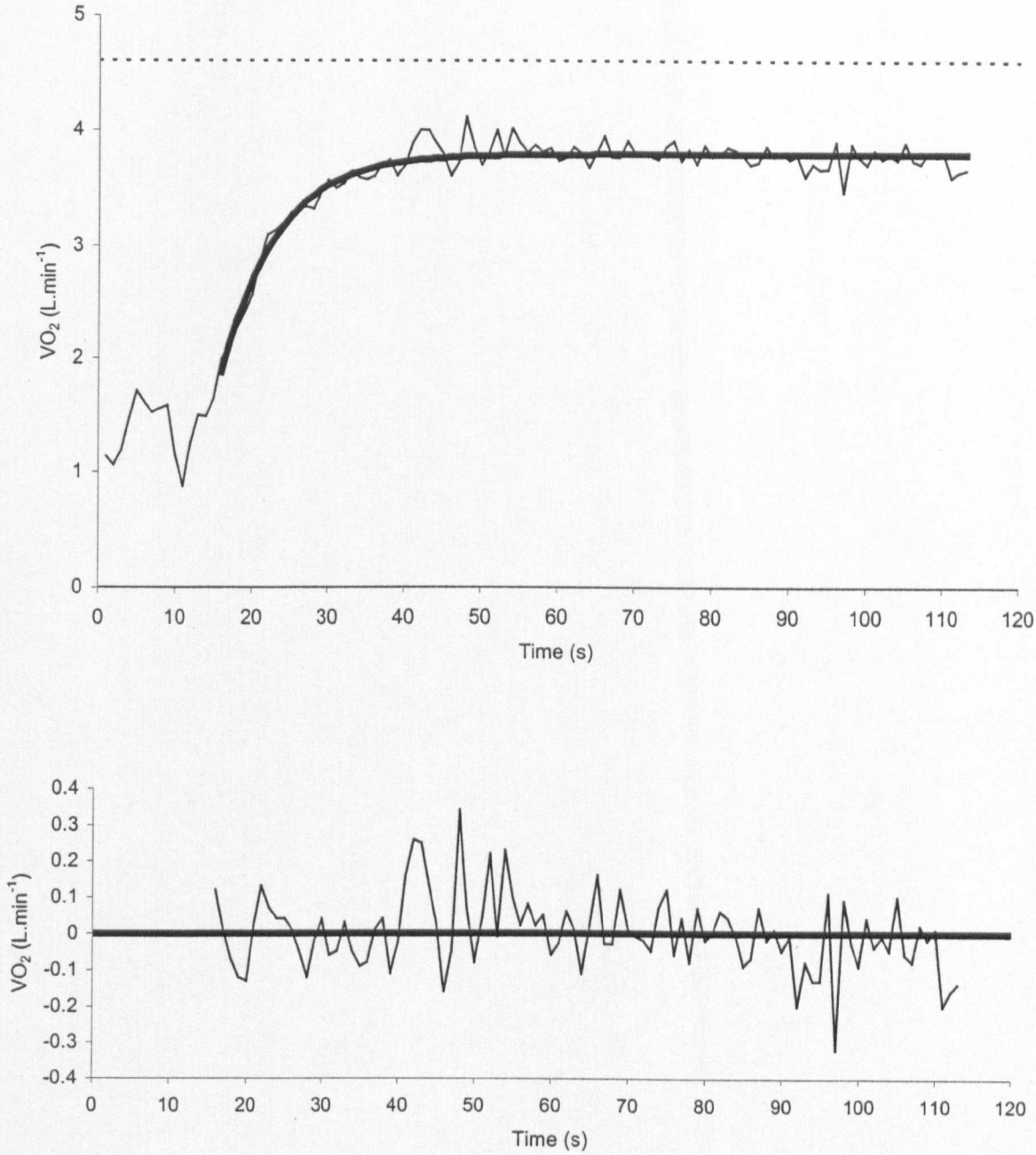

Figure 6.1: **The $\dot{V}O_2$ response to the exhaustive treadmill test at 800 m pace (upper panel) and the residual errors of the regression model (lower panel) for a representative subject**

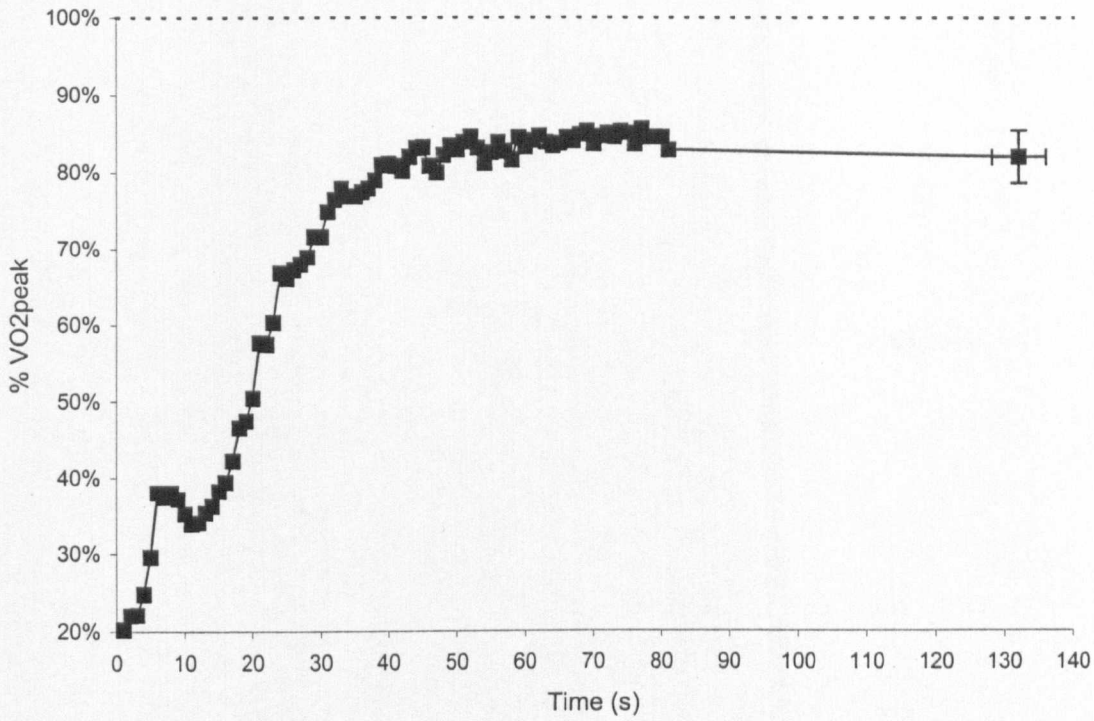

Figure 6.2: The mean $\dot{V}O_2$ response for all subjects to the exhaustive treadmill test at 800 m pace
Note: Error bars represent SEM. For clarity the error bars are omitted from all but the final data point.

The parameter estimates from the modelling of the $\dot{V}O_2$ response to the square wave run at 800 m pace are contained in Table 6.2. The asymptote expressed as a percentage of $\dot{V}O_2$peak from the incremental test is also included in this table.

Table 6.2: Individual parameter estimates, for the asymptotic value of $\dot{V}O_2$ above resting (GAIN), time constant (τ) and time delay (δ), for the $\dot{V}O_2$ response to the treadmill run at 800 m pace. The asymptote is given as a percentage of $\dot{V}O_2$peak and resting $\dot{V}O_2$ (baseline) are also given

Subject	Baseline ($ml.min^{-1}$)	GAIN ($ml.min^{-1}$)	Asymptote (% $\dot{V}O_2$peak)	τ (s)	δ (s)
1	512	3081	87%	10.9	11.8
2	680	4044	91%	11.9	11.7
3	645	3144	90%	14.6	7.6
4	416	3758	90%	16.1	10.5
5	598	3314	91%	9.8	12.4
6	568	3229	82%	7.1	12.3
7	595	3460	79%	6.9	11.6
8	745	3381	73%	8.1	13.4
MEAN	**595**	**3426**	**85%**	**10.7**	**11.4**
SD	**101**	**326**	**7%**	**3.4**	**1.8**

Oxygen uptake appeared to be reaching the plateau within 60 seconds (Figure 6.2). That $\dot{V}O_2$ was level throughout the last 30 seconds of the square wave test at 800 m pace was confirmed by the gradients contained in Table 6.3. These gradients were shown to be not different from zero ($P = 0.771$). The SD of the residuals of the same data are also shown for each individual. It was demonstrated that the process of converting data to second-to-second data itself reduced the random error.

Table 6.3: Gradients and SD of the residuals from the last 30 seconds of the square wave test at 800 m pace for all subjects

Subject	Gradient ($ml.min^{-2}$)	SD Breath-by-breath ($ml.min^{-1}$)	SD Second-to-second ($ml.min^{-1}$)
1	49	91	76
2	186	241	190
3	394	176	134
4	- 42	150	123
5	- 463	155	103
6	- 93	126	94
7	- 338	281	172
8	72	126	85
MEAN	**- 29**	**168**	**122**
SD	**275**	**63**	**41**

Pearson correlation coefficients were calculated between $\dot{V}O_2peak$ ($ml.kg^{-1}min^{-1}$) from the ramp test and each of three other variables. The three variables examined were the asymptotic value for $\dot{V}O_2$ (baseline + gain) expressed as $ml.kg^{-1}min^{-1}$, the same asymptotic value for $\dot{V}O_2$ expressed as a percentage of $\dot{V}O_2peak$ from the ramp test, and τ. No relationship was found between $\dot{V}O_2peak$ and the asymptotic $\dot{V}O_2$ expressed as $ml.kg^{-1}min^{-1}$, a moderate relationship was found between $\dot{V}O_2peak$ and τ, but a strong relationship existed between $\dot{V}O_2peak$ and the percentage of $\dot{V}O_2peak$ achieved during the treadmill run at 800 m pace. These three relationships are shown graphically in figures 6.3 to 6.5.

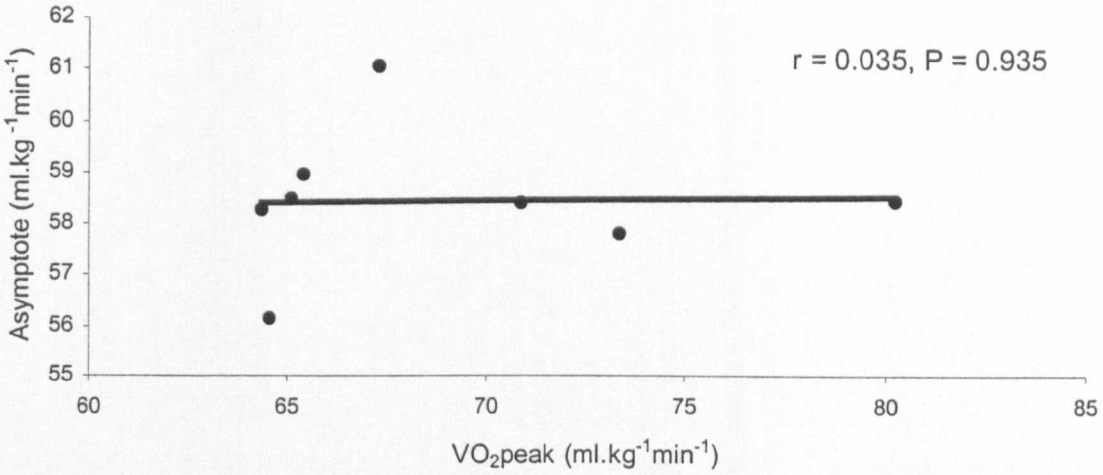

Figure 6.3: **Relationship between $\dot{V}O_2$peak from the ramp test and the asymptotic value for $\dot{V}O_2$ (baseline + gain) from the treadmill run at 800 m pace.**

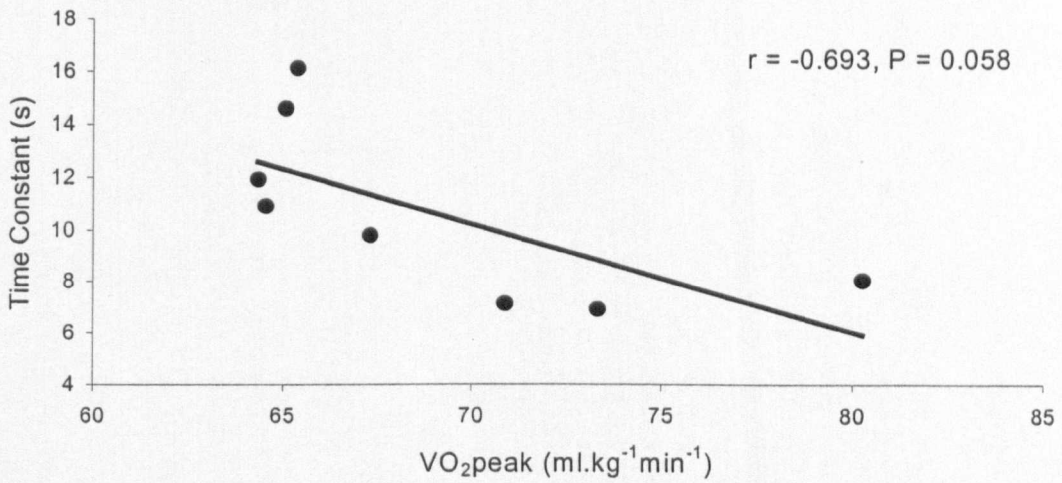

Figure 6.4: **Relationship between $\dot{V}O_2$peak from the ramp test and the time constant (τ) for the $\dot{V}O_2$ response to the treadmill run at 800 m pace.**

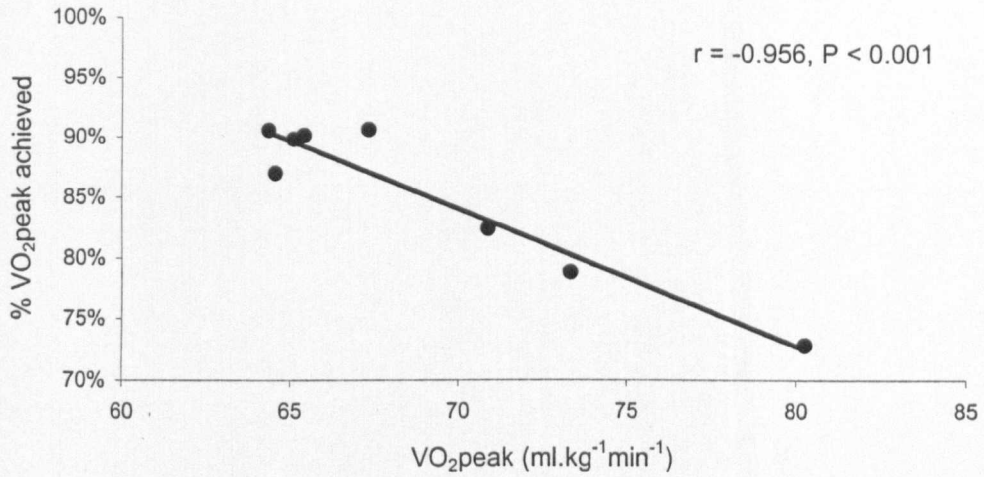

Figure 6.5: Relationship between $\dot{V}O_2$peak from the ramp test and the asymptotic value for $\dot{V}O_2$ (baseline + gain) (expressed as a percentage of $\dot{V}O_2$peak from the ramp test) from the treadmill run at 800 m pace.

Figures 6.3 to 6.5 show a cluster of subjects with a $\dot{V}O_2$peak from the ramp test of about 65 ml.kg^{-1}min^{-1} and that subject 8 represents an extreme value for this group (80 ml.kg^{-1}min^{-1}). Outliers in the data set are known to affect correlation coefficients (Atkinson and Nevill 1998). Therefore the correlation coefficients were recalculated omitting subject 8, to ensure that the reported relationship between $\dot{V}O_2$peak and asymptote % $\dot{V}O_2$peak achieved in the square wave test was not due to this extreme value. The correlation coefficients of $\dot{V}O_2$peak (ramp) (ml.kg^{-1}min^{-1}) with asymptote (ml.kg^{-1}min^{-1}), τ (s) and asymptote (% $\dot{V}O_2$peak achieved) were r = 0.058 (P = 0.901), r = -0.327 (P = 0.474) and r = -0.889 (P = 0.007) respectively. This confirmed the strength of the relationship between $\dot{V}O_2$peak and % $\dot{V}O_2$peak achieved in the square wave test.

The asymptotic (GAIN + Baseline) $\dot{V}O_2$ from the whole test data was 4021 ± 344 ml.min^{-1} compared to 4146 ± 546 ml.min^{-1} when estimated from just the initial 45 seconds of data.

There was found to be no difference between these figures (P = 0.311). Both figures were considerably less than both the $\dot{V}O_2$peak from the ramp test (4740 ± 547 ml.min^{-1}) and the theoretical O_2 demand (5448 ± 611 ml.min^{-1}) and this was the case for all subjects. There was no difference between τ for the two modelling strategies (10.7 ± 3.4 s using all data points and 11.5 ± 6.0 s using just the initial 45 s) (P = 0.681). Individual parameter estimates from the initial 45 seconds are given in Appendix 6 (Table A6.1).

Ratios of SD / GAIN, using the SD calculated from second-to-second data, ranged from 0.025 (subject 1) to 0.050 (subject 7). The underlying responses from these two subjects were used in the simulations. The parameter confidence limits from the simulations are contained in Table 6.4. Equation 6.4 resulted in a value for L of 30.0 for simulations of both ratios, using confidence limits for τ. Equation 6.2 showed that for confidence limits of ± 1 second for τ only one transition would be needed for the subject with the lowest noise to signal ratio (n = 0.55), whereas two would be necessary for the subject with the highest noise to signal ratio (n = 2.22).

Table 6.4: Confidence limits (1.96 * SD) of parameter estimates from simulations of SD / GAIN ratios of 0.025 and 0.050

SD / GAIN	Parameter	Assumed value	95% confidence limits
0.025	GAIN	3081 ml.min^{-1}	± 11.8 ml.min^{-1}
	τ	10.9 seconds	± 0.7 seconds
	δ	11.8 seconds	± 0.7 seconds
0.050	GAIN	3460 ml.min^{-1}	± 35.8 ml.min^{-1}
	τ	6.9 seconds	± 1.5 seconds
	δ	11.6 seconds	± 1.6 seconds

6.4 Discussion

A principal finding from the present study was that in exhaustive square wave running of approximately 2 minutes duration $\dot{V}O_2$ does not attain $\dot{V}O_2$max. The results from the breath-by-breath analysis techniques used in the present study were consistent with the Douglas bag data reported in Chapter 4 and with the 10 second averaged data presented by Spencer et al. (1996) and Spencer and Gastin (2001).

The tendency for $\dot{V}O_2$ to plateau below $\dot{V}O_2$max that was demonstrated in Study 1 for a group of aerobically fit individuals, was shown to be more pronounced in a specifically run trained population. Average $\dot{V}O_2$peak was found to be 12% lower in the square wave test when compared to that achieved during the ramp exercise test. Furthermore $\dot{V}O_2$ did not tend either to $\dot{V}O_2$max or the $\dot{V}O_2$ required as has been suggested by previous researchers (Katch 1973; Hughson et al. 1978; Whipp 1994a; Hughson et al. 2000). Rather $\dot{V}O_2$ tended towards a rate that was below its maximum by some margin.

The plot of the mean data (Figure 6.2) seemed to suggest that $\dot{V}O_2$ had reached its asymptote within 60 seconds of exercise. The modelling process that gave mean parameter estimates of 10.7 seconds for τ and 11.4 seconds for δ (Table 6.2) confirmed this. In this exponential model it takes five τs to reach 99.3 % of the asymptote. Five τs (plus δ) would be 65 seconds using the mean values for this subject group. The zero gradient of the $\dot{V}O_2$-time relationship for the final 30 seconds of exercise confirmed that a plateau had been achieved.

The modelling process showed an asymptote for $\dot{V}O_2$ that was well below either the $\dot{V}O_2$ required or $\dot{V}O_2$peak for the ramp test. Furthermore τ and δ were much smaller than has previously been reported for heavy intensity exercise (Barstow and Mole 1991; Gaesser and Poole 1996). That $\dot{V}O_2$ was tending towards a figure that was some way below $\dot{V}O_2$max was confirmed by the separate modelling of the response from the initial 45 seconds of the test. It appeared from the modelling of these data that $\dot{V}O_2$ was tending neither to $\dot{V}O_2$max nor to $\dot{V}O_2$ required, as has been previously assumed (Margaria 1965; Hughson 1978; Hughson et al. 2000; Hill and Stevens 2001). Some caution should be

exercised at this point however since the τ displayed by the subjects in the present study was so much quicker than that reported by Hill and Stevens (2001) who used less trained subjects. The time period of 45 seconds, which gave only 30 seconds of data in the present study after the removal of the phase-1 data points, represented little more than one τ in the Hill and Stevens study. In the present study however this same period represented approximately 3 τs and therefore the response was already close to reaching its asymptote (approximately 95%). This highlights how fast the $\dot{V}O_2$ response was in this subject group and how difficult it is to determine where this response tends to when the response is so fast.

The results of the present study challenge current mathematical models of 800 m running performance. Such models all assume that $\dot{V}O_2$ will tend towards $\dot{V}O_2max$ or the $\dot{V}O_2$ required (Wilkie 1980; Di Prampero 1986; Capelli et al. 1999). Capelli et al. (1999) assumed τ to be 24 seconds; however the models of Wilkie (1980) and Di Prampero (1986) assume τ to equal 10 seconds, as in the present study. Di Prampero's model has been shown to overestimate the speed that could be maintained for 800 m however (Di Prampero et al. 1993). A model that uses an appropriate value for τ but wrongly assumes that the $\dot{V}O_2$ response tends to $\dot{V}O_2max$ would overestimate the aerobic contribution to the energy demand and therefore overestimate the sustainable speed (assuming a fixed anaerobic capacity).

Intra-individual SDs, calculated from the final 30 seconds of the square wave treadmill test, gave an indication of the level of noise surrounding the data at this exercise intensity. The SDs for $\dot{V}O_2$ were larger for breath-by-breath data than for data that had been interpolated to second-to-second. It has been previously shown that the process of interpolation itself acts as a filter to reduce noise (Lamarra et al. 1987). Subjects varied in the level of noise on the data, but the noise was always small in comparison to the magnitude of the response. Therefore whilst further analysis of the exponential model used (Equation 6.3) is necessary, it was considered worthwhile attempting to model and evaluate the response as described above. Plots of the residual error (Figure 6.1) suggest that this model fits the data well.

A degree of caution should be exercised in the interpretation of the modelling data from the present study because the parameters were calculated using a single transition. Previous researchers have used multiple transitions and averaged the data across them to reduce the level of noise prior to modelling the response (Whipp et al. 1982; Patterson and Whipp 1991). The average level of breath-by-breath noise for severe intensity running was greater than that reported by Lamarra et al. (1987) for moderate intensity cycling (168 and 89 $ml.min^{-1}$ respectively). The confidence intervals of the various parameters are dependent on both the level of noise and the magnitude of the response (Lamarra et al. 1987), as was demonstrated by the wider confidence limits for the simulation of the noise to signal ratio (SD / GAIN) of 0.050. However even for this worst case scenario confidence limits of less than ± 2 seconds were returned. This is considered to be a reasonable level of accuracy for studies of moderate intensity exercise (Lamarra et al.1987).

The use of the equations given by Lamarra et al. (1987) showed that differences exist between exercise intensities. A value for the constant L was calculated as 30 for the subjects in the present study, whilst a value of approximately 50 was apparent in the calculations of Lamarra et al. (1987) for moderate intensity exercise. This is presumably due to the smaller τ in severe intensity exercise, and in particular in a trained population. The value for L shows that narrower confidence limits will be returned for a given number of exercise transitions in short duration exhaustive exercise compared to moderate intensity exercise. It also suggests that averaging data over only 2 transitions would be adequate for a 95% confidence limit for τ of ± 1 second in most subjects. Lamarra et al. (1987) calculated the number of transitions required for confidence limits of ± 2 seconds. Since the present study suggests that severe intensity exercise will result in a much shorter τ, a higher degree of accuracy is desirable for exercise in this intensity domain. The effect of the magnitude of τ on the confidence limits of the model parameters is discussed in detail in Chapter 7.

The relationship between $\dot{V}O_2$peak from the ramp test ($ml.kg^{-1}min^{-1}$) and τ suggested a link between the speed of $\dot{V}O_2$ kinetics in severe intensity running and aerobic capability. Such a relationship may be associated with a prevalence of type I muscle fibres. Type I fibres have been shown to demonstrate faster $\dot{V}O_2$ kinetics than Type IIa (Crow and

Kushmeric 1982; Barstow et al. 1996). However the strength of this correlation was reduced when subject 8 (80 ml. $kg^{-1}min^{-1}$) was removed from the analysis, suggesting that the strength of the initial relationship may have been due to this outlier.

That there was no relationship between $\dot{V}O_2peak$ ($ml.kg^{-1}min^{-1}$) and the asymptote for $\dot{V}O_2$ ($ml.kg^{-1}min^{-1}$) was unexpected. It appears that despite a higher aerobic potential the subjects with the highest $\dot{V}O_2peak$ were unable to realise this potential during the 800 m paced run. This is clearly demonstrated by comparing the SD for $\dot{V}O_2peak$ from the ramp test (5.6 $ml.kg^{-1}min^{-1}$) with that for the $\dot{V}O_2peak$ from the square wave test (1.2 $ml.kg^{-1}min^{-1}$). It is interesting to note that subject 8 whom possessed an extremely high $\dot{V}O_2max$ was not the fastest 800 m runner despite this advantage in aerobic potential.

The asymptotic value for $\dot{V}O_2$ ($ml.kg^{-1}min^{-1}$) from the square wave test represented only 85 ± 7 % of the $\dot{V}O_2peak$ from the ramp test. Furthermore, when expressed relative to body mass ($ml.kg^{-1}min^{-1}$), the values for all subjects were very similar (58.4 ± 1.3 $ml.kg^{-1}min^{-1}$). A strong negative correlation was shown to exist between $\dot{V}O_2peak$ from the ramp test ($ml.kg^{-1}min^{-1}$) and the percentage of this $\dot{V}O_2peak$ that was achieved in the square wave test. Those subjects with the highest $\dot{V}O_2peak$ achieved the lowest percentage of this value.

The results of the present study suggest that for running events of approximately 2 minutes duration, participants may only achieve a $\dot{V}O_2$ of approximately 60 $ml.kg^{-1}min^{-1}$. The implication is that there may be little benefit in possessing a very high $\dot{V}O_2max$, since subjects seemed unable to use this extra aerobic reserve within the duration of the event. Elite middle distance runners have been demonstrated to possess a lower $\dot{V}O_2max$ than elite distance runners (Svedenhag and Sjodin 1984), despite the higher intensity of the former's training regime. Whilst recent studies have stressed the aerobic contribution to middle distance running (Hill 1998; Spencer and Gastin 2001), the present study suggests that whilst the aerobic energy contribution is indeed high, the anaerobic contribution (or capacity) is a more important determinant of performance than $\dot{V}O_2max$.

In summary the present study supported the findings of Study 1 (Chapter 4), in that for square wave running at a speed fast enough to elicit exhaustion in approximately 2 minutes, $\dot{V}O_2$ would plateau below its maximum rate. Furthermore $\dot{V}O_2$ kinetics seem to be faster in this domain than has been reported for moderate or heavy intensity exercise. The study also suggested that there is a limit to the $\dot{V}O_2$ that can be achieved at such an intensity that seems to be independent of $\dot{V}O_2$max. The physiological mechanisms that underlie this inability to achieve $\dot{V}O_2$max are not known. The results suggest that this apparent plateau in $\dot{V}O_2$ represents the asymptote of the response. However the speed of the Phase-2 response made this analysis problematic.

Confidence limits calculated from computer simulation of noise, together with the equations of Lamarra et al. (1987), suggested that if data were averaged across two transitions confidence limits for τ of approximately $\pm$ 1 second would be achieved. There are however questions about the noise on the $\dot{V}O_2$ data which have not been answered for severe intensity exercise. First whether the noise is Gaussian, and whether therefore repeated transitions will reduce the noise in the manner described by Lamarra et al. (1987). Second, whether the level of noise is unchanged in the severe exercise intensity domain. These issues have only been investigated for moderate intensity cycling (Lamarra et al. 1987; Potter et al. 1999). These issues are examined with respect to short duration exhaustive square wave running in Chapter 7.

CHAPTER 7

STUDY 4: BREATH-BY-BREATH 'NOISE' AND PARAMETER ESTIMATION FOR $\dot{V}O_2$ KINETICS IN SEVERE INTENSITY RUNNING

7.1 Introduction

Breath-by-breath $\dot{V}O_2$ data are a composite of the underlying response to a given exercise demand and the irregularities and fluctuations that may be considered as "noise" on this signal. The level of this noise varies considerably between individuals (Lammara et al. 1987). Study 3 (Chapter 6) characterised the $\dot{V}O_2$ response to severe intensity running of a short duration using on-line breath-by-breath gas analysis. The equations presented by Lamarra et al. were used to determine the number of superimposed transitions that would be necessary to produce acceptable confidence limits. But the equations used were based on assumptions about the nature of the noise that have to date only been examined in moderate intensity cycling (Lamarra et al.1987; Potter et al. 1999). The nature of this noise in severe intensity running is as yet unknown.

The noise on breath-by-breath $\dot{V}O_2$ data has been shown to be both random and of a normal (Gaussian) distribution for moderate intensity cycling (Lamarra et al. 1987), both by visual examination of the data and using a process of auto-correlation. However, Potter et al. (1999) also tested whether the noise was Gaussian using the Chi squared (χ^2) statistic, and found that in the majority of subjects the noise was not Gaussian. Potter et al. used a child population, but this key difference between these studies may be due to the χ^2 test providing a more stringent test of the data.

The major assumption of the equations presented by Lamarra et al. (1987), used to predict the number of repeated transitions required for a given confidence limit of the parameter estimates, is that the data are Gaussian in nature. The nature of breath-by-breath noise has to date not been assessed in severe intensity cycling and never running. Since the models used to describe the $\dot{V}O_2$ responses are produced on a 'best-fit' basis, the models themselves assume that the data will be randomly and normally distributed around the underlying response. If the noise is not Gaussian this will distort the model description of

the underlying response. Furthermore, the reduction in noise as a result of repetition and averaging of exercise transitions, also depends on the noise being random and Gaussian. It is important then that the nature of this noise is investigated.
Lamarra et al. (1987) presented the following equations

$$K_l = L (SD / GAIN) \quad (7.1)$$

$$n = [(L * SD)/ (K_n * GAIN)]^2 \quad (7.2)$$

where $\pm K_l$ are the 95% confidence limits of the parameter estimate from a single transition, L is a constant, SD is the standard deviation of the noise and GAIN is the magnitude of the response (i.e. the difference between the asymptotic value of $\dot{V}O_2$ and the baseline or resting $\dot{V}O_2$). In equation 7.2, n is the number of transitions required to produce the desired confidence limits ($\pm K_n$).

In chapter 6, the value of L was derived after confidence limits had been established, using computer simulations, with the following equation derived from equation 7.1:

$$L = [K_l / (SD / GAIN)] \quad (7.3).$$

A value of 30 was derived for L for the two subjects displaying the highest and lowest level of noise compared to a value of approximately 50 for the data of Lamarra et al. (1987) for moderate intensity cycling. The ratio of noise to signal (SD / GAIN) is likely to be smaller, due to the greater magnitude of the response, at high exercise intensities. Unless the level of noise is significantly increased at severe exercise intensities therefore, the confidence limits will become narrower (for a given number of transitions), as the GAIN is bound to be of a greater magnitude at this exercise intensity. It is important therefore to understand both the nature and the magnitude of the noise in the severe exercise intensity domain. Lamarra et al. demonstrated that there is no difference in the level of this noise between cycling at 0W and 100W (moderate intensity exercise for those subjects), but moderate exercise has never been compared to higher exercise intensities.

In Study 3, the SD and GAIN of the two subjects with the highest and lowest SD / GAIN ratio were used in Equation 7.2, together with a value of 30 for L. This suggested that superimposition of just two transitions would be sufficient to give confidence limits of ± 1 second (for τ and δ) in exhaustive square wave running of about 2 minutes duration. It remains to be determined whether this is a robust equation that can be used across exercise intensities. Equations 7.1 and 7.2 imply that the level of noise on a data set is function of ($1 / \sqrt{n}$), where n is the number of transitions. The equation can be readily validated then by plotting the relationship between the SD of the noise and ($1 / \sqrt{n}$), as subsequent transitions are added.

Lamarra et al. (1987) used a series of computer simulations to assess the impact of the length of the time constant (τ) on the resulting confidence limits of the parameter estimates derived from non-linear regression. This analysis showed a widening of these confidence limits with an increasing τ. Study 3 (Chapter 6) showed that the time constant for severe intensity running was much shorter than that reported for moderate intensity exercise (Lamarra et al. 1987). The short time constant seen in severe intensity running would be expected to result in narrow confidence limits. However, no difference was found in L for the two subjects examined in Study 3 despite a difference in τ (10.9 v 6.9 seconds). Since L is to a large extent dependent upon τ this relationship warranted further investigation in the severe intensity domain.

The purpose of the present study was fivefold. First to examine the nature of breath-by-breath noise for $\dot{V}O_2$ in exhaustive severe intensity running. Second to investigate the relationship between τ and L in the severe intensity domain. Third to establish whether the equations presented by Lamarra et al. (1987) were valid for use in the severe intensity domain. Fourth if the previous two aims were satisfied, to calculate the number of transitions required for subsequent studies in the severe intensity domain. Finally, to examine the applicability of the exponential model to determine if the estimated asymptote reflected the observed plateau in $\dot{V}O_2$.

7.2 Method

7.2.1 Subjects

Four male subjects (mean ± SD: age 25.3 ± 3.9 years, height 1.82 ± 0.11 m, mass 83.4 ± 10.2 kg, and $\dot{V}O_2$peak from a ramp test 55.9 ± 2.5 $ml.kg^{-1}min^{-1}$) volunteered to take part in the study. They were physically fit and involved in regular aerobic exercise, though not specifically trained for running.

7.2.2 Study design

The subjects were required to attend the laboratory on six occasions, all on separate days. On the first visit subjects performed an exhaustive ramp test to determine $\dot{V}O_2$peak and the AT.

On each of the subsequent five visits subjects completed two constant intensity runs, one at a moderate and the other at a severe intensity. The first was an 8 minute run at a speed equivalent to 90% AT and the second was an exhaustive run at a speed that would elicit exhaustion in approximately 2 minutes. The speed for the severe intensity run was estimated from the peak speed achieved during the ramp test. If on the first occasion the run proved to be too long or too short, the speed was adjusted and subjects attended a further five sessions, completing a further five runs at the adjusted speed. The severe intensity run always started 5 minutes after the moderate intensity run. No warm up was performed prior to the moderate intensity test. However, subjects were encouraged to stretch thoroughly for up to 3 minutes between tests. They breathed through the gas collection apparatus for 2 minutes prior to each test so that a baseline $\dot{V}O_2$ could be determined.

7.2.3 Test Protocols

The speed for the ramp test was continually increased by 0.1 $km.h^{-1}$ every 5 seconds until the subject was unable to keep pace with the belt and lifted himself clear , thus ending the

test. The starting speed was estimated depending on the fitness of the subject to elicit exhaustion in approximately 12 minutes.

The moderate intensity run lasted for 8 minutes and the treadmill was then stopped. In the severe intensity run the subject ran to exhaustion on each occasion and was not given feedback on the elapsed time.

7.2.4 Expirate analysis

Expired gases were analysed on-line using the QP9000.

7.2.5 Data Analysis

Data analysis was limited to examination of the $\dot{V}O_2$ data since $\dot{V}CO_2$ and $\dot{V}_E$ were not modelled in this thesis. To examine the distribution of the noise, the final 30 seconds of breath-by-breath $\dot{V}O_2$ data from each transition, both moderate and severe, were used. Any outliers were first removed from the data. Outliers were defined as data points that were more than 3 SDs from the mean. This approach was also adopted by Lamarra et al. (1987), who suggested that certain recorded breaths were clearly artifactual, possibly the result of coughing or swallowing, and should be excluded from such an analysis. The data contained relatively few of these artefacts at either intensity. Breath-by-breath data were used in this part of the analysis since data points from interpolated second-to-second data would, by definition, be related to the value of the previous data point, particularly at low respiratory frequencies, and therefore would not represent truly random data. Thirty seconds of data were used at both exercise intensities. This is fewer data than were used by other researchers (Lamarra et al. 1987; Potter et al. 1999) examining moderate intensity exercise. For the purposes of the present study the 30 seconds of data were used because of the limited amount of steady state (plateau) data available for the severe intensity run. The final 30 second period was used for both moderate and severe exercise intensities to allow a valid comparison between the intensities.

The 30 seconds of breath-by-breath data were first regressed on time, to remove any linear trend that was sometimes present. The residuals from this analysis were considered to represent the 'noise'. These residuals were then plotted to examine the distribution of the noise. Further analysis of the normality of the data was performed using the Chi Squared (χ^2) Test. For the χ^2 analysis data from each test were divided into ten groups based on the Z-score of the data. These groups ranged from Z-scores of –3 to 3 (-3.0 to -2.0, -2.0 to -1.5, -1.5 to -1.0, -1.0 to -0.5, -0.5 to 0.0, 0.0 to 0.5, 0.5 to 1.0, 1.0 to 1.5, 1.5 to 2.0, and 2.0 to 3.0). Analysis of the random nature of this noise used a process of auto-correlation. Auto correlation is a process that involves correlating a given data set with itself, but at a given delay. For example, a 1-breath delay would pair breath 1 with breath 2, breath 2 with breath 3 etc. Auto-correlation coefficients were calculated for the data at 1, 2 and 3 breath delays. If the coefficient is near to 1 then data points are related to those around them; if the coefficient is near to zero then the noise may be considered to be random.

Breath-by-breath data from the QP9000 were then interpolated to second-to-second data using linear interpolation between breaths. Data from the ramp test were used to determine $\dot{V}O_2$peak and AT. Second-to-second data were averaged across transitions to give mean values for each second across 2, 3, 4 and 5 transitions. In the severe intensity tests, the data were only averaged and modelled up to the time at which the shortest transition was terminated. Data from each individual transition were modelled along with averaged data across 2, 3, 4 and 5 transitions to determine the effect of repeated transitions on the level of noise.

The level of noise on the data was evaluated using data from the final 30 seconds of the moderate and the severe intensity runs. The SD of the residuals of the regressed data was used, as in Study 3, to quantify the variability in the data. This SD of the residuals was calculated for second-to-second data on each individual transition as well as the averaged data across 2, 3, 4 and 5 transitions. The final 30 second-to-second data points from all five transitions were also combined to give 150 data points per subject at both exercise intensities. Differences in the level of noise between the two intensities were then compared using Levene's test for equality of variances.

In modelling the $\dot{V}O_2$ response, the data from the first phase of the kinetics were removed. For the moderate intensity exercise the initial 25 seconds of data were excluded (Whipp et al. 1982a; Lamarra et al. 1987; Gerbino et al. 1996). As in Chapter 6 only the first 15 seconds of data were excluded from the modelling of the response to the severe intensity transitions. The remaining data were modelled using the same model as Study 3:

$$\dot{V}O_2(t) = \text{baseline} + \text{GAIN} \times (1-e^{-(t-\delta)/\tau}) \quad (7.4)$$

The baseline ($\dot{V}O_2$) was calculated as the average $\dot{V}O_2$ for the 60 second period immediately preceding the constant speed test.

The assumption that the decrease in noise would be a function of $(1/\sqrt{n})$, where n is the number of transitions superimposed, was also tested. The mean SD (from the addition of each transition across subjects) was plotted against $(1/\sqrt{n})$, and this assumption was tested by linear regression. This was done for both moderate and severe intensity exercise.

As discussed in Chapter 6, confidence limits calculated during the fitting of non-linear regression cannot be used for the individual parameter estimates (Mutulsky and Ransnas 1987). Computer simulations as described by Lamarra et al. (1987) therefore were used to calculate confidence limits for GAIN, τ and δ.

The SD / GAIN ratio was calculated for each subject for each of the five tests in both moderate and severe intensity running. All ratios for severe intensity running fell between the best and worst case scenarios from Study 3 (Chapter 6) (0.025 and 0.050). The effect of this ratio on the confidence limits of the parameter estimates was investigated in Study 3. However, Lamarra et al. (1987) suggest that the constant L is also a function of the time constant (τ). A further three simulations were therefore carried by adding noise, to the underlying response of the subject from Study 3 with SD / GAIN ratio of 0.025 (see Table 6.4 (Chapter 6)). The GAIN and δ were the same for each simulation but the value for τ was changed. Five hundred data sets were generated for each of three values of τ, namely 5, 15 and 25 seconds. Each data set was then modelled by the same iterative method as the test data, and confidence limits (1.96 x SD) were calculated from the values returned from

these 500 model fits. This confidence limit was then used to calculate a value for the constant L using equation 7.3.

Finally the average $\dot{V}O_2$ from the final 30 seconds of the severe intensity exercise was compared with the parameter estimate of the asymptote from non-linear regression. This comparison was conducted for the data averaged across five transitions and used a paired t-test. The alpha level was set at 0.05 for all tests and data are mean ± SD unless otherwise stated.

7.3 Results

Breath-by-breath noise generally appeared to be normally distributed (Figure 7.1). The χ^2 test, which evaluates whether the observed distribution differs significantly from the expected normal distribution, showed that the noise on the breath-by-breath data was basically Gaussian. Of the 40 tests only one (moderate intensity test) was found to be significantly different from Gaussian (Figure 7.2). The individual χ^2 probabilities are contained in Table 7.1. Figure 7.1 shows the data set for which the χ^2 probability was highest ('most normal', P = 0.95), whereas Figure 7.2 shows the data set for which the χ^2 probability was lowest ('least normal', P = 0.01).

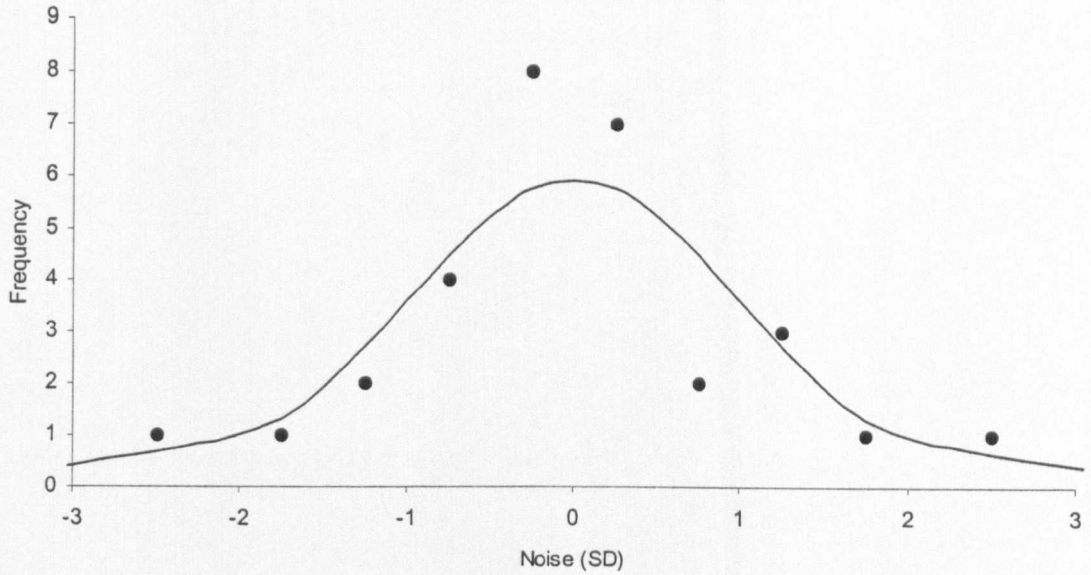

Figure 7.1: **Distribution of the breath-by-breath noise from the 5th severe intensity transition for subject 2. The line represents the normal distribution curve.**

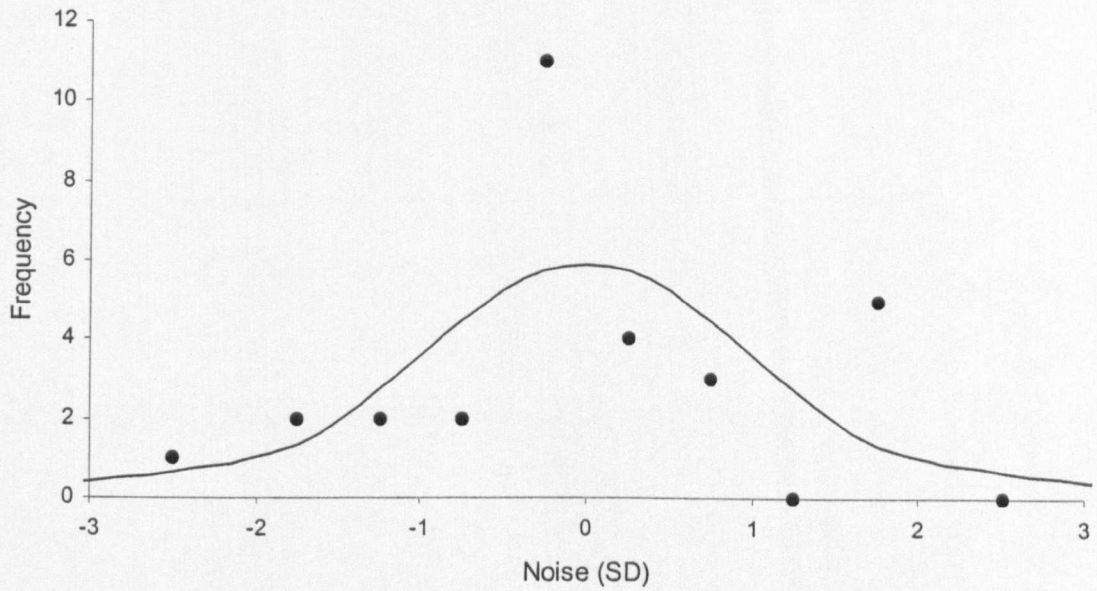

Figure 7.2: **Distribution of the breath-by-breath noise from the 4th moderate intensity transition for subject 1. The line represents the normal distribution curve.**

Table 7.1: 'Goodness of fit' of $\dot{V}O_2$ breath-by-breath noise compared to Gaussian distributions using the χ^2 test, from all transitions for each of the 4 subjects

Subject	Transition	Moderate Intensity Probability	Severe Intensity Probability
1	1	0.78	0.48
	2	0.97	0.92
	3	0.35	0.47
	4	0.01*	0.21
	5	0.75	0.90
2	1	0.83	0.76
	2	0.45	0.15
	3	0.91	0.36
	4	0.88	0.65
	5	0.49	0.95
3	1	0.83	0.42
	2	0.43	0.78
	3	0.80	0.90
	4	0.77	0.81
	5	0.59	0.80
4	1	0.79	0.61
	2	0.66	0.91
	3	0.77	0.91
	4	0.83	0.82
	5	0.06	0.33

*** denotes significant difference from Gaussian distribution**

Results of the auto correlation for all subjects and transitions are contained in Table 7.2. Auto -correlation coefficients are given for delays of 1, 2 and 3 breaths for each data set. This analysis demonstrated that the data were largely uncorrelated and therefore the noise was random.

Table 7.2: Autocorrelation coefficients for the breath-by-breath noise at breath delays of 1, 2 and 3 breaths, from all transitions for each of the 4 subjects

		Moderate Intensity			Severe Intensity		
		Breath Delay			Breath Delay		
Subject	Transition	1	2	3	1	2	3
1	1	-0.13	-0.34	0.21	0.07	0.18	0.11
	2	0.30	-0.17	-0.20	0.13	0.17	0.33
	3	0.21	-0.28	-0.13	0.20	0.14	0.14
	4	-0.34	0.09	-0.17	0.01	0.13	0.23
	5	-0.10	0.08	-0.34	0.35	0.01	-0.20
2	1	0.08	0.06	-0.06	-0.12	0.10	0.27
	2	-0.36	-0.09	0.32	-0.04	-0.09	-0.15
	3	-0.23	-0.13	0.00	0.00	0.04	0.30
	4	-0.25	-0.04	-0.02	-0.20	-0.21	-0.11
	5	0.01	0.06	0.26	-0.36	-0.02	0.11
3	1	0.37	0.08	-0.41	-0.57	0.31	-0.26
	2	-0.01	-0.01	-0.01	-0.48	0.07	0.09
	3	-0.22	0.25	-0.10	-0.44	0.36	-0.35
	4	-0.16	-0.35	0.28	-0.58	0.47	-0.22
	5	-.016	-0.21	0.07	0.11	0.08	-0.18
4	1	-0.14	0.05	0.05	-0.14	-0.36	-0.04
	2	0.25	0.35	-0.10	-0.15	-0.22	0.11
	3	-0.29	0.15	-0.06	0.07	-0.07	-0.20
	4	-0.07	-0.15	0.13	-0.14	0.23	-0.20
	5	-0.15	0.11	0.06	-0.18	0.09	0.01

The SDs of the residuals, from the regression of the interpolated second-to-second data, from the final 30 seconds, on time are contained in Table 7.3. Again data are shown for all four subjects. The mean gradient, across subjects, of this regression line was -133 ± 106 and -131 ± 269 ml.min^{-2}, for moderate and severe intensity exercise respectively. The data in Table 7.3 show that the SD decreased (i.e. the level of noise was reduced) as more transitions were superimposed. Levene's test for equality of variances showed that the level of noise was greater in moderate compared to severe intensity exercise in subjects 1, 2 and 3 ($P < 0.01$) but not in Subject 4 ($P = 0.240$).

Table 7.3: SDs of residuals for interpolated second-to-second data from the final 30 seconds of all transitions. Data are presented as ± ml.min^{-1}. Also shown is the effect of superimposing (sup) transitions to reduce the level of noise on the data.

		Transition								
Intensity	Subject	1	2	3	4	5	1 - 2 (sup)	1 - 3 (sup)	1 - 4 (sup)	1 - 5 (sup)
Moderate	1	377	271	242	163	217	242	187	138	128
	2	662	167	154	124	141	355	264	194	164
	3	336	171	767	133	215	170	276	209	180
	4	119	53	159	85	96	74	74	66	64
	Mean	**373**	**165**	**331**	**126**	**167**	**211**	**200**	**152**	**134**
	SD	**223**	**89**	**294**	**32**	**59**	**118**	**93**	**65**	**52**
Severe	1	126	141	134	136	166	88	76	63	60
	2	156	162	145	84	92	109	91	75	65
	3	104	76	98	83	86	60	51	45	42
	4	127	82	66	116	66	73	60	53	42
	Mean	**128**	**115**	**111**	**105**	**102**	**83**	**70**	**59**	**52**
	SD	**22**	**43**	**36**	**26**	**44**	**21**	**18**	**13**	**12**

Investigation into the relationship between the SD of the noise and $(1/\sqrt{n})$ confirmed that the SD was directly proportional to $(1/\sqrt{n})$, for both moderate and severe intensity exercise (figures 7.3 and 7.4). The effect of reducing noise with increasing numbers of transitions, is shown for a typical subject (subject 2) in Figure 7.5. The noise as shown by the residuals for the exponential model, is shown for a single transition, for data averaged across three transitions and for data averaged across five transitions. The underlying response (calculated from non-linear regression) is also shown.

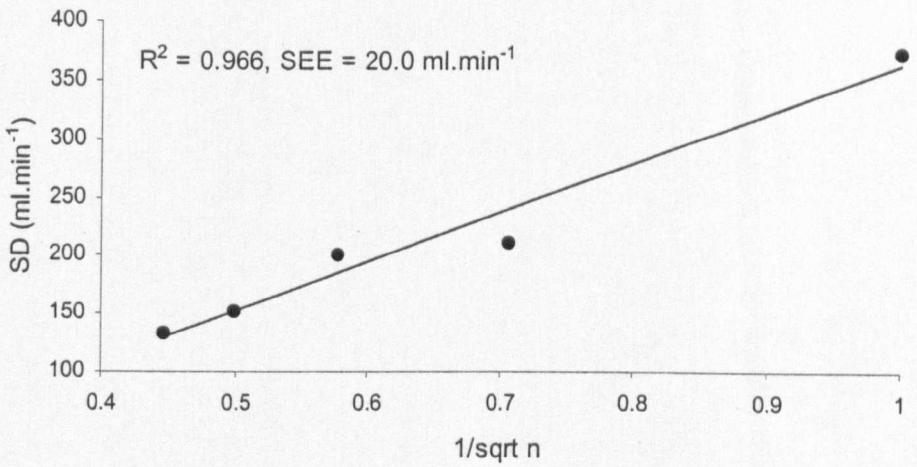

Figure 7.3: Relationship between the SD of the noise (as calculated from the superimposition of 1, 2, 3, 4 and 5 transitions) and $(1/\sqrt{n})$ (where n is the number of transitions superimposed) for the moderate intensity transitions

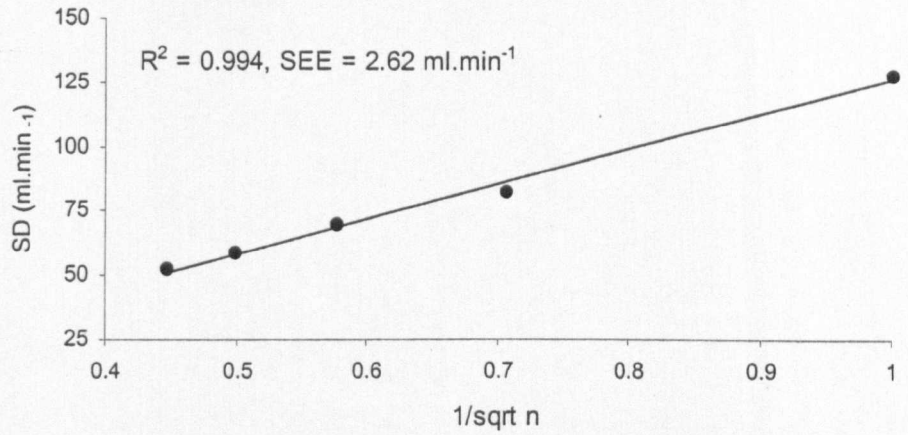

Figure 7.4: **Relationship between the SD of the noise (as calculated from the superimposition of 1, 2, 3, 4 and 5 transitions) and ($1 / \sqrt{n}$) (where n is the number of transitions superimposed) for the severe intensity transitions**

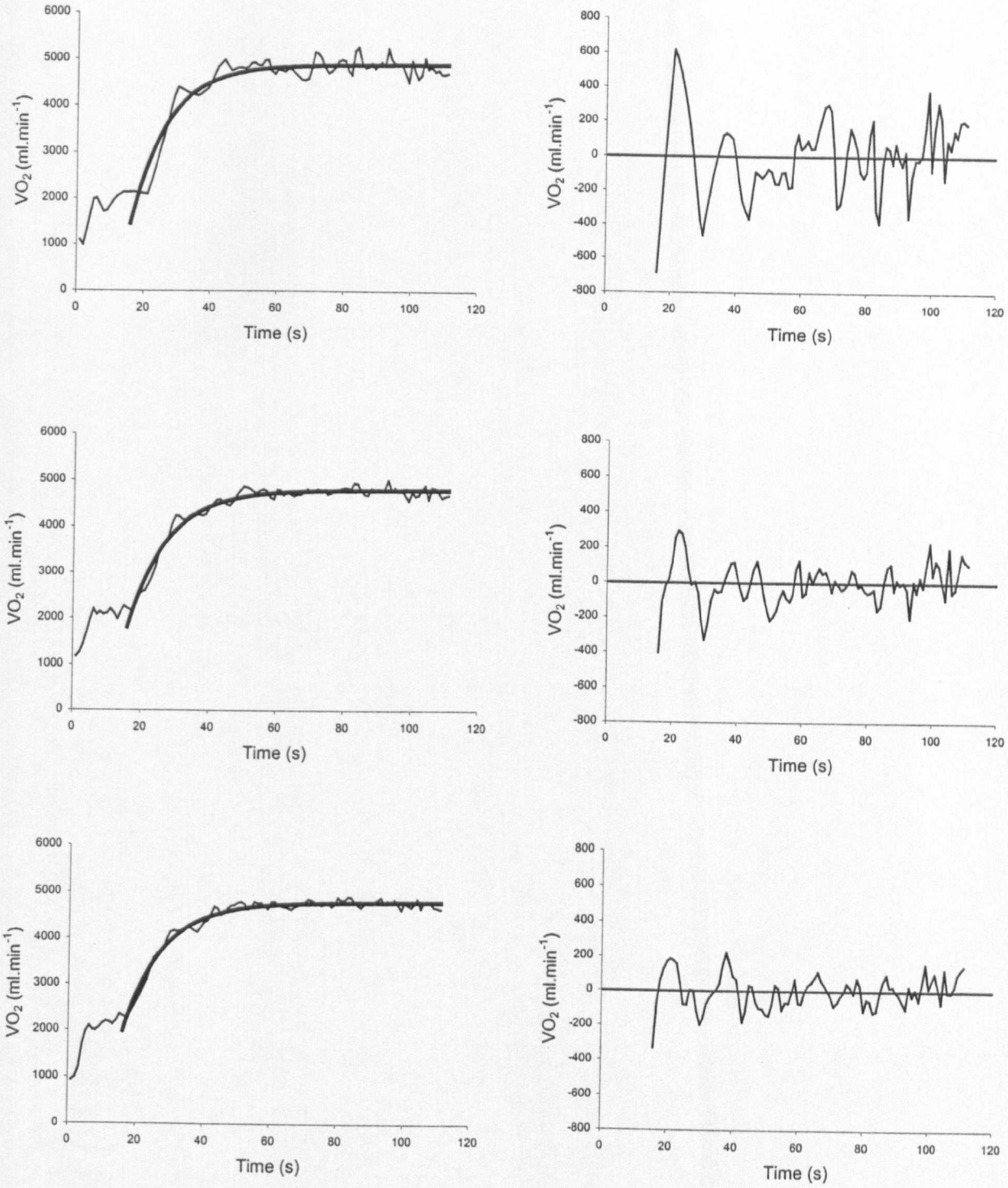

Figure 7.5: **The $\dot{V}O_2$ response (left panels) and underlying noise as shown by the residuals (right panels) around the model (heavy line) for a typical subject. The upper panels are those from a single transition, the middle panels are those averaged across three transitions and the bottom panels are those averaged across five transitions.**

Results from the modelling of the $\dot{V}O_2$ response are contained in Tables 7.4 and 7.5. Parameter estimates are presented for each transition. In addition, means across the transitions are included, along with parameter estimates calculated from the superimposed data sets. Mean values across subjects are also given.

Table 7.4: Parameter estimates for GAIN ($ml.min^{-1}$), τ (s) and δ (s), as calculated using non-linear regression, for moderate intensity exercise. Values are given for individual transitions (single) as well as mean values (average) across 2 , 3, 4, and 5 transitions and values calculated from the data superimposed across 2 , 3, 4, and 5 transitions (super).

Subject			Transition 1	2	3	4	5
		Single	2275	2304	2392	2736	2419
	GAIN	Average		2290	2324	2427	2425
		Super		2290	2323	2425	2424
		Single	9.7	7.2	14.7	11.6	12.1
1	τ	Average		8.5	10.5	10.8	11.1
		Super		8.9	9.5	10.0	10.3
		Single	21.1	25.0	12.1	17.7	16.7
	δ	Average		23.1	19.4	19.0	18.5
		Super		23.3	21.3	20.5	19.9
		Single	2500	2373	2257	2413	2443
	GAIN	Average		2437	2377	2386	2397
		Super		2456	2376	2385	2395
		Single	20.8	16.8	15.2	10.8	19.4
2	τ	Average		18.8	17.6	15.9	16.6
		Super		18.2	16.9	14.9	15.7
		Single	9.3	14.1	21.0	22.5	16.5
	δ	Average		11.7	14.8	16.7	16.7
		Super		12.4	15.9	18.1	17.9
		Single	2407	2333	2337	2248	2395
	GAIN	Average		2370	2359	2331	2344
		Super		2370	2357	2327	2342
		Single	7.1	6.9	12.1	20.7	10.4
3	τ	Average		7.0	8.7	11.7	11.4
		Super		7.0	7.6	10.5	10.5
		Single	22.5	19.7	13.9	13.8	12.6
	δ	Average		21.1	18.7	17.5	16.5
		Super		21.3	20.4	18.3	17.4
		Single	2242	1676	1987	1693	1697
	GAIN	Average		1959	1968	1899	1859
		Super		2058	2035	1950	2361
		Single	13.2	9.8	14.3	10.6	11.2
4	τ	Average		11.5	12.4	12.0	11.8
		Super		11.4	12.2	11.8	13.3
		Single	16.0	22.1	14.1	21.6	21.2
	δ	Average		19.1	17.5	18.5	19.0
		Super		19.2	17.8	18.7	17.0
		Single	**2412**	**2189**	**2210**	**2192**	**2245**
	GAIN	**Average**		**2301**	**2270**	**2251**	**2249**
		Super		**2335**	**2286**	**2262**	**2373**
		Single	**15.5**	**12.6**	**14.2**	**13.2**	**15.1**
MEAN	τ	**Average**		**14.0**	**14.1**	**13.9**	**14.1**
		Super		**13.7**	**13.4**	**13.0**	**13.8**
		Single	**14.3**	**17.5**	**17.5**	**20.1**	**16.7**
	δ	**Average**		**15.9**	**16.5**	**17.4**	**17.2**
		Super		**16.3**	**17.5**	**18.3**	**17.6**

Table 7.5: Parameter estimates for GAIN ($ml.min^{-1}$), τ (s) and δ (s), as calculated using non-linear regression, for severe intensity exercise. Values are given for individual transitions (single) as well as mean values (average) across 2 , 3, 4, and 5 transitions and values calculated from the data superimposed across 2 , 3, 4, and 5 transitions (super).

Subject			Transition 1	2	3	4	5
		Single	3824	3908	5051	3997	4586
	GAIN	Average		3866	4261	4195	4273
		Super		3867	4260	4170	4552
		Single	9.2	12.4	12.3	12.1	12.4
1	τ	Average		10.8	11.3	11.5	11.7
		Super		11.0	11.4	11.6	11.8
		Single	11.6	10.8	8.9	9.7	10.2
	δ	Average		11.2	10.4	10.3	10.2
		Super		11.0	10.2	10.2	10.2
		Single	4211	4119	4124	3974	4222
	GAIN	Average		4165	4151	4107	4130
		Super		4165	4145	4100	4125
		Single	11.0	9.9	14.1	14.0	10.8
2	τ	Average		10.5	11.7	12.3	12.0
		Super		10.6	11.5	11.9	11.7
		Single	13.8	11.9	10.2	7.9	12.2
	δ	Average		12.9	12.0	11.0	11.2
		Super		12.8	12.2	11.4	11.5
		Single	4024	3381	1997	3723	3631
	GAIN	Average		3703	3134	3281	3351
		Super		3694	3795	3777	3748
		Single	12.1	17.5	12.9	13.8	12.4
3	τ	Average		14.8	14.2	14.1	13.7
		Super		14.3	13.8	13.8	13.5
		Single	10.0	7.8	10.4	8.9	11.5
	δ	Average		8.9	9.4	9.3	9.7
		Super		9.1	9.6	9.4	9.9
		Single	3510	3327	3752	3749	3302
	GAIN	Average		3419	3530	3585	3528
		Super		3418	3523	3581	3525
		Single	14.1	13.6	18.9	17	15.5
4	τ	Average		13.9	15.5	15.9	15.8
		Super		13.8	15.4	15.9	15.8
		Single	7.2	9.6	4.9	8.6	9.8
	δ	Average		8.4	7.2	7.6	8.0
		Super		8.5	7.4	7.6	8.1
		Single	**3989**	**3737**	**3499**	**3855**	**3844**
	GAIN	**Average**		**3863**	**3742**	**3770**	**3785**
		Super		**3861**	**3902**	**3890**	**3881**
		Single	**12.1**	**12.7**	**15.0**	**14.7**	**12.4**
MEAN	τ	**Average**		**12.4**	**13.3**	**13.7**	**13.4**
		Super		**12.3**	**13.1**	**13.4**	**13.2**
		Single	**11.2**	**10.3**	**8.9**	**8.3**	**11.4**
	δ	**Average**		**10.8**	**10.2**	**9.7**	**10.0**
		Super		**10.8**	**10.4**	**10.0**	**10.3**

Results of the computer simulations are contained in Table 7.6. Also contained within this table is the resulting value for L as calculated from Equation 7.3. The increasing τ had a minor effect on the confidence limits of the parameter estimates, particularly between the simulations using a τ of 5 and those using a τ of 15 seconds. Whilst L was shown to be dependent on τ, again this difference was minor between the simulations using a τ of 5 and those using a τ of 15 seconds.

Table 7.6: Ninety five percent confidence limits (1.96 x SD) for GAIN ($ml.min^{-1}$), τ (s) and δ (s), derived from computer simulations based on a noise to signal (SD / GAIN) ratio of 0.025, but a differing τ of 5, 15 and 25 seconds. Also given is the derived value for the constant L from Equation 7.3.

Parameter	τ		
	5	15	25
GAIN	8	15	25
τ	0.7	0.9	1.2
δ	0.9	0.6	0.7
L	28.7	34.1	49.8

The paired t-test, using superimposed data across all five transitions from the last 30 seconds of severe intensity running, revealed no difference (P = 0.519) between the average $\dot{V}O_2$ for that time period (4718 ± 453 $ml.min^{-1}$) and the calculated asymptote (4778 ± 590 $ml.min^{-1}$) from non-linear regression.

7.4 Discussion

The primary objective of the present study was to examine the noise that is observed on the underlying $\dot{V}O_2$ response in breath-by-breath data collected during severe intensity running. This noise was demonstrated to be both random and Gaussain (normally distributed).

The random nature of this noise was investigated using the auto-correlation technique (Lamarra et al. 1987; Potter et al. 1999). This process returned similar results to previous research conducted at lower exercise intensities; there appeared to be little difference between the data for moderate and severe exercise intensities and the values were generally low for both exercise intensities.

In most cases the data followed closely the Gaussian curve (see Figure 7.1). There was only one test for which the data were found to be different from a Gaussian distribution (see Figure 7.2) using the χ^2 test. The normality of all 40 breath-by-breath data sets was tested (four subjects, five transitions and two exercise intensities). Only one was found to be significantly different from a Gaussian distribution and this was in moderate intensity running. The χ^2 test itself was conducted at an alpha level of 0.05. Therefore it is not unreasonable to expect the return of one positive result from 40 as the result of chance. Potter et al. (1999) also used this Chi-squared approach, but found 19 from 24 data sets were significantly different from a Gaussian distribution. However, since Potter et al. investigated moderate intensity exercise only and used very long exercise transitions they had far more data points in the steady state to use in the analysis (22 minutes of data for each of the 24 subjects). This was not possible in the present study due to the short duration of the severe intensity running. Therefore, it is likely that this difference between the findings of the present study and those of Potter et al. was due to an abundance of statistical power in the latter study. It would be desirable to have a more powerful test in the severe intensity domain, but the short duration means that there will be a limited number of data points available.

Whilst Lamarra et al. (1987) did not statistically test the distribution of the noise in their subjects, they stated that is was well described by a Gaussian distribution, and supported

this claim with typical plots. Two major assumptions that underlie the equations described in Lamarra et al.'s study are that the data are Gaussian and that they are uncorrelated (random). The results of the present study, unlike those of Potter et al. (1999), showed the data to be both Gaussian and random. It should be realised that the study of Potter et al. (1999) was specific to a child population. That may explain this, and other, differences between that study, the present study, and that of Lamarra et al. (1987).

The two equations presented by Lamarra et al. (1987), that is Equations 7.1 and 7.2, both incorporate the ratio of the level of the noise (SD) to the magnitude of the response (GAIN). It is of course the case that as exercise is performed at increasing exercise intensities, the magnitude (GAIN) of the $\dot{V}O_2$ response must increase (Whipp and Wasserman 1972). The results of the modelling of the present data (Tables 7.4 and 7.5), showed a marked increase in the magnitude of the GAIN at the severe compared to the moderate intensity. Therefore the ratio (SD / GAIN) would be lower for severe intensity exercise, unless the level of noise on the data increased as a function of exercise intensity. The trend was in fact in the opposite direction: there was a tendency for the level of noise to be lower for severe intensity exercise (as shown by Levene's test for equality of variances).

In Chapter 6, it was speculated that 30 was an appropriate value for the constant L, compared to a value of approximately 50 that was apparent from the data of Lamarra et al. (1987) for moderate intensity cycling. Lamarra et al. describe how this constant is dependent, in a complex manner, on τ. They reported a mean τ of 28 seconds, whilst a mean τ of 10.7 seconds was found for severe intensity running in Study 3, explaining this difference in the value for L. Lamarra et al. used computer simulations to assess the impact of the magnitude of τ on the resulting confidence limits of τ and δ estimated from non-linear regression for values of τ of 30, 60 and 90 seconds. The present study adopted a similar approach, using the same underlying response in each case but with values of τ of 5, 15 and 25 seconds. Confidence limits were also calculated for GAIN.

These computer simulations confirmed the relationship between L and τ. When a similar τ to that reported by Lamarra et al. (25 seconds) was used in the simulations, the calculated value for L matched that which was apparent from their data. The simulations also showed

that the suggested value of 30 was a suitable value for L for severe intensity running. The value changed little when τ was increased from 5 to 15 seconds, which represents the range of τ found for severe intensity running in this thesis. This suggests that researchers must investigate the parameter estimates for τ in addition to the SD / GAIN ratio, for a given population or exercise intensity, in order to determine the required number of transitions to return given confidence limits. The range of values for τ reported in Chapter 6 for severe intensity running was 6.9 to 14.6 seconds. The simulations reported in the present Chapter demonstrate that this difference would have little effect on the resulting confidence limits, or the value of 30 proposed for L.

In contrast to previous research (Lamarra et al. 1987; Potter et al.1999), confidence limits were also calculated for the GAIN parameter, as well as for τ and δ. A primary focus of this thesis was the apparent plateau of $\dot{V}O_2$ below its maximum; therefore the confidence limits of the GAIN parameter were pertinent. Confidence limits for the GAIN, as were those for τ and δ, were found to be narrow. All parameters demonstrated widening confidence limits as the SD / GAIN ratio was increased. The present study also supported the assertion of Lamarra et al. (1987) that confidence limits for τ and δ would be virtually identical. That confidence limits for these two parameters were wider than $\pm$ 1 second when the SD / GAIN ratio was 0.05 shows the need for two transitions for subjects with this level of noise. The confidence limits for GAIN, even when SD / GAIN was 0.05, represent only $\pm$ 0.5 $ml.kg^{-1}min^{-1}$ for a typical 70 kg subject.

Equation 7.2, incorporating this value of 30 for L, was used to calculate the required number of transitions (n) to give confidence limits of $\pm$ 1 second for τ and δ. Using the values for SD and GAIN that had been used in the computer simulations to produce ratios of 0.025 and 0.05, the desired number of transitions could then be calculated. It is important though that the effect of τ is again considered to quantify the possible error in the calculation of (n) since the value of τ will determine how far L deviates from the assumed value of 30. In the 'best case' scenario, namely a low level of noise and a short τ (a SD / ratio of 0.025 and an L of 28.1 seconds) (n) would equal 0.50 (from equation 7.2), indicating that one transition would be sufficient for a confidence limit for τ of $\pm$ 1 second. In the 'worst case' scenario, namely a high level of noise and a long τ (a SD / ratio of 0.05 and L of 34.1 seconds) n would however equal 2.86. However, such a worst scenario is

likely to be encountered only rarely, and would only occur with both a high level of noise and a slow $\dot{V}O_2$ response. This represents a scenario that is 'worse' than any encountered in either Study 3 or the present study. Furthermore, it has been clearly demonstrated that the confidence limits will narrow as a function of $1/\sqrt{n}$ (Figure 7.4), and therefore even in this 'worst case' scenario, two repeated transitions would give confidence limits of ± 1.2 seconds. The analysis of the present study shows that two transitions will be sufficient for further analysis of the $\dot{V}O_2$ response to severe intensity running.

It is normal for researchers modelling $\dot{V}O_2$ kinetics to interpolate breath-by-breath data to produce a value for every second. This procedure serves two purposes. Firstly the process of interpolation itself serves to reduce the level of noise (Lamarra et al. 1987), and secondly interpolating to one value per second allows data to be averaged across multiple transitions. As the confidence limits are dependent on the SD / GAIN ratio multiple transitions would reduce them by reducing the SD of the noise. This is similar to the narrowing of the confidence limits that occurs in the severe intensity due to the increased magnitude of the GAIN. Many transitions are often used for moderate intensity exercise. Indeed Lamarra et al. (1987) note that eight transitions may be needed and this number has in fact been used (Whipp et al. 1982). However, for the heavy and severe intensity domains, researchers have tended to use fewer transitions. Hughson et al. (2000) for example used two transitions. The assumption that higher exercise intensities will require fewer transitions is consistent throughout the literature, although it has never been established whether this is the case or what confidence limits might be assumed.

Furthermore, the assumption, inherent in Equation 7.2, that the noise would be reduced as a function of the number of transitions, (i.e. the SD would decrease as a function of $(1/\sqrt{n})$), was also shown to be valid. Linear regression produced an R^2 of close to 1 for both moderate and severe intensity exercise. This is further support for the 'effective' Gaussian nature of the noise even in second-to-second data.

Curiously, whilst Lamarra et al. (1987) used second-to-second data to model the response, and therefore to calculate the magnitude of the GAIN, the SD of the noise was calculated from breath-by-breath data. As has already been discussed, the process of interpolation will itself reduce the noise and therefore the ratio of noise to signal would more logically

be based on the SD from the second-to-second data. The present study used this latter approach and found the equations to be valid despite this change.

Another significant finding of the present study was that the single exponential model was shown to adequately describe the plateau in the $\dot{V}O_2$ response to severe intensity running. The present study showed that the calculated asymptote was no different from the average $\dot{V}O_2$ over the last 30 seconds of exercise (from the same 5 transitions).

It has been established that the SD / GAIN ratio has a major effect on the resulting confidence limits of the parameter estimates. However, whilst researchers routinely report individual parameter estimates including GAIN, the SD (i.e. level of noise) is not routinely included. Such an inclusion would perhaps give greater insight into the quality of these parameter estimates. This may be particularly relevant for three-component models where the magnitude of the GAIN of phases-1 and –3 may be small.

In summary, the noise on the $\dot{V}O_2$ data from severe intensity running was shown to be both random and Gaussain. This and the other assumptions surrounding the equations proposed by Lamarra et al. (1987) were tested and shown to be valid for severe intensity running. These equations were used to calculate that data should be averaged over two transitions to ensure confidence limits for the time parameters τ and δ of $\pm$ 1.2 seconds. Therefore it was shown that it would be necessary to perform two transitions of severe intensity running in the following studies. That there was no difference between the actual plateau in $\dot{V}O_2$ and the calculated parameter estimate for the asymptote of the exponential response suggests that the mono-exponential model used was an appropriate model to describe the $\dot{V}O_2$ response to exercise in this intensity domain.

Studies 5 and 6 (Chapter 8 and 9) investigated the cause of the shortfall in the $\dot{V}O_2$ response to severe intensity running. It was vital before commencing these studies that the modelling technique used was tested and found to be robust. Furthermore, it was now known that two repeated transitions at this intensity would be sufficient to return suitably narrow confidence limits for each of the parameter estimates. Studies 5 and 6 offered an opportunity to model and further investigate the $\dot{V}O_2$ response using these two averaged transitions.

CHAPTER 8

STUDY 5: THE EFFECT OF PRIOR MODERATE OR HEAVY INTENSITY RUNNING ON THE $\dot{V}O_2$ RESPONSE TO SEVERE INTENSITY RUNNING

8.1 Introduction

In previous chapters, it has been consistently shown that for exhaustive severe intensity running lasting approximately 2 minutes, $\dot{V}O_2$ does not tend towards $\dot{V}O_2$max or the $\dot{V}O_2$ required. Rather $\dot{V}O_2$ reaches a plateau some way below $\dot{V}O_2$max, as has been shown by Spencer et al. (1996) and Spencer and Gastin (2001), who investigated the aerobic and anaerobic energy contributions to 800 m running.

It has been shown in cycling that prior exercise at an intensity above the AT, that is heavy intensity exercise, accelerates the $\dot{V}O_2$ kinetics of a subsequent bout of exercise performed in the heavy intensity domain (Gausche et al. 1989; Gerbino et al. 1996; MacDonald et al. 1997). The suggested mechanisms for the faster $\dot{V}O_2$ response are a metabolite driven vasodilation that improves local muscle perfusion and an acidosis mediated Bohr effect that releases more O_2 at a constant PO_2. That prior moderate intensity exercise does not affect the $\dot{V}O_2$ kinetics of a subsequent exercise bout has been taken as evidence that the effect seen for prior heavy intensity exercise is due to the metabolic acidosis resulting from exercise above the AT (Gerbino et al. 1996). Using Doppler ultrasound to monitor blood flow, MacDonald et al. (2001) demonstrated an increased muscle blood flow, in conjunction with a decreased venous pH, in a second bout of heavy intensity forearm exercise.

Two recent studies (Burnley et al. 2000; Bearden and Moffatt 2001c) have revisited the findings of Gerbino et al. (1996). Both suggested that as that study used a simple mono-exponential model to evaluate the $\dot{V}O_2$ kinetics, the results did not necessarily represent an acceleration of the primary (Phase-2) kinetics. When the response was modelled using a three-component model to evaluate the different phases separately no acceleration in the phase-2 $\dot{V}O_2$ kinetics was observed. Rather, it was shown that the apparent acceleration of $\dot{V}O_2$ kinetics is due to a reduction in the magnitude of the slow component (Burnley et al.

2000). Burnley et al. concluded that the acidosis mediated improvements in O_2 delivery and uptake had no effect on $\dot{V}O_2$ kinetics during the first 2 minutes of exercise, whereas Bearden and Moffatt found that the phase-2 response was altered.

There exists then some controversy over the effect of prior supra AT exercise on the $\dot{V}O_2$ kinetics of a following bout of exercise, with the conclusions drawn being dependent of the modelling strategy employed (Jones et al. 2001; Hughson et al. 2001). The effect of prior exercise has never been investigated in the domain of severe intensity exercise. This is surprising given that when the exercise duration is short, the phase-2 response may represent the complete $\dot{V}O_2$ response, as the slow component has been shown not to become manifest until approximately 3 minutes after the onset of exercise (Gaesser and Poole 1996). Furthermore, it appears likely that for exhaustive severe intensity exercise of a short duration $\dot{V}O_2$ kinetics may be O_2 delivery limited (Hughson et al. 2000). If Hughson's assumption is correct and the Phase-2 plateau does represent an O_2 delivery limitation, metabolic acidosis (caused for example by prior heavy intensity exercise) would be expected to increase the asymptotic $\dot{V}O_2$ for this phase. Severe intensity exercise might offer an opportunity to evaluate these acidosis-mediated effects in a domain where O_2 delivery is likely to be limiting and a single exponential may be used to adequately describe the response.

This study evaluated the effects of prior sub and supra AT exercise on the $\dot{V}O_2$ kinetics of a severe intensity run at a speed that would elicit exhaustion in approximately 2 minutes. The primary aim was to test the hypothesis that an increased metabolic acidosis might improve O_2 delivery and thus increase the asymptotic $\dot{V}O_2$. The study also provided the opportunity to confirm the response established in Chapter 6 using data averaged over two transitions. The appropriateness of removing of the initial 15 seconds of data prior to modelling the phase-2 response was also analysed by using objective criteria to determine the end of the phase-1 response.

8.2 Method

8.2.1 Subjects

Ten male volunteers (mean ± SD: age 23.6 ± 4.3 years, height 1.78 ± 0.04 m, mass 70.9 ± 5.7 kg, $\dot{V}O_2$peak 62.4 ± 5.4 ml.kg^{-1}min^{-1}) participated in the study. Subjects were all trained distance- and middle-distance runners.

8.2.2 Study design

The study required the subjects to attend the laboratory on six separate days. On the first visit subjects performed an exhaustive ramp test to determine $\dot{V}O_2$peak and speed at the AT. On the next four visits subjects performed a 6 minute warm up, followed by 6 minutes rest, and finally an exhaustive run at an intensity severe enough to result in exhaustion in approximately 2 minutes. The speed for the severe intensity run was estimated from the peak speed achieved during the ramp test. If on the first occasion this run proved to be too long or short, the speed was adjusted and subjects attended a further four sessions in which the adjusted speed was used. The exhaustive severe intensity run was always performed at the same speed but the warm up was performed at a speed that was equivalent to either 90% AT or 50% of the difference between AT and $\dot{V}O_2$max (50%Δ). Each protocol was performed twice, to ensure confidence limits of ± 1.2 seconds for τ and δ (for the severe intensity run), and the order in which the subjects performed the protocols was counterbalanced.

On the sixth and final visit to the laboratory subjects repeated the exhaustive ramp test. The $\dot{V}O_2$peak was then averaged across these two tests, for comparison against that achieved during the square wave tests, to control for order effects.

8.2.3 Test Protocols

For the ramp test the speed was continually increased by 0.1 km.h^{-1} every 5 seconds (1.2 km.h^{-1}.min^{-1}) until the subject was unable to maintain the required speed. The starting

speed was estimated depending on the fitness of the subject to elicit exhaustion in approximately 12 minutes.

The square wave tests began after the subject had been standing on the treadmill and breathing through the appropriate apparatus for 2 minutes to determine a baseline $\dot{V}O_2$. The warm-up (at either 90% AT or 50%Δ) lasted 6 minutes and was followed by 6 minutes rest before the severe intensity run began. Subjects remained on the treadmill and connected to the gas analysis equipment throughout the 6 minute rest period. They were however permitted to perform stretches during this period. During the final minute of the rest period subjects were instructed to stand astride the treadmill belt while it was accelerated to the required speed. Gas analysis continued throughout the rest period so that a second baseline $\dot{V}O_2$ could be established.

8.2.4 Expirate analysis

Expired gases were analysed on-line using the QP9000.

8.2.5 Data Analysis

Breath-by-breath $\dot{V}O_2$ data were first interpolated to second-to-second data. Data from the ramp tests were used to determine $\dot{V}O_2$peak. The AT was also determined from the ramp test data, as was the $\dot{V}O_2$- running speed relationship for each subject, which was used to calculate speeds corresponding to 90% AT and 50% Δ.

Second-to-second $\dot{V}O_2$ data were averaged, for each second, across the two transitions for the square wave tests. Since the repeated exhaustive runs varied slightly in duration only time points present in both transitions were averaged, and as in previous chapters the first 15 seconds of data were removed. Non-linear regression was used to model the $\dot{V}O_2$ from the severe intensity transitions, assuming a single exponential response:

$$\dot{V}O_2(t) = \text{baseline} + \text{GAIN} \times (1-e^{-(t-\delta)/\tau}) \quad (8.1)$$

Parameter estimates were produced, through this iterative process, for GAIN, τ and δ. The overall speed of the $\dot{V}O_2$ response was evaluated using the mean response time (MRT), which was calculated as $\tau + \delta$ (Lamarra et al. 1987). Baseline $\dot{V}O_2$ was calculated as the average $\dot{V}O_2$ for the 60 second period immediately preceding the transition.

The initial phase of $\dot{V}O_2$ kinetics (phase-1) was evaluated by determining the duration of this phase. The end of this phase was determined according to criteria outlined by Mettauer et al. (2000). Phase-1 was considered to have ended when there was a simultaneous:

a) decrease in the end-tidal O_2 concentration ($F_{ET}O_2$)
b) increase in the end tidal CO_2 concentration ($F_{ET}CO_2$)
c) sudden decrease in RER
d) end of the small initial plateau in $\dot{V}O_2$.

These criteria are shown for a typical subject in Figure 8.1. The process was performed using data averaged across two transitions.

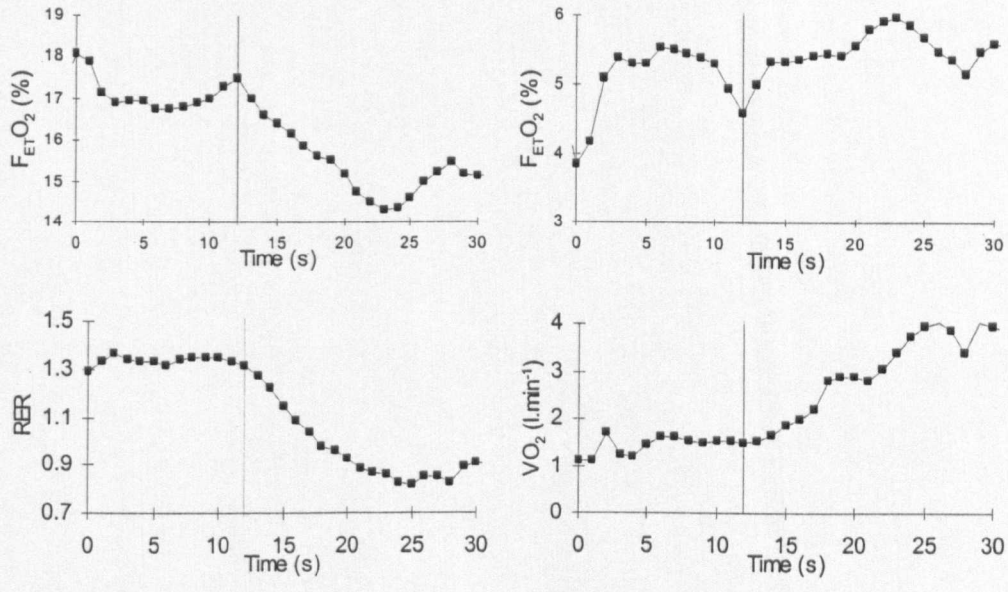

Figure 8.1: Criteria used to identify the end of phase-1 $\dot{V}O_2$ kinetics for a typical subject performing severe intensity running. The responses of $F_{ET}O_2$, $F_{ET}CO_2$, RER and $\dot{V}O_2$ are shown and the vertical line shows where phase-1 was considered to have ended (in this case at 12 s).

Comparisons (for $\dot{V}O_2$peak HRpeak and post exercise blood lactate concentration) were made between the ramp and the two severe intensity conditions using a one-way repeated measures ANOVA (protocol). Significant differences were investigated post hoc using Bonferonni corrected t-tests. The P values given for this analysis are the corrected (Bonferonni) values. In instances where this corrected P value was greater than 1, this P value is not given. Differences in baseline $\dot{V}O_2$, phase-1 duration, GAIN, asymptote, τ ,δ and MRT between the two severe intensity conditions were investigated using paired t-tests. The alpha level was set at 0.05 for all tests and data are mean ± SD unless otherwise stated.

8.3 Results

As in previous chapters, $\dot{V}O_2$ was shown to plateau during the severe intensity runs below the maximum achieved during the ramp test. Peak values from the ramp test, along with values from the two severe intensity conditions, are contained in Table 8.1. A main effect for protocol was found for $\dot{V}O_2$peak ($P < 0.001$). It was found that $\dot{V}O_2$peak was lower in both of the severe intensity conditions than the ramp test ($P<0.001$ in both instances). There was no difference in $\dot{V}O_2$peak between the two severe intensity conditions.

A main effect was also found for HRpeak ($P < 0.001$). It was shown that HRpeak was higher in the ramp test than the severe intensity run preceded by moderate intensity running ($P < 0.001$) and higher than the severe intensity run preceded by heavy intensity running ($P = 0.041$). The severe intensity run preceded by heavy intensity running produced a higher HRpeak than that preceded by moderate intensity running ($P = 0.03$). There was no main effect for post exercise blood lactate concentration ($P = 0.253$).

Table 8.1: Peak values (averages of the two transitions) for $\dot{V}O_2$, HR and lactate from the ramp test, and the very severe intensity runs (preceded by both moderate (90% AT) and heavy (50% Δ) exercise).

	$\dot{V}O_2$ ($ml.min^{-1}$)	$\dot{V}O_2$ (% ramp)	HR ($b.min^{-1}$)	lactate (Mm)
Ramp	4421 ± 450	100	189 ± 9	6.6 ± 1.5
Severe (following moderate)	4025 ± 459	91.0 ± 3.2	181 ± 9	7.1 ± 1.4
Severe (following heavy)	4066 ± 428	92.0 ± 3.9	186 ± 7	7.1 ± 1.5

The $\dot{V}O_2$ responses (averaged over 2 transitions) for a typical subject to both the warm-up and the exhaustive severe intensity run under the two warm up conditions are shown in Figure 8.2. The response to the severe intensity run together with the best fit model is shown in Figure 8.3. A clear but submaximal plateau was shown in both severe intensity runs. Results of the modelling confirmed this response with $\dot{V}O_2$ achieving an asymptote at 89 ± 4 % $\dot{V}O_2$peak when preceded by moderate intensity running and 90% ± 4 % $\dot{V}O_2$peak when preceded by heavy intensity running (P = 0.19). Figure 8.4 shows the mean response across all subjects.

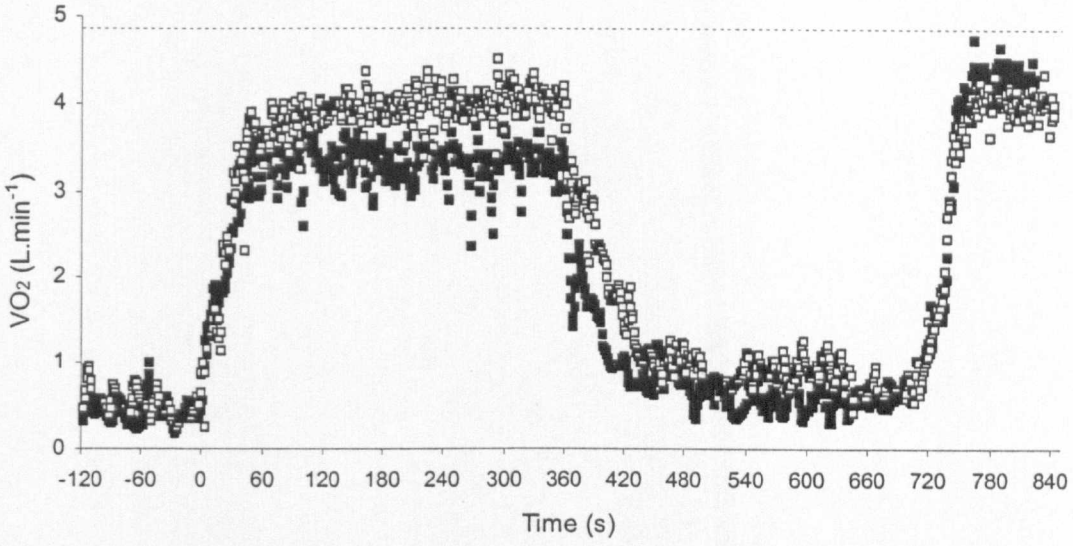

Figure 8.2: $\dot{V}O_2$ **response of a typical subject to exhaustive severe intensity running when preceded by either 6 minutes of moderate intensity running and 6 minutes rest (closed symbols) or 6 minutes of heavy intensity running and 6 minutes rest (open symbols). Each data set shows the response to the entire exercise protocol and is an average of two transitions. Also shown is** $\dot{V}O_2$**peak (broken line), averaged over the two ramp tests.**

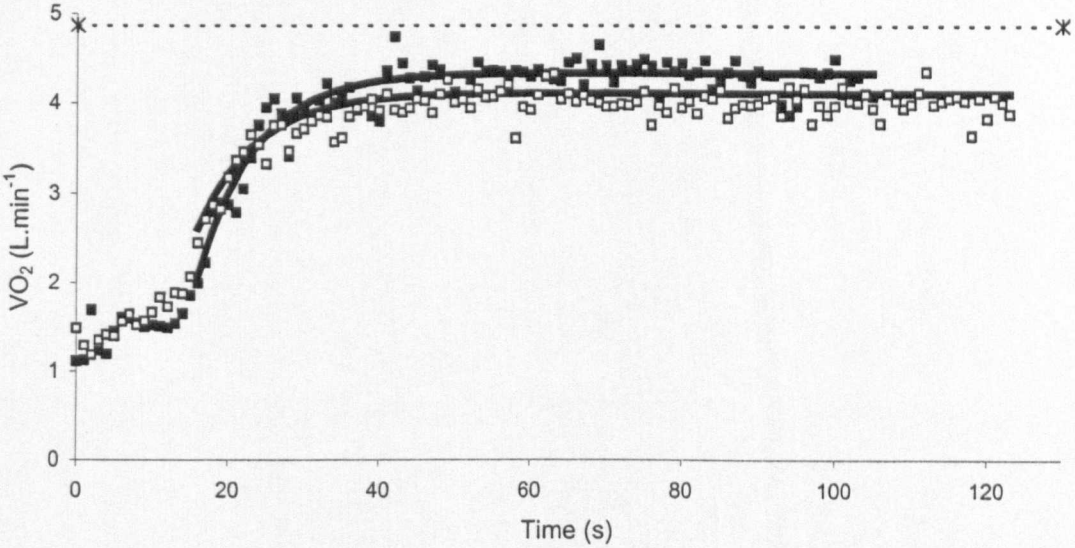

Figure 8.3: $\dot{V}O_2$ **response of the same representative subject to exhaustive severe intensity running when preceded by either 6 minutes of moderate intensity running and 6 minutes rest (closed symbols), or 6 minutes of heavy intensity running and 6 minutes rest (open symbols). Each data set shows the response just the severe intensity run and is an average of two transitions. Also shown is** $\dot{V}O_2$**peak (broken line), averaged over the two ramp tests and the best fit model for each data set (heavy lines).**

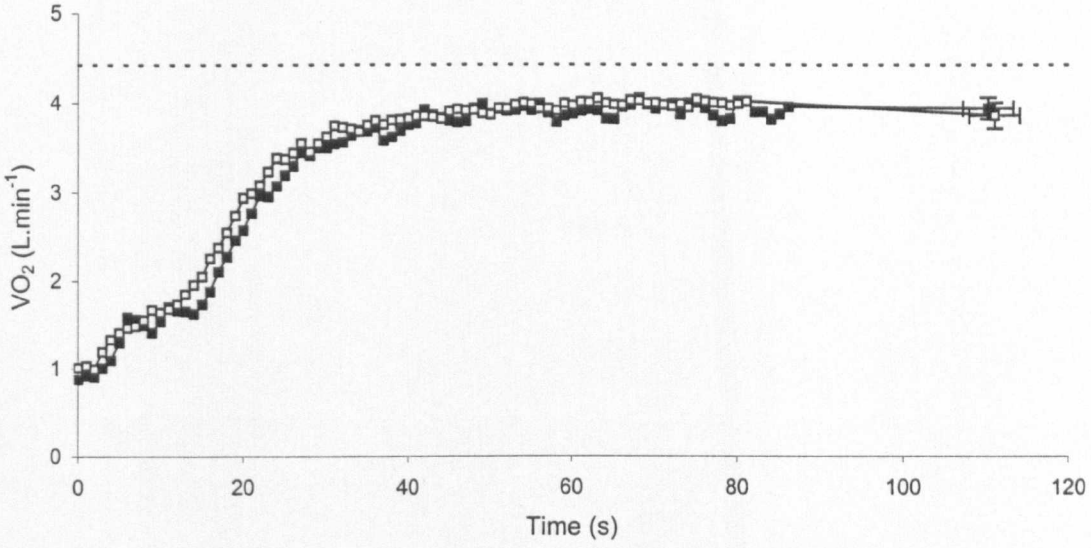

Figure 8.4: Mean $\dot{V}O_2$ response across all subjects to exhaustive severe intensity running when preceded by either 6 minutes of moderate intensity running and 6 minutes rest (closed symbols) or 6 minutes of heavy intensity running and 6 minutes rest (open symbols). Each data set shows the response just the severe intensity run. Also shown is mean $\dot{V}O_2$peak (broken line)

Note: Error bars represent SEM. For clarity error bars are omitted from all but the final data points.

The differing warm-up affected neither the $\dot{V}O_2$ attained in the severe intensity run (see Table 8.1) nor the duration of the exhaustive test itself. The test duration with prior moderate intensity exercise was 110.2 ± 9.7 seconds, whilst the duration with prior heavy intensity exercise was 111.0 ± 15.2 seconds (P = 0.81).

The results of the modelling by non-linear regression are contained in Table 8.2. Firstly the duration of the first phase of $\dot{V}O_2$ kinetics is given. This was shorter when the severe exercise intensity exercise was preceded by heavy exercise than when it was proceeded by moderate exercise.

In addition to the GAIN parameter estimate and the baseline $\dot{V}O_2$, also given is the value for the asymptote (GAIN + baseline). This was included as it is represents the asymptote of $\dot{V}O_2$ irrespective of any fluctuations in the baseline value. A significant difference was found between the two severe conditions for GAIN and baseline. However, the asymptote was not different between the two conditions.

Table 8.2 also contains values for the other two important parameter estimates, τ and δ, as well as MRT, which is the sum of the two. No difference was found between the two severe intensity conditions for τ. However, a difference was found for δ. When the speed of the $\dot{V}O_2$ kinetics were assessed as a whole by MRT, they were found to be faster following prior heavy intensity exercise.

Table 8.2: Calculated parameter estimates and test results from modelling the severe intensity runs under both warm-up conditions. In addition to the calculated parameter estimates (GAIN, τ and δ), baseline $\dot{V}O_2$, asymptote (GAIN + baseline $\dot{V}O_2$), MRT ($\tau + \delta$), and Phase-1 duration are also included. The final column contains P values from the respective difference test.

	Severe (following moderate)	Severe (following heavy)	Difference	P value (paired t-test)
Phase-1 (s)	14.1 ± 2.0	11.1 ± 1.9	3.0 ± 2.5	0.004
Baseline $\dot{V}O_2$ ($ml.min^{-1}$)	734 ± 131	652 ± 119	82 ± 56	0.001
GAIN ($ml.min^{-1}$)	3188 ± 408	3341 ± 390	-154 ± 164	0.016
Asymptote ($ml.min^{-1}$)	3922 ± 439	3993 ± 424	72 ± 174	0.226
τ (s)	8.8 ± 2.5	9.6 ± 3.5	-0.8 ± 1.9	0.222
δ (s)	12.1 ± 1.1	9.3 ± 3.9	2.8 ± 3.4	0.026
MRT (s)	20.9 ± 1.9	18.9 ± 1.0	2.1 ± 2.4	0.026

8.4 Discussion

The principal finding from the present study was that prior supra AT running did not increase the level at which $\dot{V}O_2$ reached a plateau in a subsequent severe intensity run, designed to result in exhaustion in approximately 2 minutes. There were no differences in either $\dot{V}O_2$peak or the asymptotic $\dot{V}O_2$ estimated from non-linear regression between severe intensity exercise preceded by moderate intensity exercise and severe intensity exercise preceded by heavy intensity exercise. Whilst the effects of prior exercise have not been investigated in this intensity domain, previous attempts to model the $\dot{V}O_2$ response to severe intensity exercise have pointed to a potential O_2 delivery limitation (Hughson et al. 2000). However, the present study found that potential acidosis mediated benefits to O_2 delivery did not improve the percentage of aerobic power the subjects were able to use during exhaustive severe intensity running.

The analysis did produce significant differences in both GAIN and baseline $\dot{V}O_2$. Differences in GAIN can be explained readily by the difference in baseline, since the final asymptote was not different between conditions. The difference in baseline $\dot{V}O_2$ is perhaps less easily explained, particularly as the baseline following moderate exercise was shown to be higher, in contrast to previous work using heavy intensity exercise preceded by both moderate and heavy intensity exercise (Burnley et al. 2000). The nature of the present study, being at such severe intensities and using treadmill running, necessitated subjects being given the freedom to stretch as required between the warm up and the severe intensity run (although they did remain on the treadmill bed and connected to the respiratory equipment). Such a requirement may lead to greater fluctuations in the respiratory data than merely standing stationary as would be possible at lower exercise intensities. The less severe warm-up intensity may have resulted in the subjects feeling the need to perform more and perhaps more vigorous stretches; this could explain the slightly elevated baseline $\dot{V}O_2$. The magnitude of the response was therefore examined using the asymptote (baseline $\dot{V}O_2$ + GAIN). Other investigators have also recommended the use of this parameter rather than the baseline dependent GAIN (Hughson et al. 2001).

Research into the effect of prior supra AT exercise on the $\dot{V}O_2$ response to heavy intensity cycling has proved equivocal. For example, there are reports that the amplitude of the

phase-2 response is both increased (Bearden and Moffatt 2001c) and unchanged (Burnley et al. 2000). Although this latter study has been criticised for separating the amplitude of the phase-2 response from the baseline $\dot{V}O_2$ despite an elevation of this baseline in the repeated heavy exercise condition (Hughson et al. 2001). Similarly the magnitude of the overall response (i.e. the sum of all the GAINS) has been shown to be both unchanged (Bearden and Moffatt 2001c) and decreased (Burnley et al. 2000).

A similar pattern was shown for severe intensity running (above MLSS) to that which had been previously shown for heavy intensity cycling, in that no difference was found in the time constant (τ) for the primary kinetics (phase-2) between the two conditions (Burnley et al. 2000; Beardon and Moffatt 2001c). It should be recognised though, that in these studies a slow component was modelled and so phase-2 represented only part of the overall response, whereas in exhaustive exercise of a short duration (as in this thesis) a slow component should not be manifest.

Whilst change was not established in the point at which $\dot{V}O_2$ reached its plateau, nor in τ for phase-2, differences were found in the $\dot{V}O_2$ response to severe intensity exercise following a supra AT warm-up. Both the delay (δ) in the onset of the single exponential used to model the response and the calculated duration of the initial phase of $\dot{V}O_2$ kinetics (phase-1), were found to be significantly shorter when the severe intensity run was preceded by heavy intensity exercise. It is logical to expect this difference to be present in both parameters since δ is representative of (though not necessarily equal to) the duration of this phase, where increases in $\dot{V}O_2$ are thought to be primarily due to increases in venous return (Wasserman et al. 1974). It is also possible that acidosis mediated vasodilation might enhance this initial response and therefore decrease the delay in the onset of the primary (phase-2) $\dot{V}O_2$ response. Such a change in delay, or in the duration of phase-1, has not been reported for heavy exercise preceded by heavy exercise (Gerbino et al. 1996; Burnley et al. 2000; Bearden and Moffatt 2001c). However, Burnley et al. (2000) attempted to model the phase-1 kinetics and this was done by a "goodness of fit" method rather than use any objective criteria to determine the end of this phase. There are few data points from which to model this phase (Grassi et al. 1996) and the phase-1 response has never been demonstrated to be exponential in nature (Whipp and Ozyener 1998). Since

Burnley et al. treated phase-1 as an exponential rather than a (pure) delay as in the present study, this may explain why Burnley et al. (2000) would not have detected such a change.

The end of the first phase of the $\dot{V}O_2$ response was determined according to the criteria outlined by Mettauer et al. (2000). The responses of O_2 and CO_2 concentrations at the onset of exercise represent the influence of CO_2 storage within the muscle (Whipp and Ozyener 1998), and an abrupt change in these concentrations at the mouth can be used to signal the end of the muscle to lung transit delay. The first phase of the $\dot{V}O_2$ response was not modelled in this thesis because of the technical and conceptual difficulties inherent in such a process. However, the present study gave considerable support to the approach that was used, namely to exclude the first 15 seconds of data from the modelling process and incorporate a delay (δ) into the model. The difficulty with the exclusion of any data from such a process is that ideally the data set used to model phase-2 would contain no data points from phase-1 and all of the data points from phase-2. In reality breath-by-breath noise makes it impossible to achieve such a result. The duration calculated for phase-1 in the present study was 12.6 ± 2.4 seconds (average across both conditions). This would suggest that 15 seconds is an appropriate time period to remove from the data set. The modelling strategy adopted appears to be robust as when the phase-1 data were removed on an individual basis (using the criteria outlined by Mettauer et al. 2000) and the data re-modelled no differences were found for any parameter estimate between this and the original modelling procedure ($P > 0.05$ in all cases).

Whilst there was no difference in the underlying τ, the effect of a significant shortening of δ was sufficient to produce a significant acceleration of the $\dot{V}O_2$ response. This was assessed in the present study as MRT and is shown in figure 8.4. The rising phase-2 response for the severe intensity exercise preceded by heavy intensity exercise is to the left of (i.e. quicker than) the phase-2 response for severe intensity exercise preceded by moderate intensity exercise. The MRT represents the time taken to achieve 63% of the overall (modelled) response. The mean value of ~20 seconds highlights the speed of the $\dot{V}O_2$ response in this severe intensity running. This is a much faster response than has been reported for other exercise intensities. It should be emphasised at this point that $\dot{V}O_2$ reaches its asymptote well below either $\dot{V}O_2$ required or $\dot{V}O_2$max. What has been shown

then is a response that is much faster but also much reduced in comparison with that which is predicted in the literature (Whipp 1994a).

The $\dot{V}O_2$ data were modelled as a single exponential with a delay phase (δ). This is in contrast to the only previous attempt to model data in exercise of this intensity (Hughson et al. 2000). Hughson applied the same three-component model to severe exercise intensities of 96% and 125% $\dot{V}O_2$max. Outlined above (and elsewhere; see Section 3.10 (Chapter 3)) are reasons why it is inadvisable to model the phase-1 response in this way. The third and slow phase of $\dot{V}O_2$ kinetics has been shown to become manifest after approximately 3 minutes of exercise (after a delay of approximately 120 s) (Gaesser and Poole 1996). It is illogical therefore to apply models used in heavy exercise to exercise of such a high intensity and short duration. Hughson et al. (2000) also produced (and favoured) a semi-logarithmic model to analyse the $\dot{V}O_2$ response, based on the assumption that the response must tend to the $\dot{V}O_2$ required. This approach was not adopted in the present study because no evidence has been found in this thesis to suggest that $\dot{V}O_2$ tend to a higher rate than the asymptote of the model used. Attempts to model the early part of the data and residual plots supported this view.

In the present study, subjects were connected to the gas analysis equipment throughout the entire protocol (lasting approximately 16 minutes). There was therefore a large amount of data not included in the present analysis. The recovery (off-kinetics) response was not analysed and neither was the response to moderate or heavy exercise. The warm-up data were not modelled for two reasons. Firstly, the possible need for a two-component model in the domain of heavy intensity exercise, and secondly that the severe intensity exercise (unlike the moderate and heavy intensity exercise) had been preceded by a warm-up. Therefore comparisons between the exercise intensities would be problematic. It was considered unethical to conduct severe intensity transitions, as examined in the present thesis, without the opportunity to warm-up and stretch. It was not a principle aim of the present thesis to make comparisons between all of the exercise intensities and the $\dot{V}O_2$ response to both moderate and heavy intensity exercise is well documented. The modelling of recovery kinetics was also beyond the scope of this thesis, and was likely to be affected by stretching.

In summary, prior supra –AT exercise did not increase the level at which $\dot{V}O_2$ plateaued in a subsequent severe intensity run. The assumed O_2 delivery benefits derived from this prior exercise did have the effect of speeding $\dot{V}O_2$ kinetics but did not improve either $\dot{V}O_2$peak or test duration. The speeding of kinetics was manifest in the initial phase (phase-1) rather than the primary (phase-2) kinetics themselves; that is the response was unchanged but began earlier. The mechanisms involved in this alteration of the response do not seem to be the underlying mechanisms causing the sub maximal $\dot{V}O_2$ plateau in severe intensity exercise. However, it is clear from Study 3 (Chapter 6) that the magnitude of the shortfall in the $\dot{V}O_2$ response is linked to the aerobic capability of the individual. The final study (Chapter 9) investigated the differences between sprint and endurance trained athletes in order to further investigate this relationship. It was hoped that this might give a greater insight into the underlying physiology of the response.

CHAPTER 9

STUDY 6: THE $\dot{V}O_2$ RESPONSE OF SPRINT AND ENDURANCE TRAINED RUNNERS TO SEVERE INTENSITY RUNNING

9.1 Introduction

Endurance training alters the $\dot{V}O_2$ response to both moderate and heavy intensity exercise (Hagberg 1980; Billat 2000b). Such an effect has been shown in both sedentary and moderately trained individuals (Berry and Moritani 1985; Norris and Peterson 1997). A shortened phase-2 τ for moderate intensity exercise (Norris and Peterson 1997) and a decreased slow component for heavy intensity exercise (Casaburi et al. 1987; Poole et al. 1990) have been reported following endurance training. Highly trained endurance athletes have been shown to exhibit a $\dot{V}O_2$ response that differs from less trained individuals, categorised by a decreased τ for phase-2 and a reduced slow component (Billat 2000b). Furthermore, individuals with a high $\dot{V}O_2$max have been shown to exhibit faster kinetics than individuals with lesser aerobic capabilities (Whipp and Wasserman 1972; Powers et al 1985).

Study 3 (Chapter 6) showed a link between the aerobic capability of the individual and the shortfall in the $\dot{V}O_2$ response to severe intensity running. The effect of endurance training status has never been investigated in the severe intensity domain, however. It is important therefore, in attempting to understand the nature of the $\dot{V}O_2$ response to severe intensity running, to rectify this omission.

Edwards et al. (1998) investigated whether a method of assessing $\dot{V}O_2$ kinetics could distinguish between the responses of elite endurance and sprint trained runners. The endurance runners were competing at distances of 3000-10 000 m and the sprint runners over 100 – 400 m. Edwards et al. found that $\dot{V}O_2$ kinetics were faster in endurance runners compared to sprint runners. However, this study used a pseudo random binary sequencing (PRBS) technique to evaluate the speed of the $\dot{V}O_2$ response to sinusoidal exercise in a single test. This technique does not allow the response to fully develop, and assumes first order linear kinetics in the modelling of the response.

There are a number of physiological characteristics that differentiate between endurance and sprint trained athletes. Endurance runners have been shown to possess a higher $\dot{V}O_2max$ (relative to body mass) and a higher percentage of slow oxidative (type-I) muscle fibres than sprint runners (Costill et al. 1976). The speed of the phase-2 response is increased as $\dot{V}O_2max$ increases for moderate (Whipp and Wasserman 1972; Powers et al. 1985) and heavy (Barstow et al. 1996) intensity exercise. Muscle fibre type will also have an effect on $\dot{V}O_2$ kinetics, since fast oxidative (type-IIa) fibres are less efficient (greater $\dot{V}O_2$ the for same power output) than type-I fibres (Crow and Kushmerick 1982). Indeed, the recruitment of fast twitch fibres is the most widely accepted explanation for the slow component (Barstow et al. 1996; Borrani et al. 2001).

It is likely that sprint runners, will have a lesser aerobic potential but a greater capacity for anaerobic work than distance runners. Therefore, in an exhaustive severe intensity run of short duration sprinters will exercise at a greater relative exercise intensity (% $\dot{V}O_2max$) than endurance runners. If $\dot{V}O_2$ kinetics are intensity dependent and rise faster with increasing exercise intensity (Whipp 1994a), it might be expected that sprinters would be able to get closer to their (comparatively lower) $\dot{V}O_2max$ during exhaustive square wave running lasting approximately 2 minutes.

The present study compared the $\dot{V}O_2$ responses to an exhaustive square wave run lasting approximately 2 minutes in a group of endurance trained and a group of sprint trained runners. The relationship found between aerobic capabilities ($\dot{V}O_2max$) and the shortfall in the $\dot{V}O_2$ response (% $\dot{V}O_2max$) in Study 3 (Chapter 6) was further investigated. In Study 3 this was relationship was demonstrated in a group that was reasonably homogenous for $\dot{V}O_2max$, but the present study offered a group (sprinters and endurance runners combined) that was heterogeneous for $\dot{V}O_2max$, yet capable of running at high speeds.

9.2 Method

9.2.1 Subjects

Twelve male club level runners volunteered to take part. Subjects were recruited to one of two groups (n = 6 in each). The first group (sprint) comprised trained sprint runners who were currently competing over 100 or 200 m. The second group (endurance) comprised trained distance and middle distance runners who were currently competing over distances ranging from 1500 to 10 000 m. The sprint group had a best time for 100 m of 11.1 ± 0.4 seconds and the endurance group had a best time for 10 000 m of 32.6 ± 1.0 minutes (mean ± SD). Whilst these do not represent an elite group of either sprinters or endurance runners (world best times at the time of writing were 9.79 seconds and 26.4 minutes for 100 and 10 000 m respectively), both groups were representative of trained club standard athletes. Subject characteristics are contained in Table 9.1. An independent samples t-test showed the endurance group to have a significantly higher $\dot{V}O_2$peak than the sprint group.

Table 9.1: Subject characteristics for and differences between (P value) the sprint and endurance groups

	Sprint Group	Endurance Group	P value
Age (yrs)	21.3 ± 5.1	21.6 ± 2.7	0.890
Height (m)	1.77 ± 0.03	1.77 ± 0.04	1.000
Mass (kg)	73.7 ± 6.4	69.7 ± 2.9	0.192
$\dot{V}O_2$peak ($ml.kg^{-1}min^{-1}$)	54.5 ± 8.5	67.5 ± 3.3	0.006

9.2.2 Study design

Subjects were required to attend the laboratory on four separate days. On the first visit subjects performed an exhaustive ramp test to determine $\dot{V}O_2$peak and speed at the AT. On each of the next two visits subjects performed a 6 minute warm up, followed by 6 minutes rest, and finally an exhaustive run at a speed fast enough to result in exhaustion in

approximately 2 minutes. The speed for the severe intensity run was estimated from the peak speed achieved during the ramp test. If on the first occasion this speed resulted in a test duration that was too short or too long, the speed was adjusted and the subject attended for an additional two sessions in which the adjusted speed was used. The warm up was performed at a speed equivalent to 90% AT. The constant speed protocol was performed twice, to ensure confidence limits of ± 1.2 seconds for τ (see Section 7.4 (Chapter 7)). On the fourth and final visit to the laboratory subjects repeated the ramp test. The $\dot{V}O_2$peak was then averaged between these two tests, for comparison against that achieved during the square wave tests, to control for order effect. The ramp test was performed first and last rather than alternated with the constant speed tests because the ramp test data were needed to determine the speeds for the warm-up and the severe intensity run.

9.2.3 Expirate analysis

Expired gases were analysed on-line using the QP9000.

9.2.4 Data analysis

Breath-by-breath $\dot{V}O_2$ data were first interpolated to second-to-second data. Data from the ramp tests were used to determine $\dot{V}O_2$peak and AT.

Second-to-second $\dot{V}O_2$ data were averaged, for each second, across the two transitions for the square wave tests. Since the repeated exhaustive runs varied slightly in duration only time points present in both transitions were averaged, and as in previous chapters the first 15 seconds of data were removed. Non-linear regression was used to model the $\dot{V}O_2$ from the severe intensity transitions, assuming a single exponential response:

$$\dot{V}O_2(t) = \text{baseline} + \text{GAIN} \times (1 - e^{-(t-\delta)/\tau}) \quad (9.1)$$

Parameter estimates were produced, through this iterative process, for GAIN, τ and δ. The overall speed of the $\dot{V}O_2$ response was evaluated as mean response time (MRT), calculated as $\tau + \delta$ (Lamarra et al. 1987). Baseline $\dot{V}O_2$ was calculated as the average $\dot{V}O_2$ for the 60

second period immediately preceding the transition. The data were modelled for both the severe intensity run and the moderate intensity warm up using the same mono-exponential model expression.

As in the previous chapter, the duration of the Phase-1 $\dot{V}O_2$ kinetics was determined according to criteria outlined by Mettauer et al. (2000). Phase-1 was considered to have ended when there was a simultaneous:

a) decrease in the end-tidal O_2 concentration ($F_{ET}O_2$)
b) increase in the end tidal CO_2 concentration ($F_{ET}CO_2$)
c) sudden decrease in RER
d) end of the small initial plateau in $\dot{V}O_2$.

Within group and between group comparisons between the ramp and the severe intensity exercise were made using a 2 x 2 (test x group) ANOVA. Within group and between group comparisons between the moderate and the severe intensity exercise also were also made using a 2 x 2 (intensity x group) ANOVA. In the event of a significant interaction Bonferonni corrected paired t-tests were used post hoc to identify the differences. Comparisons made solely between the two groups (subject characteristics) were made using independent samples t-tests. Relationships were investigated using the Pearson product moment correlation coefficient. The alpha level was set at 0.05% for all tests. Data are mean ± SD unless otherwise stated.

9.3 Results

Peak values from both the ramp test and the severe intensity runs are contained in Table 9.2. In the comparison between the ramp test and the severe intensity run, no interaction was found with the subject group ($P = 0.071$) for $\dot{V}O_2$peak (observed power = 0.447). Although there was a trend for the sprint group to achieve a higher percentage of the $\dot{V}O_2$peak from the ramp test in the severe intensity run (99.3 ± 10.5 v 92.0 ± 3.6 %). A main effect was found however for both test ($P = 0.023$) with the ramp test producing the higher values, and for group ($P = 0.017$) where the endurance group produced the higher values.

No interaction was found for HRpeak ($P = 0.752$) and there was no main effect for group ($P = 0.168$). A main effect was found for test however ($P < 0.001$) and a higher HRpeak was attained in the ramp test. There was no interaction ($P = 0.105$) or main effect for either group ($P = 0.756$) or test ($P = 0.276$) for post exercise blood lactate concentration.

Table 9.2: Peak values from the ramp test and the severe intensity run. Values are means from the two transitions of each test.

	Sprint group		Endurance group	
	Ramp	Severe	Ramp	Severe
HR ($b.min^{-1}$)	196 ± 8	188 ± 11	190 ± 7	180 ± 6.1
Lactate ($mmol.l^{-1}$)	6.4 ± 1.3	7.6 ± 1.2	6.9 ± 1.5	6.6 ± 1.6
$\dot{V}O_2$ ($ml.min^{-1}$)	3900 ± 481	3846 ± 393	4614 ± 279	4243 ± 265

The parameter estimates from the non-linear regression used for modelling the warm-up (90% AT) and the severe intensity data are contained in Table 9.3. In addition, the variables baseline $\dot{V}O_2$ and duration of Phase-1 duration are given. The mean response for each group is shown in Figure 9.1.

Table 9.3: Calculated parameter estimates from the modelling of the moderate (90 % AT) and the severe intensity data. Also given are baseline $\dot{V}O_2$ and Phase-1 duration. Asymptote (Baseline + GAIN) is also expressed a % of $\dot{V}O_2$peak from the ramp test.

	Sprint Group		Endurance Group	
	Moderate	Severe	Moderate	Severe
Baseline $\dot{V}O_2$ ($ml.min^{-1}$)	522 ± 27	683 ± 111	523 ± 130	797 ± 134
Phase-1 duration (s)	16.0 ± 3.2	13.3 ± 2.1	17.3 ± 2.9	14.0 ± 1.7
GAIN ($ml.min^{-1}$)	1844 ± 367	3049 ± 342	2312 ± 323	3356 ± 243
Asymptote ($ml.min^{-1}$)	2366 ± 427	3733 ± 403	2835 ± 299	4153 ± 230
% $\dot{V}O_2$peak (%)	60.7 ± 7.9	96.2 ± 9.0	61.5 ± 6.1	90.1 ± 3.2
τ (s)	16.6 ± 6.4	11.2 ± 1.1	12.3 ± 2.3	9.3 ± 1.9
δ (s)	14.7 ± 7.3	10.8 ± 2.7	12.7 ± 2.7	12.0 ± 0.8
MRT (s)	31.3 ± 2.3	21.9 ± 2.4	25.0 ± 1.1	21.3 ± 1.6

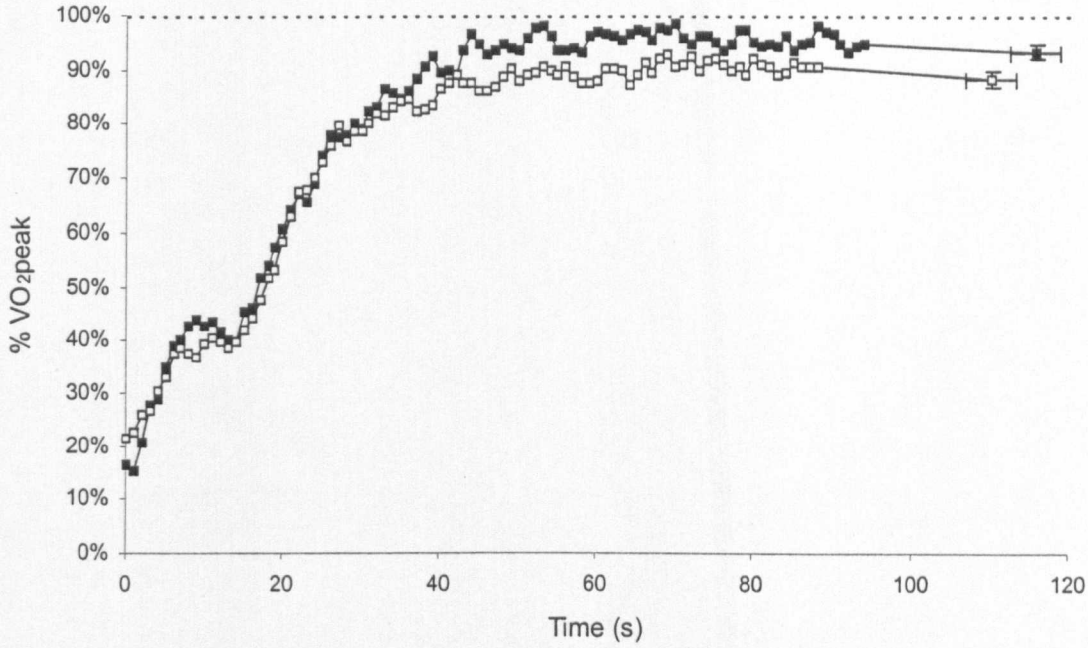

Figure 9.1: **Mean $\dot{V}O_2$ response for both the sprint group (closed symbols) and endurance group (open symbols) to exhaustive severe intensity running. Data are presented as a percentage of $\dot{V}O_2$peak from the ramp test.**
Note: Error bars represent SEM. For clarity error bars are omitted from all but the final data points

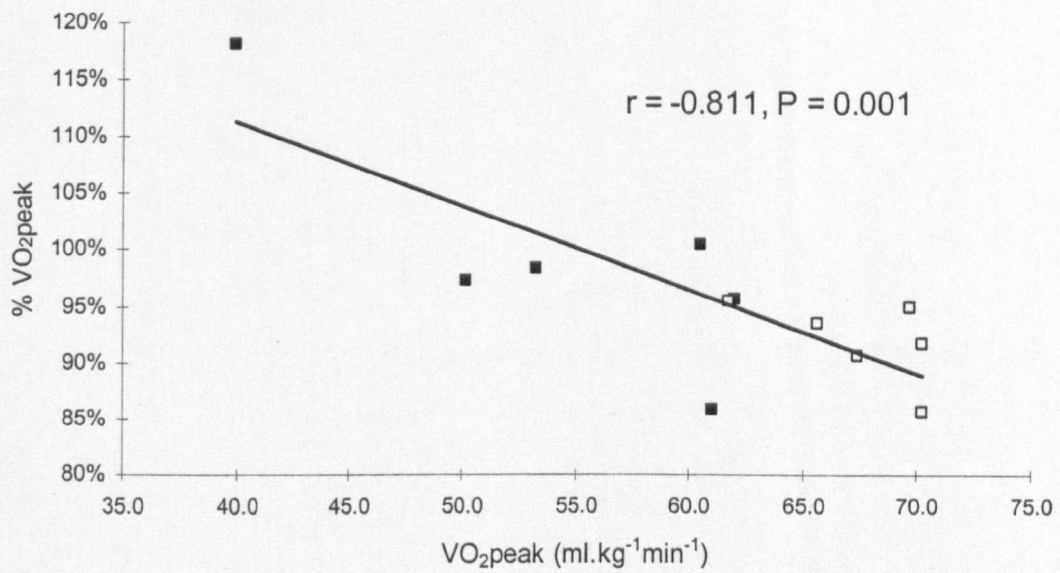

Figure 9.2: **Relationship between $\dot{V}O_2$peak and the percentage of this peak that subjects were able to attain during the severe intensity run. Sprint subjects are shown as closed symbols and endurance subjects are shown as open symbols.**

There was no interaction (intensity x group) for baseline $\dot{V}O_2$ ($P = 0.236$) and no main effect for group ($P = 0.341$). However, there was a main effect for intensity ($P = 0.001$) which was higher in the severe intensity. There was no interaction (intensity x group) for Phase-1 duration ($P = 0.791$) or main effect for group ($P = 0.239$). However, there was a main effect for intensity ($P = 0.035$) and this was longest in the sprint group.

No interaction (intensity x group) was found for the parameter estimate for GAIN ($P = 0.459$). A main effect was found for both group ($P = 0.030$) where the endurance group produced higher values, and intensity ($P < 0.001$) where the severe intensity GAIN was largest. No interaction was found for the asymptote ($P = 0.806$), but main effects for both group ($P = 0.030$) where the endurance group were highest, and intensity ($P < 0.001$) where the severe intensity was highest.

No interaction ($P = 0.400$) or main effect for group ($P = 0.076$) was found for τ. However, a main effect was found for intensity ($P = 0.011$) and τ was longer in the moderate intensity exercise. No interaction ($P = 0.228$) or main effect for either group ($P = 0.860$) or intensity ($P = 0.108$) was found for δ.

The only significant interaction was found for MRT (the sum of τ and δ) ($P = 0.003$). Main effects were also found for both group ($P = 0.002$) where the endurance group demonstrated the faster response, and intensity (0.001) where the severe intensity produced a faster response than the moderate intensity. Post hoc tests showed that the groups differed for MRT only in the moderate intensity condition and that there was greater increase in MRT between exercise intensities for the sprint group.

There was no difference in test duration ($P = 0.43$) for the severe intensity run between the groups. Test duration was 115.6 ± 15.1 and 109.9 ± 7.8 seconds for the sprint and endurance groups respectively.

The relationship between $\dot{V}O_2$peak from the ramp test and the percentage of this peak achieved in the severe intensity test is shown, for all 12 subjects, in Figure 9.2. A strong and significant correlation was found between the two variables.

9.4 Discussion

The subjects recruited for the present study were all club athletes. The groups were shown to be different from each other both in terms of aerobic capability ($\dot{V}O_2$peak) and in their $\dot{V}O_2$ response to moderate intensity exercise. The present study supported the findings of Costill et al. (1976) in showing the endurance group to have a higher $\dot{V}O_2$peak than the sprinters. The data also showed that the endurance group demonstrated a faster $\dot{V}O_2$ response (MRT) to moderate intensity exercise than the sprinters, as has been reported previously (Edwards et al. 1999). The calculated τ for moderate intensity running was similar to previously reported values (Carter et al. 2000b). No difference was found in the component parameters of the MRT, that is τ and δ, largely because of the increased variance at the lower exercise intensity. The SD was higher for both τ and δ for moderate than for severe intensity exercise. More exercise transitions would have been necessary to confidently establish this response in moderate intensity exercise (see Chapter 7). The use of only two transitions for moderate intensity exercise would have resulted in wide confidence limits for both τ and δ. However, the variance in MRT was small even at moderate intensities since any decrease in either variable due to the effect of noise on the non-linear regression fit would result in an increase in the other. The MRT then, whilst being a crude measure of the overall (phase-1 and phase-2) response does represent a useful and more robust measure when confidence limits are wide. Data from Study 4 suggest that six transitions would be necessary to give confidence limits of $\pm$ 1.2 seconds for τ and δ for the moderate intensity exercise.

An important finding from the present study was that whilst the endurance group again exhibited the same response to the severe intensity exercise as described in previous chapters there was a different response from some of the sprint trained group (Figure 9.1). However, owing to the heterogeneity and small size of the subject groups the interaction between test and group for $\dot{V}O_2$peak was only close to significance. The trend was however for the endurance group to plateau some way below $\dot{V}O_2$peak, whilst the sprint group (with two exceptions) plateaued very close to their $\dot{V}O_2$peak from the ramp test (see figure 9.1). The endurance group's $\dot{V}O_2$ response supported that which has been consistently shown in this thesis that $\dot{V}O_2$ would reach a plateau below $\dot{V}O_2$max. The

sprint group supported the widely held belief that severe intensity exercise will result in the achievement of $\dot{V}O_2$max (Whipp 1994a).

That those subjects with the lesser aerobic capabilities failed to demonstrate the sub $\dot{V}O_2$max plateau may explain why this has not been reported in previous research (Williams et al. 1998; Hill and Ferguson 1999; Hughson et al. 2000; Hill and Stevens 2001). The only work that has clearly demonstrated such a plateau used highly aerobically trained subjects (Spencer et al. 1996; Spencer and Gastin 2001).

The $\dot{V}O_2$ response in the sprint group was not a consistent one (as reflected in large SDs). Indeed it was for this reason that, unlike the previous studies, data from a typical subject was not included in this analysis. In Chapter 6, the extent of the shortfall in the $\dot{V}O_2$ response was linked to the aerobic capabilities of the athlete. The sprint group was extremely heterogeneous for $\dot{V}O_2$peak. Some of these subjects competed over distances of up to 400m, and possessed a relatively high $\dot{V}O_2$peak (Figure 9.2). Subject 5 in the sprint group, who had the highest $\dot{V}O_2$peak of that group (62 $ml.kg^{-1}min^{-1}$), achieved only 86% of this rate in the severe intensity run. Subject 2 on the other hand was the fastest 100 m runner and had limited aerobic capabilities ($\dot{V}O_2$peak from the ramp test of just 40 $ml.kg^{-1}min^{-1}$). This subject achieved a $\dot{V}O_2$ during the severe intensity run, that was even higher than that achieved during the ramp test. The individual data are contained in Appendix 9.

The data from this latter subject (subject 2 from the sprint group) warrant closer scrutiny. They represent something of an anomaly since this subject achieved 118% of his $\dot{V}O_2$peak from the ramp test in the severe intensity test. There appear to be few problems with this subject's data, however. He achieved very similar values for the two ramp tests (difference of 47 $ml.min^{-1}$) and demonstrated a $\dot{V}O_2$ plateau in both of those tests. There was little difference in the duration of the two severe intensity runs (4 seconds), and although there was some difference in the $\dot{V}O_2$peak from the two severe intensity runs (275 $ml.min^{-1}$) both tests exceeded the values from the ramp tests. The full extent of the relationship between both aerobic and anaerobic capabilities and the $\dot{V}O_2$ response for severe intensity exercise warrants further research.

The strong relationship between the percentage of $\dot{V}O_2$peak achieved in a 2 minute exhaustive severe intensity run and $\dot{V}O_2$peak (shown first in a group of trained middle distance runners in Chapter 6) was again demonstrated in this study. For the present study the 12 subjects (representing a more heterogeneous sample) were compared as a single group and again a strong relationship was evident. This relationship (shown in Figure 9.2) highlights again why previous research may have failed to demonstrate the $\dot{V}O_2$ response seen in this thesis. The relationship suggests that subjects with modest aerobic capabilities ($\dot{V}O_2$max < 55ml.kg^{-1}min^{-1}) may be expected to reach their $\dot{V}O_2$max in exhaustive severe intensity running. Previous published work has often used subjects of limited aerobic potential as shown by modest figures for $\dot{V}O_2$peak (Hill and Ferguson 1999; Hill and Stevens 2001). Furthermore, this relationship has important performance implications for the highly trained. If those with the highest $\dot{V}O_2$max are unable to make use of this advantage, anaerobic training may be far more important than has been suggested for the 800 m (Gamboa et al. 1996).

The modelling of the severe intensity data revealed no differences between the two groups for any of the parameter estimates. This is in contrast to results reported for heavy exercise where a training effect on the speed of the $\dot{V}O_2$ kinetics has been reported (Cerretelli et al. 1979). The speed of the response was consistent (as indicated by small SDs within the group for all the time parameter estimates) across the subject group (n=12) with a τ of approximately 10 seconds and a MRT of approximately 21 seconds. These values are far quicker than have been reported at lower exercise intensities (Gaesser and Poole 1996). Baseline $\dot{V}O_2$ for the severe intensity run was elevated above that recorded for the moderate intensity warm-up. However, this may have been due to subjects stretching on the treadmill between the warm-up and the severe intensity run.

Oxygen uptake kinetics were shown to be accelerated in the very severe intensity run compared to the moderate intensity warm up. Parameter estimates for both τ and MRT were smaller in the severe intensity compared to the moderate intensity run, despite a larger GAIN for the severe intensity run.

In summary, the present study showed that sprint trained individuals were more likely to achieve $\dot{V}O_2$max in an exhaustive test at a speed that results in exhaustion in 2 minutes

than endurance trained individuals. The plateau of $\dot{V}O_2$ below its maximum rate appeared to be more closely linked to the aerobic capabilities of the individual than to the type of training they were involved in however. The implications of these findings for athletic performance are further discussed in Chapter 10.

CHAPTER 10

GENERAL DISCUSSION

10.1 The $\dot{V}O_2$ response to severe intensity running

The principal finding of this thesis was that in severe intensity running at a speed that resulted in fatigue in approximately 2 minutes aerobically trained individuals exhibited a $\dot{V}O_2$ response that differed from that which had been suggested (Whipp 1994a) or reported (Hill and Ferguson 1999) previously. The literature indicates that all exercise intensities performed in the severe intensity domain result in the achievement of $\dot{V}O_2max$. The first study contained in this thesis challenged these findings as subjects clearly reached a plateau in $\dot{V}O_2$ at a rate that was below that achieved during a ramp test and other square wave tests performed at lower work rates. This phenomenon was demonstrated clearly only in treadmill running (which was the primary focus of this thesis). The $\dot{V}O_2$ response to cycling was similar in that subjects were unable to attain $\dot{V}O_2max$ but a clear plateau was not shown. It was possible therefore that the cycling response was tending towards $\dot{V}O_2max$, however such a response would have been very slow.

The domain of severe intensity exercise is defined as all exercise intensities that are greater than the MLSS or critical power/speed (Poole et al. 1988). It is also apparent that there is a consensus of opinion within the literature that all square wave exercise performed in this intensity domain results in $\dot{V}O_2max$ being attained prior to fatigue (Ward 1999). This consensus is echoed in review papers on $\dot{V}O_2$ kinetics (Gaesser and Pooole 1996, Xu and Rhodes 1999). The results of the Study 1 question the validity of this consensus. It should be highlighted however that the results of the severe intensity transitions lasting approximately 5 and 8 minutes showed exactly the expected response. The $\dot{V}O_2peak$ from these transitions was no different from that achieved in a ramp test and a third phase in $\dot{V}O_2$ appeared to be manifest. It was only at exercise intensities where exhaustion was reached in a short duration (~2 minutes) that the sub maximal plateau in $\dot{V}O_2$ was demonstrated.

The $\dot{V}O_2$ response to exercise at intensities where the theoretical $\dot{V}O_2$ requirement is greater than maximum aerobic power has been reviewed (Whipp 1994a). Whipp suggests that at such intensities the primary (phase-2) response will tend towards energy the $\dot{V}O_2$ required but that the response will be cut-off at $\dot{V}O_2max$. Moreover there is research evidence to support this view (Hill and Ferguson 1999 and Hill and Smith 2001). The 'conventional' view of kinetics for this high intensity exercise, as expressed by Whipp (1994a), is contrasted with the response found in this thesis in Figure 10.1. This example uses a theoretical subject who has a $\dot{V}O_2max$ of 5 $L.min^{-1}$ and performs 2 minutes of exhaustive exercise at 120% of $\dot{V}O_2max$ ($\dot{V}O_2$ required = 6 $L.min^{-1}$). Both traces have an identical δ (10 seconds) but a τ of 30 seconds was used for the 'conventional response' as this is representative of the majority of the data from the literature. A τ of 10 seconds was used for the typical response as demonstrated in this thesis.

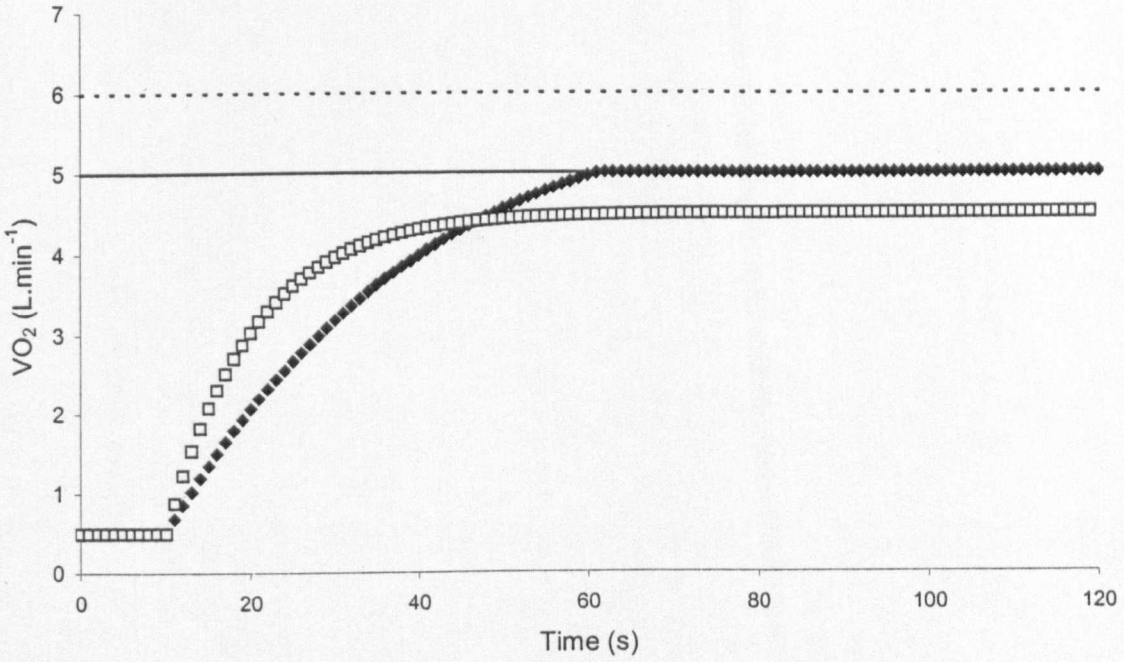

Figure 10.1: 'Conventional' Phase-2 $\dot{V}O_2$ response to exercise in the severe intensity domain (adapted from Whipp 1994a) compared with the response demonstrated in this thesis. The 'conventional' response (closed symbols) tends towards the $\dot{V}O_2$ required but is cut off at $\dot{V}O_2max$. The response shown in this thesis (open symbols) displays a short τ (~10 seconds) and tends to an asymptote at 90% $\dot{V}O_2max$. $\dot{V}O_2max$ (solid line) and the $\dot{V}O_2$ required (broken line) are also shown.

This illustration of the conventional view of $\dot{V}O_2$ kinetics in the severe intensity domain highlights how both the asymptote and the speed of the response may have an influence on running performance. It is clear that the closed symbols trace overestimates the final $\dot{V}O_2$ and thus potentially the aerobic energy contribution to this exercise. However since the thesis response is faster (reduced τ), the O_2 deficit is in fact only slightly higher (by ~4 %) for this response.

Whilst there are published data that support the response outlined by Whipp (1994a), there are also data that support the response shown in this thesis. Spencer et al. (1996) and Spencer and Gastin (2001) showed a similar response to that in this thesis in trained middle distance runners running at 800 m race pace. The aim of their research however was not to model the $\dot{V}O_2$ response; rather it was to quantify (via O_2 deficit) the relative aerobic and anaerobic contributions to this exercise. Consequently the trials were not genuine square wave exercise and the speed was decreased if necessary to enable the subject to match the duration of the treadmill run to that the subject's 800 m track time. Study 1 (Chapter 4) was therefore necessary to establish whether a shortfall in the expected $\dot{V}O_2$ response would occur in genuine square wave exercise transitions. This phenomenon has been shown most clearly in treadmill exercise and so it was also important to determine whether the shortfall in $\dot{V}O_2$ was particular to the mode of exercise or the exercise intensity. Study 1 showed that the $\dot{V}O_2$ response to exhaustive square wave exercise lasting approximately 2 minutes differed between cycling and running. A similar shortfall in the $\dot{V}O_2$ response occurred in both cycling and running exercise but the clear plateau at this sub maximal $\dot{V}O_2$ was only demonstrated in running. The following five studies therefore concentrated on severe intensity running.

An important question arising from Study 1 was why the data supported some published data (Spencer and Gastin 2001) and yet conflicted with others (Hill and Ferguson 1999). These two studies both examined exhaustive severe intensity running. Spencer and Gastin (2001) found that $\dot{V}O_2$ reached a clear plateau below $\dot{V}O_2$max during a run at 800 m race pace (as demonstrated in this thesis). However Hill and Ferguson (1999) concluded that $\dot{V}O_2$peak is independent of test duration. There are two major differences between these studies that may explain the conflicting results. The first is the subject groups that participated. Spencer and Gastin (2001) used Australian 800 m runners who were

competing at state level; this was reflected in high yet homogenous values for $\dot{V}O_2max$ (67 ± 2 $ml.kg^{-1}min^{-1}$). Hill and Ferguson (1999) however, despite all subjects being "involved in regular fitness programs and were running 40 $km.wk^{-1}$" (p. 291), used subjects who recorded relatively low and heterogeneous values for $\dot{V}O_2max$ (52.7 ± 14.5 $ml.kg^{-1}min^{-1}$). Secondly Spencer and Gastin (2001) used exercise intensities that would result in exhaustion in a given time period (approximately 2 minutes), whereas Hill and Ferguson (1999) used exercise intensities that represented given percentages of speed at $\dot{V}O_2max$ (termed Vmax). Hill and Ferguson (1999) report a large standard deviation for time to exhaustion at the highest intensity (129 ± 37 seconds).

It is possible then to explain how Hill and Ferguson (1999) concluded that $\dot{V}O_2max$ was achieved at all exercise intensities above critical speed. The present thesis (Chapters 6 and 9) showed a strong relationship between the aerobic ability of the subject and the magnitude of the shortfall in $\dot{V}O_2$ in a severe intensity run. Furthermore such a shortfall in the $\dot{V}O_2$ response has only been reported in trained subjects (Spencer et al. 1996, Spencer and Gastin 2001). It is apparent that Hill and Ferguson's subject group was of limited aerobic ability and that there was a large variation in time to exhaustion. Study 1 showed that with increased test duration (5 and 8 minutes) the subjects achieved $\dot{V}O_2max$. Finally it should be emphasised that the mean $\dot{V}O_2peak$ achieved in Hill and Ferguson's 110% Vmax test was only 95% of that achieved during a test at 100% Vmax (for which the duration was 4.8 ± 2.4 minutes). Hill and Ferguson failed to demonstrate a statistically significant difference between these values using a repeated measures ANOVA however. Five repeated measures were included in this analysis, which coupled with the large variance in both the aerobic capabilities of the subjects and the time to exhaustion, would make identification of a difference in $\dot{V}O_2peak$ unlikely.

The view that $\dot{V}O_2$ will achieve its maximum rate at all intensities in the severe domain is widely held (Whipp 1994a; Gaesser and Poole 1994; Ward 1999; Xu and Rhodes 1999). That this incorrect assumption has been widely supported may be due to the training status of the subjects that have been tested and the protocols used to test them being fixed percentages of $\dot{V}O_2max$ (rather than fixed duration). Seeking to apply findings from untrained subjects to the highly trained is a common yet dangerous practice within the field

of exercise physiology. Using percentages of $\dot{V}O_2max$ as the exercise intensities in research in the severe exercise domain is curious. Hill and Ferguson (1999) state that all exercise intensities performed above critical speed will result in $\dot{V}O_2max$, yet they reference all severe exercise intensities to the speed at which $\dot{V}O_2max$ occurred in a ramp test. The implication of the first statement is that there is a range of speeds that will all result in $\dot{V}O_2max$. From this it follows that the speed at which $\dot{V}O_2max$ occurs is entirely protocol dependent.

The close link between the concepts of a maximum steady state and critical speed / power is widely accepted and both are considered to represent the lower boundary of the domain of severe intensity exercise (Gaesser and Poole 1996). Poole et al. (1998) further demonstrated that critical power coincided with the maximum work rate that would not result in $\dot{V}O_2max$. Hill and Ferguson (1999), despite concluding that $\dot{V}O_2peak$ is independent of exercise intensity (across the range of exercise intensities they studied), highlight that the upper limit of the severe exercise intensity domain has never been established. Those subjects in Study 1 achieved $\dot{V}O_2max$ in the 5 and 8 minute trials (i.e. they demonstrated the expected response in the severe intensity domain). It may be therefore that a higher exercise intensity domain exists, in particular for the aerobically trained, where it is no longer possible to reach $\dot{V}O_2max$. The intensity or exhaustive test duration that would equal the upper limit of the severe intensity domain remains to be established however. It should be emphasised that Hill and Ferguson believe that this upper limit to severe intensity exercise is where there is insufficient time (because exhaustive exercise duration is short) to achieve $\dot{V}O_2max$. This thesis has shown that there are severe intensities where there is time for the $\dot{V}O_2$ response to fully develop but it tends to a sub maximal asymptote.

10.2 Breath-by-breath analysis of the response

The findings from Study 1 strongly suggested that current understanding of the $\dot{V}O_2$ response to exhaustive square wave exercise of a short duration was flawed. Whilst this suggestion was not without some support from previous literature (Spencer et al. 1996;

Spencer and Gastin 2001), further analysis and description of the response was not possible with Douglas bag data. Douglas bag analysis of respiratory gases constitutes a simple and reliable method for gaining mean values for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ over a reasonably long time period (> 30 seconds unless small bags are used). Such analysis, owing to the small number of data points, does not allow modelling of the overall response and assessment of non-steady state exercise (Lamarra and Whipp 1995). The remainder of the analysis of the $\dot{V}O_2$ response contained in this thesis was conducted using breath-by-breath equipment.

Study 2 therefore dealt with the validation procedures used to ensure that the QP9000 was a suitable instrument to assess the $\dot{V}O_2$ response to treadmill running. It was important that this validation work was carried out across an appropriate range of exercise intensities as whilst the focus of this thesis was severe intensity exercise it was important that the measurement of $\dot{V}O_2$ was accurate for the other domains. The resting (baseline) $\dot{V}O_2$ was vital to the modelling of the response, as was the $\dot{V}O_2$ in the early part of the exercise transition before high rates of $\dot{V}O_2$ were attained. Later chapters that made comparisons with moderate intensity exercise were also dependent on the reliability of the system at all these intensities. The QP9000 was found to be valid and reliable for the measurement of $\dot{V}O_2$ at rest, at moderate and severe exercise intensities, and for the assessment of $\dot{V}O_2$max.

The primary aims of Study 3 were to confirm the sub maximal $\dot{V}O_2$ plateau that had been seen with off-line Douglas bag data using breath-by-breath equipment and to begin to describe this response with a simple model. The subjects participating in the initial Douglas bag study were aerobically trained but not specialist runners. In contrast the subjects recruited in Study 3 were trained distance and middle distance runners. The 800 m was chosen as an event since this most closely matched the duration of the running transitions from Study 1. The plateau in $\dot{V}O_2$ below $\dot{V}O_2$max was demonstrated even more clearly in this highly trained group, confirming the results of Study 1. The training status of the subject appeared to be an important factor in determining this response. Not only was the shortfall in $\dot{V}O_2$ seen in this group greater than had been seen in Study 1, but also a negative correlation was identified between $\dot{V}O_2$max ($ml.kg^{-1}min^{-1}$) and the percentage of $\dot{V}O_2$max achieved. This showed that the aerobic capabilities of the individual were important in determining the $\dot{V}O_2$ response to 800 m running. Moreover

the data suggest that training should be geared towards increasing the anaerobic contribution to the exercise since the athletes with the higher $\dot{V}O_2$max figures were unable to make use of this reserve in aerobic power.

As has been reviewed elsewhere in this thesis (Chapter 2), many different approaches have been taken to modelling the $\dot{V}O_2$ response to square wave exercise. The exercise transitions described in this thesis were too short to include a third (slow) component (which is generally manifest after approximately 3 minutes of supra AT exercise) (Gaesser and Poole 1996). That no slow component was present was confirmed by the finding that the slope of the $\dot{V}O_2$ – time relationship was not different from zero for the last 30 seconds of the exercise transition. This was an important finding since previous research has attempted to use a three-component exponential model to describe exhaustive exercise of a similar duration (Hughson et al. 2000). Two important decisions remain for the researcher in producing a model of the $\dot{V}O_2$ response in this exercise intensity domain. Firstly, how should phase-1 be dealt with; and secondly should the model be constrained to tend to a given asymptote ($\dot{V}O_2$max or the $\dot{V}O_2$ required).

The Phase-1 response was not modelled in the present thesis. There are many arguments against including this initial phase in a model of the $\dot{V}O_2$ response and very few to justify its inclusion. Modelling the first phase is appealing in that all data points may be included in the analysis. However there are two major objections to the inclusion of phase-1. Firstly, whilst phase-1 is routinely modelled as an exponential function (identical to the phase-2 response except beginning at time 0), there is no evidence to suggest that the response is in fact exponential (Whipp and Ozyener 1998). Second, whilst $\dot{V}O_2$ certainly increases during phase-1, it is not yet understood exactly what causes this increase. The major contribution seems to be an increase in cardiac output (Wasserman et al. 1974); however other factors also contribute (Casaburi et al. 1989a). Phase-2 kinetics have consistently been shown to be exponential and closely mirror the rate of phosphocreatine degradation (Whipp 1994a). Research evidence supporting an exponential phase-1 response and identifying the underlying physiological mechanisms has been provided. The current convention in modelling phase-1 kinetics as an exponential (Burnley et al. 2000, Carter et al. 2002) therefore appears to lack any real justification.

It is however important that the correct amount of data is removed from the data set, since when modelling phase-2, removal of some of the phase-2 data or inclusion of part of the phase-1 data would affect the calculated τ and δ parameters. There seems to be considerable variation in these parameters between subjects: a τ of approximately 10 seconds was seen in the present thesis using trained subjects whereas a τ of around 40 seconds has previously been reported for a similar exercise intensity (Hill and Stevens 2001). That this was a function of the trained status of the subjects rather than the exercise intensity was shown, by a similar τ being returned in moderate intensity running (Chapter 7) to that found for severe intensity running. A τ of around 30 seconds has been reported previously for moderate intensity cycling exercise using less trained subjects. The removal of 15 seconds of data in this thesis was shown to be a reasonable method of separating the phase-1 data, once the duration of the initial phase was assessed. However this was specific to trained subjects and the high intensity exercise that was the focus of this thesis. Researchers should therefore assess this phase-1 duration since it will be dependent on the subject group and exercise intensity investigated.

The plateau below $\dot{V}O_2$max was confirmed in all studies and thus was shown to be a robust phenomenon. The question remained however of whether this plateau represented a genuine asymptote or whether the response was cut off at this sub maximal rate of $\dot{V}O_2$ while tending towards some higher figure. An attempt was made in Chapter 6 to answer this question by modelling the initial 45 seconds of data separately to the full data set as previously described by Hill and Stevens (2001). This analysis, contrary to that of Hill and Stevens (2001), showed a $\dot{V}O_2$ response that was never tending towards a higher $\dot{V}O_2$ than the eventual (sub maximal) plateau. The analysis was problematic however as $\dot{V}O_2$ kinetics were very fast in the trained runners studied. In the study of Hill and Stevens (2001), 45 seconds represented little more than one τ, whereas for the subjects used in this thesis the same period represented approximately three τs. Three τs represents the time to reach approximately 95 % of the modelled response. Therefore the detection of a difference in the GAIN parameter was unlikely. Hill and Stevens (2001) failed to see any difference in GAIN when more data were included in their analysis. In the present work, attempts to model the data from 15 – 30 seconds either resulted in a failure to fit a model to the data (i.e. the model could not be solved with positive parameter estimates by iteration) or yielded an asymptote that was much lower than the final measured $\dot{V}O_2$. Since in many

cases this analysis was unsuccessful in even fitting the regression to these data (i.e. the parameter estimates could not be determined by iteration), it was not reported in this thesis. Until more is known about the mechanisms underlying and the nature of the Phase-1 response it is unlikely that this issue can be resolved in severe intensity exercise using trained subjects.

10.3 Implications for modelling the $\dot{V}O_2$ response to severe intensity running

In Study 3, a model was established that described the $\dot{V}O_2$ response to severe intensity exercise. Further analysis of this model was then carried out in Study 4 (Chapter 7). This was necessary to determine the confidence limits for the parameter estimates, and also to determine how many repeated transitions needed to be performed to produce acceptable confidence limits. The use of repeated transitions and the averaging of the interpolated second-to-second data is an established practice and is necessary to reduce the effect of the breath-by-breath noise on the underlying $\dot{V}O_2$ response (Lamarra et al. 1987). The number of transitions required for any experiment will depend on three factors: the desired confidence limits, the level of noise present on the data, and the magnitude of the response (Lamarra et al.1987).

The analysis conducted in Study 4 was necessary because the nature of the noise had never been assessed in severe intensity exercise or in running. It has been shown that the level of breath-by-breath noise is unchanged with increasing exercise intensity (Lamarra et al. 1987), although this was only been shown within the moderate intensity domain (0 and 100 W cycling). It should be stressed however that the level of this noise varies considerably between subjects (Potter et al. 1999). Any assessment of the effect of this noise must therefore include subjects who display high levels of this noise on their breath-by-breath data. It is clear from the literature that researchers use a smaller number of transitions when working at higher exercise intensities (Carter et al. 2000b; Hughson et al. 2000). Such a convention makes the reasonable assumption that the magnitude of the response will be increased at higher exercise intensities, but also assumes that the level of noise is

no greater for heavy and severe exercise intensities in comparison to moderate intensity exercise. This second assumption has not been investigated previously.

Increasing exercise intensity will result in an overall $\dot{V}O_2$ response of increasing magnitude. However, researchers who choose to fit a three-component model (Barstow et al. 1996; Burnley et al. 2000; Carter et al. 2002) should be aware that the magnitude of each separate response may not be great enough to achieve reasonable confidence in the calculated parameters. This is a strong argument for the omission of the Phase-1 data from any modelling since the magnitude of this phase is small yet the level of noise is as great as during moderate intensity exercise (Lamarra et al. 1987). In the study of Lamarra et al. (1987) it was established that for the highest level of noise eight repeated transitions would be required to achieve 95 % confidence limits of ± 2 seconds for the phase-2 τ. If the Phase-1 (or Phase 3) response during heavy and severe exercise were of a smaller magnitude than this primary response to moderate exercise, a very large number of transitions would need to be performed to achieve reasonable confidence limits for the parameter estimates.

A curious anomaly of the study of Lamarra et al. (1987) is that the SD of the noise was calculated from the breath-by-breath data, whilst the data were actually modelled using the interpolated second-to-second data. The interpolation of the data to second-to-second values itself acts as a filter to reduce the magnitude of the noise (Lamarra et al 1987). If the SD from the breath-by-breath data were incorporated into the calculation of the number of transitions necessary for a given confidence limit, this might return a value that was too stringent. Researchers should therefore use the SD (or the SEE) from the interpolated data in such a calculation, since it is those data that must be used in order to model using repeated transitions. Analysis of the SD / GAIN ratio should be performed since this ratio, and therefore the number of transitions required, will vary greatly between subjects and exercise intensities. Subjects themselves vary considerably in the level of noise they exhibit (SD) and the magnitude of the GAIN will be greater at high exercise intensities. The current convention of using two exercise transitions for all exercise intensities above AT lacks any real justification and needs to be addressed.

10.4 Potential mechanisms that might explain this response

Studies 1 and 3 had demonstrated a $\dot{V}O_2$ response that was very different from that presented in the majority of the literature (Whipp 1994a). The model used to describe this response had been tested and it was established that narrow confidence limits could be achieved if two identical exercise transitions were interpolated and averaged on a second-to-second basis. The final two studies (5 and 6) provided an opportunity to use two transitions for the modelling of this response. The fundamental question remained that of why $\dot{V}O_2$ did not tend to $\dot{V}O_2max$ in exercise where the $\dot{V}O_2$ required was greater than $\dot{V}O_2max$.

Research on the $\dot{V}O_2$ response to severe intensity exercise is scarce, and most of the debate has centred on how to model such a response (Hughson et al. 2000; Hill and Stevens 2001). This research has been limited and has made no reference to research that has suggested a different response (Spencer et al. 1996). Consequently there has been no explanation offered for why a plateau in $\dot{V}O_2$ could occur below $\dot{V}O_2max$.

Two factors that have been shown to alter $\dot{V}O_2$ kinetics for heavy intensity exercise are a prior bout of heavy intensity exercise and aerobic training. Hughson et al. (2000) suggest that a shortfall in the Phase-2 response to severe intensity exercise is due to an O_2 delivery limitation. The suggested mechanisms whereby prior heavy intensity exercise might accelerate the $\dot{V}O_2$ response to a second bout of heavy exercise are a metabolic acidosis mediated Bohr effect and vasodilation (Gerbino et al. 1996). If O_2 delivery were the underlying reason why $\dot{V}O_2$ was unable to attain its maximum, prior heavy intensity exercise might also alter $\dot{V}O_2$ kinetics in severe intensity exercise.

The other factor that has been shown to affect $\dot{V}O_2$ kinetics in the heavy intensity exercise domain is aerobic training (Hagberg 1980; Powers 1985). Oxygen uptake kinetics have been shown to alter following a period of endurance training (Casaburi et al. 1987). It seems that the majority of this effect is due to a reduction in the slow component (Womack et al 1995). A considerable amount of this effect however may also be due to the researchers using the same absolute work-rates despite an increase in the intensity at which the AT occurred (Poole et al. 1990). What is clear from the results contained in this thesis,

and from other studies (Billat et al. 1998a; Lucia et al. 2000), is that the $\dot{V}O_2$ response of trained endurance athletes is different to that of other subject groups. Apart from the shortfall in $\dot{V}O_2$ in severe intensity exercise, the subjects participating in the present thesis showed a $\dot{V}O_2$ response that was much faster than that reported in previous research (Gaesser and Poole 1996).

Fast oxidative (Type IIa) muscle fibres are known to exhibit slower $\dot{V}O_2$ kinetics than slow oxidative (Type I) fibres (Crow and Kushmerick 1982). Indeed the recruitment of these less efficient fibres is considered to be responsible for the majority of the elevated and delayed third phase of $\dot{V}O_2$ kinetics (Slow component) (Whipp 1994a). In addition to the slow component, an experiment in which pedal cadence was manipulated during cycle exercise, in order to affect the fibre recruitment patterns, suggested that fibre type would also alter the Phase-2 response (Barstow et al. 1996). Since endurance athletes are likely to possess a higher percentage of Type I fibres than untrained or sprint trained subjects it is very difficult to determine whether changes in $\dot{V}O_2$ kinetics are due to the genetic endowment of fibre type or an adaptation to endurance training.

Study 5 therefore investigated the influence of prior heavy intensity exercise on a subsequent bout of severe intensity exercise, in order to see if the metabolic status of the individual would affect the $\dot{V}O_2$ response. Study 6 investigated the differences in the $\dot{V}O_2$ response to severe intensity exercise between a group of competitive middle distance and distance runners and a group of competitive sprint runners. This final study was to determine whether the response seen in the preceding chapters was only seen in the aerobically trained. The confidence limits for the parameter estimates were narrowed in these chapters as two transitions were averaged for each model. This reduced the variance in the parameter estimates of the model and therefore improved the statistical power of any tests.

The $\dot{V}O_2$ response to severe intensity running was found to be dependent on the intensity of the warm-up, but this did not affect the sub maximal plateau. It was hypothesised that the elevated metabolic acidosis, following supra AT exercise, might improve O_2 delivery and reduce the apparent shortfall in the $\dot{V}O_2$ response. This was not demonstrated and the plateau in $\dot{V}O_2$ below its maximum rate was again manifest in both conditions;

furthermore no difference was found in this final $\dot{V}O_2$ between conditions. No difference was shown in τ for the phase-2 response; however a difference in the MRT ($\tau + \delta$) showed that overall the response was faster following heavy exercise. This difference was due to a reduced duration of the Phase-1 response and not a speeding of the primary (phase-2) kinetics. It is possible that the proposed vasodilation aids venous return during this initial phase.

Whilst the reduction in the delay of the onset of the primary phase of $\dot{V}O_2$ kinetics was small, there are potential implications for performance. If a theoretical example is again examined (using a hypothetical subject, who has a $\dot{V}O_2max$ of 5 $L.min^{-1}$ but achieves only 90% of this rate during an exhaustive test at 120% $\dot{V}O_2max$ lasting 2 minutes), the effect of a change in δ can be seen. Figure 10.2 shows the difference in the response if the δ is altered from 12 to 9 seconds, as occurred following a supra AT warm-up.

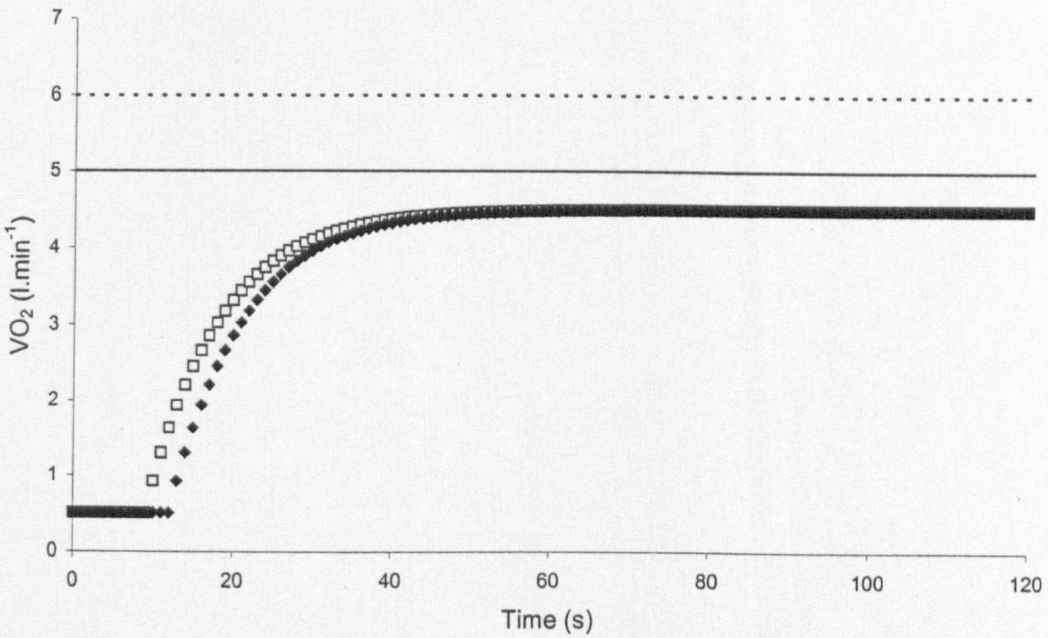

Figure 10.2: Theoretical change in the $\dot{V}O_2$ response and O_2 deficit for severe intensity exercise lasting 2 minutes when the δ is reduced from 12 s (closed symbols) to 9 s (open symbols). The τ and GAIN of phase-2 are assumed to be equal in both traces. $\dot{V}O_2max$ (solid line) and $\dot{V}O_2$ required (broken line) are also shown.

Both traces (in figure 10.2) reach the same asymptotic $\dot{V}O_2$ yet the reduced δ results in a reduction in the O_2deficit of 0.20 L (4.5%). Such an effect is still small (approximately 2.8 $ml.kg^{-1}$ for a subject mass of 70 kg), yet any shift in the aerobic / anaerobic energy contribution to exercise metabolism will theoretically have some impact on performance. Within Chapter 8 this difference in the response and the potential benefits to performance did not result in an increased time to exhaustion, although a fairly high degree of variability was apparent in test duration.

Study 5 further supported the model used to describe the $\dot{V}O_2$ response to severe intensity running and showed that prior supra AT exercise altered the initial part of this response. The shortfall in $\dot{V}O_2$ was unchanged however and the mechanism responsible for this shortfall was still unknown. This was further investigated in Study 6 with separate groups of sprinters and middle distance and distance runners. This was a necessary next step since previous research had shown that both aerobic training (Casaburi et al. 1987; Womack et al.1995) and fibre type (Crow and Kushmerick 1982; Barstow et al 1996) affect the $\dot{V}O_2$ response to heavy intensity exercise. In addition the breath-by-breath data presented in Study 3 showed a strong correlation between the magnitude of this shortfall in $\dot{V}O_2$ and $\dot{V}O_2$max itself. Theoretically the sprinters might be expected to possess a higher capacity to do anaerobic work and a lower $\dot{V}O_2$max than the endurance-trained group (Medbo and Burgers 1990). Conversely endurance athletes have been shown to possess no greater capacity for anaerobic work (as estimated from O_2deficit) than untrained subjects (Medbo and Burgers 1990). These differences in both aerobic and anaerobic capabilities would presumably result in differences in the relative aerobic and aerobic contributions to exercise, as the sprinters would compensate for a limited $\dot{V}O_2$max with a greater capacity for anaerobic energy provision. This would result in the sprinters maintaining a greater $\dot{V}O_2$ required relative to their $\dot{V}O_2$max.

Study 6 however failed to demonstrate any differences in the $\dot{V}O_2$ response to severe intensity running between the two groups. This result was probably due to the limitations of the study. The study recruited from local club athletes who competed at sprint events or middle distance / distance events. Unfortunately, owing to the sub elite nature of the subject groups (coupled with the practice of participation in several events in club athletics), the subject groups were of mixed aerobic capabilities. The range of $\dot{V}O_2$max

values from the sprint group reflected this variance. The groups were however sufficiently different for the endurance group to demonstrate a faster $\dot{V}O_2$ response to moderate exercise than the sprint group. Both groups demonstrated a similar and fast phase-2 response to the severe intensity exercise.

It appeared that the shortfall in $\dot{V}O_2$ had more to do with the aerobic capability of the subject than with the involvement in sprint and endurance competition and training. When the data from this chapter were examined as a single group, a strong correlation was again present between the shortfall in $\dot{V}O_2$ and $\dot{V}O_2$peak from the ramp test. The fastest of the sprint group, with the lowest $\dot{V}O_2$peak from the ramp test, was able to exceed this $\dot{V}O_2$peak during the 2 minute test. Linear regression suggested that any $\dot{V}O_2$max greater than 57 ml.kg^{-1}min^{-1} would result in a shortfall in $\dot{V}O_2$ during severe intensity running (R^2=0.66, SEE ± 5 ml.kg^{-1}min^{-1}). Whilst there is a high degree of variance in these data it is apparent that for subjects who possess a high $\dot{V}O_2$max (as would be expected for elite 800 m runners), there will be a considerable discrepancy between achieved and maximum aerobic power. The subject with the highest $\dot{V}O_2$max of those tested in this thesis (80 ml.kg^{-1}min^{-1}) (subject 8 in Study 3) achieved only 73% of this $\dot{V}O_2$max during an exhaustive run at 800 m race pace despite exhibiting extremely fast $\dot{V}O_2$kinetics (τ = 7.8 seconds). As shown in Study 3, the asymptotic $\dot{V}O_2$, for an exhaustive run at 800 m pace varies little among trained subjects. Thus whilst the anaerobic contribution to 800 m running has generally been overestimated (Spencer et al. 1996), this contribution is vital to success. That is, if two runners have an identical capacity for anaerobic work, the runner with the higher $\dot{V}O_2$max will derive little benefit from this aerobic potential. When this situation is reversed however and $\dot{V}O_2$max is equal, the runner with the greater anaerobic capacity will have a considerable advantage since $\dot{V}O_2$ will plateau at a similar rate.

Identification of the physiological mechanisms that might cause $\dot{V}O_2$ to plateau below its maximum is of course complex. No consensus yet exists on what may limit $\dot{V}O_2$ at the onset of exercise at any exercise intensity (Hughson et al. 2001a; Grassi 2001) or is responsible for the slow component of $\dot{V}O_2$ (Poole et al 1994a), despite considerable research into both areas. The factors that might potentially influence both O_2 delivery and O_2 consumption are numerous (Richardson 1998). The matching of O_2 delivery to O_2

requirement is a complex process even for moderate intensity exercise and requires both feed forward and feedback control mechanisms (Hughson et al. 2001a). It is apparent that there is some feed forward control since heart rate and cardiac output are known to increase very early in the exercise transition (Phase-1 $\dot{V}O_2$ kinetics), largely due to a reduction in parasympathetic activity (Rowell 1993). In addition the muscle pump itself will increase O_2 supply to the working musculature at the onset of exercise, although not enough to meet the $\dot{V}O_2$ required even at moderate exercise intensities (Shoemaker and Hughson 1999). Further increases arise from the activation of the sympathetic nervous system and appropriate arterial vasodilation to increase blood flow. Arterial blood pressure needs to be maintained however and this vasodilatation must be accompanied by vasoconstriction of non-working regions (Hughson 2001a). Since subjects achieved a higher $\dot{V}O_2$ in the ramp tests it is apparent that further adaptation is possible; what is unclear is at what point this complex process may fail during severe intensity exercise, or alternatively what time period is necessary for its full response.

10.5 Implications for existing models of middle-distance running

Mathematical models of running performance were reviewed in detail in chapter 2. The contemporary models, or more specifically the part of these models that describes the $\dot{V}O_2$ response (and therefore the aerobic and anaerobic contribution) to middle distance running, all share two major assumptions. Firstly, and most importantly, it is assumed that $\dot{V}O_2$ will tend towards $\dot{V}O_2max$. Secondly it is assumed, that the $\dot{V}O_2$ response is a simple exponential (no time delay or Phase-1 response is included in the model) (Wilkie 1980; DiPrampero 1986; Perronet and Thibault 1989; Capelli 1999). The models differ however in the τ assigned to this response. Some have taken the value for τ from published research into $\dot{V}O_2$ kinetics and assigned values of 24 seconds (Capelli 1999) or 30 seconds (Perronet and Thibault 1989); others have arrived at a τ of 10 seconds. The figure of 10 seconds has been derived from the best fit the model of the performance data rather than based on previous research (Wilkie 1980; DiPrampero 1986). Whilst a similar τ was demonstrated in this thesis, it should be realised that a delay (δ) was also incorporated, making the thesis response much slower than that described by these models.

The methods employed by existing models of middle-distance running performance to describe the $\dot{V}O_2$ response only really differ then in the assigned value of τ. Since the $\dot{V}O_2$ response demonstrated in this thesis is different from that proposed in the existing literature (Whipp 1994a), this section will compare the $\dot{V}O_2$ response to severe intensity running (2 minutes duration) with the response suggested by the contemporary models of 800 m running performance. Since all of the models share the assumption that $\dot{V}O_2$ will tend to $\dot{V}O_2max$ in middle distance events, this will be done by examining a model which assigns a τ of 10 s and one which assigns a τ of 30 s. It is perhaps curious that all models assume that $\dot{V}O_2$ will tend to $\dot{V}O_2max$ when the popular assumption is that the response will tend to the $\dot{V}O_2$ required in this intensity domain (Whipp 1994a; Hughson et al. 2000; Hill and Stevens 2001). This is perhaps because it would be impossible to describe such a response with a simple exponential function.

In order to compare these models, the example used was again of an individual with a $\dot{V}O_2max$ of 5 $L.min^{-1}$, performing 2 minutes of exhaustive running at 120% $\dot{V}O_2max$. Figure 10.3 shows the response assumed in Perronet and Thibault's (1989) model where τ is 30 s and the response tends to $\dot{V}O_2max$. This is similar to the response suggested by Capelli (1999), except that Capelli used a τ of 24 seconds. The thesis response that tends to only 90% $\dot{V}O_2max$, assumes a τ of 10 seconds but also incorporates a delay of 10 seconds. Figure 10.4 shows the difference between the $\dot{V}O_2$response proposed in this thesis to that used in models assuming that $\dot{V}O_2$will tend to $\dot{V}O_2max$ with a τ of 10 s (Wilkie 1980, DiPrampero 1986). The thesis response is as in Figure 10.3.

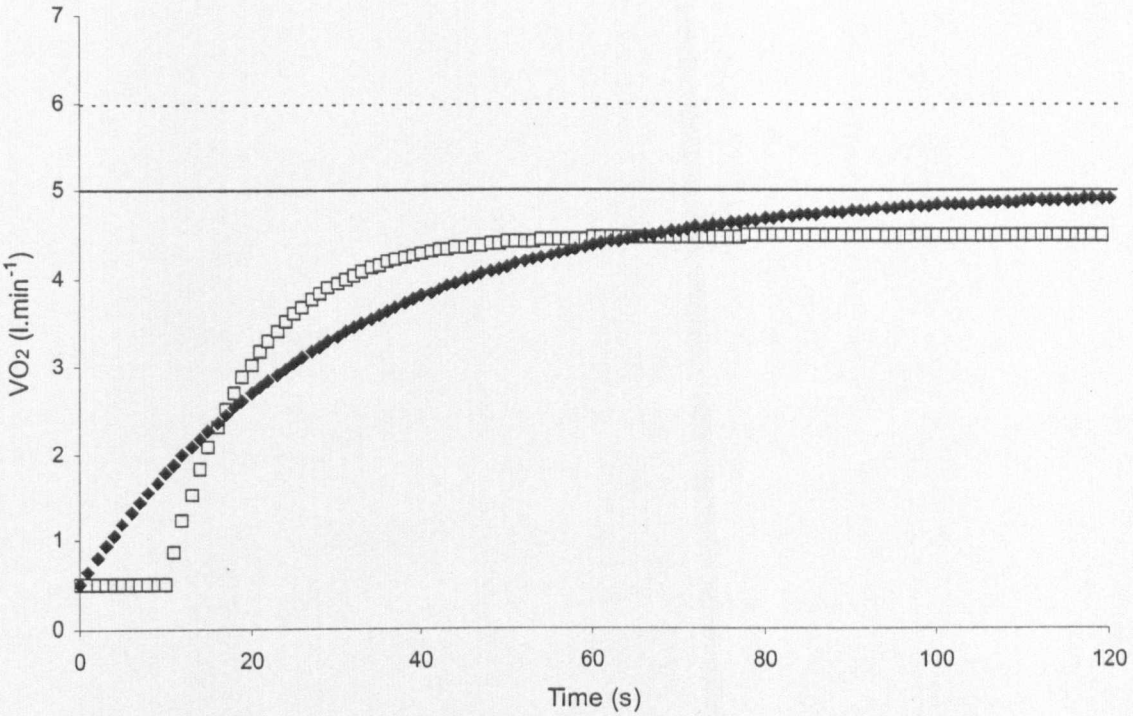

Figure 10.3: **Schematic comparing the typical $\dot{V}O_2$ response shown in this thesis (open symbols) assuming a τ of 10 seconds, a δ of 10 seconds and that $\dot{V}O_2$ will tend to only 90% $\dot{V}O_2max$ with that generated from a model (closed symbols) which assumes a τ of 30 seconds, no delay and that $\dot{V}O_2$ will tend to 100% $\dot{V}O_2max$. $\dot{V}O_2max$ (solid line) and $\dot{V}O_2$ required (broken line) are also shown.**

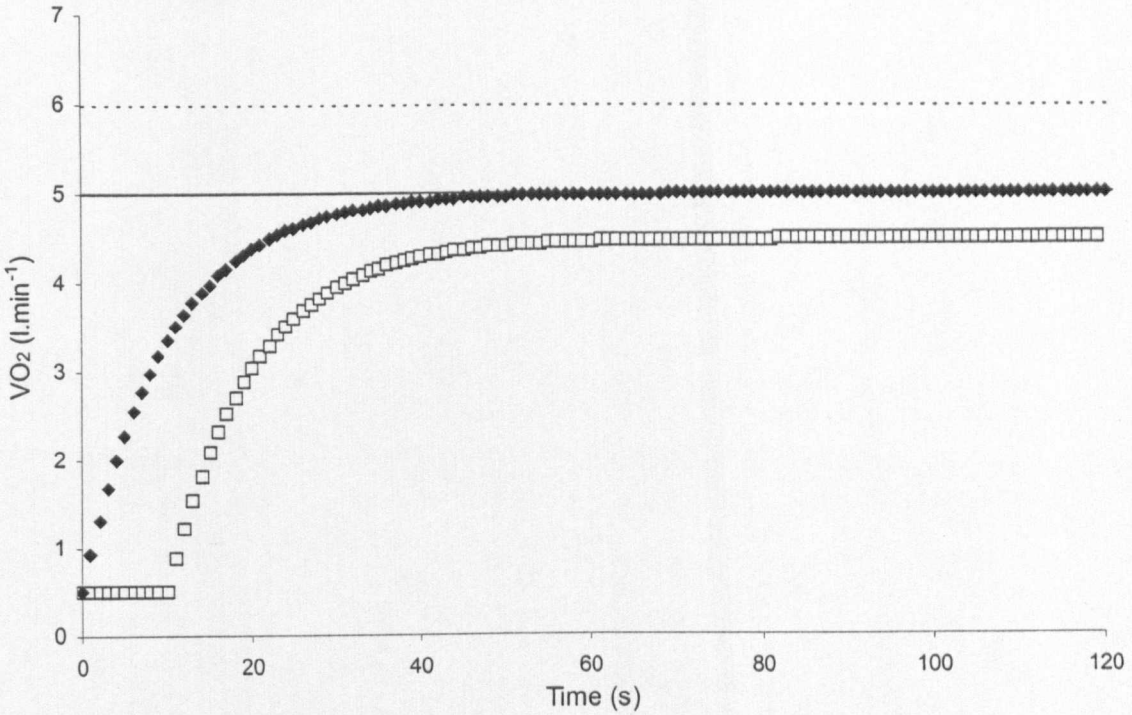

Figure 10.4: Schematic comparing the typical $\dot{V}O_2$ response shown in this thesis (open symbols) assuming a τ of 10 seconds, a δ of 10 seconds and that $\dot{V}O_2$ will tend to only 90% $\dot{V}O_2$max with that generated from a model (closed symbols) which assumes a τ of 10 seconds, no delay and that $\dot{V}O_2$ will tend to 100% $\dot{V}O_2$max. $\dot{V}O_2$max (solid line) and $\dot{V}O_2$ required (broken line) are also shown.

These comparisons illustrate the differences between the actual $\dot{V}O_2$ responses shown in trained runners and the assumed responses used in contemporary models of running performance. Comparisons can be made both in terms of how well the two responses are matched and perhaps more importantly in the differences in the relative aerobic and anaerobic energy contributions to the exercise.

The model that assumes a τ of 30 s (Figure 10.3) shows a very gradual $\dot{V}O_2$ response that is very different to the very rapid response shown in trained runners in this thesis. In terms of the relative aerobic and anaerobic energy contributions, the difference in O_2 deficit is however small (~2 % higher in the thesis response). This apparent agreement is achieved since the O_2 deficit is overestimated early in the exercise due to the slow kinetics, yet is

underestimated during the last phase of exercise, in which $\dot{V}O_2$ is wrongly assumed to reach $\dot{V}O_2max$.

The model that assumed a τ of 10 seconds, which agrees with the speed of the response demonstrated in this thesis, showed a closer agreement with the actual response. The lack of any delay component, to account for venous return, resulted in a trace that rose earlier and again the assumption was that the response would tend to $\dot{V}O_2max$. These two factors both resulted in an underestimation of the O_2 deficit. A difference was found of 1.6 L (22.5 $ml.kg^{-1}$ for a 70 kg subject) in O_2 deficit. A typical value for the maximal accumulated O_2 deficit (MAOD) is 45 $ml.kg^{-1}$ in aerobically trained individuals (Medbo and Burgers 1990; Spencer et al. 1996). Therefore such an error is a major one.

The model assuming this fast τ of 10 seconds performs reasonably well in predicting running performance at longer distances but has been shown to be poor in predicting performance at 800 m and 1000 m (DiPrampero 1986). The model has tended to overestimate the sustainable running speed over these distances. DiPrampero (1986) explained this problem by suggesting that perhaps the duration of the event was insufficient to fully exhaust the capacity to do anaerobic work. Such a suggestion is problematic however, since evidence suggests that the anaerobic capacity is exhausted within 2 minutes (Medbo et al. 1988), which is less than it takes to complete the 1000 m even at world record pace. The results of this thesis suggest that the overestimation of running speed (DiPrampero 1986) may in fact be the result of the incorrect assumption that the $\dot{V}O_2$ response will achieve $\dot{V}O_2max$.

None of the current mathematical models of middle distance running performance adequately describes the $\dot{V}O_2$ response, and therefore the aerobic contribution, to exhaustive severe intensity running such as the 800 m. The current models are based on flawed assumptions regarding both the speed of the $\dot{V}O_2$ response and the asymptote of this response. A model that would adequately describe this response would, in addition to a τ of approximately 10 seconds, need to incorporate a δ and reference the $\dot{V}O_2$ response to an asymptote that is somewhere below $\dot{V}O_2max$. This asymptote would need to be estimated based on the $\dot{V}O_2max$ of the individual.

Perhaps part of the problem with the current models is that they attempt to predict performance across a large range of race distances (and hence exercise intensities). Since however the $\dot{V}O_2$ response differs greatly across intensities, particularly in the severe intensity domain, a less global approach may be necessary.

10.6 Recommendations for future research

This thesis aimed to investigate the $\dot{V}O_2$ response to exhaustive severe intensity running in an appropriately trained population. The investigation demonstrated that this response is very different to that which has been previously proposed. The thesis however raises as many questions as it answers, as the physiological mechanisms responsible for this different response are unknown. Furthermore the investigations examined the response during exhaustive exercise lasting 2-, 5- and 8 minutes only. The 5- and 8 minute tests exhibited a $\dot{V}O_2$ response consistent with previous literature, whilst the 2 minute tests did not. To further understanding of the response it would be necessary to investigate a range of exhaustive intensities where the exercise time would perhaps range from 30 seconds to 5 minutes.

Subjects with a high $\dot{V}O_2max$ showed the greatest shortfall in $\dot{V}O_2$ during the 2 minute test. Conversely subjects with a limited $\dot{V}O_2max$ were able to reach this maximum during such a test. The correlation between $\dot{V}O_2max$ ($ml.kg^{-1}min^{-1}$) and the magnitude of the shortfall in $\dot{V}O_2$ (as a % $\dot{V}O_2max$) (Figure 9.2) supported this. Most of the analysis in the thesis was performed however with subjects who were reasonably homogenous for $\dot{V}O_2max$. A study that examined a subject group that was as heterogeneous for $\dot{V}O_2max$ as possible might more fully explain the nature of this relationship. If subjects with the highest $\dot{V}O_2max$ figures cannot achieve a higher rate of $\dot{V}O_2$ during a square wave run than those possessing a lower $\dot{V}O_2max$, there may exist an optimum $\dot{V}O_2max$ beyond which there is no performance benefit.

In terms of identifying the mechanisms responsible for the sub maximal plateau in $\dot{V}O_2$, there are of course many manipulations that might be attempted in order to answer this

question in whole body exercise (other than those used in this thesis). Hypoxic and hyperoxic gases might be breathed to manipulate O_2 saturation and delivery, or beta-blockade could be used to limit heart-rate (and thus cardiac output) kinetics. Study 6 (sprint versus endurance runners) could be repeated if a very elite group of subjects could be recruited, or a biopsy based study might be a more sophisticated means to assess the influence of fibre-type on the $\dot{V}O_2$ response to severe intensity running.

The above manipulations and interventions may represent little more than a best guess however. Greater scrutiny and understanding of the $\dot{V}O_2$ response itself might be necessary before such studies are undertaken. Two important questions remain about the model used to describe the response in this thesis. Firstly, does the primary (Phase-2) response tend towards the sub maximal asymptote or is it that it tends to a higher asymptote but gets cut-off at this sub maximal level? Secondly, how should the Phase-1 response to this exercise be dealt with?

Answering the first of these questions is problematic since the response is so fast. No evidence was found to support the assumption that $\dot{V}O_2$ was tending to $\dot{V}O_2$ required. Therefore, whilst the analysis used by Hill and Stevens (2001) could show no differences in this asymptote, this may not be conclusive evidence.

Inclusion of the Phase-1 response is not a simple matter. Indeed many of the objections to its inclusion have already been outlined in this chapter. A primary concern is that whilst $\dot{V}O_2$ does increase during this initial phase, the mechanisms behind the increase are not well understood (Casaburi et al. 1989a). The comparatively small magnitude of this response means that a very large number of transitions must be averaged to model this response. Moreover it must not be assumed that the response follows the same exponential model as the Phase-2 response. Finally, the use of a Haldane transformation based breath-by-breath calculation of $\dot{V}O_2$ is likely to be particularly problematic during this phase since changes in lung gas stores are likely to be large in the early stages of a rest to exercise transition. Therefore a measurement of inspired volume and a correction for estimated alveolar $\dot{V}O_2$ should be made (Beaver et al. 1981). Current methods for making this correction however assume a constant lung volume and would need to be improved for this purpose. The turbine device used in conjunction with the QP9000 mass spectrometer is bi-

directional. It should be possible therefore (albeit with considerable developmental work to overcome technical problems with measuring the inspired flow signal) to calculate $\dot{V}O_2$ using this equipment. It is the contention of this author that a major review of the modelling of Phase-1 at all intensities should be conducted, as current methods are unsound.

10.7 Summary and conclusions

In summary the primary finding from this thesis was that in aerobically trained individuals the $\dot{V}O_2$ response to running performed at an intensity that is severe enough to result in exhaustion in approximately 2 minutes differs considerably from that which has previously been proposed. This response tends neither to the $\dot{V}O_2$ required nor to $\dot{V}O_2$max but rather to an asymptote that is some way below $\dot{V}O_2$max. Furthermore this response appears to be much faster than has previously been reported for any exercise intensity, with a τ of approximately 10 seconds. This $\dot{V}O_2$ plateau below its maximum was shown to be a robust and repeatable phenomenon in running, amongst the aerobically trained, and this was demonstrated using both on-line and off-line gas analysis systems.

The effect of mode of exercise was investigated and showed that the resulting response was different between running and cycling. The sub maximal plateau in $\dot{V}O_2$ was clearly demonstrated in running. A similar response was shown in cycling, in that subjects were unable to attain $\dot{V}O_2$max but a clear plateau was not seen. This was important to establish because it was unclear whether the response reported by Spencer et al. (1996) was an effect of aerobic training or the mode of exercise employed.

The $\dot{V}O_2$ response was modelled as a single exponential after removing the initial 15 seconds of data to exclude Phase-1. If lesser trained individuals were used this would need to be reviewed, as it is uncertain whether the length of phase-1 is the same. The model resulted in a τ and δ both of approximately 10 seconds. Since it was demonstrated that the noise on the $\dot{V}O_2$ data was Gaussian, confidence limits could be calculated. It was shown that if interpolated second-to-second data was averaged across two exercise transitions the

95% confidence limits of these parameters was <± 1.2 seconds for τ and δ in severe intensity exercise. It was also concluded that these confidence limits were applicable only to a single exponential model and that researchers using exponential models containing two- or three- exponential terms would require many more transitions to achieve the same level of reliability.

No evidence was found to suggest that the $\dot{V}O_2$ response was at any time tending to either $\dot{V}O_2max$ or the $\dot{V}O_2$ required; however the speed of the response made any analysis of the early part of exercise difficult. Previous research (Hill and Stevens 2001) had analysed the first 45 seconds of severe intensity exercise to determine the initial asymptote; however this analysis involved a $\dot{V}O_2$response for which τ was ~40 seconds.

Attempts to identify the mechanisms that might result in a shortfall in the $\dot{V}O_2$ response were largely unsuccessful. An increased metabolic acidosis at the onset of exercise, caused by a prior bout of heavy intensity exercise, failed to affect the asymptotic $\dot{V}O_2$. This did however speed the overall response (MRT), although this was largely due to a reduction in the Phase-1 duration, presumably due to vasodilation improving venous return. There was no difference in the $\dot{V}O_2$ response of sprint and endurance trained subjects (although the heterogeneous nature of these groups may have masked such a difference). What was again apparent the strong link between $\dot{V}O_2max$ ($ml.kg^{-1}min^{-1}$) and the magnitude of the shortfall in the response (asymptotic $\dot{V}O_2$ as a $\%\dot{V}O_2max$). Whilst further investigation is required into this phenomenon it appears that there may be little benefit to the performer in possessing a very high $\dot{V}O_2max$. Furthermore, since the $\dot{V}O_2$ response appears to be an important determinant of 800 m running performance, any laboratory assessment of middle-distance athletes should include an assessment of $\dot{V}O_2$ kinetics. What is critical to 800 m performance will be the achievable $\dot{V}O_2$ not $\dot{V}O_2max$ and the assessment of the athlete should reflect this.

The thesis also reviewed current mathematical models of running performance and concluded than none adequately describe the $\dot{V}O_2$ response of trained runners in the shorter middle distance events. Many of these models over-estimate running speed since the incorrect assumption is made that $\dot{V}O_2$ will tend to and reach $\dot{V}O_2max$. It is perhaps

for this reason that such models have struggled to adequately describe 800 m running performance (DiPrampero 1986).

In conclusion, the current view of $\dot{V}O_2$ kinetics for exhaustive severe intensity exercise of a short duration is incorrect. In subjects with high aerobic capabilities $\dot{V}O_2$ will tend, in a very fast response, to an asymptote which is below $\dot{V}O_2max$.

APPENDIX 1

Abstract of initial findings from Study 1: Presented at European College of Sport Science Annual Congress, Rome 1999

THE $\dot{V}O_2$ RESPONSE TO EXHAUSTIVE SUPRAMAXIMAL EXERCISE OF DIFFERING DURATION IN CYCLING AND RUNNING EXERCISE

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The majority of research into the kinetics of $\dot{V}O_2$ kinetics has been concerned with the moderate and heavy exercise domains, and using cycle exercise. The $\dot{V}O_2$ response to treadmill exercise particularly in the supramaximal domain is perhaps less well understood. Williams et al (1998), found that in exhaustive treadmill exercise of duration's between 2 and 5 mins, there was no difference between final $\dot{V}O_2$ and $\dot{V}O_2$max. However Spencer et al, (1996) showed a final $\dot{V}O_2$ which was lower than $\dot{V}O_2$max in a treadmill run at 800m race pace.

Six physically fit male subjects (mean ± SD: age 26.0 ± 5.2 yrs; height 1.82 ± 0.07 m; mass 79.4 ± 10.3 kg) completed a $\dot{V}O_2$peak test, and exhaustive constant intensity tests at speeds estimated to elicit exhaustion in 8, 5, and 2 minutes, on both cycle ergometer and treadmill. Cycle tests were completed on a modified Monark ergometer at 80/90 r.p.m., whilst all treadmill tests were completed on a zero gradient. Ramp rates for the $\dot{V}O_2$peak tests were 22.0/24.7 $W.min^{-1}$ and 1.2 $km.h^{-1}.min^{-1}$ respectively. Once the initial $\dot{V}O_2$peak test was completed the order for the subsequent constant intensity tests was randomised. The constant intensity tests began either with the subject jumping onto a moving treadmill or the experimenter dropping the test load after a short period of unloaded cycling at the desired cadence, tests continued until volitional exhaustion. Thirty-second gas collections were made continuously throughout each test into Douglas bags.

There was no difference in $\dot{V}O_2$peak for the two ramp tests, 4.56 ± 0.55 $l.min^{-1}$ (cycling), 4.75 ± 0.57 $l.min^{-1}$ (running), and no difference was found in test duration between cycling and running for the constant intensity tests ($P>0.05$). $\dot{V}O_2$peak values for the constant intensity tests are contained in Table 1, together with the percentage of$\dot{V}O_2$peak, from the ramp test, that this represented. For treadmill exercise, the 2 min test produced a lower $\dot{V}O_2$peak ($P<0.01$) than the 5 and 8 min tests, and only reached 93% ± 1% of the $\dot{V}O_2$peak from the ramp test. The cycling tests followed a similar trend but the results were not significant ($P>0.05$). Interestingly $\dot{V}O_2$ seemed to reach a plateau below $\dot{V}O_2$peak for both conditions in the two minute test, with a delta $\dot{V}O_2$ between the last two collections of 0.19 ± 0.18 $l.min^{-1}$ (cycle) and 0.004 ± 0.13 $l.min^{-1}$ (running). There was found to be no significant difference between the last two gas collections in either condition ($P<0.05$), again this trend was stronger in treadmill exercise.

Table 1: $\dot{V}O_2$peak values (mean ± SD) for the constant intensity tests

	8 min cycle	5 min cycle	2 min cycle	8 min run	5 min run	2 min run
$\dot{V}O_2$peak ($l.min^{-1}$)	4.60 ± 0.5	4.65 ± 0.63	4.37 ± 0.66	4.82 ± 0.58	4.75 ± 0.60	4.44 ± 0.54 *#
% $\dot{V}O_2$peak (ramp)	101% ± 3%	102% ± 5%	96% ± 6%	101% ± 3%	100 ± 2%	93% ± 1% *#

*$p<0.01$ vs 8 min, #$p<0.01$ vs 5min.

The results of the present study show a levelling of $\dot{V}O_2$ below $\dot{V}O_2$peak in constant intensity treadmill exercise at an intensity severe enough to reach volitional exhaustion in approximately 2 mins. Cycling exercise showed a similar trend but results were not significant.

REFERENCES

Spencer,M.R., Gastin, P.B., Payne, W.R. (1996) New Studies in Athletics. 11:4 59-65.
Williams, C.S., Ehler, K.L., Ramirez, C.P., Poole, D.C., Smith, J.C., Hill, D.W. (1998) Official Jnl of the ACSM. 30:5 s55.

APPENDIX 2

Statement of informed consent

UNIVERSITY COLLEGE
CHICHESTER
Bishop Otter Campus
College Lane Chichester
West Sussex P019 4PE
T: 01243 816000
F: 01243 816080

INFORMED CONSENT FOR PHYSIOLOGICAL TESTING PROCEDURES

I (print name and date)

_____

hereby give my consent to participate in the exercise test(s) explained to me. I am satisfied that I understand the procedures involved and accept the possible health risks due to the nature of strenuous exercise testing.

In particular I am aware of the possible dangers of certain blood borne diseases (H.I.V., HEPATITIS B etc) associated with blood sampling. Also I recognise that I am at liberty to withdraw my involvement at any stage of the work.

Subject's full signature:

_____

Experimenter's signature:

_____

Supervisor's signature:

_____

Head of section's signature:

_____

A REGISTERED CHARITY

APPENDIX 3

Health history questionnaire

UNIVERSITY COLLEGE
CHICHESTER
Bishop Otter Campus
College Lane Chichester
West Sussex P019 4PE
T: 01243 816000
F: 01243 816080

Before we can carry out any physiological tests on you we have to check that you are in a satisfactory condition to undergo strenuous exercise. We would therefore like you to fill in the following questionnaire about yourself. All information given will be treated as strictly confidential

Name:_____ **Date of Birth:** _____

Specialist Sport: _____

Sex (M/F): ☐ **Age:** ☐

1. How would you describe your present level of activity in both your work and recreation?

Sedentary ☐ Moderately Active ☐ Active ☐ Highly Active ☐

2. In terms of fitness how would you describe your present level of fitness?

Very unfit ☐ Moderately fit ☐ Trained ☐ Highly trained ☐

3. How do you view your current body weight? Are you:

Underweight ☐ Ideal weight ☐ Slightly overweight ☐ Very overweight ☐

4. Are you, or have you ever been a smoker? Yes ☐ No ☐

If yes how many did / or do you smoke a day? ☐

5. Do you drink alcohol? Yes ☐ No ☐

If you do, do you consider yourself to be a:

Very light drinker ☐ Light drinker ☐ Heavy drinker ☐ Very heavy drinker ☐

6. Have you had to consult your doctor during the last six months? If so, briefly say why:

_____

_____

7. Have you suffered from a bacterial or viral infection in the last 2 weeks? Yes ☐ No ☐

If yes, give details:

_____

_____

8. Are you presently taking any form of medication? Yes ☐ No ☐

If yes, give details:

_____

_____

9. Do you suffer or have you ever suffered from Diabetes? Yes ☐ No ☐

If yes, give details:

_____

_____

10. Do you suffer or have you ever suffered from Asthma? Yes ☐ No ☐

If yes, give details:

_____

_____

11. Do you suffer or have you ever suffered from Bronchitis? Yes ☐ No ☐

If yes, give details:

_____

_____

12. Do you suffer or have you ever suffered from any form of Heart Complaint? Yes ☐ No ☐

If yes, give details:

_____

_____

13. Is there a history of Heart Disease in your family? Yes ☐ No ☐

If yes, give details:

_____

_____

14. Do you currently suffer from any form of Muscular or Joint Injury? Yes ☐ No ☐

If yes, give details:

_____

_____

15. Have you ever suffered from Hepatitis? Yes ☐ No ☐

16. Have you ever had a blood transfusion? Yes ☐ No ☐

17. Are you a member of a social grouping which is considered to be particularly at risk from Acquired Immune Deficiency Syndrome? Yes ☐ No ☐

18. Have you for any reason, had to suspend your normal training for the past two weeks prior to this test? Yes ☐ No ☐

If yes, give details:

_____

_____

19. Lastly, is there anything to your knowledge that may prevent you from successfully completing the tests that have been outlined to you? Yes ☐ No ☐

If yes, give details:

_____

_____

Please sign:_____ **Date:**_____

APPENDIX 4

Individual data and statistical output for Study 1 (Chapter 4)

Table A4.1: Physiological responses to all tests contained in Study 1 (individual data)

			Subject								
			1	2	3	4	5	6	7	8	9
Age	(yrs)		21	22	21	31	32	29	23	27	23
Height	(m)		1.73	1.79	1.80	1.86	1.84	1.95	1.76	1.78	1.88
Mass	(kg)		70.2	80.9	88.8	63.9	82.8	89.7	79.0	75.1	85.4
$\dot{V}O_2$peak (ramp)	($l.min^{-1}$)	Cycle	4.05	4.48	4.95	3.85	4.71	5.32	3.90	3.79	4.95
		Run	4.38	5.10	4.96	3.78	4.96	5.36	4.47	4.09	4.81
$\dot{V}O_2$peak (8 min)	($l.min^{-1}$)	Cycle	4.10	4.51	4.95	4.06	5.05	5.18	4.23	3.88	4.77
		Run	4.32	5.13	4.85	3.96	5.11	5.56	4.55	4.09	4.95
$\dot{V}O_2$peak (5 min)	($l.min^{-1}$)	Cycle	4.23	4.73	5.18	3.59	4.92	5.24	4.42	3.75	4.54
		Run	4.34	4.99	4.93	3.84	4.85	5.59	4.48	4.36	4.83
$\dot{V}O_2$peak (2 min)	($l.min^{-1}$)	Cycle	3.82	4.62	5.03	3.39	4.36	5.03	3.65	3.44	4.38
		Run	4.15	4.66	4.63	3.50	4.65	5.06	4.29	3.89	4.52
HRpeak (ramp)	($b.min^{-1}$)	Cycle	186	184	185	185	169	180	191	173	192
		Run	207	206	189	188	174	193	190	177	205
HRpeak (8 min)	($b.min^{-1}$)	Cycle	182	190	177	180	170	193	184	173	199
		Run	203	204	183	200	168	195	187	176	207
HRpeak (5 min)	($b.min^{-1}$)	Cycle	182	182	185	171	165	185	186	175	191
		Run	195	196	184	180	167	190	186	177	202
HRpeak (2 min)	($b.min^{-1}$)	Cycle	171	186	180	170	160	174	183	163	185
		Run	190	188	179	176	170	188	175	177	200
Lac (post) (ramp)	(mM)	Cycle	8.4	6.4	7.4	10.3	7.0	8.4	9.1	9.0	8.1
		Run	8.1	7.9	5.9	8.0	4.5	7.2	8.1	6.4	6.4
Lac (post) (8 min)	(mM)	Cycle	6.8	6.6	7.7	11.4	7.4	9.9	9.3	9.2	9.5
		Run	8.5	7.8	5.3	6.9	5.0	9.6	7.4	6.8	8.1
Lac (post) (5 min)	(mM)	Cycle	7.4	7.4	7.8	8.2	7.9	6.8	9.4	9.1	9.5
		Run	8.9	8.5	6.7	6.6	5.2	7.8	7.3	7.6	7.9
Lac (post) (2 min)	(mM)	Cycle	8.3	6.4	7.7	8.3	5.9	7.6	8.6	8.7	6.6
		Run	9.3	7.5	5.6	6.6	4.8	6.8	8.1	8.7	6.8
RERpeak (ramp)		Cycle	1.14	1.12	1.21	1.27	1.12	1.14	1.36	1.24	1.28
		Run	1.14	1.21	1.12	1.27	1.11	1.16	1.23	1.09	1.16
RERpeak (8 min)		Cycle	1.08	1.11	1.12	1.19	1.21	1.28	1.24	1.14	1.25
		Run	1.08	1.10	1.16	1.12	1.06	1.19	1.25	1.11	1.11
RERpeak (5 min)		Cycle	1.10	1.21	1.16	1.31	1.22	1.30	1.33	1.24	1.39
		Run	1.19	1.17	1.08	1.19	1.17	1.20	1.27	1.18	1.21
RERpeak (2 min)		Cycle	1.24	1.21	1.33	1.47	1.29	1.33	1.53	1.47	1.42
		Run	1.35	1.36	1.16	1.37	1.20	1.35	1.34	1.22	1.36
$\Delta\dot{V}O_2$ (2 min)	($l.min^{-1}$)	Cycle	0.04	0.43	0.17	-0.09	0.27	0.12	0.26	0.30	0.17
		Run	-0.06	-0.17	-0.07	0.05	0.15	0.07	0.03	0.01	0.00
Slow-comp (8 min)	($l.min^{-1}$)	Cycle	0.36	0.61	0.35	0.63	0.86	0.69	0.61	0.70	0.52
		Run	0.42	0.32	0.30	0.35	0.38	0.50	0.58	0.48	0.43
Slow-comp (5 min)	($l.min^{-1}$)	Cycle	0.44	0.12	0.16	0.20	0.29	0.20	0.42	0.46	0.03
		Run	0.17	0.00	0.27	0.17	0.01	0.37	0.12	0.24	0.14

Output A4.1: Two-way RM ANOVA (ergometry x duration) for $\dot{V}O_2$peak from square wave tests in Study 1

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
ERGO	1.000	.000	0	.	1.000	1.000	1.000
DURATION	.625	3.291	2	.193	.727	.847	.500
ERGO * DURATION	.943	.410	2	.815	.946	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
ERGO	Sphericity Assumed	.471	1	.471	8.106	.022	.503	8.106	.704
	Greenhouse-Geisser	.471	1.000	.471	8.106	.022	.503	8.106	.704
	Huynh-Feldt	.471	1.000	.471	8.106	.022	.503	8.106	.704
	Lower-bound	.471	1.000	.471	8.106	.022	.503	8.106	.704
Error(ERGO)	Sphericity Assumed	.465	8	5.815E-02					
	Greenhouse-Geisser	.465	8.000	5.815E-02					
	Huynh-Feldt	.465	8.000	5.815E-02					
	Lower-bound	.465	8.000	5.815E-02					
DURATION	Sphericity Assumed	1.329	2	.664	32.931	.000	.805	65.862	1.000
	Greenhouse-Geisser	1.329	1.454	.913	32.931	.000	.805	47.897	1.000
	Huynh-Feldt	1.329	1.694	.784	32.931	.000	.805	55.796	1.000
	Lower-bound	1.329	1.000	1.329	32.931	.000	.805	32.931	.999
Error(DURATION)	Sphericity Assumed	.323	16	2.017E-02					
	Greenhouse-Geisser	.323	11.636	2.774E-02					
	Huynh-Feldt	.323	13.555	2.381E-02					
	Lower-bound	.323	8.000	4.034E-02					
ERGO * DURATION	Sphericity Assumed	9.710E-04	2	4.855E-04	.022	.978	.003	.044	.053
	Greenhouse-Geisser	9.710E-04	1.892	5.131E-04	.022	.974	.003	.042	.053
	Huynh-Feldt	9.710E-04	2.000	4.855E-04	.022	.978	.003	.044	.053
	Lower-bound	9.710E-04	1.000	9.710E-04	.022	.886	.003	.022	.052
Error(ERGO*DURATION)	Sphericity Assumed	.352	16	2.197E-02					
	Greenhouse-Geisser	.352	15.138	2.322E-02					
	Huynh-Feldt	.352	16.000	2.197E-02					
	Lower-bound	.352	8.000	4.395E-02					

Observed power computed using alpha = .05

Output A4.2: Two-way RM ANOVA (ergometry x duration) for HRpeak from square wave tests in Study 1

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
DURATION	.445	5.674	2	.059	.643	.713	.500
ERGO	1.000	.000	0	.	1.000	1.000	1.000
DURATION * ERGO	.451	5.572	2	.062	.646	.717	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
DURATION	Sphericity Assumed	677.333	2	338.667	15.482	.000	.659	30.964	.997
	Greenhouse-Geisser	677.333	1.286	526.753	15.482	.002	.659	19.908	.970
	Huynh-Feldt	677.333	1.426	475.067	15.482	.001	.659	22.074	.980
	Lower-bound	677.333	1.000	677.333	15.482	.004	.659	15.482	.930
Error(DURATION)	Sphericity Assumed	350.000	16	21.875					
	Greenhouse-Geisser	350.000	10.287	34.024					
	Huynh-Feldt	350.000	11.406	30.685					
	Lower-bound	350.000	8.000	43.750					
ERGO	Sphericity Assumed	748.167	1	748.167	14.116	.006	.638	14.116	.907
	Greenhouse-Geisser	748.167	1.000	748.167	14.116	.006	.638	14.116	.907
	Huynh-Feldt	748.167	1.000	748.167	14.116	.006	.638	14.116	.907
	Lower-bound	748.167	1.000	748.167	14.116	.006	.638	14.116	.907
Error(ERGO)	Sphericity Assumed	424.000	8	53.000					
	Greenhouse-Geisser	424.000	8.000	53.000					
	Huynh-Feldt	424.000	8.000	53.000					
	Lower-bound	424.000	8.000	53.000					
DURATION * ERGO	Sphericity Assumed	12.444	2	6.222	.345	.714	.041	.689	.096
	Greenhouse-Geisser	12.444	1.291	9.637	.345	.625	.041	.445	.086
	Huynh-Feldt	12.444	1.434	8.677	.345	.646	.041	.494	.088
	Lower-bound	12.444	1.000	12.444	.345	.573	.041	.345	.081
Error(DURATION*ERGO)	Sphericity Assumed	288.889	16	18.056					
	Greenhouse-Geisser	288.889	10.330	27.965					
	Huynh-Feldt	288.889	11.474	25.179					
	Lower-bound	288.889	8.000	36.111					

Observed power computed using alpha = .05

Output A4.3: Two-way RM ANOVA (ergometry x duration) for Lac (post) from square wave tests in Study 1

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
DURATION	.629	3.246	2	.197	.729	.851	.500
ERGO	1.000	.000	0	.	1.000	1.000	1.000
DURATION * ERGO	.971	.206	2	.902	.972	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
DURATION	Sphericity Assumed	3.534	2	1.767	1.509	.251	.159	3.018	.274
	Greenhouse-Geisser	3.534	1.459	2.423	1.509	.255	.159	2.201	.231
	Huynh-Feldt	3.534	1.701	2.077	1.509	.254	.159	2.567	.251
	Lower-bound	3.534	1.000	3.534	1.509	.254	.159	1.509	.192
Error(DURATION)	Sphericity Assumed	18.740	16	1.171					
	Greenhouse-Geisser	18.740	11.670	1.606					
	Huynh-Feldt	18.740	13.610	1.377					
	Lower-bound	18.740	8.000	2.343					
ERGO	Sphericity Assumed	8.307	1	8.307	2.757	.135	.256	2.757	.310
	Greenhouse-Geisser	8.307	1.000	8.307	2.757	.135	.256	2.757	.310
	Huynh-Feldt	8.307	1.000	8.307	2.757	.135	.256	2.757	.310
	Lower-bound	8.307	1.000	8.307	2.757	.135	.256	2.757	.310
Error(ERGO)	Sphericity Assumed	24.104	8	3.013					
	Greenhouse-Geisser	24.104	8.000	3.013					
	Huynh-Feldt	24.104	8.000	3.013					
	Lower-bound	24.104	8.000	3.013					
DURATION * ERGO	Sphericity Assumed	2.327	2	1.164	3.417	.058	.299	6.834	.557
	Greenhouse-Geisser	2.327	1.944	1.197	3.417	.060	.299	6.641	.548
	Huynh-Feldt	2.327	2.000	1.164	3.417	.058	.299	6.834	.557
	Lower-bound	2.327	1.000	2.327	3.417	.102	.299	3.417	.370
Error(DURATION*ERGO)	Sphericity Assumed	5.448	16	.341					
	Greenhouse-Geisser	5.448	15.549	.350					
	Huynh-Feldt	5.448	16.000	.341					
	Lower-bound	5.448	8.000	.681					

Observed power computed using alpha = .05

Output A4.4: Two-way RM ANOVA (ergometry x duration) for RERpeak from square wave tests in Study 1

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
DURATION	.658	2.934	2	.231	.745	.876	.500
ERGO	1.000	.000	0	.	1.000	1.000	1.000
DURATION * ERGO	.425	5.988	2	.050	.635	.701	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
DURATION	Sphericity Assumed	.293	2	.147	66.947	.000	.893	133.894	1.000
	Greenhouse-Geisser	.293	1.490	.197	66.947	.000	.893	99.744	1.000
	Huynh-Feldt	.293	1.753	.167	66.947	.000	.893	117.325	1.000
	Lower-bound	.293	1.000	.293	66.947	.000	.893	66.947	1.000
Error(DURATION)	Sphericity Assumed	3.506E-02	16	2.192E-03					
	Greenhouse-Geisser	3.506E-02	11.919	2.942E-03					
	Huynh-Feldt	3.506E-02	14.020	2.501E-03					
	Lower-bound	3.506E-02	8.000	4.383E-03					
ERGO	Sphericity Assumed	4.930E-02	1	4.930E-02	7.503	.025	.484	7.503	.671
	Greenhouse-Geisser	4.930E-02	1.000	4.930E-02	7.503	.025	.484	7.503	.671
	Huynh-Feldt	4.930E-02	1.000	4.930E-02	7.503	.025	.484	7.503	.671
	Lower-bound	4.930E-02	1.000	4.930E-02	7.503	.025	.484	7.503	.671
Error(ERGO)	Sphericity Assumed	5.256E-02	8	6.571E-03					
	Greenhouse-Geisser	5.256E-02	8.000	6.571E-03					
	Huynh-Feldt	5.256E-02	8.000	6.571E-03					
	Lower-bound	5.256E-02	8.000	6.571E-03					
DURATION * ERGO	Sphericity Assumed	1.019E-03	2	5.097E-04	.140	.870	.017	.280	.068
	Greenhouse-Geisser	1.019E-03	1.270	8.027E-04	.140	.775	.017	.178	.064
	Huynh-Feldt	1.019E-03	1.401	7.276E-04	.140	.797	.017	.196	.065
	Lower-bound	1.019E-03	1.000	1.019E-03	.140	.718	.017	.140	.063
Error(DURATION*ERGO)	Sphericity Assumed	5.821E-02	16	3.638E-03					
	Greenhouse-Geisser	5.821E-02	10.159	5.730E-03					
	Huynh-Feldt	5.821E-02	11.208	5.193E-03					
	Lower-bound	5.821E-02	8.000	7.276E-03					

Observed power computed using alpha = .05

Output A4.5: Two-way RM ANOVA (ergometry x duration) for test duration from square wave tests in Study 1

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
ERGO	1.000	.000	0	.	1.000	1.000	1.000
DURATION	.342	7.504	2	.023	.603	.652	.500
ERGO * DURATION	.957	.305	2	.861	.959	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
ERGO	Sphericity Assumed	9.757E-02	1	9.757E-02	.099	.761	.012	.099	.059
	Greenhouse-Geisser	9.757E-02	1.000	9.757E-02	.099	.761	.012	.099	.059
	Huynh-Feldt	9.757E-02	1.000	9.757E-02	.099	.761	.012	.099	.059
	Lower-bound	9.757E-02	1.000	9.757E-02	.099	.761	.012	.099	.059
Error(ERGO)	Sphericity Assumed	7.878	8	.985					
	Greenhouse-Geisser	7.878	8.000	.985					
	Huynh-Feldt	7.878	8.000	.985					
	Lower-bound	7.878	8.000	.985					
DURATION	Sphericity Assumed	368.304	2	184.152	141.485	.000	.946	282.971	1.000
	Greenhouse-Geisser	368.304	1.206	305.267	141.485	.000	.946	170.702	1.000
	Huynh-Feldt	368.304	1.304	282.450	141.485	.000	.946	184.492	1.000
	Lower-bound	368.304	1.000	368.304	141.485	.000	.946	141.485	1.000
Error(DURATION)	Sphericity Assumed	20.825	16	1.302					
	Greenhouse-Geisser	20.825	9.652	2.158					
	Huynh-Feldt	20.825	10.432	1.996					
	Lower-bound	20.825	8.000	2.603					
ERGO * DURATION	Sphericity Assumed	.276	2	.138	.288	.754	.035	.576	.088
	Greenhouse-Geisser	.276	1.918	.144	.288	.745	.035	.552	.087
	Huynh-Feldt	.276	2.000	.138	.288	.754	.035	.576	.088
	Lower-bound	.276	1.000	.276	.288	.606	.035	.288	.076
Error(ERGO*DURATION)	Sphericity Assumed	7.680	16	.480					
	Greenhouse-Geisser	7.680	15.346	.500					
	Huynh-Feldt	7.680	16.000	.480					
	Lower-bound	7.680	8.000	.960					

Observed power computed using alpha = .05

APPENDIX 5

Individual data and statistical output for Study 2 (Chapter 5)

Table A5.1: Individual data from Study 2. Data are averages of 2 transitions.

				Subject					
				1	2	3	4	5	6
Age	(yrs)			26	32	30	22	28	31
Height	(m)			1.79	1.84	1.95	1.77	1.79	1.84
Mass	(kg)			78.6	63.0	92.3	91.1	82.3	79.1
$\dot{V}O_2$	($l.min^{-1}$)	Rest	DB	0.39	0.33	0.50	0.42	0.35	0.46
			BxB	0.45	0.24	0.56	0.44	0.40	0.56
		Moderate	DB	2.32	2.14	3.10	2.82	2.43	2.35
			BxB	2.51	2.22	3.23	2.86	2.50	2.22
		Severe	DB	4.09	3.74	5.39	4.27	3.74	4.00
			BxB	4.13	3.74	5.42	4.15	3.80	3.85
		Max	DB	3.97	3.84	5.56	4.17	3.61	3.79
			BxB	4.06	3.75	5.56	4.19	3.54	3.85
$\dot{V}CO_2$	($l.min^{-1}$)	Rest	DB	0.37	0.26	0.39	0.32	0.31	0.41
			BxB	0.39	0.20	0.50	0.35	0.35	0.48
		Moderate	DB	2.13	1.91	2.78	2.64	2.21	2.11
			BxB	2.18	1.94	2.81	2.51	2.28	1.99
		Severe	DB	4.63	4.34	6.14	5.00	3.37	4.30
			BxB	4.49	4.18	5.93	4.93	4.36	3.96
		Max	DB	4.64	4.53	6.31	4.87	4.07	4.06
			BxB	4.38	4.23	6.03	4.80	3.97	4.01
$\dot{V}_{E(BTPS)}$	($l.min^{-1}$)	Rest	DB	9.3	14.6	12.9	9.6	10.7	15.1
			BxB	9.9	13.7	17.4	9.3	9.4	13.8
		Moderate	DB	60.4	58.0	80.0	65.8	63.9	65.9
			BxB	60.5	52.5	77.1	63.3	60.0	63.2
		Severe	DB	138.3	144.8	192.6	137.7	127.1	144.6
			BxB	133.3	136.1	186.6	130.2	124.3	128.5
		Max	DB	134.1	143.1	192.8	137.7	125.0	123.1
			BxB	125.2	133.8	183.5	133.1	120.6	123.7

Output A5.1: Two-way RM ANOVA (intensity x method) for $\dot{V}O_2$ from the constant intensity tests in Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENS	.385	3.823	2	.146	.619	.721	.500
METHOD	1.000	.000	0	.	1.000	1.000	1.000
INTENS * METHOD	.458	3.122	2	.207	.649	.781	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENS	Sphericity Assumed	85.608	2	42.804	239.857	.000	.980	479.714	1.000
	Greenhouse-Geisser	85.608	1.238	69.149	239.857	.000	.980	296.948	1.000
	Huynh-Feldt	85.608	1.443	59.331	239.857	.000	.980	346.087	1.000
	Lower-bound	85.608	1.000	85.608	239.857	.000	.980	239.857	1.000
Error(INTENS)	Sphericity Assumed	1.785	10	.178					
	Greenhouse-Geisser	1.785	6.190	.288					
	Huynh-Feldt	1.785	7.214	.247					
	Lower-bound	1.785	5.000	.357					
METHOD	Sphericity Assumed	5.650E-03	1	5.650E-03	1.026	.358	.170	1.026	.132
	Greenhouse-Geisser	5.650E-03	1.000	5.650E-03	1.026	.358	.170	1.026	.132
	Huynh-Feldt	5.650E-03	1.000	5.650E-03	1.026	.358	.170	1.026	.132
	Lower-bound	5.650E-03	1.000	5.650E-03	1.026	.358	.170	1.026	.132
Error(METHOD)	Sphericity Assumed	2.754E-02	5	5.508E-03					
	Greenhouse-Geisser	2.754E-02	5.000	5.508E-03					
	Huynh-Feldt	2.754E-02	5.000	5.508E-03					
	Lower-bound	2.754E-02	5.000	5.508E-03					
INTENS * METHOD	Sphericity Assumed	1.211E-02	2	6.053E-03	1.981	.188	.284	3.962	.316
	Greenhouse-Geisser	1.211E-02	1.297	9.332E-03	1.981	.210	.284	2.570	.243
	Huynh-Feldt	1.211E-02	1.562	7.750E-03	1.981	.202	.284	3.094	.271
	Lower-bound	1.211E-02	1.000	1.211E-02	1.981	.218	.284	1.981	.210
Error(INTENS*METHOD)	Sphericity Assumed	3.055E-02	10	3.055E-03					
	Greenhouse-Geisser	3.055E-02	6.486	4.710E-03					
	Huynh-Feldt	3.055E-02	7.809	3.912E-03					
	Lower-bound	3.055E-02	5.000	6.110E-03					

Observed power computed using alpha = .05

Output A5.2: Two-way RM ANOVA (intensity x method) for $\dot{V}CO_2$ from the constant intensity tests in Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENS	.164	7.234	2	.026	.545	.580	.500
METHOD	1.000	.000	0	.	1.000	1.000	1.000
INTENS * METHOD	.909	.380	2	.829	.917	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENS	Sphericity Assumed	114.386	2	57.193	235.524	.000	.979	471.048	1.000
	Greenhouse-Geisser	114.386	1.089	105.011	235.524	.000	.979	256.551	1.000
	Huynh-Feldt	114.386	1.160	98.626	235.524	.000	.979	273.161	1.000
	Lower-bound	114.386	1.000	114.386	235.524	.000	.979	235.524	1.000
Error(INTENS)	Sphericity Assumed	2.428	10	.243					
	Greenhouse-Geisser	2.428	5.446	.446					
	Huynh-Feldt	2.428	5.799	.419					
	Lower-bound	2.428	5.000	.486					
METHOD	Sphericity Assumed	1.624E-02	1	1.624E-02	3.520	.119	.413	3.520	.332
	Greenhouse-Geisser	1.624E-02	1.000	1.624E-02	3.520	.119	.413	3.520	.332
	Huynh-Feldt	1.624E-02	1.000	1.624E-02	3.520	.119	.413	3.520	.332
	Lower-bound	1.624E-02	1.000	1.624E-02	3.520	.119	.413	3.520	.332
Error(METHOD)	Sphericity Assumed	2.306E-02	5	4.612E-03					
	Greenhouse-Geisser	2.306E-02	5.000	4.612E-03					
	Huynh-Feldt	2.306E-02	5.000	4.612E-03					
	Lower-bound	2.306E-02	5.000	4.612E-03					
INTENS * METHOD	Sphericity Assumed	5.693E-02	2	2.847E-02	7.271	.011	.593	14.541	.838
	Greenhouse-Geisser	5.693E-02	1.834	3.104E-02	7.271	.014	.593	13.334	.809
	Huynh-Feldt	5.693E-02	2.000	2.847E-02	7.271	.011	.593	14.541	.838
	Lower-bound	5.693E-02	1.000	5.693E-02	7.271	.043	.593	7.271	.583
Error(INTENS*METHOD)	Sphericity Assumed	3.915E-02	10	3.915E-03					
	Greenhouse-Geisser	3.915E-02	9.170	4.270E-03					
	Huynh-Feldt	3.915E-02	10.000	3.915E-03					
	Lower-bound	3.915E-02	5.000	7.831E-03					

Observed power computed using alpha = .05

Output A5.3: Two-way RM ANOVA (intensity x method) for $\dot{V}_E$ from the constant intensity tests in Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENS	.225	5.960	2	.050	.564	.615	.500
METHOD	1.000	.000	0	.	1.000	1.000	1.000
INTENS * METHOD	.618	1.923	2	.380	.724	.941	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENS	Sphericity Assumed	105283.71	2	52641.857	197.269	.000	.975	394.538	1.000
	Greenhouse-Geisser	105283.71	1.127	93418.845	197.269	.000	.975	222.323	1.000
	Huynh-Feldt	105283.71	1.230	85627.749	197.269	.000	.975	242.552	1.000
	Lower-bound	105283.71	1.000	105283.71 5	197.269	.000	.975	197.269	1.000
Error(INTENS)	Sphericity Assumed	2668.533	10	266.853					
	Greenhouse-Geisser	2668.533	5.635	473.561					
	Huynh-Feldt	2668.533	6.148	434.066					
	Lower-bound	2668.533	5.000	533.707					
METHOD	Sphericity Assumed	106.296	1	106.296	16.449	.010	.767	16.449	.895
	Greenhouse-Geisser	106.296	1.000	106.296	16.449	.010	.767	16.449	.895
	Huynh-Feldt	106.296	1.000	106.296	16.449	.010	.767	16.449	.895
	Lower-bound	106.296	1.000	106.296	16.449	.010	.767	16.449	.895
Error(METHOD)	Sphericity Assumed	32.310	5	6.462					
	Greenhouse-Geisser	32.310	5.000	6.462					
	Huynh-Feldt	32.310	5.000	6.462					
	Lower-bound	32.310	5.000	6.462					
INTENS * METHOD	Sphericity Assumed	95.117	2	47.558	11.264	.003	.693	22.528	.959
	Greenhouse-Geisser	95.117	1.448	65.709	11.264	.008	.693	16.305	.889
	Huynh-Feldt	95.117	1.882	50.544	11.264	.003	.693	21.197	.949
	Lower-bound	95.117	1.000	95.117	11.264	.020	.693	11.264	.764
Error(INTENS*METHOD)	Sphericity Assumed	42.221	10	4.222					
	Greenhouse-Geisser	42.221	7.238	5.834					
	Huynh-Feldt	42.221	9.409	4.487					
	Lower-bound	42.221	5.000	8.444					

Observed power computed using alpha = .05

Output A5.4: One-way RM ANOVA (comparison) on the intra-individual SDs of $\dot{V}O_2$ from the three comparisons of the incremental test data from Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
COMPARISON	.667	1.623	2	.442	.750	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
COMPARE	Sphericity Assumed	2183.373	2	1091.687	2.351	.146	.320	4.702	.367
	Greenhouse-Geisser	2183.373	1.500	1455.752	2.351	.165	.320	3.526	.306
	Huynh-Feldt	2183.373	2.000	1091.904	2.351	.146	.320	4.701	.367
	Lower-bound	2183.373	1.000	2183.373	2.351	.186	.320	2.351	.240
Error(COMPARISON)	Sphericity Assumed	4643.133	10	464.313					
	Greenhouse-Geisser	4643.133	7.499	619.156					
	Huynh-Feldt	4643.133	9.998	464.406					
	Lower-bound	4643.133	5.000	928.627					

Observed power computed using alpha = .05

Note: Individual data are contained in Table 5.2 (Chapter 5)

Output A5.5: One-way RM ANOVA (comparison) on the intra-individual SDs of $\dot{V}CO_2$ from the three comparisons of the incremental test data from Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
COMPARISON	.454	3.157	2	.204	.647	.777	.500

Tests of Within-Subjects Effects

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
COMPARE	Sphericity Assumed	1287.768	2	643.884	.322	.732	.061	.645	.088
	Greenhouse-Geisser	1287.768	1.294	995.332	.322	.645	.061	.417	.080
	Huynh-Feldt	1287.768	1.555	828.188	.322	.682	.061	.501	.083
	Lower-bound	1287.768	1.000	1287.768	.322	.595	.061	.322	.075
Error(COMPARISON)	Sphericity Assumed	19973.239	10	1997.324					
	Greenhouse-Geisser	19973.239	6.469	3087.514					
	Huynh-Feldt	19973.239	7.775	2569.033					
	Lower-bound	19973.239	5.000	3994.648					

Observed power computed using alpha = .05

Note: Individual data are contained in Table 5.2 (Chapter 5)

Output A5.6: One-way RM ANOVA (comparison) on the intra-individual SDs of $\dot{V}_E$ from the three comparisons of the incremental test data from Study 2

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
COMPARISON	.650	1.725	2	.419	.741	.979	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
COMPARE	Sphericity Assumed	1.623	2	.811	.339	.720	.064	.678	.090
	Greenhouse-Geisser	1.623	1.481	1.096	.339	.662	.064	.502	.084
	Huynh-Feldt	1.623	1.957	.829	.339	.716	.064	.664	.090
	Lower-bound	1.623	1.000	1.623	.339	.586	.064	.339	.077
Error(COMPARISON)	Sphericity Assumed	23.919	10	2.392					
	Greenhouse-Geisser	23.919	7.406	3.230					
	Huynh-Feldt	23.919	9.786	2.444					
	Lower-bound	23.919	5.000	4.784					

Observed power computed using alpha = .05

Note: Individual data are contained in Table 5.2 (Chapter 5)

Output 5.7: One sample t-tests for slope, of the regression of differences, between each of the three comparisons and for all three variables

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
VO2 bxb v db	-1.498	5	.194	-6.9833E-02	-.1897	5.000E-02
CO2 bxb v db	-2.077	5	.092	-8.6333E-02	-.1932	2.051E-02
VE bxb v db	-1.089	5	.326	-2.7573E-02	-9.2676E-02	3.753E-02
VO2 bxb1 v bxb2	1.220	5	.277	4.067E-02	-4.4996E-02	.1263
CO2 bxb1 v bxb2	-.184	5	.862	-7.1667E-03	-.1075	9.321E-02
VE bxb1 v bxb2	1.213	5	.279	6.133E-02	-6.8684E-02	.1914
VO2 db1 v db2	-.063	5	.952	-4.1667E-03	-.1731	.1648
CO2 db1 v db2	.158	5	.881	9.333E-03	-.1425	.1612
VE db1 v db2	.437	5	.681	2.146E-02	-.1048	.1477

APPENDIX 6

Individual data and statistical output for Study 3 (Chapter 6)

Table A6.1: Individual data from Chapter 6

			Subject 1	2	3	4	5	6	7	8
	Age	(yrs)	32	22	24	21	24	18	18	20
	Height	(m)	1.84	1.80	1.73	1.74	1.82	1.78	1.79	1.90
	Mass	(kg)	64.0	81.1	64.8	70.8	64.1	65.0	70.2	70.6
Incremental	Duration	(s)	723	698	701	695	724	775	799	783
	$\dot{V}O_2peak$	($L.min^{-1}$)	4.13	5.22	4.23	4.63	4.32	4.61	5.14	5.67
	$\dot{V}O_2peak$	($ml.kg^{-1}min^{-1}$)	64.5	64.3	65.1	65.4	67.3	70.9	73.3	80.2
	HRpeak	($b.min^{-1}$)	196	193	185	199	190	193	194	188
800 m (track)	Duration	(s)	139.1	141.7	145.7	135.0	140.7	119.8	118.9	119.0
	Av. speed	($km.h^{-1}$)	20.7	20.3	19.8	21.3	20.5	24.1	24.2	24.2
	HRpeak	($b.min^{-1}$)	183	188	184	201	187	217	179	182
800 m (lab)	Duration	(s)	136.7	134.4	119.7	139.4	124.3	114.7	82.4	93.1
	Speed	($km.h^{-1}$)	21.7	213	20.8	22.3	21.5	25.1	25.2	25.2
	$\dot{V}O_2peak$	($L.min^{-1}$)	3.94	4.86	3.94	4.25	3.98	3.87	4.15	4.14
	$\dot{V}O_2peak$	($ml.kg^{-1}min^{-1}$)	61.6	60.0	60.8	60.0	62.1	59.5	59.2	58.6
	HRpeak	($b.min^{-1}$)	189	181	175	192	187	210	169	184
(initial 45s)	Asymptote	($L.min^{-1}$)	3.82	4.96	3.39	4.91	3.85	3.90	4.25	4.09
	τ	(s)	13.9	13.2	5.0	24.5	8.9	8.6	9.7	7.8

Table A6.2: Test duration from the pilot tests at the average track speed (n = 4) and at the speed adjusted for air resistance

Test duration (s)	
Adjusted speed	Average track speed
76	171
49	147
71	158
71	156

Output A6.1: One-way RM ANOVA (protocol) for HRpeak Chapter 5

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
PROTOCOL	.570	3.377	2	.182	.699	.820	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
PROTOCOL	Sphericity Assumed	168.583	2	84.292	1.595	.238	.186	3.189	.281
	Greenhouse-Geisser	168.583	1.398	120.573	1.595	.246	.186	2.229	.231
	Huynh-Feldt	168.583	1.640	102.812	1.595	.243	.186	2.615	.251
	Lower-bound	168.583	1.000	168.583	1.595	.247	.186	1.595	.195
Error(PROTOCOL)	Sphericity Assumed	740.083	14	52.863					
	Greenhouse-Geisser	740.083	9.787	75.617					
	Huynh-Feldt	740.083	11.478	64.478					
	Lower-bound	740.083	7.000	105.726					

Observed power computed using alpha = .05

Output A6.2: One-sample t-test for slope of $\dot{V}O_2$ response for final 30 s of laboratory based 800 m pace run

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
SLOPE	-.302	7	.771	-29.3825	-259.2680	200.5030

Note: Individual data are contained in Table 6.3

Output A6.3: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.276177626	.025144152	3.226298391	3.326056860
DELAY	6.974134049	1.342492829	4.310989403	9.637278695
CONSTANT	14.563863282	1.289185348	12.006466338	17.121260226

Output A6.4: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	4.043509804	.030934916	3.982233689	4.104785918
DELAY	11.713118428	.864246949	10.001211554	13.425025301
CONSTANT	11.929152850	.997786136	9.952730548	13.905575152

Output A6.5: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.143718226	.025144131	3.093839033	3.193597419
DELAY	7.575208453	1.296226545	5.003843669	10.146573237
CONSTANT	14.563854917	1.289183043	12.006462546	17.121247287

Output A6.6: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.760805792	.021941938	3.717343036	3.804268548
DELAY	10.417381016	.687541244	9.055494134	11.779267897
CONSTANT	16.163875206	.842003621	14.496028084	17.831722327

Output A6.7: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.314206349	.019847201	3.274853016	3.353559682
DELAY	12.357115706	.606829374	11.153885154	13.560346258
CONSTANT	9.806379224	.688787789	8.440640275	11.172118172

Output A6.8: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.229356512	.012506413	3.204528143	3.254184880
DELAY	12.293588308	.426297949	11.447279878	13.139896738
CONSTANT	7.138914910	.421642530	6.301848655	7.975981165

Output A6.9: Non-linear regression to model the $\dot{V}O_2$ response of subject 7 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.459700460	.033179040	3.393397440	3.526003480
DELAY	11.644785005	.965241809	9.715903166	13.573666845
CONSTANT	6.921884572	.903987849	5.115409003	8.728360140

Output A6.10:Non-linear regression to model the $\dot{V}O_2$ response of subject 8 to the laboratory based 800 m paced run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
ASSYM	3.381027825	.023093931	3.335001688	3.427053963
DELAY	13.369732188	.453231642	12.466442828	14.273021549
CONSTANT	8.082979131	.555015425	6.976834984	9.189123277

Output A6.11: Correlation matrix from Chapter 6

Correlations

		VO2peak	Asymptotic VO2	% VO2peak	Constant
VO2peak / kg (ramp)	Pearson Correlation	1.000	.035	-.951	-.690
	Sig. (2-tailed)	.	.935	.000	.058
	N	8	8	8	8
Asymptotic VO2 / kg	Pearson Correlation	.035	1.000	.263	.074
	Sig. (2-tailed)	.935	.	.529	.861
	N	8	8	8	8
% VO2peak achieved	Pearson Correlation	-.951	.263	1.000	.713
	Sig. (2-tailed)	.000	.529	.	.047
	N	8	8	8	8
Time constant	Pearson Correlation	-.690	.074	.713	1.000
	Sig. (2-tailed)	.058	.861	.047	.
	N	8	8	8	8

Output A6.12: Correlation matrix from Chapter 6 (Subject 8 removed)

Correlations

		VO2peak	Asymptotic VO2	% VO2peak	Constant
VO2peak / kg (ramp)	Pearson Correlation	1.000	.058	-.889	-.327
	Sig. (2-tailed)	.	.901	.007	.474
	N	7	7	7	7
Asymptotic VO2 / kg	Pearson Correlation	.058	1.000	.400	-.079
	Sig. (2-tailed)	.901	.	.374	.866
	N	7	7	7	7
% VO2peak achieved	Pearson Correlation	-.889	.400	1.000	.244
	Sig. (2-tailed)	.007	.374	.	.597
	N	7	7	7	7
Time constant	Pearson Correlation	-.327	-.079	.244	1.000
	Sig. (2-tailed)	.474	.866	.597	.
	N	7	7	7	7

APPENDIX 7

Individual data and statistical output for Study 4

Table A7.1: Individual subject data from Study 4

		Subject 1	2	3	4
Age	(yrs)	31	24	24	22
Height	(m)	1.94	1.88	1.78	1.69
Mass	(kg)	95.9	85.4	80.8	71.4
$\dot{V}O_2peak$	($ml.kg^{-1}min^{-1}$)	59.1	56.7	53.3	54.6

Output A7.1: Correlation of $1/\sqrt{n}$ with SD in the moderate domain

Correlations

		1/sqrt n	SD
1/sqrt n	Pearson Correlation	1.000	.990
	Sig. (2-tailed)	.	.001
	N	5	5
SD	Pearson Correlation	.990	1.000
	Sig. (2-tailed)	.001	.
	N	5	5

Output A7.2: Correlation of $1/\sqrt{n}$ with SD in the severe intensity domain

Correlations

		1/sqrt n	SD
1/sqrt n	Pearson Correlation	1.000	.996
	Sig. (2-tailed)	.	.000
	N	5	5
SD	Pearson Correlation	.996	1.000
	Sig. (2-tailed)	.000	.
	N	5	5

APPENDIX 8

Individual data and statistical output for Study 5 (Chapter 8)

Table A8.1: Individual subject and test data from Study 5

			Subject									
			1	2	3	4	5	6	7	8	9	10
	Age	(yrs)	24	23	23	19	20	25	34	19	25	24
	Height	(m)	1.78	1.83	1.75	1.78	1.71	1.77	1.77	1.78	1.83	1.84
	Mass	(kg)	70.1	76.2	69.2	67.0	70.7	71.8	60.6	74.6	67.5	81.6
Ramp	$\dot{V}O_2peak$	($l.min^{-1}$)	4.24	4.35	4.69	4.54	4.96	3.91	3.51	4.87	4.38	4.76
	$\dot{V}O_2peak$	($ml.kg^{-1}min^{-1}$)	60.5	57.1	67.8	67.7	70.2	54.4	58.0	65.2	64.9	58.3
	HRpeak	($b.min^{-1}$)	189	193	198	189	197	204	177	177	188	179
	Lac	(mM)	5.7	8.0	6.7	5.0	7.4	7.0	5.0	9.5	5.6	5.8
Moderate /	$\dot{V}O_2peak$	($l.min^{-1}$)	4.05	4.00	4.45	3.89	4.55	3.39	3.14	4.41	4.10	4.28
severe	$\dot{V}O_2peak$	(%ramp)	95.5	91.0	94.9	85.7	91.7	86.7	89.3	90.6	93.6	90.0
	Phase-1	(s)	14	17	13	16	13	12	16	12	16	12
	Baseline	($l.min^{-1}$)	0.98	0.63	0.75	0.65	0.94	0.63	0.70	0.75	0.72	0.60
	Gain	($l.min^{-1}$)	2.98	3.33	3.65	3.25	3.44	2.69	2.34	3.55	3.28	3.38
	Asymp	($l.min^{-1}$)	3.96	3.96	4.40	3.89	4.38	3.31	3.04	4.30	3.99	3.98
	τ	(s)	7.6	12.5	7.9	11.7	10.0	9.0	7.2	7.5	11.1	4.2
	δ	(s)	12.4	11.0	12.6	12.6	11.2	10.4	13.4	12.6	11.0	13.7
	MRT	(s)	20.0	23.5	20.5	24.2	21.2	19.3	20.6	20.1	22.1	17.9
	HRpeak	($b.min^{-1}$)	183	188	184	178	190	195	175	169	178	170
	Lac	(mM)	6.2	8.6	7.6	6.8	5.0	8.5	6.1	9.2	5.4	8.0
	Duration	(s)	119.5	104.5	103.0	102.0	104.0	128.0	113.0	113.5	117.5	96.5
Heavy /	$\dot{V}O_2peak$	($l.min^{-1}$)	4.02	4.00	4.59	3.91	4.73	3.59	3.29	4.14	4.05	4.35
severe	$\dot{V}O_2peak$	(%ramp)	94.7	92.0	97.8	86.2	95.2	92.0	93.7	85.0	92.5	91.4
	Phase-1	(s)	11	9	14	11	10	10	15	10	11	10
	Baseline	($l.min^{-1}$)	0.83	0.51	0.65	0.50	0.86	0.61	0.59	0.67	0.68	0.62
	Gain	($l.min^{-1}$)	3.06	3.49	3.89	3.41	3.78	2.89	2.65	3.34	3.28	3.64
	Asymp	($l.min^{-1}$)	3.89	4.00	4.54	3.90	4.64	3.50	3.23	4.01	3.96	4.26
	τ	(s)	6.6	16.4	8.2	13.8	8.9	12.4	6.6	7.7	9.8	5.8
	δ	(s)	11.7	2.0	12.3	4.0	10.1	6.7	11.8	9.8	10.1	14.1
	MRT	(s)	18.3	18.4	20.4	17.8	18.9	19.1	18.4	17.5	19.9	20.0
	HRpeak	($b.min^{-1}$)	189	191	186	187	197	193	176	177	181	180
	Lac	(mM)	6.7	9.4	6.2	8.3	6.7	7.8	4.7	9.0	5.7	6.8
	Duration	(s)	119.5	104.5	92.5	108.5	106.0	132.5	91.5	129.5	126.5	98.5

Output A8.1: One-way RM ANOVA (protocol) for $\dot{V}O_2$peak between the ramp test and constant intensity tests in Study 5

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
PROTOCOL	.797	1.815	2	.401	.831	.997	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
PROTOCOL	Sphericity Assumed	.945	2	.473	37.248	.000	.805	74.497	1.000
	Greenhouse-Geisser	.945	1.663	.568	37.248	.000	.805	61.926	1.000
	Huynh-Feldt	.945	1.993	.474	37.248	.000	.805	74.245	1.000
	Lower-bound	.945	1.000	.945	37.248	.000	.805	37.248	1.000
Error(PROTOCOL)	Sphericity Assumed	.228	18	1.269E-02					
	Greenhouse-Geisser	.228	14.963	1.526E-02					
	Huynh-Feldt	.228	17.939	1.273E-02					
	Lower-bound	.228	9.000	2.537E-02					

Observed power computed using alpha = .05

Output A8.2: One-way RM ANOVA (protocol) for HRpeak between the ramp test and constant intensity tests in Study 5

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
PROTOCOL	.871	1.100	2	.575	.886	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
PROTOCOL	Sphericity Assumed	343.817	2	171.908	22.259	.000	.712	44.518	1.000
	Greenhouse-Geisser	343.817	1.772	193.999	22.259	.000	.712	39.449	1.000
	Huynh-Feldt	343.817	2.000	171.908	22.259	.000	.712	44.518	1.000
	Lower-bound	343.817	1.000	343.817	22.259	.001	.712	22.259	.987
Error(PROTOCOL)	Sphericity Assumed	139.017	18	7.723					
	Greenhouse-Geisser	139.017	15.950	8.716					
	Huynh-Feldt	139.017	18.000	7.723					
	Lower-bound	139.017	9.000	15.446					

Observed power computed using alpha = .05

Output A8.3: One-way RM ANOVA (protocol) for lactate between the ramp test and constant intensity tests in Study 5

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
PROTOCOL	.978	.175	2	.919	.979	1.000	.500

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
PROTOCOL	Sphericity Assumed	2.222	2	1.111	1.483	.253	.141	2.965	.275
	Greenhouse-Geisser	2.222	1.958	1.135	1.483	.254	.141	2.903	.272
	Huynh-Feldt	2.222	2.000	1.111	1.483	.253	.141	2.965	.275
	Lower-bound	2.222	1.000	2.222	1.483	.254	.141	1.483	.194
Error(PROTOCOL)	Sphericity Assumed	13.486	18	.749					
	Greenhouse-Geisser	13.486	17.618	.765					
	Huynh-Feldt	13.486	18.000	.749					
	Lower-bound	13.486	9.000	1.498					

Observed power computed using alpha = .05

Output A8.4: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.975116557	.024187793	2.926875525	3.023357588
CONSTANT	7.548825378	.726960202	6.098948973	8.998701783
DELAY	12.410339797	.684384452	11.045378048	13.775301546

Output A8.5: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.331554269	.017211257	3.297227500	3.365881038
CONSTANT	12.498461014	.516185084	11.468962327	13.527959702
DELAY	10.967679815	.415842683	10.138307736	11.797051895

Output A8.6: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.651725760	.012729453	3.626398142	3.677053378
CONSTANT	7.914760847	.331190937	7.255794769	8.573726924
DELAY	12.610755708	.303005352	12.007870104	13.213641313

Output A8.7: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.244696458	.014610670	3.215556395	3.273836522
CONSTANT	11.646813642	.394796491	10.859416869	12.434210415
DELAY	12.582857996	.289592277	12.005284412	13.160431580

Output A8.8: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.435418020	.029939323	3.375890625	3.494945414
CONSTANT	9.949871501	.990449237	7.980593069	11.919149934
DELAY	11.204773544	.938305040	9.339171746	13.070375341

Output A8.9: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.684933689	.009688214	2.665727934	2.704139444
CONSTANT	8.946684796	.521606817	7.912660116	9.980709476
DELAY	10.366768291	.579184356	9.218602856	11.514933726

Output A8.10: Non-linear regression to model the $\dot{V}O_2$ response of subject 7 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.339381333	.014046228	2.311484311	2.367278355
CONSTANT	7.225376485	.551913740	6.129228159	8.321524811
DELAY	13.380812744	.483096021	12.421342346	14.340283141

Output A8.11: Non-linear regression to model the $\dot{V}O_2$ response of subject 8 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.553615147	.022944663	3.508010145	3.599220149
CONSTANT	7.501483196	.636954894	6.235466373	8.767500019
DELAY	12.639707547	.599489811	11.448156634	13.831258460

Output A8.12:Non-linear regression to model the $\dot{V}O_2$ response of subject 9 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.278670227	.015439512	3.248014714	3.309325739
CONSTANT	11.094718614	.578336615	9.946417709	12.243019520
DELAY	10.970631567	.535264689	9.907850980	12.033412155

Output A8.13:Non-linear regression to model the $\dot{V}O_2$ response of subject 10 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.380156393	.041753593	3.296838396	3.463474390
CONSTANT	4.190026371	.880902885	2.432212033	5.947840709
DELAY	13.703844494	.890903784	11.926073672	15.481615317

Output A8.14:Non-linear regression to model the $\dot{V}O_2$ response of subject 1 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.059060185	.018254825	3.022804527	3.095315843
CONSTANT	6.551728150	.697524330	5.166384621	7.937071678
DELAY	11.744129143	.784631204	10.185783844	13.302474441

Output A8.15:Non-linear regression to model the $\dot{V}O_2$ response of subject 2 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.486953071	.020836840	3.445523851	3.528382290
CONSTANT	16.432569562	1.166423126	14.113407893	18.751731230
DELAY	1.945495816	1.373334201	-.785060502	4.676052134

Output A8.16:Non-linear regression to model the $\dot{V}O_2$ response of subject 3 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.887738804	.017375895	3.853026464	3.922451145
CONSTANT	8.162206152	.384108608	7.394860995	8.929551308
DELAY	12.252827571	.348603810	11.556411402	12.949243740

Output A8.17:Non-linear regression to model the $\dot{V}O_2$ response of subject 4 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.404502532	.014090221	3.376487399	3.432517666
CONSTANT	13.759260678	.780486854	12.207443714	15.311077642
DELAY	4.002914116	.966347515	2.081556365	5.924271866

Output A8.18:Non-linear regression to model the $\dot{V}O_2$ response of subject 5 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.778031729	.030287524	3.717831996	3.838231462
CONSTANT	8.868767718	1.063396378	6.755152269	10.982383166
DELAY	10.059629744	1.196243733	7.681965793	12.437293696

Output A8.19: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.893992843	.010276143	2.873633950	2.914351737
CONSTANT	12.438035326	.687486483	11.076000609	13.800070043
DELAY	6.692609750	.823287535	5.061528656	8.323690845

Output A8.20: Non-linear regression to model the $\dot{V}O_2$ response of subject 7 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.644558631	.015443675	2.613696910	2.675420353
CONSTANT	6.585408961	.542755738	5.500798165	7.670019756
DELAY	11.807506740	.585022774	10.638432013	12.976581467

Output A8.21: Non-linear regression to model the $\dot{V}O_2$ response of subject 8 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.341781151	.015007559	3.312023935	3.371538368
CONSTANT	7.687403606	.714932301	6.269824919	9.104982292
DELAY	9.794183253	.911097066	7.987646075	11.600720430

Output A8.22: Non-linear regression to model the $\dot{V}O_2$ response of subject 9 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.277958323	.017215496	3.243834224	3.312082422
CONSTANT	9.790587651	.771904383	8.260539252	11.320636051
DELAY	10.119295038	.834215653	8.465734891	11.772855185

Output A8.23: Non-linear regression to model the $\dot{V}O_2$ response of subject 10 to the severe runs with prior moderate intensity exercise

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.636871565	.021921519	3.593182040	3.680561090
CONSTANT	5.842120009	.421251276	5.002567427	6.681672592
DELAY	14.123490883	.348896373	13.428141415	14.818840351

APPENDIX 9

Individual data and statistical output for Study 6 (Chapter 9)

Table A9.1: Individual subject and test data from Sprint group in Study 6

			Subject					
			1	2	3	4	5	6
	Age	(yrs)	23	24	24	27	15	15
	Height	(m)	1.76	1.81	1.73	1.76	1.76	1.81
	Mass	(kg)	79.2	76.0	76.9	74.9	61.2	74.1
	100m (PB)	(s)	11.7	10.9	10.6	11.0	11.3	11.1
Ramp	$\dot{V}O_2peak$	($l.min^{-1}$)	3.83	4.18	3.05	4.42	3.76	4.15
	$\dot{V}O_2peak$	($ml.kg^{-1}min^{-1}$)	50.2	60.5	40.0	62.0	61.0	53.3
	HRpeak	($b.min^{-1}$)	191	196	205	183	204	197
	Lac	(mM)	5.7	8.5	4.6	6.5	7.0	6.4
Moderate	Phase-1	(s)	14	13	18	13	17	21
	Baseline	($l.min^{-1}$)	0.56	0.58	0.29	0.66	0.50	0.55
	Gain	($l.min^{-1}$)	1.69	1.45	1.76	2.38	1.59	2.20
	Asymp	($l.min^{-1}$)	2.24	2.04	2.04	3.04	2.08	2.75
	τ	(s)	24.3	14.5	11.0	24.4	16.0	9.5
	δ	(s)	3.7	18.6	19.2	7.0	18.6	20.9
	MRT	(s)	28.0	33.1	30.2	31.4	34.6	30.4
Severe	$\dot{V}O_2peak$	($l.min^{-1}$)	3.73	4.20	3.60	4.23	3.24	4.08
	$\dot{V}O_2peak$	(%ramp)	97.2	100.5	118.1	95.7	85.9	98.3
	Phase-1	(s)	10	16	14	14	14	12
	Baseline	($l.min^{-1}$)	0.74	0.69	0.70	0.83	0.50	0.64
	Gain	($l.min^{-1}$)	2.91	3.38	2.70	3.28	2.64	3.39
	Asymp	($l.min^{-1}$)	3.65	4.08	3.40	4.11	3.14	4.02
	τ	(s)	12.6	12.2	11.5	9.7	11.0	10.1
	δ	(s)	6.4	12.6	12.3	11.4	8.6	13.2
	MRT	(s)	19.0	24.8	23.8	21.1	19.6	23.3
	HRpeak	($b.min^{-1}$)	178	193	195	171	197	192
	Lac	(mM)	7.7	7.0	5.4	8.7	8.2	8.2
	Duration	(s)	132.5	134.5	114.0	98.0	112.0	102.5

Table A9.2: Individual subject and test data from Endurance group in Study 6

			Subject					
			1	2	3	4	5	6
	Age	(yrs)	19	19	23	25	24	20
	Height	(m)	1.78	1.78	1.75	1.83	1.78	1.71
	Mass	(kg)	74.6	67.0	69.2	67.5	70.1	70.7
	10km (PB)	(min)	31.7	32.1	31.5	32.9	33.9	33.8
Ramp	$\dot{V}O_2$peak	($l.min^{-1}$)	4.87	4.54	4.69	4.38	4.24	4.96
	$\dot{V}O_2$peak	($ml.kg^{-1}min^{-1}$)	65.2	67.7	67.8	64.9	60.5	70.2
	HRpeak	($b.min^{-1}$)	177	189	198	188	189	197
	Lac	(mM)	9.5	5.0	6.7	5.6	5.7	7.4
Moderate	Phase-1	(s)	22	17	17	14	15	19
	Baseline	($l.min^{-1}$)	0.46	0.36	0.75	0.53	0.57	0.48
	Gain	($l.min^{-1}$)	2.87	2.49	2.23	1.98	2.22	2.08
	Asymp	($l.min^{-1}$)	3.33	2.85	2.98	2.52	2.79	2.56
	τ	(s)	10.7	11.7	10.8	16.2	10.6	14.0
	δ	(s)	15.8	13.7	14.0	8.0	12.9	11.5
	MRT	(s)	26.5	25.4	24.8	24.2	23.5	25.5
Severe	$\dot{V}O_2$peak	($l.min^{-1}$)	4.41	3.89	4.45	4.10	4.05	4.55
	$\dot{V}O_2$peak	(%ramp)	90.6	85.7	94.9	93.6	95.5	91.7
	Phase-1	(s)	12	16	13	16	14	13
	Baseline	($l.min^{-1}$)	0.75	0.65	0.75	0.72	0.98	0.94
	Gain	($l.min^{-1}$)	3.55	3.25	3.65	3.28	2.98	3.44
	Asymp	($l.min^{-1}$)	4.30	3.89	4.40	3.99	3.96	4.38
	τ	(s)	7.5	11.7	7.9	11.1	7.6	10.0
	δ	(s)	12.6	12.6	12.6	11.0	12.4	11.2
	MRT	(s)	20.1	24.2	20.5	22.1	20.0	21.2
	HRpeak	($b.min^{-1}$)	169	178	184	178	183	190
	Lac	(mM)	9.2	6.8	7.6	5.4	6.2	5.0
	Duration	(s)	113.5	102.0	103.0	117.5	119.5	104.0

Output A9.1: Two way RM ANOVA (test x group) for HR peak in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
TEST	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
TEST	Sphericity Assumed	463.760	1	463.760	58.137	.000	.853	58.137	1.000
	Greenhouse-Geisser	463.760	1.000	463.760	58.137	.000	.853	58.137	1.000
	Huynh-Feldt	463.760	1.000	463.760	58.137	.000	.853	58.137	1.000
	Lower-bound	463.760	1.000	463.760	58.137	.000	.853	58.137	1.000
TEST * GROUP	Sphericity Assumed	.844	1	.844	.106	.752	.010	.106	.060
	Greenhouse-Geisser	.844	1.000	.844	.106	.752	.010	.106	.060
	Huynh-Feldt	.844	1.000	.844	.106	.752	.010	.106	.060
	Lower-bound	.844	1.000	.844	.106	.752	.010	.106	.060
Error(TEST)	Sphericity Assumed	79.771	10	7.977					
	Greenhouse-Geisser	79.771	10.000	7.977					
	Huynh-Feldt	79.771	10.000	7.977					
	Lower-bound	79.771	10.000	7.977					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	851455.010	1	851455.010	6632.453	.000	.998	6632.453	1.000
GROUP	283.594	1	283.594	2.209	.168	.181	2.209	.270
Error	1283.771	10	128.377					

Observed power computed using alpha = .05

Output A9.2: Two way RM ANOVA (test x group) for lactate in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
TEST	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
TEST	Sphericity Assumed	1.086	1	1.086	1.325	.276	.117	1.325	.181
	Greenhouse-Geisser	1.086	1.000	1.086	1.325	.276	.117	1.325	.181
	Huynh-Feldt	1.086	1.000	1.086	1.325	.276	.117	1.325	.181
	Lower-bound	1.086	1.000	1.086	1.325	.276	.117	1.325	.181
TEST * GROUP	Sphericity Assumed	2.597	1	2.597	3.169	.105	.241	3.169	.363
	Greenhouse-Geisser	2.597	1.000	2.597	3.169	.105	.241	3.169	.363
	Huynh-Feldt	2.597	1.000	2.597	3.169	.105	.241	3.169	.363
	Lower-bound	2.597	1.000	2.597	3.169	.105	.241	3.169	.363
Error(TEST)	Sphericity Assumed	8.194	10	.819					
	Greenhouse-Geisser	8.194	10.00	.819					
	Huynh-Feldt	8.194	10.00	.819					
	Lower-bound	8.194	10.00	.819					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	413.420	1	413.420	1804.155	.000	.994	1804.155	1.000
GROUP	1.854	1	1.854	8.089	.017	.447	8.089	.727
Error	2.291	10	.229					

Observed power computed using alpha = .05

Output A9.3: Two way RM ANOVA (test x group) for $\dot{V}O_2$peak in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
TEST	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
TEST	Sphericity Assumed	.268	1	.268	7.143	.023	.417	7.143	.674
	Greenhouse-Geisser	.268	1.000	.268	7.143	.023	.417	7.143	.674
	Huynh-Feldt	.268	1.000	.268	7.143	.023	.417	7.143	.674
	Lower-bound	.268	1.000	.268	7.143	.023	.417	7.143	.674
TEST * GROUP	Sphericity Assumed	.153	1	.153	4.080	.071	.290	4.080	.447
	Greenhouse-Geisser	.153	1.000	.153	4.080	.071	.290	4.080	.447
	Huynh-Feldt	.153	1.000	.153	4.080	.071	.290	4.080	.447
	Lower-bound	.153	1.000	.153	4.080	.071	.290	4.080	.447
Error(TEST)	Sphericity Assumed	.376	10	3.759E-02					
	Greenhouse-Geisser	.376	10.000	3.759E-02					
	Huynh-Feldt	.376	10.000	3.759E-02					
	Lower-bound	.376	10.000	3.759E-02					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	851455.010	1	851455.010	6632.453	.000	.998	6632.453	1.000
GROUP	283.594	1	283.594	2.209	.168	.181	2.209	.270
Error	1283.771	10	128.377					

Observed power computed using alpha = .05

Output A9.4: Two way RM ANOVA (intensity x group) for baseline in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	283620.0	1	283620.04	23.990	.001	.706	23.990	.992
	Greenhouse-Geisser	283620.0	1.000	283620.04	23.990	.001	.706	23.990	.992
	Huynh-Feldt	283620.0	1.000	283620.04	23.990	.001	.706	23.990	.992
	Lower-bound	283620.0	1.000	283620.04	23.990	.001	.706	23.990	.992
INTENSITY * GROUP	Sphericity Assumed	18760.04	1	18760.042	1.587	.236	.137	1.587	.207
	Greenhouse-Geisser	18760.04	1.000	18760.042	1.587	.236	.137	1.587	.207
	Huynh-Feldt	18760.04	1.000	18760.042	1.587	.236	.137	1.587	.207
	Lower-bound	18760.04	1.000	18760.042	1.587	.236	.137	1.587	.207
Error(INTENSITY)	Sphericity Assumed	118224.4	10	11822.442					
	Greenhouse-Geisser	118224.4	10.000	11822.442					
	Huynh-Feldt	118224.4	10.000	11822.442					
	Lower-bound	118224.4	10.000	11822.442					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	9564700.042	1	9564700.042	483.602	.000	.980	483.602	1.000
GROUP	19780.042	1	19780.042	1.000	.341	.091	1.000	.148
Error	197780.417	10	19778.042					

Observed power computed using alpha = .05

Output A9.5: Two way RM ANOVA (intensity x group) for phase-1 duration in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	54.000	1	54.000	5.978	.035	.374	5.978	.598
	Greenhouse-Geisser	54.000	1.000	54.000	5.978	.035	.374	5.978	.598
	Huynh-Feldt	54.000	1.000	54.000	5.978	.035	.374	5.978	.598
	Lower-bound	54.000	1.000	54.000	5.978	.035	.374	5.978	.598
INTENSITY * GROUP	Sphericity Assumed	.667	1	.667	.074	.791	.007	.074	.057
	Greenhouse-Geisser	.667	1.000	.667	.074	.791	.007	.074	.057
	Huynh-Feldt	.667	1.000	.667	.074	.791	.007	.074	.057
	Lower-bound	.667	1.000	.667	.074	.791	.007	.074	.057
Error(INTENSITY)	Sphericity Assumed	90.333	10	9.033					
	Greenhouse-Geisser	90.333	10.000	9.033					
	Huynh-Feldt	90.333	10.000	9.033					
	Lower-bound	90.333	10.000	9.033					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	5520.667	1	5520.667	1440.174	.000	.993	1440.174	1.000
GROUP	6.000	1	6.000	1.565	.239	.135	1.565	.205
Error	38.333	10	3.833					

Observed power computed using alpha = .05

Output A9.6: Two way RM ANOVA (intensity x group) for GAIN in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	7590375.3	1	7590375.3	115.348	.000	.920	115.348	1.000
	Greenhouse-Geisser	7590375.3	1.000	7590375.3	115.348	.000	.920	115.348	1.000
	Huynh-Feldt	7590375.3	1.000	7590375.3	115.348	.000	.920	115.348	1.000
	Lower-bound	7590375.3	1.000	7590375.3	115.348	.000	.920	115.348	1.000
INTENSITY * GROUP	Sphericity Assumed	38962.04	1	38962.042	.592	.459	.056	.592	.107
	Greenhouse-Geisser	38962.04	1.000	38962.042	.592	.459	.056	.592	.107
	Huynh-Feldt	38962.04	1.000	38962.042	.592	.459	.056	.592	.107
	Lower-bound	38962.04	1.000	38962.042	.592	.459	.056	.592	.107
Error(INTENSITY)	Sphericity Assumed	658044.0	10	65804.408					
	Greenhouse-Geisser	658044.0	10.000	65804.408					
	Huynh-Feldt	658044.0	10.000	65804.408					
	Lower-bound	658044.0	10.000	65804.408					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	167286240.3	1	167286240.3	1181.661	.000	.992	1181.661	1.000
GROUP	899775.375	1	899775.375	6.356	.030	.389	6.356	.624
Error	1415687.750	10	141568.775					

Observed power computed using alpha = .05

Output A9.7: Two way RM ANOVA (intensity x group) for asymptote in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	10808468	1	10808468.1	188.139	.000	.950	188.139	1.000
	Greenhouse-Geisser	10808468	1.000	10808468.1	188.139	.000	.950	188.139	1.000
	Huynh-Feldt	10808468	1.000	10808468.1	188.139	.000	.950	188.139	1.000
	Lower-bound	10808468	1.000	10808468.1	188.139	.000	.950	188.139	1.000
INTENSITY * GROUP	Sphericity Assumed	3650.667	1	3650.667	.064	.806	.006	.064	.056
	Greenhouse-Geisser	3650.667	1.000	3650.667	.064	.806	.006	.064	.056
	Huynh-Feldt	3650.667	1.000	3650.667	.064	.806	.006	.064	.056
	Lower-bound	3650.667	1.000	3650.667	.064	.806	.006	.064	.056
Error(INTENSITY)	Sphericity Assumed	574493.1	10	57449.317					
	Greenhouse-Geisser	574493.1	10.000	57449.317					
	Huynh-Feldt	574493.1	10.000	57449.317					
	Lower-bound	574493.1	10.000	57449.317					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	256852008.1	1	256852008.1	1379.673	.000	.993	1379.673	1.000
GROUP	1186370.667	1	1186370.667	6.373	.030	.389	6.373	.625
Error	1861687.167	10	186168.717					

Observed power computed using alpha = .05

Output A9.8: Two way RM ANOVA (intensity x group) for asymptote (as a % $\dot{V}O_2$peak) in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	.615	1	.615	184.877	.000	.949	184.877	1.000
	Greenhouse-Geisser	.615	1.000	.615	184.877	.000	.949	184.877	1.000
	Huynh-Feldt	.615	1.000	.615	184.877	.000	.949	184.877	1.000
	Lower-bound	.615	1.000	.615	184.877	.000	.949	184.877	1.000
INTENSITY * GROUP	Sphericity Assumed	7.212E-03	1	7.212E-03	2.169	.172	.178	2.169	.266
	Greenhouse-Geisser	7.212E-03	1.000	7.212E-03	2.169	.172	.178	2.169	.266
	Huynh-Feldt	7.212E-03	1.000	7.212E-03	2.169	.172	.178	2.169	.266
	Lower-bound	7.212E-03	1.000	7.212E-03	2.169	.172	.178	2.169	.266
Error(INTENSITY)	Sphericity Assumed	3.325E-02	10	3.325E-03					
	Greenhouse-Geisser	3.325E-02	10.000	3.325E-03					
	Huynh-Feldt	3.325E-02	10.000	3.325E-03					
	Lower-bound	3.325E-02	10.000	3.325E-03					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	14.281	1	14.281	2298.835	.000	.996	2298.835	1.000
GROUP	4.330E-03	1	4.330E-03	.697	.423	.065	.697	.118
Error	6.212E-02	10	6.212E-03					

Observed power computed using alpha = .05

Output A9.9: Two way RM ANOVA (intensity x group) for τ in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	107.950	1	107.950	9.795	.011	.495	9.795	.805
	Greenhouse-Geisser	107.950	1.000	107.950	9.795	.011	.495	9.795	.805
	Huynh-Feldt	107.950	1.000	107.950	9.795	.011	.495	9.795	.805
	Lower-bound	107.950	1.000	107.950	9.795	.011	.495	9.795	.805
INTENSITY * GROUP	Sphericity Assumed	8.520	1	8.520	.773	.400	.072	.773	.125
	Greenhouse-Geisser	8.520	1.000	8.520	.773	.400	.072	.773	.125
	Huynh-Feldt	8.520	1.000	8.520	.773	.400	.072	.773	.125
	Lower-bound	8.520	1.000	8.520	.773	.400	.072	.773	.125
Error(INTENSITY)	Sphericity Assumed	110.214	10	11.021					
	Greenhouse-Geisser	110.214	10.000	11.021					
	Huynh-Feldt	110.214	10.000	11.021					
	Lower-bound	110.214	10.000	11.021					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	3663.010	1	3663.010	249.585	.000	.961	249.585	1.000
GROUP	57.350	1	57.350	3.908	.076	.281	3.908	.431
Error	146.764	10	14.676					

Observed power computed using alpha = .05

Output A9.10:Two way RM ANOVA (intensity x group) for δ in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	30.827	1	30.827	3.112	.108	.237	3.112	.358
	Greenhouse-Geisser	30.827	1.000	30.827	3.112	.108	.237	3.112	.358
	Huynh-Feldt	30.827	1.000	30.827	3.112	.108	.237	3.112	.358
	Lower-bound	30.827	1.000	30.827	3.112	.108	.237	3.112	.358
INTENSITY * GROUP	Sphericity Assumed	16.335	1	16.335	1.649	.228	.142	1.649	.214
	Greenhouse-Geisser	16.335	1.000	16.335	1.649	.228	.142	1.649	.214
	Huynh-Feldt	16.335	1.000	16.335	1.649	.228	.142	1.649	.214
	Lower-bound	16.335	1.000	16.335	1.649	.228	.142	1.649	.214
Error(INTENSITY)	Sphericity Assumed	99.068	10	9.907					
	Greenhouse-Geisser	99.068	10.000	9.907					
	Huynh-Feldt	99.068	10.000	9.907					
	Lower-bound	99.068	10.000	9.907					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	3765.015	1	3765.015	153.544	.000	.939	153.544	1.000
GROUP	.807	1	.807	.033	.860	.003	.033	.053
Error	245.208	10	24.521					

Observed power computed using alpha = .05

Output A9.11:Two way RM ANOVA (intensity x group) for MRT in Study 6

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
INTENSITY	1.000	.000	0	.	1.000	1.000	1.000

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
INTENSITY	Sphericity Assumed	254.150	1	254.150	78.480	.000	.887	78.480	1.000
	Greenhouse-Geisser	254.150	1.000	254.150	78.480	.000	.887	78.480	1.000
	Huynh-Feldt	254.150	1.000	254.150	78.480	.000	.887	78.480	1.000
	Lower-bound	254.150	1.000	254.150	78.480	.000	.887	78.480	1.000
INTENSITY * GROUP	Sphericity Assumed	48.450	1	48.450	14.961	.003	.599	14.961	.935
	Greenhouse-Geisser	48.450	1.000	48.450	14.961	.003	.599	14.961	.935
	Huynh-Feldt	48.450	1.000	48.450	14.961	.003	.599	14.961	.935
	Lower-bound	48.450	1.000	48.450	14.961	.003	.599	14.961	.935
Error(INTENSITY)	Sphericity Assumed	32.384	10	3.238					
	Greenhouse-Geisser	32.384	10.000	3.238					
	Huynh-Feldt	32.384	10.000	3.238					
	Lower-bound	32.384	10.000	3.238					

Observed power computed using alpha = .05

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power
Intercept	14855.350	1	14855.350	3562.933	.000	.997	3562.933	1.000
GROUP	71.760	1	71.760	17.211	.002	.633	17.211	.961
Error	41.694	10	4.169					

Observed power computed using alpha = .05

Output A9.12: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	12.585594682	1.502845334	9.607899294	15.563290069
DELAY	6.442512931	1.810844229	2.854557214	10.030468648
GAIN	2.908227465	.022341768	2.863960116	2.952494814

Output A9.13: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	12.221074498	.765049746	10.705077354	13.737071642
DELAY	12.647732248	.604848878	11.449183817	13.846280679
GAIN	3.382646715	.021864400	3.339320947	3.425972484

Output A9.14: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	11.519923948	.897569737	9.737778195	13.302069702
DELAY	12.349455359	.725545013	10.908868728	13.790041989
GAIN	2.698274690	.022274089	2.654048965	2.742500415

Output A9.15: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	9.667430116	.967870619	7.741306201	11.593554031
DELAY	11.393289267	.906969271	9.588362897	13.198215637
GAIN	3.276719279	.029475843	3.218060482	3.335378077

Output A9.16: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	11.013746213	.836132687	9.352368374	12.675124051
DELAY	8.561894585	.928650080	6.716686656	10.407102514
GAIN	2.644121166	.014966101	2.614383841	2.673858490

Output A9.17: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 of the Sprint group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
CONSTANT	10.073188441	.710021193	8.659058679	11.487318203
DELAY	13.175331289	.541812482	12.096218209	14.254444370
GAIN	3.384782492	.027636133	3.329740364	3.439824621

Output A9.18: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.553615147	.022944663	3.508010145	3.599220149
CONSTANT	7.501483196	.636954894	6.235466373	8.767500019
DELAY	12.639707547	.599489811	11.448156634	13.831258460

Output A9.19: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.244696458	.014610670	3.215556395	3.273836522
CONSTANT	11.646813642	.394796491	10.859416869	12.434210415
DELAY	12.582857996	.289592277	12.005284412	13.160431580

Output A9.20: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.651725760	.012729453	3.626398142	3.677053378
CONSTANT	7.914760847	.331190937	7.255794769	8.573726924
DELAY	12.610755708	.303005352	12.007870104	13.213641313

Output A9.21: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.278670227	.015439512	3.248014714	3.309325739
CONSTANT	11.094718614	.578336615	9.946417709	12.243019520
DELAY	10.970631567	.535264689	9.907850980	12.033412155

Output A9.22: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.975116557	.024187793	2.926875525	3.023357588
CONSTANT	7.548825378	.726960202	6.098948973	8.998701783
DELAY	12.410339797	.684384452	11.045378048	13.775301546

Output A9.23: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 of the Endurance group to the severe run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	3.435418020	.029939323	3.375890625	3.494945414
CONSTANT	9.949871501	.990449237	7.980593069	11.919149934
DELAY	11.204773544	.938305040	9.339171746	13.070375341

Output A9.24: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 of the Sprint group to the moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	1.684954746	.018192975	1.649166707	1.720742785
CONSTANT	24.271973680	6.248694951	11.979946782	36.564000577
DELAY	3.719837982	8.834887865	-13.65957966	21.099255624

Output A9.25: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 of the Sprint group to the moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	1.450880823	.009085803	1.433007822	1.468753823
CONSTANT	14.459911270	1.905910561	10.710727790	18.209094750
DELAY	18.566755765	2.029969597	14.573531408	22.559980123

Output A9.26: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 of the Sprint group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	1.757423158	.011145612	1.735498235	1.779348082
CONSTANT	11.039848792	1.862536528	7.375987892	14.703709691
DELAY	19.241518357	2.146431769	15.019197176	23.463839537

Output A9.27: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 of the Sprint group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.384445165	.015585234	2.353786905	2.415103425
CONSTANT	24.400247397	3.292357472	17.923735594	30.876759201
DELAY	7.044264535	4.225622039	-1.268104714	15.356633784

Output A9.28: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 of the Sprint group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	1.586284662	.008514763	1.569534973	1.603034350
CONSTANT	16.011616238	1.628478455	12.808179180	19.215053297
DELAY	18.603236192	1.657869262	15.341983446	21.864488937

Output A9.29: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 of the Sprint group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.199703233	.011881751	2.176330225	2.223076242
CONSTANT	9.522965680	1.360442191	6.846792154	12.199139207
DELAY	20.912861360	1.470089067	18.020997620	23.804725099

Output A9.30: Non-linear regression to model the $\dot{V}O_2$ response of subject 1 of the Endurance group to the moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.874447776	.012415234	2.850026133	2.898869419
CONSTANT	10.715644095	1.329717598	8.099995688	13.331292501
DELAY	15.822993658	1.601428075	12.672871558	18.973115757

Output A9.31: Non-linear regression to model the $\dot{V}O_2$ response of subject 2 of the Endurance group to the moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.494340599	.007756275	2.479084271	2.509596927
CONSTANT	11.653643956	.747788188	10.182770215	13.124517698
DELAY	13.658438548	.705539434	12.270666657	15.046210439

Output A9.32: Non-linear regression to model the $\dot{V}O_2$ response of subject 3 of the Endurance group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.232188385	.009884010	2.212746876	2.251629893
CONSTANT	10.761212500	1.022662855	8.749669706	12.772755294
DELAY	13.991219148	.964052950	12.094960030	15.887478267

Output A9.33: Non-linear regression to model the $\dot{V}O_2$ response of subject 4 of the Endurance group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	1.978445198	.013307083	1.952271440	2.004618956
CONSTANT	16.160595300	2.024337867	12.178916483	20.142274118
DELAY	8.024912765	2.010215253	4.071011778	11.978813752

Output A9.34: Non-linear regression to model the $\dot{V}O_2$ response of subject 5 of the Endurance group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.221424620	.012868734	2.196112789	2.246736451
CONSTANT	10.572800863	1.230013692	8.153456618	12.992145108
DELAY	12.889501257	1.078873279	10.767438816	15.011563699

Output A9.35: Non-linear regression to model the $\dot{V}O_2$ response of subject 6 of the Endurance group to the very moderate run

Parameter	Estimate	Asymptotic Std. Error	Asymptotic 95 % Confidence Interval Lower	Upper
GAIN	2.077864294	.017540299	2.043362397	2.112366191
CONSTANT	14.042480979	2.795707714	8.543303467	19.541658492
DELAY	11.527175143	3.202063516	5.228692797	17.825657489

Output A9.36: Correlation between $\dot{V}O_2$peak from the ramp test and % $\dot{V}O_2$peak achieved in the severe intensity run

		VO2PEAK	PERCENT
VO2PEAK	Pearson Correlation	1.000	-.811
	Sig. (2-tailed)	.	.001
	N	12	12
PERCENT	Pearson Correlation	-.811	1.000
	Sig. (2-tailed)	.001	.
	N	12	12

** Correlation is significant at the 0.01 level (2-tailed).

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