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THE PHYSIOLOGY  
OF THE  
PURSUIT CYCLE RACE

by

PETER KEEN

A thesis submitted for the degree of

MASTER OF PHILOSOPHY  
(Human Biology)

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## ABSTRACT

The principal aim of this thesis was to develop a greater understanding of the physiological mechanisms underlying performance in the individual pursuit cycle race. Three separate topics were addressed: The suitability of various laboratory measures of physiological function, the relationship between pursuit performance and selected physiological indices, and the longitudinal training responses of pursuit cyclists.

The aerobic power of national squad pursuit cyclists was assessed in two ways.  $\dot{V}O_{2max}$  was measured on a specially modified ergometer using a protocol optimized for cyclists. Submaximal blood lactate responses to incremental were evaluated with the 4 mmol.l<sup>-1</sup> lactate threshold measure. A pilot study on 11 cyclists found this to be an accurate index of the maximum power output that could be sustained under true steady state conditions. No suitable test of anaerobic power could be found so this variable was not directly measured.

Performance in the 1987 British Championships was correlated with laboratory data measured just prior to competition for 9 pursuit cyclists. Significant relationships were found between race speed and absolute values of  $\dot{V}O_{2max}$  ( $r = 0.63$ ,  $p < 0.05$ ), and Power output at 4 mmol.l<sup>-1</sup> lactate ( $r = 0.93$ ,  $p < 0.01$ ). However, when these variables were related to body mass, body mass<sup>-0.667</sup> or body surface area reduced correlations were observed. No relationship between post race blood lactate levels and performance was found.

It was concluded that pursuit racing performance is primarily limited by the metabolic acidosis arising from the failure to deliver sufficient oxygen to the mitochondria of the exercising musculature. At elite levels of competition, heavier cyclists appear to possess an advantage over their lighter rivals due to a higher absolute work capacity. The measurement of power output at 4 mmol.l<sup>-1</sup> lactate was found to be the most appropriate measure of pursuit performance potential, and the most sensitive index of long term training responses in competitive cyclists.

This thesis is the result of an attempt to integrate two challenges which have fascinated me for some time, the quest for sporting excellence and the search for a greater knowledge of the mechanisms underlying human performance.

Although humble in its goals and limited in its conclusions this work nevertheless represents the achievement of an important personal goal, one that could not have been contemplated, much less attempted without the continuous support and inspiration of one man. It is therefore with a deep sense of personal gratitude that I dedicate this thesis to Gordon Wright, a revered friend and educator.

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A study such as this could not have succeeded without the excellent co-operation of the subjects, who were not only willing to travel long distances to participate in experiments but also demonstrated great tolerance during data collection, even when this occurred in competition. I am deeply indebted to them all.

The West Sussex Institute of Higher Education has provided a warm and flexible environment in which to work and I am very grateful for the support of the staff and all my colleagues at the Institute.

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PART ONE

STATEMENT OF THE PROBLEM

AND

REVIEW OF LITERATURE

## Chapter One

### OVERVIEW

The significance modern society attaches to International Sport was clearly demonstrated by the spectacle of the recent Olympic Games in Seoul, South Korea. New levels of excellence were achieved across the spectrum of disciplines, in terms of both world records and quality of competition. Sports Scientists can claim some of the credit for the remarkable rise in athletic performances witnessed in recent years. However, in many events standards have improved at a much greater rate than our scientific knowledge of the limitations to specific forms of human performance. Consequently, areas of concern such as athlete suitability, excessive training regimes and drug abuse cannot yet be fully addressed by researchers. One sport in which this lack of understanding of limiting factors is apparent is Cycle racing.

This thesis describes a programme of research designed to increase the understanding of performance limitations in a single cycling event, the individual pursuit track race. The aims of the project were:

1. To investigate the physiological mechanisms underlying individual pursuit race performance.
2. To evaluate the suitability and effectiveness of the training programmes and race strategies of pursuit cyclists.
3. To provide an experimental model on which subsequent investigations of other cycling events or similar sports can be based.

The structure of the thesis is intended to reflect the logical progression of the project from the construction of a research strategy, through the phases of data collection to the practical application of the findings to athletic competition.

The introduction describes the physical nature of the pursuit race and reviews the findings of a pilot study. The physiological

mechanisms requiring investigation are identified in broad terms, and discussed. A statement of the research objectives is made, together with a brief description of the approach to data collection.

Physiological research relating to athletic performance is discussed in the review of literature. The information drawn from this exercise, together with previous observations was used to formulate the series of investigations carried out. The section concludes with a statement of the hypotheses to be tested.

Experimental work was divided into a three discrete phases. For the sake of clarity each investigation is reported and discussed separately, in chronological order.

A general discussion of the results follows, from which a number of conclusions about the physiology of pursuit racing are made. The thesis ends with an examination of the practical implications of the findings, both for training and competition.

## Chapter Two

### INTRODUCTION

Organised competitive cycling originated well over a century ago and has been an Olympic sport since the first modern Games in Athens, 1896. The individual track pursuit appeared as an international event in 1896 and is therefore a highly evolved form of cycle competition. The format of the race is as follows. Two riders are positioned on the straights of the track diametrically opposite each other. The objective is to either catch one's opponent before completing the distance or record a faster time. The former rarely occurs today in top competition unless the track is unusually small, thereby reducing the time gap between competitors. Women and juniors (<19 years of age) compete over a distance of 3000m, senior amateurs 4000m and professionals 5000m. Table 1 shows the current world records and best times for these various categories, together with the best times achieved by British competitors. It should be noted that world records can only be set in time trials, due to the possibility that an opponent could either hinder or aid a rider. Times recorded in competition are therefore classified "worlds best".

TABLE 1. Current World and British pursuit records and fastest competition times (min.sec).

Category (distance)	World Record	Best time in competition	fastest time by british rider
Juniors (3000m)	-----	3.21.53	3.34.62*
Women (3000m)	3.46.23	3.39.20*	3.53.23**
Amateurs (4000m)	4.37.61*	4.27.02**	4.30.85**
Professionals (5000m)	5.44.70*	5.40.33*	5.40.33*

\* Denotes time established at an altitude above 2000m

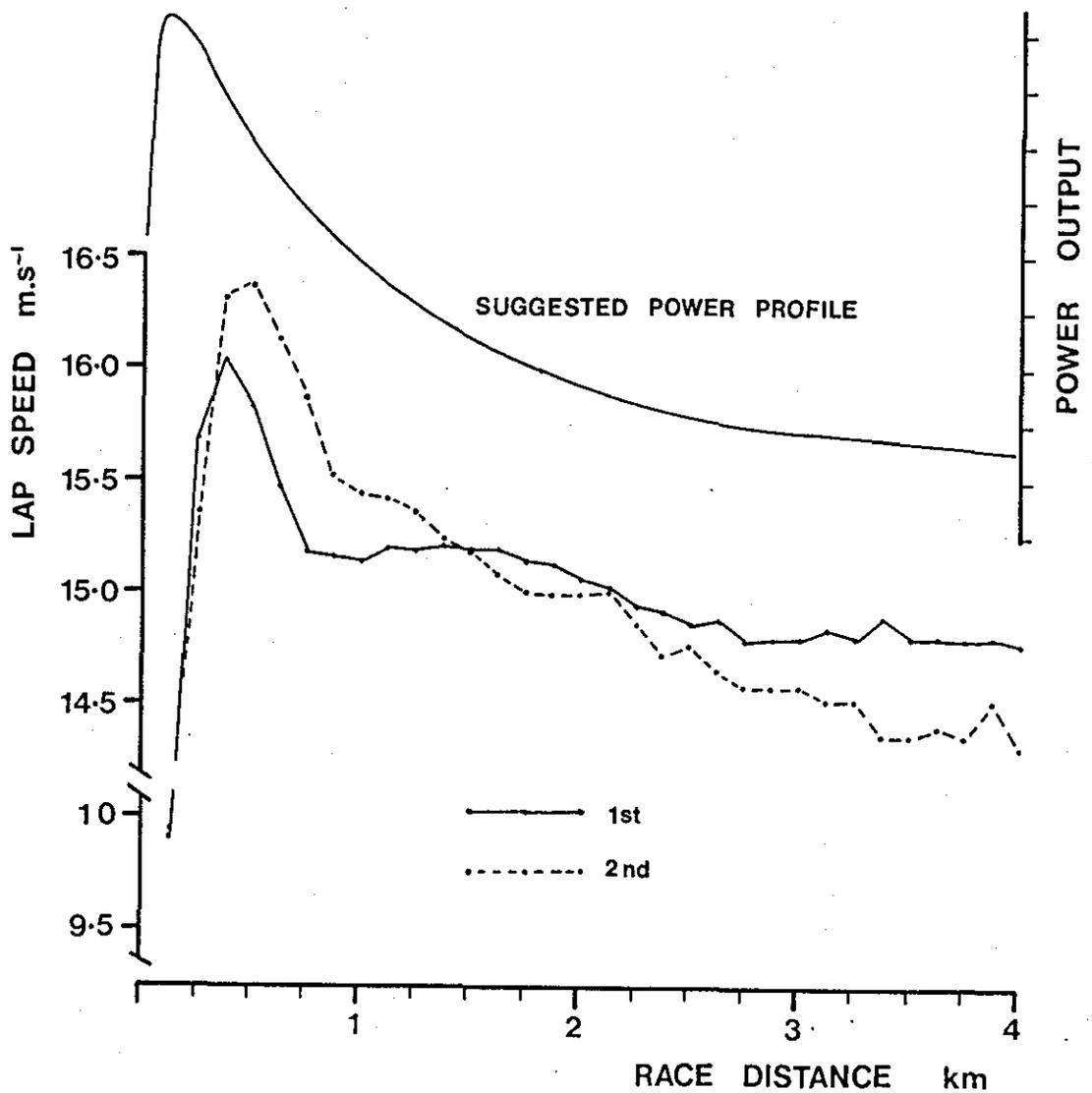
\*\* Denotes time established on an indoor velodrome

The physical stress of pursuit racing is best described diagrammatically. Figure 1 shows a plot of lap speed against distance for the riders Guintautas Umaras and Viatcheslav Ekimov (both USSR) as they raced in the final of the 1987 World Championship. This profile is typical of pursuit racing at elite levels (Wilberg and Pratt, 1988). Both riders achieved peak speeds within 60 seconds of the start followed by a gradual decline in speed reflecting fatigue. Final times (min.sec) for the respective riders were 4.29.58 and 4.30.52.

Also shown in figure 1 is a theoretical plot of energy production (power output) corresponding to the speed profile of the Russian riders (the Y-axis is intentionally unitless). Peak power output will occur soon after the start as the rapid acceleration of the high inertial load of rider and bicycle (typically 85 kg) requires a rapid production of energy. The power decay appears exaggerated in relation to the speed plot. This is due to the curvilinear relationship between cycling speed and power output (Sjogaard et.al., 1982) and the fact that the kinetic energy stored during acceleration will be expended during deceleration.

The primary objective of this thesis was to investigate the physiological mechanisms that enabled the pursuit riders to produce the power required for propulsion around the track in the manner described above. A thorough review of literature failed to reveal a study in which this question has been directly addressed. Clearly, pursuit racing bears little resemblance, other than duration, to middle distance running where pace is dictated by bunch tactics and peak power output occurs in the final stages of the race. Furthermore, the energy cost of running is directly related to body mass. This may not be the case in pursuit cycling where air resistance provides the major resistance to motion (Sjogaard et al. 1982). The findings of studies on running are therefore of limited value. It was thus necessary to commence this research by considering the basic concepts concerned with performance of muscular exercise in humans.

FIGURE 1. Speed and theoretical power output profiles of the two finalists in the World amateur pursuit championship, 1987.



Our knowledge of the apparatus that enables skeletal muscles to generate external force and therefore locomotion is now quite advanced. The sliding filament theory of muscle contraction, whilst still incomplete, is universally accepted (Bagshaw, 1982). Research interest has now focussed on two major issues; the mechanisms responsible for regenerating the adenosine tri-phosphate (ATP) hydrolysed to fuel the interactions between actin and myosin filaments in cross-bridge formation, and the causes of muscular fatigue.

The duration of a pursuit race (4 to 6 minutes) suggests that the majority of the ATP required to maintain force production in the contracting muscle fibers will be supplied by the complete oxidation of muscle glycogen to water and carbon-dioxide (Newsholme, 1986). However, Burke et al. (1981) has reported a mean post race blood lactate level of  $15.2 \text{ mmol.l}^{-1}$  on riders competing in the US national pursuit championships. This suggests that the demand for ATP considerably exceeds the rate at which it can be produced by oxidative mechanisms, the additional ATP required being generated by the anaerobic degradation of glycogen or glucose to lactate. The relative role of these two energy systems will be key a factor determining performance and clearly requires analysis.

The cause of fatigue in muscular exercise is an area of great debate in exercise physiology. It was beyond the scope of this thesis to directly address this question with respect to pursuing. However, it would be inappropriate to discuss limitations to pursuit performance without reference to the subject of fatigue. Edwards (1981, p.1) defined fatigue as "the failure to maintain the required or expected force (of muscular contraction)". This definition is most applicable to isometric muscle contractions, the form of exercise favoured by fatigue researchers. If the definition is applied to the graphs in figure 1 it could be argued that a degree of fatigue is apparent within the first minute of the race as the reduction in speed must be due to a decrease in the force required to maintain the pace set by the rider.

This statement is intended to highlight the difficulty of applying

rigid theories of fatigue to a practical sports situation. It is conceivable that the initial reduction in speed is due to different physiological mechanisms than those responsible for the symptoms of exhaustion at the end of a pursuit race. For example, depletion of phosphagen stores (Karlsson et al., 1972) or an initial reduction of intra-cellular pH (Stainsby, 1986) could possibly explain the former, whereas the end of race exhaustion may, for example, be the result of mitochondrial oxygen deficiency (Katz and Sahlin, 1988), exhausted buffering capacity resulting in metabolic acidosis (Sutton et al., 1977), failure of ventilatory apparatus (Dempsey, 1986) or even heat accumulation. It is also possible that all of these factors combine, in degrees dependent on individual and environmental factors, to cause the fatigue experienced by pursuiter. A major purpose of the review of literature was to untangle this confusion and identify the most likely single cause of fatigue, bearing in mind the metabolic demands of the event.

A pilot study on 8 pursuit cyclists (Keen et al., 1985) attempted to quantify the significance of aerobic and anaerobic power in pursuit racing. It examined the relationship between standard laboratory measures ( $\dot{V}O_{2max}$  and the Wingate anaerobic power test), a laboratory race simulation, and performance in a track time-trial. The findings were equivocal in that although the cyclists possessed high levels of both aerobic and anaerobic fitness (as measured by the above tests) neither factor correlated with time-trial performance. However, a number of important observations were made from the study.

It was clear that a re-appraisal of the techniques used for measuring physiological parameters was required. The validity of the Wingate anaerobic test has been questioned recently by Coleman et al. (1986) who point out that the method used to calculate power ignores the acceleratory components inherent in most ergometer systems. Other groups have reported a significant aerobic energy contribution of up to 27% and these data do not include the possible contribution of  $O_2$  bound to myoglobin (for a comprehensive review see Bar-Or, 1988). The main weakness of the test, however, is its dependence on subject motivation. Although

strong test-retest correlations have been reported, (Bar-Or, 1988) no information is available on repeatability in elite competitors. Such individuals can be difficult to motivate to a level where maximal effort is assured in a laboratory setting, a fact underlined by the frequently reported observation that athletes achieve higher blood lactate levels during competition than in "maximal" laboratory tests (Svedenhag and Sjodin, 1984). After careful consideration it was concluded that no suitable objective measure of anaerobic capacity was available and the development of such a test was beyond the scope of this project.

Traditionally,  $\dot{V}O_{2max}$  has been thought of as the principal determinant of endurance capacity (Saltin and Astrand, 1967). A large volume of research has therefore focussed on the factors thought to limit maximal oxygen uptake, particularly the cardiovascular system (Blomqvist and Saltin, 1983) and the uptake of oxygen in working tissues (Kajiser, 1970). A consequence of this pre-occupation with factors limiting  $\dot{V}O_{2max}$  has been a tendency to ignore the question of what limits athletic performance in endurance events. The fact that the work intensity required to elicit  $\dot{V}O_{2max}$  can only be sustained for a matter of seconds rather than minutes suggests that other factors may be of greater significance in the majority of endurance disciplines.

The concept of the "anaerobic threshold", a sub-maximal exercise intensity above which a progressive accumulation of lactate in muscle and blood occurs, has received considerable attention in more recent literature. The ability to identify the onset of metabolic acidosis is an attractive concept as the accumulation of hydrogen ions ( $H^+$ ) is widely believed to be a primary cause of fatigue in high intensity exercise (Kindermann and Keul, (1977). Although great controversy surrounds the causes of the abrupt rise in blood lactate observed during incremental aerobic work (Brooks, 1986; Mader and Heck, 1985; Wasserman et al., 1986; Yeh et al., 1983), the predictive power of this parameter is well established. Despite the variety of indices used to describe the anaerobic threshold (AT), correlations reported between endurance running performance and functional capacity at AT are consistently higher

than those reported for  $\dot{V}O_{2\max}$  (Fohrenbach et al., 1987; Sjodin and Jacobs, 1981; Tanaka and Matsuura, 1984). This approach to the assessment of endurance capacity thus appeared to offer an additional tool with which to examine pursuit cyclists.

The pilot study also pointed to the need to use experienced pursuit cyclists as subjects and to correlate laboratory measurements with performance in a major competition. It was felt that data recorded from a controlled time-trial was not truly reflective of pursuit ability, an important consideration bearing in mind the aims of this thesis.

Finally, it was felt that expressing physiological variables simply as a function of body mass may not be appropriate for high speed cycling events. Air drag, which accounts for nearly all the energy expended cycling at  $50\text{km}\cdot\text{h}^{-1}$ , will theoretically be determined primarily by the combined frontal area of rider and machine, not body mass (Kyle, 1986). More suitable indices therefore needed to be considered.

It would be incorrect to assume that the performance limiting factors in pursuing can be discussed purely in terms of mechanics and the physiology of muscle fatigue. It is acknowledged that psychological factors such as motivation can have a profound influence on human performance. This thesis was, however, only concerned with physiological variables that can be objectively quantified.

When all these considerations were taken into account the following research objectives emerged, which formed the basis of a series of investigations.

- 1) To apply the concept of the "anaerobic threshold" to racing cyclists and develop a suitable method of evaluating their responses to incremental sub-maximal exercise.

ii) To examine the relationship between the performance of elite pursuiter in the British championships and a variety of physiological indices, including  $\dot{V}O_{2max}$  and the "anaerobic threshold".

iii) To monitor the longitudinal changes of these parameters in pursuit cyclists.

iv) To apply the findings of the pursuit investigation to the competitive environment.

## Chapter Three

### REVIEW OF LITERATURE

The energy cost of pursuit cycling.

The mechanical energy required to propel a bicycle on a flat surface is determined by two main factors.

1. The energy exchanged at the tyre-surface interface, commonly termed rolling resistance. This is a function of the road or track surface, tyre composition, dimensions and pressure, wheel diameter and the mass of rider and bike. (Sjogaard et al., 1982).
2. Air resistance, which is dependent on air density, air velocity (head or tail wind) and the aerodynamic profile of both rider and cycle, (Di Prampero et al., 1979). The last is a complex factor influenced by the total frontal area of rider and cycle, clothing design and cycle construction (particularly wheels).

Rolling resistance demonstrates a linear function against speed and accounts for the majority of the energy expended up to a speed of approximately  $5\text{m.s}^{-1}$  (Di Prampero et al. 1979). Beyond this speed air resistance, which increases at approximately the square of the speed, is the dominant resistive force and will account for around 90% of the energy expended at elite pursuit racing speeds (Sjogaard et al., 1982)

A number of researchers have attempted to evaluate the energetics of cycling (e.g. Dill 1953; Van Baak and Binkhorst, 1981), but most have been concerned with standard utility bicycling at low speeds. As rolling resistance will be the dominant factor in such experiments the extrapolation of the energy cost of pursuing from these data would be invalid.

Several studies have examined the energy expenditure of racing cyclists riding at higher speed on competition bicycles. Both Pugh (1974) and Swain et al. (1987) measured the oxygen uptake ( $\text{VO}_2$ ) of

cyclists riding on flat roads by collecting expired gases with respiratory apparatus supported by an following car. Marion and Leger (1988) studied track cyclists on an indoor velodrome. Expired gases were collected immediately following each load using lightweight spirometry equipment attached to the subject.  $\dot{V}O_2$  was calculated by backward extrapolation to zero time (Leger et al., 1980). Di Prampero (1979) measured total tractional resistance by towing racing cyclists with a 25m long 0.003m diameter nylon cable attached to a vehicle via a dynamometer. Estimations of power output and oxygen consumption were derived from the forces registered in cable. Nonweiler (1957) measured the total drag of a stationary racing cyclist and bicycle suspended in a wind tunnel and calculated the power output corresponding to a range of air speeds. Whitt (1971) and Sjogaard et al.(1982) both used data previously reported on rolling and air resistance to produce equations describing the energy demands of racing cyclists.

Because of the technical difficulties involved there have been no reports of attempts to specifically measure the energy expenditure during a pursuit race. The speed of the rider and the geometry of cycle tracks would make collection of expired gases very difficult and the increased drag created by the apparatus would invalidate readings. Measurement of pedal force application using a telemetric link is perhaps the most promising solution but, although such equipment has been developed (Massagrande, 1983), no data on power output in pursuing has appeared in the literature.

In order to compare the results of the studies described above and to estimate the energy cost of pursuing, regression equations linking speed and power were generated from the data reported. Table 2 shows the theoretical power outputs corresponding to a speed of  $13.9 \text{ m.s}^{-1}$  ( $50 \text{ km.h}^{-1}$ ) extrapolated from these equations. Also listed in Table 2 are the assumptions made and the values used in order to standardize the data as much as possible. A speed of  $13.9 \text{ m.s}^{-1}$  was chosen as this is representative of elite speeds for pursuing at sea level prior to the introduction of aerodynamic clothing and cycles. Recent research has shown that these

**TABLE 2.** Estimate of power output required to cycle at  
13.9 m.s<sup>-1</sup> from various authors.

AUTHOR	POWER @ 13.9 m.s <sup>-1</sup> (Watts)
Di Prampero et al., (1979)	593
Marion and Leger (1988)	692
Nonweiler (1957)	560
Pugh (1974)	618
Sjogaard et al., (1983)	623
Swain et al., (1987)	661
Whitt (1971)	661
	---
	Mean = 640

Where possible the above data were standardized using the following values:

Barometric pressure	760 mmHg
ambient temperature	20°C
Mass of rider and cycle	80 kg
Height of cyclist	1.8 m
Total frontal area	0.35 m <sup>2</sup>

advances considerably reduce the drag co-efficient of the cyclist and therefore the power output at a given speed (Kyle, 1986). As no such equipment was used in the above studies it was thought inappropriate to compare current elite race speeds which are considerably higher ( $14.8 \text{ m.s}^{-1}$ ). The wide variation in predicted power output (Table 2) probably reflects the difficulty of standardizing field conditions and the variety of methods employed.

At an average speed of  $13.9 \text{ m.s}^{-1}$  a 4000m pursuit race would last 288 seconds (4min. 48sec.). If the power output required at this speed is 640W, taken as the mean value from Table 2, the total energy released in the race would be about 740kJ, assuming a mechanical efficiency of 25% ( $640\text{J} \times 288\text{s} \times 4$ ). Available data indicate that 25% represents the upper limit of net mechanical efficiency in cycling exercise (Suzuki, 1979). Net efficiency is known to decrease above pedal frequencies of  $90 \text{ min}^{-1}$  even in elite cyclists (Boning et al., 1984; Buchanan and Weltman, 1985; Hagberg et al., 1981; Jordan and Merrill, 1979). Calculations based on the speeds achieved in high level pursuing and the gear ratios used indicate that pursuited usually race at pedal rates above  $110 \text{ min}^{-1}$  so it seems doubtful that efficiencies above 25% occur in competition.

An energy yield of 740kJ over a duration of 4min. 48sec. would require a  $\dot{V}O_2$  of approximately  $7.7 \text{ l.min}^{-1}$  if produced purely by aerobic metabolism, as 1 litre  $O_2$  consumed = 21kJ energy liberated when glycogen is the sole substrate (Astrand and Rodahl, 1986). This figure is some 30% greater than  $5.5 \text{ l.min}^{-1}$ , a value typically reported in the literature as the  $\dot{V}O_{2\text{max}}$  of elite international cyclists (Hahn et al., 1986; Hermansen, 1973; Sjogaard, 1984; Stromme et al., 1977). Thus, if an elite rider utilised his entire aerobic capacity throughout the event, an anaerobic energy yield in excess of 210kJ would be needed to satisfy the predicted demand for energy. This cannot occur however as pursuit races are preceded by upto 5 minutes of rest. The inertia of the cardiorespiratory system is such that maximal levels of oxygen consumption are not reached until at least 60 seconds after the start of intense exercise (Di Prampero et al., 1970; Hughson and

Morrissey, 1982), even if a strenuous warm up has taken place (Martin et al., 1975).

Astrand and Rodahl (1986, p325) suggest that elite sprint athletes are capable of a maximal anaerobic energy production of approximately 200kJ from the combined yield of the breakdown of high energy phosphagens and anaerobic glycolysis. (For simplicity the term glycolysis will be used in this thesis to describe the degradation of both glucose and glycogen to pyruvate). Thus, even if a pursuit rider possessed a highly developed anaerobic capacity it would only be capable of supplying around 27% of the total estimated energy requirement of elite performance ( $200\text{kJ}/740\text{kJ} = 0.27$ ). It is highly unlikely, however, that the anaerobic energy yield in pursuing can be as high as 200kJ for the following reasons.

It is clear from the discussion so far that pursuit cyclists must possess a very high aerobic capacity. Ivy et al., (1980b) have shown that  $\dot{V}O_{2\text{max}}$  in active men is strongly correlated with muscle respiratory capacity (the rate of pyruvate oxidation in a muscle homogenate preparation) and the percentage of slow twitch muscle fibres. Reviews by Holloszy and Coyle (1984), Karlsson (1979) and Lindstedt et al. (1988) also concluded that composition of locomotor muscles, in terms of twitch type and oxidative capacity, is a primary determinant of aerobic capacity. By inference, elite aerobic athletes are therefore unlikely to be characterized by a high percentage of fast twitch (FT) glycolytic muscle fibres (Brooks and Fahey, 1984; Costill, et al., 1976). As anaerobic capacity is strongly related to the number and size of FT glycolytic fibres (Froese and Houston, 1987) it seems reasonable to conclude that elite pursuit cyclists will not possess an anaerobic capacity comparable to that found in elite sprint athletes, although no direct evidence is available to support this statement.

Astrand et al. (1986) studied the aerobic and anaerobic energy production of 4 large (<80kg) endurance trained men during an all-out arm and leg ergometer performance task lasting on average 348s (5min. 48s). The total anaerobic yield, calculated by

subtracting the measured aerobic yield ( $470 \pm 74$  kJ) from the total work performed ( $593 \pm 80$  kJ), was  $125 \pm 16$  kJ, equivalent to 21% of the total energy expended. It could be argued that a somewhat larger anaerobic yield is possible in exercise using both arms and legs than in pure leg cycling due to the greater muscle mass involved. It is interesting to note however, that the peak blood lactate level of  $15.8 \text{ mmol.l}^{-1}$  reported is comparable with the mean of  $15.2 \text{ mmol.l}^{-1}$  reported for pursuit riders following competition in the US championships (Burke et al., 1981). This suggests that a similar stress was placed on the anaerobic system in both groups.

Gollnick and Hermansen (1973) reviewed several attempts to assess anaerobic energy capacity and proposed a figure of 126kJ as the upper limit in exercise lasting between 1 and 5 minutes. They also pointed out that methodological problems prevent satisfactory measurements from being made so this value is only an approximation. Although no firm conclusions can be drawn from these data it would seem reasonable to suppose that the maximum anaerobic energy yield during a pursuit race will not exceed 125kJ.

An anaerobic yield of 125kJ would only account for about 15% of the total estimated energy required (740kJ), considerably below the 30%+ needed in addition to the expected aerobic yield to balance the energy equation. This strongly suggests that the formulae used to generate the data in Table 2 considerably over-estimate the actual energy demand of cycling. The following example serves to further illustrate this point.

The world record for 1 hour unpaced track cycling at sea level stood at 48.08km prior to the introduction of aerodynamic equipment. On the basis of the above data this would require a power output of 570W, equivalent to a  $\dot{V}O_2$  of  $6.8 \text{ l.min}^{-1}$ . It is well established that highly trained endurance athletes can exercise at up to 85% of their  $\dot{V}O_{2\text{max}}$  for such a prolonged period (Kindermann and Keul, 1979; Sjodin and Jacobs, 1981). On this basis, the record holder would have to possess a  $\dot{V}O_{2\text{max}}$  in the region of  $7.8 \text{ l.min}^{-1}$ . The highest  $\dot{V}O_{2\text{max}}$  reported in the literature for a elite track cyclist is  $6.5 \text{ l.min}^{-1}$  (Sjogaard et al.

1982). Within a month of the measurement of this value the individual concerned established a new amateur world hour record of 48.20km at an altitude of 2230m (Mexico City) using standard equipment (Beyerholm, 1987).

It appears likely from the above information that the true energy demand of elite pursuing is between 80 and 85% of the mean value derived from the data in Table 2, i.e. in the region of 600kJ, equivalent to a power output of 520W. The most probable source of error lies in extrapolating data from a nonlinear function such as cycling speed versus power output. Unfortunately, none of the studies quoted measured speeds above  $11.9 \text{ m.s}^{-1}$  so extrapolation to  $13.9 \text{ m.s}^{-1}$  (racing speed) was unavoidable. Also, it seems certain that the various methods employed to measure the energy cost of cycling created additional loads on the cyclists.

To summarize the above discussion, a world class 4000m pursuit race will require a total energy production of around 600kJ. The maximum contribution from anaerobic metabolism is unlikely to be greater than 125kJ, and will therefore contribute no more than 20% of the total energy demand of elite level races. Thus it can be concluded that the aerobic energy system must exert the primary influence on pursuit performance and therefore requires careful analysis if a greater understanding of the event is to be achieved. A further observation is that optimal achievement in competition will require both the aerobic and anaerobic energy systems to be maximally taxed.

## Muscular fatigue and performance impairment.

A fundamental question concerning this thesis is what actually limits the pursuit rider's capacity to produce mechanical power during the race? A competitor would almost certainly answer this question by describing the discomfort of physical exhaustion, so evident at the finish of a pursuit. Unfortunately, subjective perceptions of fatigue provide few clues to understanding the mechanisms that have determined performance (Lollgen et al., 1980).

A review of the possible causes of fatigue in pursuit racing cannot be made without firstly constructing a working definition of the problem. It has already been pointed out that both the aerobic and anaerobic energy production systems must be fully taxed for optimal performance. Maximal values for heart rate, oxygen uptake, ventilation and lactic acid accumulation are therefore to be expected, but these factors may only be symptomatic of the stress imposed.

Fundamentally, fatigue in pursuing can be thought of as the failure of the locomotor muscles to generate or maintain the desired force application pattern at the foot-pedal interface, the result of which must be a drop in mechanical power output and therefore speed (after Edwards, 1981). The speed of muscle contraction will be a direct function of the force generated, as the cyclists limbs move in a fixed plane and a single gear ratio must be used in all track racing.

The term "force application pattern" is included in the above definition for the following reason. It is conceivable that a reduction in mechanical power (speed) could be due to a change in the pattern of forces applied at the pedal as well as a simple reduction in the magnitude of muscular force generated. Such an effect would be characterized by a decrease in effective force normal to the crank, and an increase in ineffective force parallel with the crank (Cavanagh and Sanderson, 1986). A possible cause of this could be a change to a less efficient or adapted fibre recruitment pattern due to fatigued motor units.

A voluntary muscle contraction is dependent on a complex chain of events commencing at the motor cortex of the brain and ending with the interaction of actin and myosin filaments in the innervated muscle fibres (Bagshaw, 1982). A reduction in the force of contraction is the result of a failure at one or more of the potentially limiting stages along this chain. Traditionally, these stages have been classified as central if they are concerned with the generation and propagation of neural stimuli, or peripheral if the mechanism concerned is responsible for the excitation or contraction of the muscle (Bigland-Ritchie, 1981).

Karlsson (1979) suggests that fatigue in exercise lasting more than one hour is peripherally located. If the exercise is of a relatively high intensity, depletion of muscle glycogen stores will almost certainly impair performance (Bergstrom and Hultman, 1967) although the precise mechanism for this phenomenon remains a mystery (Conlee, 1987). At work intensities where oxidation of fats can supply the bulk of the energy required, disturbed intracellular homeostasis due to dehydration, thermal stress, reduced cell membrane integrity or electrolyte imbalance appears to limit work capacity (Armstrong et al., 1985; Karlsson, 1979).

In exercise of a relatively short duration where depletion of carbohydrate stores is not limiting the position is less clear. A wealth of evidence supporting both central and peripheral theories of fatigue exists. The work of Mosso at the start of the century, (described by Edwards, 1983), first demonstrated a possible central limitation. Using a simple finger ergograph he showed that psychological arousal can dramatically effect dynamic work performance. Asmussen (1979), has investigated the role of central mechanisms by studying the effects of a variety of mental and physical diverting activities performed between repeated bouts of exhaustive arm exercise. He found that activities as diverse as counting backwards and exercising the fingers of the collateral hand all enhanced work output compared to passive rest. He suggested that inhibitory feedback from the fatiguing muscles must in some way diminish the voluntary effort; and that diverting activity, by generating facilitatory feedback to the brain, serves to

depress this inhibitory state. Thus, although the proposed mechanism is centrally located, the actual source of the inhibition is the exercising muscles. Asmussen concluded that muscle fatigue is primarily peripheral in origin, but the resulting decrease in performance capacity is mediated by a central component reflecting inhibitory feedback.

A common observation in studies of isometric work during sustained maximal voluntary contractions (MVC's) is that the reduction in electromyogram (EMG) activity roughly parallels the decrease in muscle force (Bigland-Ritchie, 1981). This is often suggested to indicate central fatigue due to a reduced drive from the motor cortex or impaired neuronal transmission. However, Merton et al., (1981) have provided evidence that the motor pathway remains fully functional during a sustained MVC. Using EEG electrodes to directly stimulate the area of the motor cortex controlling forearm muscle contractions, they showed that normal action potentials can be generated in the adductor pollicis even when the muscle is severely fatigued following a 4min. sustained contraction. This group also reported that direct electrical stimulation of the fatigued muscle using a method that bypasses the neuromuscular junction does not restore performance in the same experimental situation. Bigland-Ritchie (1981) has shown that the progressive reduction in motor neuron firing rate observed during sustained MVC's may actually serve to optimize muscle performance by matching the firing rate to the decreased contractility of fatiguing muscle.

The aetiology of fatigue that appears to be emerging from the literature can be summarized as follows. The primary site of performance failure in short to medium duration physical activity almost certainly lies within the exercising muscles. As a consequence of inhibitory feedback reflecting this undesirable situation a secondary central mechanism will exert a restraining influence on the muscles. This scenario is similar to the model of fatigue proposed by Wilkie (1981). He concluded that although a shortage of the chemical fuel required to maintain contraction was primarily the cause of fatigue in short term exhaustive exercise, a secondary action controlling activation prevents the muscle from

self destruction through the complete depletion of ATP. Karlsson (1979) also proposes that suitably located receptors may enable exercise intensity to be regulated with respect to an individuals maximal work capacity.

Edwards (1983), in proposing an integrated theory of fatigue, suggested that it may prove impossible to assign fatigue to a single failing mechanism. He concluded that describing the events that immediately influence the failure of the contractile system may provide the closest explanation of the phenomenon.

Clearly, no firm conclusion can be made regarding the exact cause of fatigue in pursuit racing, but it is certainly possible to speculate about the factors that appear to exert a primary influence. From the above evidence it seems unlikely that a failure will occur in the neural pathway from the motor cortex to the terminal end plates of the working musculature. As centrally mediated inhibition is almost certainly the result of negative feedback from fatiguing muscle the fundamental limitation to pursuit performance must lie within the locomotor muscles of the cyclist. The remainder of this discussion will therefore focus on the potential causes of fatigue beyond the neuromuscular junction.

Research into muscle fatigue past the neuromuscular junction has highlighted three possible sources of impaired muscular performance during athletic events lasting up to 10 minutes (Karlsson, 1979; Edwards, 1983). They are:-

1. A shortage of chemical fuel.
2. The accumulation of metabolites.
3. The failure of excitation/contraction coupling.

The free energy required to drive the cross-bridge interactions in contracting muscle is supplied by the hydrolysis of ATP to adenosine diphosphate (ADP) and inorganic phosphate (Pi). Wilkie (1981) has pointed out that the complete utilization of the muscle ATP store would result in rigor mortis, a statement which is supported by a wealth of evidence that dramatic reductions in ATP

levels do not occur in short-term exhaustive exercise (Jacobs et al., 1982; Karlsson, 1971; Sahlin, 1978). Therefore, if a shortage of chemical fuel is the cause of muscle fatigue the limitation/s must occur in the mechanisms by which ATP is resynthesized.

The immediate re-synthesis of ATP through the splitting of creatine phosphate (CP) will provide the first support for the ATP system, but the muscle store of CP is only thought to be capable of liberating approximately 15kJ (Astrand and Rodahl, 1986) and therefore cannot make a quantitatively significant energy contribution in pursuing. It has been suggested in the past that exhaustion of the CP store is directly linked to fatigue in high intensity exercise on the basis that biopsy data on muscle CP levels indicate virtual depletion following exhaustion in work lasting between 2 and 15 minutes (Bergstrom et al., 1971; Karlsson, 1979). However, the introduction of the non-invasive Nuclear Magnetic Resonance technique for quantifying phosphorus metabolites in intact muscle has raised potentially serious doubts about the accuracy of the needle biopsy method in this type of investigation. Dawson, (1983) has presented data that indicates a large artifactual error in the needle biopsy assay of muscle CP, due in part to the delay in sample freezing. The conclusions of studies that have employed this technique must therefore be treated with caution. What does seem certain is that CP values will fall rapidly to a fixed level in the first stages of a pursuit and remain at that level until the finish (Bergstrom et al., 1971). Whilst this change may have significance for cellular regulation it appears unlikely to directly result in fatigue.

It has already been established that a large majority of the energy produced during the event will result from aerobic metabolism. The intensity of a pursuit race is such that carbohydrate, or more specifically muscle glycogen, will almost certainly be the sole substrate metabolized through the oxidative pathway (Newsholme, 1986). Although the exhaustion of the muscle glycogen store has been shown to reduce maximal aerobic performance (Conlee, 1987), available data indicate that the degree of depletion that occurs during a maximal effort like a pursuit race is not sufficient to slow

the rate of ATP production from this substrate (Saltin and Karlsson, 1971). A reduction of 50% in muscle glycogen following 6 minutes of exhaustive ergometer exercise has been reported by Astrand et al., (1986). Glycogen levels returned to 75% of the resting value following 60 min. passive recovery in this study. It therefore appears that substrate availability will not be limiting in a single race unless the level of muscle glycogen has been significantly reduced beforehand. Whilst this should not occur in a well prepared rider prior to competition, pursuit riders occasionally have to race 3 rounds in the space of 12 hours and semi-finals and finals are rarely more than 3 hours apart. Under such circumstances the position may be less clear.

It is an accepted practice in pursuit racing to actively recover from a race and therefore prepare for the next round by riding for up to 20 minutes on cycle rollers. This is logical as elevated lactate levels have been shown to impair maximal performance (Hogan and Welch, 1984) and the clearance of lactate is greatly enhanced by submaximal exercise (McLellan and Skinner, 1982). However, this practice may effect the rate of muscle glycogen resynthesis following the race. Hermansen and Vaage, (1977) and Astrand et al., (1986) both concluded that conversion of lactate to glycogen within the active muscles accounted for the bulk of the muscle glycogen resynthesized following maximal exercise. However, these studies employed completely passive recovery strategies. Mazzeo et al., (1986) has presented data strongly suggesting that moderate recovery exercise will result in the vast majority of accumulated lactate being oxidized, principally in the slow twitch muscle fibres. It is therefore conceivable that muscle glycogen could become significantly depleted during of a pursuit series unless carbohydrate intake between rounds occurs.

The limitation to aerobic energy production in the absence of glycogen depletion must be either the attainment of a maximal rate of substrate flux through the respiratory chain, the inhibition of this process by the accumulation of metabolites or a lack of molecular oxygen in the mitochondria of the activated muscle fibres.

The flow of substrate through the respiratory chain is thought to be primarily a function of the concentration of substrate and the volume of enzymes responsible for catalyzing the reactions of the chain (Gollnick and Saltin, 1982). In reviewing the subject of enzymatic profiles and athletic performance Holloszy and Coyle (1984), concluded that the oxidative enzyme concentration in locomotor muscles does not directly limit oxygen consumption. They point to the fact that oxidative enzyme levels vary greatly between individuals of similar aerobic power and demonstrate much greater changes of magnitude than  $\dot{V}O_{2max}$  during periods of training or de-training (Favier et al., 1986; Hoppeler et al, 1985; Sjodin et al., 1982). It has recently been suggested that the remarkably high concentration of oxidative enzymes found in elite endurance athletes is primarily linked to the increased levels of fat combustion during sub-maximal exercise observed in such athletes (Gollnick and Saltin, 1982; Sjogaard, 1984).

The end metabolites of the complete oxidation of glycogen:  $CO_2$ ,  $H_2O$ , and heat, are unlikely to present a direct threat to the active cells in the type of exercise under consideration here. Arterial  $CO_2$  levels remain essentially constant or drop during maximal work, suggesting that the mechanisms responsible for removing the  $CO_2$  from active tissue are sufficient to prevent respiratory acidosis (Grimby, 1969; Dempsey, 1986). Although intracellular  $H_2O$  levels are known to increase by up to 15% during strenuous exercise (Hermansen and Vaage, 1977), this increase is mainly due to an influx of  $H_2O$  from the extracellular space in response to an increased osmotic pressure resulting from the accumulation of lactate and other metabolites (Hultman and Sahlin, 1980).

Metabolic heat production is potentially a danger in middle distance athletic events in certain climatic conditions. Brown et al., (1982), reported a 30% reduction in time to exhaustion working at 100%  $\dot{V}O_{2max}$  on a cycle ergometer in an ambient temperature of 35°C compared to 20°C. Performance times were  $4.7 \pm 0.7$  min. and  $3.3 \pm 0.2$  min. respectively. They concluded that compromised blood flow due to the increased need to dissipate heat resulted in greater muscle hypoxia. Cyclists have an advantage over most other

athletes in this respect however, as the convective heat losses are greatly enhanced by the high air flow experienced.

Katz and Sahlin (1988) have recently reported data that supports the popular view that a lack of molecular oxygen at the appropriate intracellular locations limits the volume of aerobic energy production. They have shown that at high exercise levels mitochondrial redox balance is disturbed by the accumulation of NADH (reduced nicotinamide adenine dinucleotide). They attribute this condition to a relative lack of molecular oxygen which must be present to accept the electrons carried by the NAD. A similar conclusion was reached by Wasserman et al., (1985) by studying the extracellular effects of varying tissue O<sub>2</sub> supply.

It therefore seems that the most likely limitation to the rate of aerobic energy production is an inability to supply sufficient oxygen to the active mitochondria of the exercising muscles. The factors limiting the supply and consumption of oxygen are discussed in the following section. The cause of muscle fatigue however, does not appear to be directly attributable to maximal rates of aerobic metabolism. The necessity to generate ATP via anaerobic glycolysis as a result of the inevitable shortfall in aerobic capacity, on the other hand, presents the exercising tissue with a number of problems.

The most significant aspect of anaerobic glycolysis is the 2 protons (H<sup>+</sup>) and 2 lactate anions produced per glucosyl unit consumed (Parkhouse and McKenzie, 1984). Classically, these protons were thought to result entirely from the dissociation of lactic acid, which in turn was produced from pyruvic acid by the mass action of lactate dehydrogenase (LDH) when oxygen was not available for its combustion in the mitochondria (Wasserman et al., 1973). Although the equimolar volume of H<sup>+</sup>, lactate and ATP generated by anaerobic glycolysis is not disputed, it is now known that the hydrolysis of ATP results in a net production of protons in anaerobic work, the volume of which is dependent on the pH and concentration of other ions in the cell (Hochachka and Mommsen, 1983). Furthermore, the conversion of pyruvate to lactate is an H<sup>+</sup>

consuming reaction and is vital to the preservation of cytosolic redox balance, as this reaction results in the oxidation of the accumulating NADH to NAD (Sahlin, 1978). NAD is required for the oxidation of 3-Phosphoglyceralddehyde to 1,3-diphosphoglycerate in order to maintain glycolytic flow (Parkhouse and McKenzie, 1984). Thus, although the classical explanation of proton production in anaerobic glycolysis now appears incorrect, the end result is the same.

The production of ATP from anaerobic glycolysis is the only other major energy source available to the pursuiter. As the initial substrate for this pathway will be the same as that for the oxidative process, i.e. muscle glycogen, this fuel source is unlikely to be rate limiting in the single race situation. Jacobs, (1981) has reported that performance of a 60 second high intensity leg extension task was unchanged across a wide range of muscle glycogen levels. Significant reductions in power output only occurred when muscle glycogen levels fell below 40% of the normal resting value. This finding is interesting in the context of the above discussion regarding repeated performances.

The metabolic acidosis that accompanies the accumulation of lactate and protons has long been thought of as a cause of fatigue (Hermansen, 1971). A wealth of empirical evidence has linked changes of pH and lactate in both muscle and blood with performance impairment. In an exemplary study, Hogan and Welch (1984) manipulated the muscle pH of subjects before an ergometer performance trial by administering hypoxic and hyperoxic gases during standardized prior exercise. Time to exhaustion increased from 9 to 15 minutes between the respective treatments but blood pH and lactate levels were not significantly different, suggesting an equal degree of acidosis at the termination of exercise.

The adjustment of acid-base balance by the ingestion of alkaline solutions has frequently been used to investigate the effects of acidosis in exercise. Significant improvements in exhaustive running lasting up to 7 minutes, coupled with higher blood lactate levels have been reported following ingestion of sodium bicarbonate

(Gledhill, 1984; Jones et al., 1977). This improvement has been attributed to an increased buffering of protons. It appears however, that the effect is most marked in exercise lasting minutes rather than seconds. Studies by Inbar et al. (1983) and McCartney et al. (1983) showed little or no change in 30 seconds of maximal cycle ergometer exercise after increasing bicarbonate levels. A possible explanation is that in short bursts of maximal effort the acidosis is mostly confined to the intracellular environment. Only moderate changes in blood pH and lactate have been reported following such exercise (Jacobs, 1982). Bicarbonate ingestion is thought to only modify extracellular pH and buffering potential (McCartney et al., 1983). In sustained efforts where blood pH and lactate demonstrate the largest changes (Karlsson, 1979), an increase in extracellular buffering may enhance the efflux of protons and lactate from muscle cells by helping to maintain a concentration gradient between the intra- and extra-cellular compartments (MacClaren, 1986).

Further empirical evidence of the link between acidosis and fatigue lies in the fact that comparable values for muscle pH in man following a variety of forms of exhaustive maximal exercise have been reported in the literature. Hermansen and Osnes (1972) reported a pH of 6.41 after ergometer exercise lasting 1-2 minutes and Sahlin (1978) found values of 6.56 and 6.60 following a fatiguing isometric contraction and 5-10 minutes of cycle ergometer work respectively. This suggests that a critical pH may exist at which regulatory mechanisms restrict cellular activity to prevent further pH reductions.

A number of mechanisms by which acidosis may exert a restrictive influence on muscle contraction have been proposed, although direct experimental evidence in support of them is scarce. For glycolytic ATP production to make a significant energy contribution the process will have to occur rapidly as the ATP yield per glucosyl unit consumed is low (Newsholme, 1986). Any bottlenecks occurring in the glycolytic chain may therefore result in a reduction in ATP production and a consequent drop in power output.

Protons are highly reactive and are capable of combining with the ionizable sites on protein structures (Newsholme, 1986). This will have the effect of changing the properties of specific protein combinations such as enzymes, thereby altering their potential to link with substrates (Parkhouse and McKenzie, 1984). Also, a reduction in pH can change the ionic state of substrates, products and activators/inhibitors along metabolic pathways (Hultman and Sahlin, 1980). The glycolytic enzymes phosphorylase and phosphofructokinase (PFK) catalyze reactions that have been identified as particularly sensitive to pH adjustments (Sahlin, 1978). PFK has been shown to be virtually inactive at a pH of 6.4 (Trividi and Danforth, 1966). These pH mediated effects would serve to diminish the flow of carbon through the glycolytic chain with a consequent drop in ATP and proton production. A protective servo mechanism therefore appears to exist.

A second mechanism by which a low pH may cause fatigue is the direct interference of protons in the contractile process itself. It is thought that protons may compete with calcium ions for the binding sites on troponin molecules, causing a reduction in the number of cross-bridge formations and therefore force (Donaldson and Hermansen, 1978). Donaldson (1983) recently concluded that the most likely cause of pH induced fatigue is through a direct inhibition of actomyosin ATPase. This would explain the difference in fatigability observed between fast- and slow-twitch muscle fibres as the wide variation that occurs in myofibrillar ATPase between fibres is well known. Indeed, it is this distinction that has typically been used to identify a fibre-type (Pette, 1984).

The high charge density of protons may also result in disturbance to the movement of ions across membranes and the location of molecules in the intracellular environment (Hultman and Saltin, 1980). One such effect is the increase in extracellular potassium observed during acidosis (Sahlin, 1983). This would have serious implications for excitation-contraction coupling as the rapid exchange of potassium and sodium ions across the cell membrane is necessary for the conduction of the excitatory impulse from the terminal end plate to the sarcoplasmic reticulum. It has also been

suggested that a low pH results in a suppressed release of calcium ions from the excited sarcoplasmic reticulum as more calcium becomes bound to proteins within it. (Tesch, 1980).

One further potentially limiting consequence of decreased pH is a reduction in the free energy change per unit ATP hydrolysed. Wilkie (1981) and Sahlin (1983) have both suggested that the reduction in free energy may be great enough to pass a critical level for an ATP consuming reaction. This could influence contraction directly through a failure to achieve cross-bridge linkage, and indirectly by reducing the effectiveness of the various ionic pumps responsible for controlling both contraction and relaxation.

To summarize, fatigue in pursuing is most probably a reflection of the sensitivity of the intracellular environment to decreases in pH which result from the necessity to generate ATP via anaerobic glycolysis. The accumulating protons responsible for this may inhibit the contractile process directly, slow the rate of ATP production causing a fuel shortage, and/or disturb the electrical balance within the cell.

Pursuit cyclists are therefore presented with two major physiological challenges. Firstly, they must have a very large capacity for the combustion of muscle glycogen in order to generate the mechanical power required. The highest levels of aerobic power are typically found in endurance athletes who compete over long periods of time (Svedenhag and Sjodin, 1984). Secondly, they must have the capability to absorb the large volume of protons generated by anaerobic glycolysis without incurring detrimental decreases in muscle pH, a characteristic thought to be essential for successful anaerobic sprint performance (Newsholme, 1986). The latter will be principally be a function of the buffering capacity of the active muscles.

## Aerobic capacity - limitations and assessment.

The significance attached to the measurement of  $\dot{V}O_{2\max}$  in the context of athletic endurance performance can be traced back to the work of A. V. Hill and associates in the early part of this century. They pioneered the concept that a level of exercise intensity is reached in incremental work beyond which no increase in oxygen consumption occurs, thus reflecting a limitation at some stage of the oxygen transport pathway (Hill and Lupton, 1923). Hill attributed the exhaustion that occurs in exercise above this critical intensity to an accumulation in lactic acid resulting from the limitation to oxygen supply.

These findings, together with the knowledge that an approximately linear relationship exists between oxygen consumption and exercise intensity in most forms of physical activity, have led physiologists to conclude that  $\dot{V}O_{2\max}$  must be the primary determinant of performance capacity in exercise lasting more than 2 minutes (Astrand and Rodahl, 1986). The discussion of the energetics of the pursuit race at the start of this review serves as an example of this logic. The determination of  $\dot{V}O_{2\max}$  in pursuit cyclists is thus an important element of this thesis.

The Fick principle, described by Astrand and Rodahl (1986), states that the rate of oxygen consumption ( $\dot{V}O_2$ ) will be the product of cardiac output ( $\dot{Q}$ ) and the arteriovenous oxygen difference ( $Ca - C\bar{v}O_2$ ), or :-

$$\dot{V}O_2 = \dot{Q} \times (Ca - C\bar{v}O_2)$$

As  $\dot{Q}$  is a function of heart rate (fc) and stroke volume (Vs) the above equation can be written as:

$$\dot{V}O_2 = (fc \times Vs) \times (Ca - C\bar{v}O_2)$$

The quest to determine the factors that limit oxygen uptake in man has perhaps attracted more research than any other single topic in exercise physiology. Most of this work can be divided into one of

two categories which reflect the two components of Fick's equation. The first is concerned with the transport of oxygen from the lung bed to the muscle fibres and is thus dominated by cardiovascular function. The second category encompasses the factors dictating the rate of oxygen consumption within active muscle tissue. This scenario is somewhat analogous to the fatigue debate, and the terms central and peripheral are often ascribed to these respective categories.

The rate of oxygen delivered to the contracting muscle fibres will be a function of the volume of arterial blood perfusing the muscles in a given time and the arterial oxygen concentration. The volume of blood perfusing a muscle is determined primarily by cardiac output and the degree of peripheral vasodilation at the arteriole level (Blomqvist and Saltin, 1983). There is powerful evidence that this first variable is the principle source of limitation to oxygen uptake. Firstly, a strong correlation exists between cardiac output and  $\dot{V}O_{2max}$ , measured absolutely in  $l \cdot min^{-1}$  (Hermansen, 1973). Secondly, no differences in cardiac output at identical sub-maximal workloads are found between sedentary individuals and athletes with high  $\dot{V}O_{2max}$  values, or in individuals who have increased their  $\dot{V}O_{2max}$  through training (Saltin, 1969). Thirdly, similar values for  $Ca-CvO_2$  during maximal work in athletes and non-athletes are frequently reported (Hermansen, 1973; Rowell, 1969; Saltin, 1969). Thus, differences in  $\dot{V}O_{2max}$  appear to result mostly from an ability to pump more blood.

Maximal heart rate is usually lower in endurance athletes than untrained individuals so the higher cardiac output of an athlete must be the result of a greatly increased stroke volume. This has been attributed to a higher ventricular volume (Cohen and Segal, 1985), decreased systemic resistance to blood flow and increased ventricular pre-load, possibly as a result of the higher blood volume possessed by athletes (Blomqvist and Saltin, 1983).

The gas exchanging potential of the lung is widely believed to be sufficient to adequately saturate haemoglobin with oxygen during maximal exercise at sea level (Saltin, 1969). If this is true arterial

oxygen tension will primarily be a function of the concentration of the oxygen binding pigment haemoglobin. Haemoglobin levels in subjects with a high  $\dot{V}O_{2\max}$  do not appear to differ from those found in sedentary individuals (Clement and Asmundson, 1982; Hermansen, 1973), suggesting that blood composition does not limit  $\dot{V}O_{2\max}$ . However, the results of experiments that have manipulated haemoglobin levels by venesection or transfusion conflict with this view (Gledhill, 1982). Celsing and co-workers (1987) recently reported linear increases of  $\dot{V}O_{2\max}$  in subjects following stepwise re-infusion of blood.

Another approach to investigating the role of arterial oxygen content has been to increase it by raising the partial pressure of oxygen in the alveoli, usually by inspiring hyperoxic gas mixtures. In a study typical of many, Ekblom et al. (1975) found a 13% increase in  $\dot{V}O_{2\max}$  when their subjects breathed 50% oxygen, again implicating oxygen transport as the limiting factor. However, the validity of experiments such as this has been questioned recently by Welch and Pederson (1981) who suggest that the conventional douglas bag technique is highly susceptible to volumetric errors when high oxygen fractions are employed. They concluded that unless special precautions are taken an overestimation of  $\dot{V}O_{2\max}$  is likely in such experiments.

The more technically demanding method of raising arterial oxygen levels by increasing barometric pressure and inspired oxygen fraction was used by Kaijser (1970) to investigate the factors limiting aerobic performance. Subjects performed ergometer performance tests lasting approximately 6 minutes in an ambient pressure of 3 atmospheres breathing 100% oxygen, and in normal atmospheric conditions (control). Arterial and venous oxygen tensions were determined via indwelling catheters. The role of local blood flow was investigated by examining arm-ergometer work. A 30% increase in arterial  $O_2$  did not result in a higher  $Ca-C\bar{V}O_2$  or a longer time to exhaustion, which led Kaijser to conclude that supply of oxygen was not limiting in this case. When the same experimental conditions were applied to leg-ergometer exercise he found that the subjects exhausted before reaching maximal cardiac

output and had considerably higher femoral-venous  $O_2$  levels than in the control condition. Time to exhaustion was not significantly longer in the hyperoxic condition.

These findings appear to conflict with the notion that oxygen uptake is purely centrally mediated. However, for technical reasons ventilatory variables were not actually measured in Kaijser's study, so these data cannot be said to conclusively demonstrate a peripheral limitation to  $\dot{V}O_{2max}$ . A further weakness was the use of volitional exhaustion as the principle criterium for determining the effects of varying experimental conditions. Hesser et al. (1981) have reported significantly impaired pulmonary function in healthy subjects during maximal exercise in an ambient pressure of 3 atmospheres, and concluded that inspiratory muscle fatigue resulting from the increased air density may limit exercise under such conditions. It is thus possible that the cause of fatigue in Kaijser's study varied between the normo- and hyperbaric conditions.

The function of cardiac output in limiting  $\dot{V}O_{2max}$  is also questioned by the finding that, with the exception of racing cyclists, most athletes achieve slightly higher peak  $\dot{V}O_2$  levels in treadmill running than ergometer cycling (Hermansen, 1969). This suggests that peripheral mechanisms such as fibre recruitment or exercising muscle mass as well as cardiac output influence  $\dot{V}O_{2max}$ .

The role of skeletal muscle morphology and metabolic profile in determining  $\dot{V}O_{2max}$  is unclear. Significant correlations between  $\dot{V}O_{2max}$  and both the percentage of ST muscle fibres and muscle respiratory capacity have been reported (Ivy et al., 1980). Lindstedt et al. (1988) have presented data that demonstrates a linear relationship between total skeletal muscle mitochondria and  $\dot{V}O_{2max}$  across a wide range of exercising animals, including humans. However, it was pointed out earlier in this review that skeletal muscle oxidative enzyme profiles do not demonstrate a uniform relationship with  $\dot{V}O_{2max}$ .

The debate on limiting factors has been widened by recent suggestions that the ability of the lung to fully saturate venous

blood with oxygen may not in fact be adequate in athletes with particularly high  $\dot{V}O_{2max}$  values (Dempsey, 1986). Williams et al., (1986) have reported a significant correlation between the degree of arterial desaturation observed in athletes and their  $\dot{V}O_{2max}$ . They suggested that the large cardiac output required for high rates of oxygen consumption may result in the transit time of red cells through the lung capillaries dropping below the minimum required for successful diffusion of  $O_2$ .

In view of the equivocal evidence surrounding the factors that actually limit oxygen consumption it is somewhat surprising that the measurement of  $\dot{V}O_{2max}$  is widely considered by sports physiologists to be the gold standard of exercise testing procedures (Astrand, 1984; Foster, 1983). Furthermore, there are some conceptual problems concerning the relationship between  $\dot{V}O_{2max}$  and athletic performance. It is well known that the exercise intensity eliciting  $\dot{V}O_{2max}$  cannot be sustained for prolonged periods. Studies that have examined responses to such exercise typically report exhaustion occurring in well under 10 minutes (Brown et al., 1982; Buono and Roby, 1982; Hermansen and Saltin, 1969). From the earlier discussion it is highly likely that the cause of fatigue in this instance will be the disturbance of acid-base balance resulting from anaerobic energy liberation. Studies on elite endurance runners have shown that only 80 to 90% of  $\dot{V}O_{2max}$  can be utilised in races lasting more than 30 minutes (Allen et al., 1985; Sjodin and Jacobs 1981; Tanaka et al., 1984).

Another challenge to the pre-eminence of  $\dot{V}O_{2max}$  as the sole factor in endurance performance comes from the observation that significant endurance performance increases have been shown to occur without changes in aerobic capacity following training (Foster, 1983). The phenomenon is most marked in elite athletes who have been training many years (Roth et al., 1981; Sjodin et al., 1982; Sjogaard, 1984). These findings indicate that factors other than  $\dot{V}O_{2max}$  determine performance in sustained efforts. In the case of running, variation in running economy has frequently been cited as the reason for this discrepancy, but studies by Allen et al., (1985) and Noakes, (1988) found that this factor alone cannot

explain the performance variations in individuals of equal aerobic power. Furthermore, mechanical efficiency, which is the cycling equivalent of running economy, has not been shown to vary between cyclists and non-cyclists (Boning et al., 1984; Suzuki, 1979; Withers et al., 1981) and only minor changes following training have been reported (Denis et al., 1982). The notion of aerobic economy thus appears less applicable to cycling than running.

The observation that lactate appears in muscle and blood at exercise levels below  $\dot{V}O_{2max}$  has frequently been reported (Karlsson and Jacobs, 1982). In the past this was thought to simply reflect a transient increase in lactate production resulting from a temporary imbalance between the rates of supply and consumption of oxygen (Saiki et al., 1967). In recent years however, data has emerged that clearly demonstrates a net production and accumulation of lactate in muscle and blood during submaximal work. Muscle lactate has been shown to rise abruptly when high levels of sub-maximal exercise are reached (Jorfeldt et al., 1978; Jacobs, 1981), and a number of studies on athletes have shown progressive rises in blood lactate leading to exhaustion in sustained submaximal exercise lasting 30 minutes (Hermansen and Stensvold, 1972; Heck et al., 1985; Stegmann and Kindermann, 1982).

The knowledge that lactate starts increasing systematically beyond a particular workload during incremental exercise has resulted in the concept of an "Anaerobic Threshold" - a level of sub-maximal work at which anaerobic metabolism appears to begin contributing to ATP production. This hypothesized metabolic threshold was expounded by Wasserman and co-workers (1973), who developed non-invasive techniques, based on respiratory gas exchange parameters, for detecting the appearance of lactate in blood during incremental work. Central to this group's work was the observed increase in ventilatory drive they attributed to raised  $CO_2$  levels resulting from the buffering of lactic acid by blood  $HCO_3^-$ . Since the early work of Wasserman et al. a multitude of different "threshold" terms have appeared in the literature, most of which reflect the individual approaches of researchers to this subject. As all of these are essentially concerned with the same concept the

term Anaerobic Threshold (AT) will henceforth be used as a general term and will not refer to a specific protocol or definition.

Despite the wide range of protocols now used to measure the AT, and the fierce debate about the physiological mechanisms underlying the phenomenon, correlations reported between this parameter and endurance running performance are remarkably high, and typically exceed the predictive power of  $\dot{V}O_{2\max}$  in this respect. (For a comprehensive review of research on this topic see Jacobs, 1986). Furthermore, this variable appears to be more sensitive to training effects than  $\dot{V}O_{2\max}$  (Davis et al., 1979; Denis et al., 1982; Hurley et al., 1984; Roth et al., 1981; Sjodin et al., 1982). Measurement of the AT thus appears to offer significant information about the functional capacity of an endurance athlete and is therefore a potentially useful tool with which to investigate the physical attributes of pursuit cyclists.

Lactate accumulation in sub-maximal work was originally thought to reflect inadequate oxygen supply to areas of the exercising musculature resulting in a degree of anaerobic ATP production (Wasserman et al., 1973). This concept is supported by the finding that administration of hyperoxic gas mixtures results in lower blood lactate levels during sub-maximal exercise (Hogan et al., 1983). A wealth of data exists, however, that suggests the rise in blood lactate reflects changes in cellular metabolism that are independent of oxygen supply. The depletion of muscle glycogen has been shown to significantly reduce sub-maximal blood lactate levels (Hughes et al., 1982). Similar effects were demonstrated by Ivy et al. (1981) through the manipulation of blood born free fatty acids and glucose. These findings imply that choice of substrate influences lactate production. Factors such as mode of exercise have also been shown to influence sub-maximal blood lactate responses. Buchanen and Weltman (1985) and Hughes et al., (1982) have shown that lactate starts to accumulate at a lower power output at high pedal frequencies in cycle ergometry.

The production of lactate through the overstimulation of glycolysis in response to increased sympathetic drive, rather than simply as a

result of a lack of oxygen is now widely thought to be the major source of lactate production during incremental work (Gollnick et al., 1986). This notion helps to explain the effect of glycogen depletion as the rate of glycolysis will be slowed by the lack of substrate leading to a reduction in lactate production (and a higher AT). Strong support for this concept is provided by the observation that plasma adrenaline and noradrenaline levels demonstrate a remarkably similar profile to blood lactate during incremental work (Lehmann et al., 1981). Furthermore, FT glycolytic muscle fibres, which possess little oxidative capacity but have a high potential for lactate formation, are increasingly recruited as work intensity rises (Citterio and Agostino, 1984). Attempts to link a lactate "break point" with abrupt changes in fibre recruitment profiles have, however, been unsuccessful (Helal et al., 1987).

Although somewhat discredited in recent years, the "lack of oxygen" argument has recently received support from the work of Katz and Sahlin, (1988) who found clear evidence of disturbed mitochondrial redox balance at sub-maximal work rates corresponding to the initial rise in blood lactate. As mentioned earlier in this review, the authors deduced that such a condition must result from a lack of molecular oxygen in these organelles.

It thus seems probable that lactate production during sub-maximal exercise results from both an overstimulation of glycolysis, principally in FT fibres, and insufficient mitochondrial O<sub>2</sub>. Although organs such as the liver, kidney and heart are capable of removing lactate from circulation, the major removal process appears to be oxidation in muscle fibres with a high respiratory capacity and low glycolytic potential (Mazzeo et al., 1986).

Most current hypotheses in the literature regarding the AT attempt to account for the above phenomena by discussing blood lactate in terms of a relationship between the volume of lactate production and lactate removal (Brooks, 1986; Hagberg, 1984; Karlsson and Jacobs, 1982; Mader and Heck, 1986; Stegmann et al., 1981). Thus, in a situation where the rate of lactate production equals the rate of its removal, no net accumulation of lactate in muscle or blood

should occur, i.e. a true steady state condition. The changes in blood lactate observed in incremental work are therefore thought to reflect a breakdown in this balance.

No universally accepted definition or method of assessing the AT has yet emerged in the literature. Many groups have focussed on the detection of an initial rise in systemic blood lactate during incremental exercise in order to measure the start of lactate accumulation (Ivy et al., 1980; Poole and Gaesser, 1985; Tanaka et al., 1984b; Yeh et al., 1983). Such tests typically employ short (60-120s) work increments and rely on subjective judgement of the lactate inflection point, although mathematical tools for this purpose have been proposed (Beaver, et al., 1985).

The use of ventilatory parameters to indirectly measure the same initial rise in blood lactate is also popular (Bunc et al., 1987; Davis et al., 1979; Dwyer and Byrnes, 1983), but serious doubts about the validity of this approach have been raised. Variations in substrate availability have been shown to uncouple ventilatory and lactate responses (Hughes et al., 1982) and ventilatory thresholds have been detected in McArdles syndrome patients who, through a lack of muscle phosphorylase, are incapable of significant lactate production (Hagberg, 1984).

It is unclear what the significance of the initial rise in blood lactate under these test conditions is, as it has been shown that prolonged exercise can be sustained without a progressive rise in blood lactate at workloads above those associated with this parameter (Scheen et al., 1981; Stegmann and Kindermann, 1982). Hagberg (1984), and Karlsson and Jacobs (1982) suggest that initial rises in blood lactate during incremental work may be transient and simply represents a period of metabolic adjustment to higher workloads. These authors thus advocate an increment duration of at least 4 minutes to avoid this problem.

The fact that elevated lactate levels do not necessarily infer a progressive accumulation has lead a number of researchers to develop AT measures based on responses to steady state exercise

(Heck et al., 1985; LaFontaine et al., 1981; Sjodin and Jacobs, 1981; Stegmann et al., 1981). The measure that has received most attention is the determination of the exercise intensity corresponding to a blood lactate level of  $4 \text{ mmol.l}^{-1}$ , first described by Mader et al. (1976). They found a mean blood lactate level of  $4 \text{ mmol.l}^{-1}$  at the highest swimming velocity that could be sustained without progressive acidosis. Kindermann et al. (1979) and Sjodin and Jacobs, (1981) extended this work to a 4 minute increment progressive treadmill test for determining the running velocity at  $4 \text{ mmol.l}^{-1}$ , which they termed the onset of blood lactate accumulation (OBLA).

The ability of the  $4 \text{ mmol.l}^{-1}$  protocol to accurately predict the maximum running velocity that can be sustained in prolonged exercise without acidosis was confirmed by Heck et al., 1985. Using a series of 30 minute fixed pace treadmill trials, this group measured the highest running speed attainable without progressive lactate accumulation (MSS), and the running velocity at  $4 \text{ mmol.l}^{-1}$  using a 5 minute increment test, in 16 runners. They found the lactate value in the incremental test that corresponded to MSS running speed averaged  $4.05 \text{ mmol.l}^{-1} (\pm 0.8)$ .

The validity of the  $4 \text{ mmol.l}^{-1}$  AT was challenged by Stegmann and Kindermann (1982), who found that progressive lactate accumulation leading to fatigue occurred in 15 of the 19 rowers they exercised at the power output corresponding to  $4 \text{ mmol.l}^{-1}$ . These authors advocated the use of a protocol for measuring the individual anaerobic threshold (IAT), a mathematical measure based on the lactate-power relationship during exercise and recovery (Stegmann et al., 1981). However, the continuous test they used to determine power at  $4 \text{ mmol.l}^{-1}$  employed 2 min. 50W increments, a work-rate increase which may be too high to enable subjects to reach a true steady state condition at each work level. McLellan (1985) has studied the effect of increment duration on a number of different threshold indices. He found that 1 and 3 min. 30W increment tests yielded significantly lower power outputs for the IAT than the  $4 \text{ mmol.l}^{-1}$  AT but in 5 min. tests no differences were found.

The measurement of the exercise level at  $4 \text{ mmol.l}^{-1}$  lactate thus appears to offer an accurate method of detecting the highest steady state work intensity that can be sustained without lactic acidosis occurring, in other words the AT. The fact that this work rate is found by graphic interpolation is another attraction; this procedure removes the need for the subjective judgement of data, a feature of many alternative threshold measures (Yeh et al., 1983; Conconi et al., 1982). Unfortunately, no validation trials in line with those described above have been reported on racing cyclists undergoing cycle ergometer exercise. One section of this thesis is thus concerned with examining the applicability of the  $4 \text{ mmol.l}^{-1}$  AT to cyclists.

## Skeletal muscle buffering capacity.

Research into the physiology of short duration maximal exercise has recently focussed on muscle buffer capacity ( $\beta$ ) in an attempt to explain the high performance capability demonstrated by sprint athletes (Parkhouse and McKenzie, 1984). McKenzie et al., (1983) found that  $\beta$ , measured by titrating muscle homogenates with acid, was 50% greater in 800m runners than marathoners, the latter have slightly lower  $\beta$  levels than untrained subjects. This group also found a significant relationship between  $\beta$  and both time to exhaustion in a paced anaerobic speed test and percentage fast-twitch fibres. Sahlin and Henriksson (1984), who calculated  $\beta$  from the ratio of released acid to the change in pH of biopsy samples, also found this to be significantly higher in well trained games players than sedentary individuals.

The effect of a high intensity interval training programme on  $\beta$  in previously untrained individuals was studied by Sharp et al., (1986). They found that  $\beta$  was initially the same as a control group of highly trained endurance cyclists, but increased by 37% following eight weeks of training. A particularly interesting finding in this study was the significant increase in  $\dot{V}O_{2max}$  that accompanied the rise in  $\beta$  following sprint training. It thus appears that high quality interval training does not necessarily have a detrimental effect on endurance capacity, a observation that has important implications for pursuing.

Parkhouse and McKenzie (1984) defined a pH buffer as "a substance or mixture of substances that permit solutions to resist large changes in pH upon the addition of small amounts of  $H^+$ ". A variety of methods for buffering protons exist in living cells. Siesjo and Messeter (1971), have classified these into three categories: physio-chemical buffering, consumption or production of non-volatile acids, and exchanges of  $H^+$  and bicarbonate ions ( $HCO_3^-$ ) across membranes.

The most significant intracellular physio-chemical buffering system is thought to be the combining of protons with  $HCO_3^-$  to form

carbonic acid, which then dissociates to  $H_2O$  and  $CO_2$  (Sahlin, 1978). The other major physio-chemical buffering mechanism is the combining of protons with intracellular proteins, particularly carnosine and histidine (Burton, 1978). Parkhouse et al., (1983) has examined the links between carnosine levels,  $\beta$  and anaerobic performance in sprinters, oarsmen, marathon runners and untrained individuals. They found a strong correlation between carnosine concentration and  $\beta$ , the latter being approximately 60% higher in both sprinters and oarsmen than in the other groups. Marathon runners possessed the lowest  $\beta$  value. This group also found a positive relationship between carnosine levels and % FT fibres, which may explain the low  $\beta$  found in marathon runners who typically demonstrate predominantly ST fibre profiles.

The splitting of CP to creatine and inorganic phosphate is a proton consuming reaction and accounts for the bulk of protons buffered through the production or consumption of weak acids or bases (Hultman and Sahlin, 1980). Sahlin (1980), concluded that CP accounts for around 30% of the total muscle  $\beta$ . The importance of this buffering mechanism suggests that the depletion of CP that occurs during maximal exercise may be linked to fatigue, as the exhaustion of this component represents a major reduction in buffering potential. It is interesting to note however, that the study of Sharp et al. cited above failed to find an increase in resting CP levels following sprint training as have other authors (Karlsson et al., 1972).

The transmembrane flux of protons represents an important buffering mechanism to the pursuit cyclist. The high levels of aerobic energy production will necessitate maximal blood flow through the exercising muscles. This will assist in the movement of protons from the intracellular compartment into the blood where they can react with  $HCO_3^-$  to form carbonic acid, or link with plasma proteins. The  $CO_2$  that results from the former is then removed at the lung. The rate of proton release from muscle tissue has been shown to be influenced by plasma  $HCO_3^-$  levels (Hirche, et al., 1975). During maximal aerobic work, blood  $HCO_3^-$  levels drop progressively (Buono and Roby, 1982), indicating a decrease in

extracellular buffering capacity and a potential drop in proton uptake from muscle tissue.

The above findings seem to indicate that endurance training alone has no positive effect on the ability to tolerate the production of a large volume of protons, but this factor can be enhanced through repeated maximal efforts without interfering with aerobic conditioning. This may partly explain the observation that elite endurance athletes often fail to attain high levels of lactate accumulation during maximal exercise. The physiological adaptations responsible for the increase in  $\beta$  are unclear from the studies reviewed but changes in protein buffers appear likely.

Although buffering capacity may well play an important factor in determining pursuit performance, quantification of  $\beta$  requires technically demanding invasive procedures and was not considered as a variable for assessment in this thesis.

## Units of expression for physiological data.

The units in which physiological measures of functional capacity are expressed should ideally reflect the nature of the exercise under investigation. As most forms of athletic activity involve the movement of body mass variables such as oxygen consumption and power output are frequently reported as the absolute value divided by body mass i.e. in  $\text{ml.kg}^{-1}\text{min}^{-1}$  and  $\text{W.g}^{-1}$  respectively. Conditioning relative to body mass appears critical in running where the vertical displacement of body mass during each stride accounts for most of the energy expended. In the case of the pursuit cyclist however, the position is less clear.

It has already been stated that the major resistive force during pursuing is air resistance. This has lead a number of authors to express metabolic data related to body surface area (BSA) (Di Prampero et al., 1979; Marion and Leger, 1988; Van Baak an Binkhorst, 1981). Swain et al., (1987) determined the frontal area (FA) of racing cyclists of varying masses and found that the ratio of FA to mass was lower in heavier riders. They concluded that heavier riders have an advantage over their smaller counterparts in level cycling due to a lower  $\dot{V}O_2/\text{FA}$  at a given sub-maximal cycling speed.

A similar conclusion was made by Secher (1983) following a series of studies on rowing. Here the major resistive force, water drag, is not dramatically effected by body mass so heavier oarsmen have an advantage due to their greater absolute power. Further support for the use of absolute measures comes from a study by Katch (1974) which examined the relationship between total work done in a 2 min. ergometer performance test and various indices of leg and body mass in an homogenous group of male college athletes. He found a strong correlation between total body mass and work done, which suggest that sustained maximal power output is simply a function of body mass in similarly conditioned athletes.

Bergh (1987), has investigated the relationship between body dimensions,  $\dot{V}O_{2\text{max}}$  and levels of success in elite cross-country

skiers. Although the world class skiers possessed similar  $\dot{V}O_{2max}$  values to the less successful elite group when related to bodyweight (83.8 to 79.6 ml.kg.<sup>-1</sup>min<sup>-1</sup>) the former group were, on average, 9.8 Kg heavier and thus had considerably higher absolute  $\dot{V}O_{2max}$  values (6.54 to 5.50 l.min<sup>-1</sup>). However, the smallest co-efficient of variation in  $\dot{V}O_{2max}$  within the groups was found when values were related to body mass<sup>-0.667</sup>, an expression based on the theoretical relationship between aerobic power and body mass (Astrand and Rodahl, 1986). The use of this index for metabolic measurements on cyclists was recommended by Marion and Leger (1988) in preference to either absolute or bodyweight<sup>-1</sup> on the basis that the unit ml.kg.<sup>-0.667</sup>min<sup>-1</sup> best describes the evolution of  $\dot{V}O_2$  with cycling speed.

On the basis of the above information it would seem appropriate to investigate the value of all the indices discussed when examining the relationship between laboratory measures of aerobic performance capacity and pursuing.

PART TWO

EXPERIMENTAL DATA COLLECTION

## Chapter Four

### A GENERAL DESCRIPTION OF DATA COLLECTION PROCEDURES

For simplicity, the experimental and analytical procedures used throughout this programme of research are described in detail in the following section and will only be referred to briefly in the chapters that follow.

#### CYCLE ERGOMETER

The cycle ergometer used for all exercise testing was a Monark 864 drop load, friction braked ergometer. This machine was modified in a number of ways to enable the cyclists to assume a position that closely resembled that achieved on their own cycles. There were two major reasons for this. First, the posture adopted by subjects has been shown to have marked effects on the physiological responses to cycle ergometry (Hamley and Thomas, 1967), so it was important that the cyclists could assume a posture approximating that used in competition. Second, it was thought that such modifications would enhance subject compliance with the exercise protocols. (For a comprehensive description of specific ergometer modifications for racing cyclists see Somerville and Quinney, 1987).

The cranks and bottom bracket bearings were discarded and replaced with conventional racing components. This permitted the use of a various pedal designs and in exceptional cases longer cranks were fitted for tall riders. A solid steel seat post together with a conventional racing saddle and clip enabled saddle position to be set with precision. In all cases this dimension was taken from the cyclists own bicycle.

The canopy covering the pulley and weight cradle arrangement at the front of the ergometer was removed together with the speedometer and cable drive (The latter was found to offer considerable resistance). This permitted the use of dropped handlebars which would otherwise foul the cover, and easy visual inspection of the weight-pulley configuration. A selection of

handlebar stem lengths provided adjustment of reach. Information on pedal cadence was provided for the subject by a digital cycle computer employing a reed switch/magnet arrangement mounted on one crank. Flywheel revolutions were monitored via a second reed switch/magnet couple attached to the flywheel. Power output was calculated from the average flywheel velocity over a 60 second period and the resistive force applied to the flywheel. The latter was provided by suspending calibrated weights of 0.1, 0.5 and 1.0 kg on the cradle attached to the braking belt. Acceleration due to gravity was taken as  $9.81 \text{ m.s}^{-1}$ .

All exercise testing was performed at a fixed pedal rate of  $90 \text{ min}^{-1}$ . This frequency is widely thought to be optimal for exercise testing racing cyclists (Hagberg et al., 1978; Hahn et al., 1986;). A large electric fan placed 1 m in front of the subjects was used to increase evaporative heat loss during strenuous exercise. This was thought to be particularly important during the sustained exercise tests where the more powerful individuals were required to maintain power outputs in excess of 300 W. The conflicting demands on blood flow due to the metabolic heat produced under such circumstances poses a threat to the validity of data.

#### RESPIRATORY GAS EXCHANGE MEASUREMENTS

All gas exchange parameters were measured using standard open circuit procedures. Expired gases were collected in 200l Douglas via a low resistance breathing valve (Jakeman and Davies, 1978) and 1 m of lightweight tubing (internal diameter 0.03 m). Gas fractions were determined with paramagnetic oxygen and infra-red carbon dioxide gas analyzers (Morgan). These machines were calibrated prior to each analysis with known gas mixtures, verified with a Haldane apparatus. Expired volumes were determined with a Parkinson-Cowan CD4 dry gasmeter or a Morgan turbine ventilometer. The accuracy of these devices was checked frequently by evacuating Douglas bags filled with air from a 1 litre calibration syringe, the volume of which had previously been confirmed by water displacement. Oxygen consumption and carbon dioxide production were calculated using the Haldane transformation.

The validity of the respiratory data collected for this thesis was examined by comparing the mean oxygen uptake - power output relationship observed with that measured on cyclists pedalling at frequency of 90 min<sup>-1</sup> by other authors. For simplicity, oxygen uptake at 300 W was compared. The results are listed below in Table 3.

TABLE 3 A comparison of the O<sub>2</sub> cost of cycling at 300 W from various sources.

STUDY	$\dot{V}O_2$ AT 300 W (l.min <sup>-1</sup> )	DATA POINTS
Present study	4.24	123
Hardman and Williams (1985)	4.17	7
Coast and Welch (1985)	4.24	5
Hermansen (1973)	4.34	>100

Although  $\dot{V}O_2$  at 300 W ranged from 3.90 and 4.55 l.min<sup>-1</sup> between individuals it can be seen from the above data that the mean value compares favourably with data from other sources. It is interesting to note that Hardman and Williams (1985) also found a wide range in  $\dot{V}O_2$  at 300 W in the 7 professional cyclists they studied (3.83 to 4.61 l.min<sup>-1</sup>). This suggests that considerable differences in efficiency exist even in highly trained cyclists.

## DETERMINATION OF BLOOD LACTATE

All blood lactate assays were performed on a Roche 640 Lactate Analyzer. Lactate concentration is measured as the background current resulting from the oxidation of lactate to pyruvate by cytochrome c in the presence of an electron acceptor (hexocyanoferrate III). The reliability of this electrochemical method compared with classical assay techniques has been reported to be very high (Poortmans et al., 1978; Tanaka et al., 1984). Arterialized capillary blood was drawn into glass capillaries (50  $\mu$ l, Corning) from a dry, sterile thumb-prick wound. Samples were immediately emptied into vials containing a haemolyzing agent and vigorously mixed. The lysed blood was then diluted 1:10 with the appropriate reagent from a calibrated Oxford micropipetter. Although samples remain stable in room temperature for 24 hours all assays were performed, in duplicate, within 2 hours. The machine was calibrated regularly against 5.0 and 10.0  $\text{mmol.l}^{-1}$  standard solutions and a maximum tolerance of  $\pm 0.1 \text{ mmol.l}^{-1}$  was applied to both calibration and test-retest procedures. The use of arterialized capillary blood samples appears justified as they have been shown to provide comparable acid-base data to arterial blood during exercise (Forster et al., 1972; McEnoy et al., 1975). Furthermore, this procedure is easily performed in the field.

The reproducibility of this assay technique was examined statistically on 100 duplicate measurements of blood lactate samples in the range 1 - 14  $\text{mmol.l}^{-1}$ . A standard method error of 0.063  $\text{mmol.l}^{-1}$  and of coefficient of variation of 3.1 % together with a correlation co-efficient of 0.9994 was found.

## Chapter Five

### EXPERIMENT I: THE RELATIONSHIP BETWEEN BLOOD LACTATE RESPONSES TO SUSTAINED POWER OUTPUT AND INCREMENTAL EXERCISE IN COMPETITIVE CYCLISTS.

#### INTRODUCTION

The applicability of the anaerobic threshold (AT) concept to short duration aerobic performance is a major issue in this thesis. It was thus important to select the most appropriate method of determining the AT of cyclists. The review of literature highlighted the need to identify the work intensity at which a progressive accumulation of lactate will occur during sustained steady state exercise, as this appears to be the only theoretically sound method of establishing that a true net lactate production has taken place. The review revealed weaknesses in AT protocols based on rapid incremental work as these do not appear to predict accurately true blood lactate responses to sustained steady state exercise (Hagberg, 1984). The use of a series of prolonged power output tests to determine an individual's AT was, however, an impractical approach for most of the data collection in this thesis, due mostly to the logistics of testing a widely dispersed national team on a regular basis.

The primary objective of this study therefore, was to develop a single incremental worktest capable of determining the power output at which a maximal steady state lactate (MSSL) level is reached in cyclists, and above which, a progressive lactate accumulation will occur in sustained exercise. Three discrete points on the lactate-power output graph, chosen to reflect the most common AT parameters reported in the literature, were examined: the power output corresponding to the initial rise in blood lactate above a baseline level, often termed the lactate threshold or LT (Buchanan and Weltman, 1985), power output at 2 mmol.l<sup>-1</sup> and 4 mmol.l<sup>-1</sup> lactate. It was hypothesized that the power output at 4 mmol.l<sup>-1</sup> lactate during incremental work would most closely approximate the MSSL power output.

## METHODS

11 male racing cyclists took part in this study. Performance levels varied from recreational veteran to world class professional. Their mean ( $\pm$  SD) age, mass and height were 24.2 (8.9) years, 74.9 (5.8) kg and 1.84 (0.06) m respectively. (See appendix 1 for individual data).

The subjects underwent an initial  $\dot{V}O_{2\max}$  test on the cycle ergometer. Following a warm up of at least 5 minutes a power output of between 200 and 250 W was applied on the ergometer and increased by 35 W every 90 seconds. The starting work load was selected on the basis of known performance capability. A short increment duration was chosen as this has been shown to yield higher  $\dot{V}O_{2\max}$  values in trained cyclists than 3 or 5 minute loads (Hahn et al., 1986; Mclellan, 1982). Tests were terminated when pedal frequency dropped by more than 5  $\text{min}^{-1}$  or upon volitional fatigue. Strong verbal encouragement was given towards the end of the test. Expired gas was collected continuously during the final stages using douglas bags connected in a series circuit, and analyzed in the manner outlined previously. Flywheel revolutions were monitored over 60 second periods for the determination of power output. On completion of a test the braking load was immediately removed and the subject encouraged to continue pedalling slowly. Thumb-prick blood samples were drawn 3 minutes post test for the determination of peak blood lactate concentration (Saiki et al., 1967; Svedenhag and Sjodin, 1984).

In the absence of a clear plateau in oxygen uptake with increasing power  $\dot{V}O_{2\max}$  was taken to be the highest oxygen uptake measured over a 60 second period.

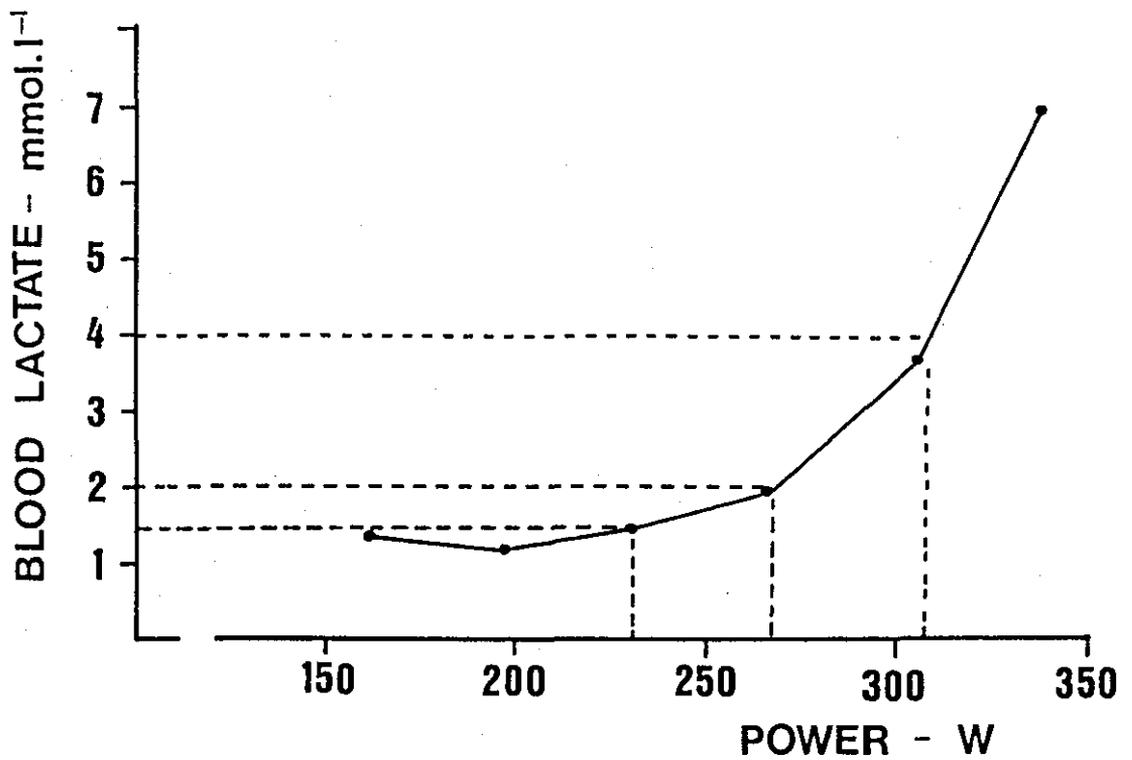
A protocol similar to that described by Sjodin and Jacobs (1981) was used to measure blood lactate responses to incremental cycle ergometer exercise. Following a 5 minute warm up at approximately 100 W an initial work load of between 120 and 170 W, depending on the conditioning of the subject, was applied to the flywheel. Power output was increased by 35 W every 4th minute. Blood samples were

drawn during the last 30 s of each increment and analyzed in the manner described earlier. Subjects continued to pedal at the required rate throughout blood sampling. Expired gases were collected during the 3rd minute of each workload for the determination of oxygen uptake.

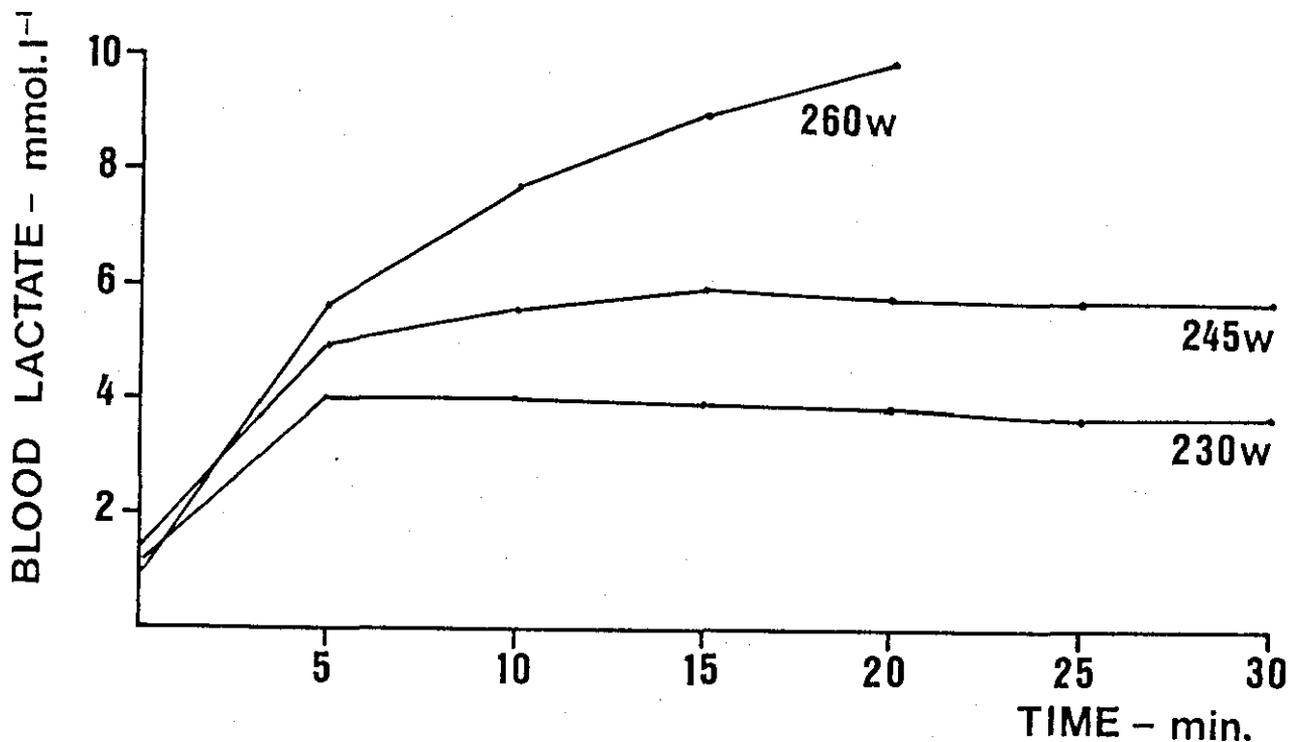
The power outputs corresponding to 2 and 4  $\text{mmol.l}^{-1}$  were determined by interpolation from the graphs of power output and blood lactate. The power output at which blood lactate began to rise systematically (LT) was determined visually by subjective judgement. Figure 2 shows the location of all the threshold indices on a typical power/lactate graph.

The power output at which a MSSL level could be identified was determined by a series of constant power output work bouts lasting 30 minutes. At least 48 hours was allowed between each test and all testing was completed within 3 weeks of the initial incremental protocol. Subjects were discouraged from hard training on the day before visiting the laboratory. Before the 30 minute test commenced a gradually increasing intensity warm up lasting 10 minutes was performed in an attempt to avoid the effects of the transient lactate formation that occurs at the onset of exercise or following abrupt rises in power output (Karlsson and Jacobs, 1982). Blood samples were drawn every 5 minutes and flywheel revolutions were monitored every 2 minutes to ensure that subjects maintained the correct power output. The first test was performed at a power output between 10 and 20 W below that corresponding to 4  $\text{mmol.l}^{-1}$  lactate. On subsequent tests power was raised approximately 15 W until a continuous rise in blood lactate was observed. Tests were terminated if the subject allowed pedal rate to drop by more than 2  $\text{min}^{-1}$ . The MSSL level was defined as the highest power output that could be sustained for 30 minutes without a continuous rise in blood lactate, or an elevation of more than 1.0  $\text{mmol.l}^{-1}$  between the 5th and 30th minute (after Heck et al., 1985).

**FIGURE 2.** Example of a typical blood lactate response to incremental exercise showing the location of the three threshold indices. (Data from subject 7)



**FIGURE 3.** Example of typical blood lactate responses to sustained exercise at varying power outputs. (data from subject 8)



Relationships between the three threshold indices, power output at MSSL and  $\dot{V}O_{2max}$  were examined with a Pearson Product Moment correlation test.

## RESULTS

$\dot{V}O_{2max}$  ( $\pm$  standard deviation) averaged  $5.2 \text{ l}\cdot\text{min}^{-1}$  ( $\pm 0.9$ ) with a range of  $4.0 - 6.7 \text{ l}\cdot\text{min}^{-1}$ ; individual values are given in table 4. Seven subjects demonstrated a clear plateau in oxygen uptake (less than  $0.15 \text{ l}$  increase between last 2 workloads) but although the remaining 3 achieved heart rates within  $5 \text{ b}\cdot\text{min}^{-1}$  of age predicted maximum and respiratory exchange ratios above 1.05 no levelling off in oxygen uptake was visible. One subject did not undergo a  $\dot{V}O_{2max}$  test, as he was over 50 years old and medical supervision of the test could not be arranged.

The power outputs corresponding to the 3 threshold indices measured in the incremental threshold test, LT, 2 and  $4 \text{ mmol}\cdot\text{l}^{-1}$ , are also given shown in table 4. Mean values ( $\pm$  SD) were  $214 \text{ W}$  ( $\pm 50$ ),  $237 \text{ W}$  ( $\pm 64$ ) and  $283 \text{ W}$  ( $\pm 58$ ) respectively.

All subjects demonstrated a identifiable MSSL power output (Power MSSL) which averaged  $279 \text{ W}$  ( $\pm 55$ ). When this workload was exceeded in sustained exercise progressive lactate accumulation was observed. All but 3 subjects exhausted before completing 30 minutes when exercising at one work level ( $15 \text{ W}$ ) above Power MSSL. Figure 3 shows typical responses to varying steady state workloads. The remaining 3 subjects who completed a 30 minute test with accumulating lactate all showed signs of considerable distress during the latter stages but were sufficiently motivated to finish. The mean blood lactate level over the MSSL 30 minute test (mean lactate @ MSSL) averaged  $3.75 \text{ mmol}\cdot\text{l}^{-1}$  ( $\pm 0.9$ ) and the oxygen uptake at Power MSSL corresponded to  $81.5\%$  ( $\pm 4.5$ ) of  $\dot{V}O_{2max}$  ( $n = 10$ ). All individual MSSL data is shown in table 5.

The blood lactate level during the incremental test corresponding to Power MSSL (MSSL lactate) determined by interpolation, was  $3.77 \text{ mmol}\cdot\text{l}^{-1}$  ( $0.3$ ) and a significant correlation of  $R = 0.67$  ( $P < 0.05$ ) ...

**TABLE 4.**  $\dot{V}O_{2max}$  ( $l \cdot min^{-1}$ ) and power output (W) corresponding to LT, 2  $mmol \cdot l^{-1}$  and 4  $mmol \cdot l^{-1}$  lactate determined from incremental exercise.

SUBJECT	$\dot{V}O_{2max}$	POWER LT	POWER 2 $mmol \cdot l^{-1}$	POWER 4 $mmol \cdot l^{-1}$
1	6.75	315	334	388
2	4.45	166	158	224
3	5.93	268	280	338
4		152	160	212
5	5.33	238	260	311
6	4.05	150	150	200
7	5.95	232	268	308
8	4.48	198		246
9	5.22	224	240	296
10	4.52	185	220	270
11	5.69	232	302	318
$\bar{X}$	5.24	214	237	283
S.D	0.9	50	64	58

**TABLE 5.** MSSL power (W), mean lactate @ MSSL, and the lactate concentration during incremental work corresponding to the MSSL power output (MSSL lactate).

SUBJECT	POWER MSSL	MEAN LACTATE @ MSSL	MSSL LACTATE
1	375	2.47	3.45
2	235	4.88	4.55
3	327	3.76	3.45
4	203	2.80	3.50
5	309	2.98	3.90
6	195	4.02	3.70
7	297	2.77	3.30
8	245	5.58	4.00
9	297	4.10	4.05
10	265	4.02	3.55
11	318	3.85	4.00
$\bar{X}$	279	3.75	3.77
S.D.	55	0.9	0.3

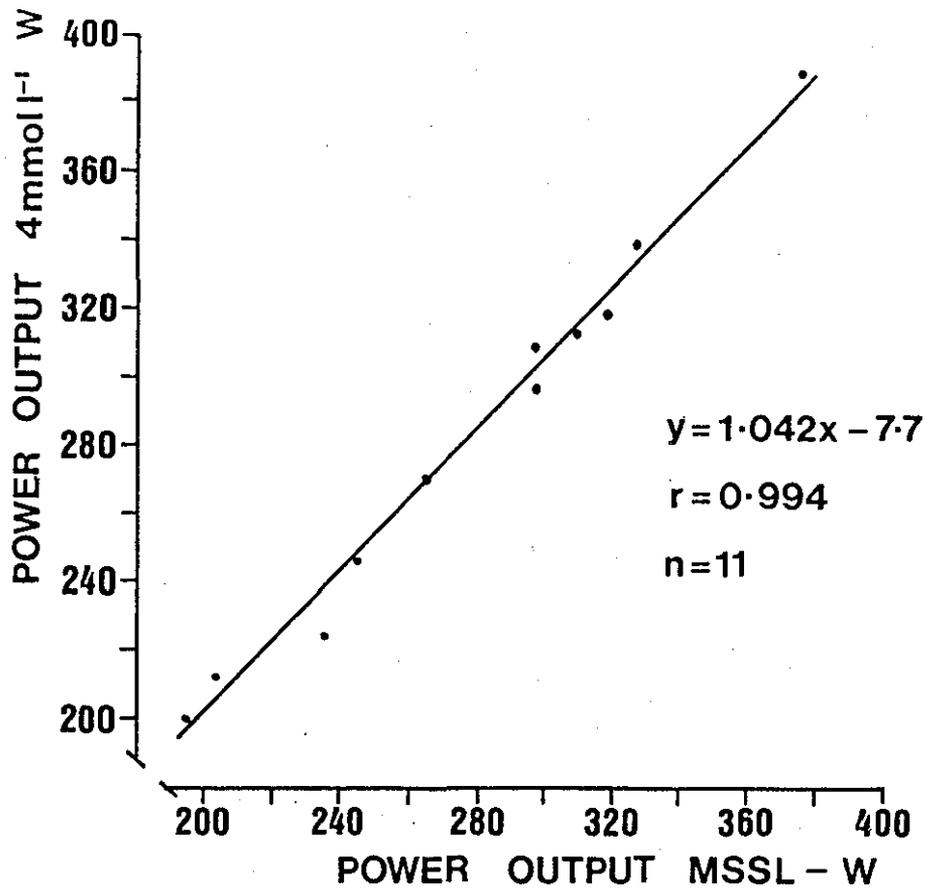
... was found between this lactate value and the mean lactate @ MSSL. All three threshold indices were found to correlate significantly with Power MSSL (see table 6), with Power 4 mmol.l<sup>-1</sup> demonstrating the strongest relationship ( R = 0.994; p < 0.001). This relationship is shown graphically in figure 4. Mean Power 4mmol.l<sup>-1</sup> was 4 W higher than Power MSSL, whereas Power 2 mmol.l<sup>-1</sup> and Power LT were 43 W and 65 W lower than this parameter, respectively.

**TABLE 6.** Correlation coefficients between various anaerobic threshold indices and MSSL power output.

	1	2	3	4	5
1 POWER MSSL	-----				
2 POWER LT	0.75*	-----			
3 POWER 2 mmol.l <sup>-1</sup>	0.97**	0.73*	-----		
4 POWER 4 mmol.l <sup>-1</sup>	0.99**	0.76*	0.98**	-----	
5 MSSL LACTATE	-0.42	-0.43	-0.51	-0.48	-----

\* = P<0.05, \*\* = P<0.001

**FIGURE 4.** The relationship between Power 4 mmol.l<sup>-1</sup> (W) measured during incremental exercise and the maximum steady state lactate power output (Power MSSL).



## DISCUSSION

These data provide considerable support for the existence of a sub-maximal metabolic threshold above which exercise cannot be performed without the continuous accumulation of lactate in blood. Jacobs (1981) found that muscle lactate concentration is considerably greater than that of blood during steady state bicycle ergometer work in which blood lactate was accumulating, and Sahlin (1978) has shown that total muscle pH is linearly related to muscle lactate concentration following 5-10 min. of exhaustive cycling. It thus seems reasonable to assume that the lactate accumulation observed in these cyclists when performing sustained work above Power MSSL was indicative of a marked metabolic acidosis. The fact that Power MSSL occurred, on average, at 81.3% of  $\dot{V}O_{2max}$  leads to the conclusion that metabolic acidosis does occur in steady state sub-maximal exercise even in highly trained individuals, despite an apparent oxygen uptake reserve. This finding is in agreement with many other studies (e.g. Hermansen and Stensvold, 1972; Kindermann et al., 1979; Scheen et al., 1981) but conflicts with the observations of Saiki et al., 1967. The last named authors claimed that sustained exercise at between 90 and 100% of  $\dot{V}O_{2max}$  did not result in lactate accumulation. It is difficult to explain the latter when, for example, Saltin and Karlsson have reported mean muscle lactate concentrations of 20 mmol.kg<sup>-1</sup> wet weight following exercise at  $\dot{V}O_{2max}$ .

It would be tempting to conclude that acidosis was the cause of fatigue in the 8 cyclists who failed to complete the 30 min. test above MSSL, but this view is not supported by the evidence that changes in muscle pH following exhaustive exercise of this duration are typically lower than those following maximal work (Karlsson, 1979).

The observation of an MSSL exercise level in trained cyclists is in agreement with the findings of authors who have studied other groups of athletes. Heck et al. (1985) identified a clear MSSL running velocity in 16 endurance runners using a series of 30 minute treadmill runs. This group also found a close relationship

between running velocity at 4 mmol.l<sup>-1</sup> and MSSL velocity. LaFontaine et al. (1981) also refer to a MSSL running velocity in runners but claim this occurs at a venous lactate level of 2.2 mmol.l<sup>-1</sup>. This does not appear to apply to the cyclists studied in this investigation however, as the power output at 2 mmol.l<sup>-1</sup> was considerably lower than Power MSSL (237 W vs 279 W). The 2 mmol.l<sup>-1</sup> parameter thus appears to be inappropriate for predicting the MSS condition in racing cyclists.

Stegmann and Kindermann (1982) identified a MSSL power output in 19 rowers through a series of 50 min. ergometer trials but found that this was poorly related to the power output at 4 mmol.l<sup>-1</sup> measured in incremental work. 15 of their 19 subjects demonstrated a rapid and progressive lactate accumulation leading to exhaustion in approximately 15 minutes when exercising at Power 4 mmol.l<sup>-1</sup>. This group also observed that the higher the conditioning of the athlete the lower the lactate level at MSSL, and consequently the greater the over-estimation of MSSL by the 4 mmol.l<sup>-1</sup> parameter. Such a relationship was not found in the current data as neither VO<sub>2max</sub> nor Power MSSL were correlated to MSSL lactate (R = -0.467 and -0.416 respectively). The explanation for these discrepancies probably lies in the fact that Stegmann employed a 2 min. 50 W increment work test. Such a rapid work rate increase is unlikely to yield true steady state responses at each power level. This point is underlined by the findings of Heck et al. (1985) and McClellan (1985) who both found that blood lactate levels were lower at a given power output in 1 and 3 minute increment work tests compared with a 5 minute protocol. Furthermore, Jacobs (1986) has shown that variations in both increment magnitude and duration have marked effects on the blood lactate-power response.

The strong inter-correlations between all 3 threshold measures and Power MSSL (see table 6) indicate that almost any clearly identifiable point on the blood lactate/power output curve will yield useful information regarding the anaerobic threshold of a racing cyclist. This may explain why consistently high correlations are reported between endurance performance and AT, regardless of threshold index employed (Jacobs, 1986). The 4 mmol.l<sup>-1</sup> point

however, most closely approximated the MSSL power (283 W vs 279 W, see tables 5,6 and figure 4) and therefore must be considered the most appropriate measure for this particular group of athletes. Although Power 2  $\text{mmol.l}^{-1}$  was equally closely correlated to Power MSSL ( $R = 0.979$ ) and would require a less strenuous test for its determination a problem with this parameter occurs when a subject does not demonstrate blood lactate levels below this value throughout a work test. This phenomenon was observed in one subject.

A further advantage of the 4  $\text{mmol.l}^{-1}$  AT parameter is the little impact errors or variations in blood sampling techniques should have on the measure. This is because the lactate value of 4  $\text{mmol.l}^{-1}$  usually lies on the steep portion of the lactate/power graph so even errors of  $\pm 0.5 \text{ mmol.l}^{-1}$  will only result in small variations in the interpolated power output. The impact of the same relative error on the 2  $\text{mmol.l}^{-1}$  parameter would be considerably greater as this value is typically located on the relatively flat or slowly rising phase of the graph. The fact that venous and arterial blood sampling methods have been shown to yield slightly different lactate values (Poortmans et al., 1978; Yeh et al., 1983) provides additional support for the use of the 4  $\text{mmol.l}^{-1}$  measure as this would enable more accurate comparisons of data generated in different laboratories to be made.

Although this experiment has provided more empirical support for the existence of an anaerobic threshold coinciding with the attainment of a blood lactate level of approximately 4  $\text{mmol.l}^{-1}$ , no clear theoretical basis for this phenomenon has yet emerged from the literature. The finding of Jorfeldt et al. (1978) that a maximal rate of lactate release from exercising leg muscle into blood is reached at a muscle lactate concentration of 4 to 5  $\text{mmol.Kg}^{-1}$ , has been proposed as a possible mechanism (Karlsson and Jacobs, 1982). This explanation has, however, been challenged by Mader and Heck (1986) who re-examined Jorfeldt's data. They found that when the rate of lactate release from muscle was plotted against the difference between muscle and blood lactate (i.e. the concentration gradient) a linear relationship was observed. This suggests that a

saturation point for the movement of lactate across the muscle membrane had not in fact been reached.

It has been stated already that a key concept in the theory of the anaerobic threshold is the relationship between the rate of lactate production and removal. As human skeletal muscle is essentially heterogenous in terms of fibre composition (Pette, 1984), it is likely that a significant volume of lactate turnover occurs purely within a isolated muscle through the movement of lactate between adjacent muscle fibres of varying respiratory capacity (Karlsson and Jacobs, 1982). It is thus possible to apply the anaerobic threshold concept to a single muscle: lactate accumulation will occur when the capacity of oxidative fibres to consume lactate is surpassed by the rate at which it is produced (by glycolytic fibres).

Saltin and Karlsson (1971) have observed an abrupt rise in muscle lactate (V. Lateralis), determined by needle biopsy, during increasing intensity steady state cycle exercise (10 minute work loads). In the trained subjects this rapid increase occurred at a muscle lactate concentration of between 3 and 4 mmol.Kg<sup>-1</sup> (wet weight). The blood lactate concentrations reported parallel the wet muscle values up to this level but beyond it a concentration gradient develops between the two compartments. A blood lactate value of 3 - 4 mmol.l<sup>-1</sup> may therefore indicate the attainment of the "muscle AT" - a metabolic rate beyond which skeletal muscle is unable to maintain an effective lactate steady state. Jacobs (1981) has also presented data on simultaneous muscle and blood lactate measurement during 4 minute incremental work. He found a concentration gradient between muscle and blood at all work levels including those eliciting blood lactate levels below 4 mmol.l<sup>-1</sup>, suggesting that no equilibrium is achieved between the two compartments. A possible explanation for this discrepancy may be that Jacob's subjects did not achieve true steady state conditions. Firstly, the work load increment used was 50 watts, which represents a massive increase in exercise intensity for the subjects who were neither highly trained or particularly large (68 ± 2 kg). Secondly, the subjects had to stop exercising at the end of each 4 minute bout to enable a biopsy sample to be taken. This would

entail having to commence increasingly strenuous workloads following a short period of complete inactivity. Both factors are likely to lead to transient lactate formation within the muscle. This will result in a muscle-blood concentration gradient unless each work level was sustained for a period long enough to allow equilibrium to occur, assuming this was below the AT (MSSL intensity). Further studies are clearly needed to verify the relationship between muscle and blood lactate during steady state sub-maximal exercise.

Mader and Heck (1986) have developed a mathematical model which is claimed to predict the nature of lactate kinetics during exercise using a 2 compartment model. Briefly, this model attempts to describe the relationship between the rates of lactate production and lactate removal within the active muscle compartment, the passive distribution space (mainly blood volume), and the aerobic power of an individual. The activation rates of glycolysis and oxidative phosphorylation are derived from the concentration of CP and ADP (the de-phosphorylation state of muscle tissue) which is in turn predicted for a given exercise intensity and  $\dot{V}O_{2\max}$ . The AT is taken as the point at which the enzyme pyruvate dehydrogenase (PDH) becomes saturated with pyruvate, presumably referring to a total net effect within the active muscle compartment. The authors claim that this saturation point corresponds to a steady state blood lactate level of approximately  $4 \text{ mmol.l}^{-1}$ , but no clear explanation is given for this.

Much of the logic underlying Mader and Heck's theory is based on the notion that ADP is the primary activator of glycolysis. However, recent studies of phosphorous metabolites using nuclear magnetic resonance have raised serious doubts about the role of ADP in regulating glycolysis. Dawson (1983) found that the high levels of ADP produced in ischemic human forearms failed to significantly activate glycolysis; muscle contraction itself appeared to be the critical activator of glycogen catabolism.

Thus it appears that although there is strong empirical evidence for the existence of a maximal steady state work level corresponding to

a blood lactate level in the region of  $4 \text{ mmol.l}^{-1}$ , the mechanisms responsible for this phenomenon remain poorly understood.

In summary, this experiment has clearly demonstrated that an anaerobic threshold can be detected in racing cyclists. When sub-maximal exercise is performed above this intensity a progressive accumulation of lactate occurs. The location of this threshold is accurately determined by interpolating the power output corresponding to  $4 \text{ mmol.l}^{-1}$  blood lactate during a progressive incremental work test comprising 4 min. 35 W loads.

## Chapter Six

### EXPERIMENT II: AN EXAMINATION OF THE RELATIONSHIPS BETWEEN SELECTED LABORATORY INDICES OF AEROBIC CONDITIONING AND PURSUIT PERFORMANCE IN NATIONAL COMPETITION.

#### INTRODUCTION

The aims of this investigation were to examine the relationship between pursuit racing ability and the  $\dot{V}O_{2max}$  and AT (Power 4 mmol.l<sup>-1</sup>) of experienced pursuit cyclists, and to identify the most appropriate method of expressing physiological data regarding pursuit cyclists.

One of the observations made in the pilot study that preceded this thesis was the need to use performance data from actual competitions, rather than non-competitive trials, when attempting to investigate the relationship between laboratory measures of exercise capacity and athletic success (Keen et al., 1985). However, the structure of a major pursuit competition raises problems regarding the collection of such performance data. A championship is typically composed of an initial qualification round to select the fastest 16 riders, followed by 4 rounds of match racing in which riders are matched on the basis of fastest against slowest (from the previous round). It is therefore possible for top riders to progress to the latter stages of the competition without the need to fully extend themselves. Furthermore, tactical riding frequently occurs. For example, a rider will often only race hard enough to just defeat a weak opponent, either to preserve energy for the next round or to gain a psychological advantage.

A solution to this problem would be to run an individual time trial. As the pursuit race is an event performed in isolation from other competitors it could be argued that this would provide an accurate simulation. Unfortunately, the demands of actual match competition invariably produce higher levels of performance. This observation is confirmed by the data in table 1 (page 12) which

shows that official world records, which have to be set under strict time trial conditions, are all slower than the best times achieved during competition.

As the primary objective of this thesis was to develop a greater understanding of the pursuit race it was concluded that the most appropriate measure of pursuit performance would be the fastest time achieved by a rider during a actual pursuit competition rather than in a non-competitive time trial.

#### METHODS

9 male pursuit cyclists, all of whom were of national squad standard or above, took part in this investigation. Mean ( $\pm$  S.D.) age, height and weight were  $20.2 \pm 3.2$  years,  $75.4 \pm 5.7$  kg and  $1.80 \pm .07$  M respectively. Individual anthropometric data is given in appendix 3.

The performance of all subjects was monitored throughout the 1987 British Pursuit Championships, staged at the 333.3 M hardwood outdoor velodrome in Leicester. Although it was originally intended to only use data from senior amateurs who competed in the 4000 M event, a number of prospective subjects failed to race in the championships through injury or illness. As a result of this, data from two junior males, who raced over 3000 M, and one professional (5000 M) were included. Performance data was therefore expressed as average race speed rather than time, due to the different race distances.

Thumb-prick blood samples were drawn 3 minutes after completion of each pursuit race and analyzed for lactate concentration in the manner outlined earlier. This delay time appears appropriate for estimating peak blood lactate concentration in endurance athletes following maximal physical exercise lasting 5 to 10 minutes (Sharp et al., 1986). The riders were not allowed to actively warm down before blood sampling took place.

Subjects attended the laboratory within 2 weeks of competing in

the Track Championships and underwent tests to determine power output at 4 mmol.l<sup>-1</sup> lactate and  $\dot{V}O_{2max}$ . The exercise test protocols were identical to those described in the previous chapter. All subjects were fully habituated to the testing procedures, having undergone similar evaluations on a number of previous occasions as part of a routine assessment programme for the National Squad. Most of the cyclists were tested in the week before competition and were not informed of their results until after the championship. It was thought that knowledge of test data might influence the performance of individuals who were aware of their previous test results, and more importantly, those of their colleagues.

$\dot{V}O_{2max}$  and Power 4 mmol.l<sup>-1</sup> lactate values were expressed as absolute values, and related to body mass, body mass<sup>-0.667</sup> and body surface area (BSA). The formula of Du Bois and Du Bois (1916) was used to estimate BSA:

$$BSA (M^2) = 0.007184 \times \text{Height}^{0.725} (\text{cm}) \times \text{Weight}^{0.425} (\text{kg})$$

The relationships between these data and pursuit speed were statistically examined with a Pearson Product Moment correlation test.

## RESULTS

All the subjects performed with distinction in the pursuit championships. The winners of the junior, senior and professional titles were members of the subject group, which also contained four of the first 5 riders in the senior race. The first and second placed riders in the womens pursuit also underwent prior laboratory assessment but it was not thought appropriate to include these data with those from the male cyclists in the statistical analyses.

The Power 4  $\text{mmol.l}^{-1}$  lactate values expressed in the units of W, W.  $\text{M}^{-2}$  BSA, W.  $\text{kg}^{-0.667}$  body mass, and W.  $\text{kg}^{-1}$  body mass are shown in table 7, together with pursuit speed. Significant Correlations were found between all the Power 4  $\text{mmol.l}^{-1}$  indices and pursuit speed (see figures 5-8). The highest correlation, ( $r = 0.93$ ;  $P < 0.01$ ) occurred for the absolute power output figure and the lowest ( $r = 0.69$ ;  $P < 0.05$ ) for the bodyweight related value. Power 4  $\text{mmol.l}^{-1}$  expressed as W.  $\text{M}^{-2}$  and W.  $\text{kg}^{-0.667}$  yielded correlations of 0.92 and 0.84 respectively.

The results of the  $\dot{V}\text{O}_{2\text{max}}$  test, again expressed in the 4 different units, are given in table 8, and the performance correlations for this parameter are shown graphically in figures 5-8. The highest correlation between  $\dot{V}\text{O}_{2\text{max}}$  and pursuit speed was found when the absolute value,  $\text{l.min}^{-1}$ , was used ( $r = 0.69$ ;  $P < 0.05$ ). A similar correlation of  $r = 0.64$  ( $p < 0.05$ ) existed for the BSA related figures but non-significant  $r$  values of 0.59 of 0.36 were found when the  $\text{ml.kg}^{-0.667}.\text{min}^{-1}$  and  $\text{ml.kg}^{-1}.\text{min}^{-1}$  indices were employed.

Post race blood lactate levels averaged  $14.1 \pm 0.9 \text{ mmol.l}^{-1}$ . A non-significant correlation of  $r = 0.13$  was found between pursuit speed and post race blood lactate.

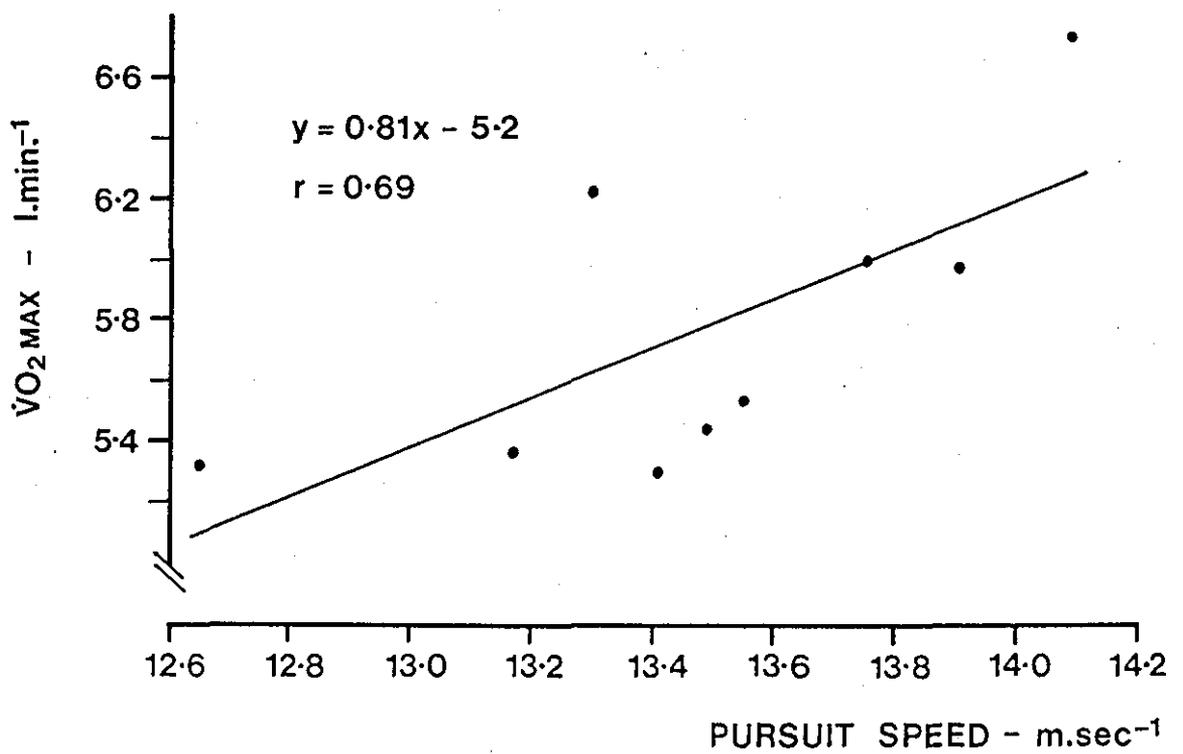
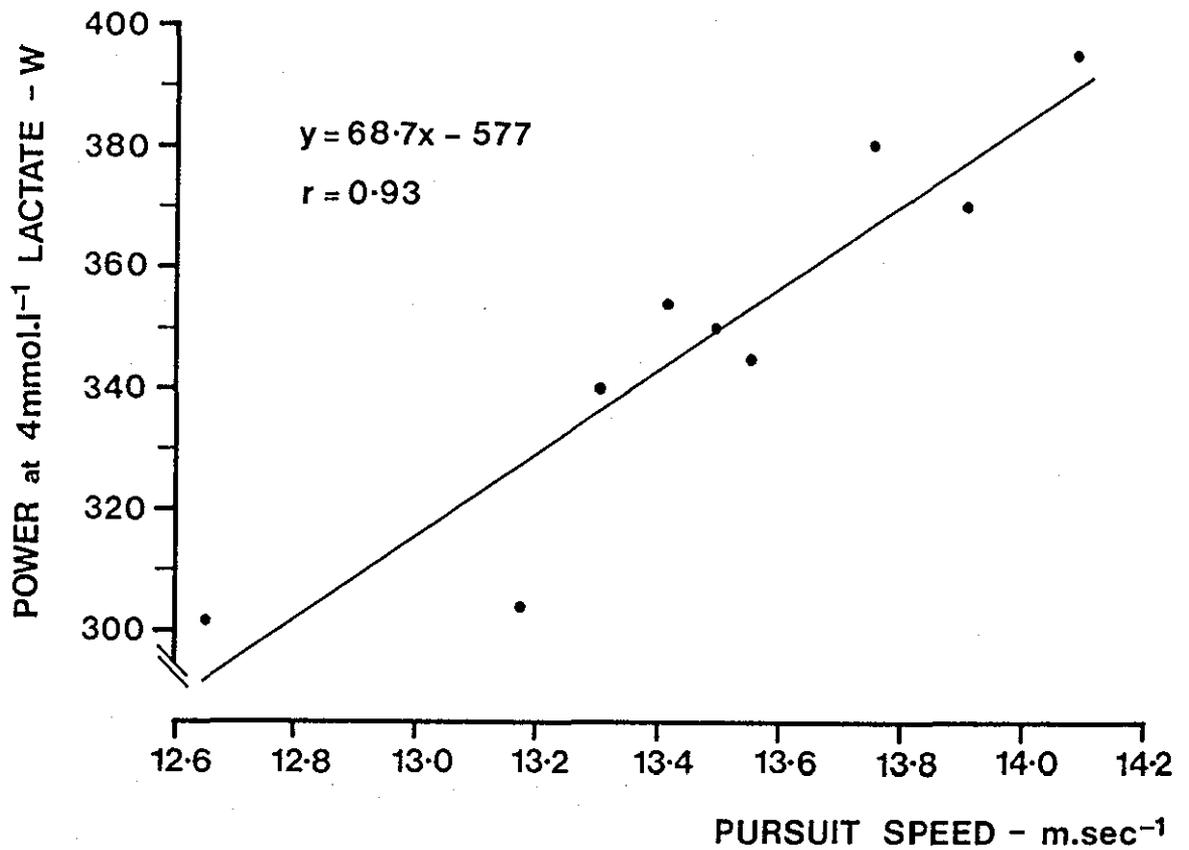
**TABLE 7.** Pursuit race speed ( $\text{m}\cdot\text{s}^{-1}$ ) and Power 4  $\text{mmol}\cdot\text{l}^{-1}$  data expressed in various units.

SUBJECT	RACE SPEED	-----POWER 4 $\text{mmol}\cdot\text{l}^{-1}$ Lactate-----			
		Watts	$\text{W}\cdot\text{kg}^{-1}$	$\text{W}\cdot\text{kg}^{-0.667}$	$\text{W}\cdot\text{m}^{-2}$ BSA
1	12.65	302	4.13	17.3	159
2	13.17	304	4.48	18.8	167
3	13.30	340	4.23	18.7	166
4	13.41	354	4.62	19.7	179
5	13.49	350	4.75	20.5	180
6	13.55	345	5.17	21.6	191
7	13.75	380	4.78	20.5	189
8	13.90	370	4.71	20.2	188
9	14.08	395	4.79	20.9	206
$\bar{X}$	13.47	348	4.63	19.9	180
S.D.	0.42	31	0.3	1.4	14

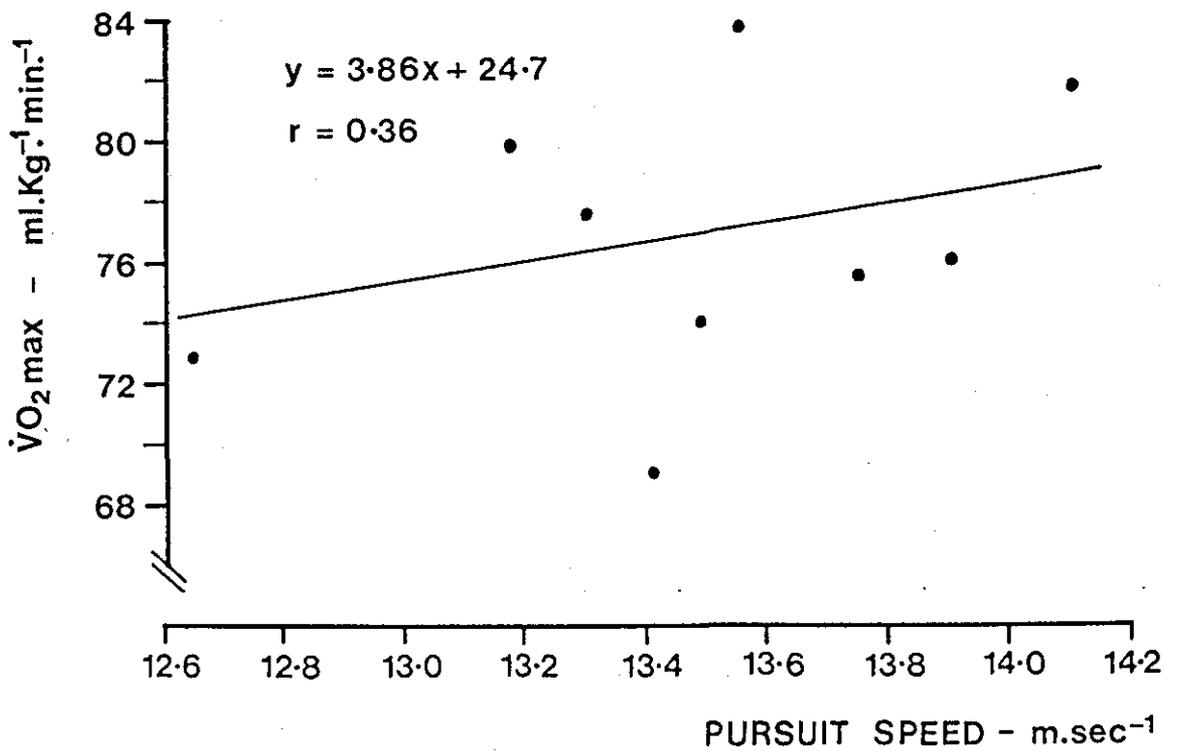
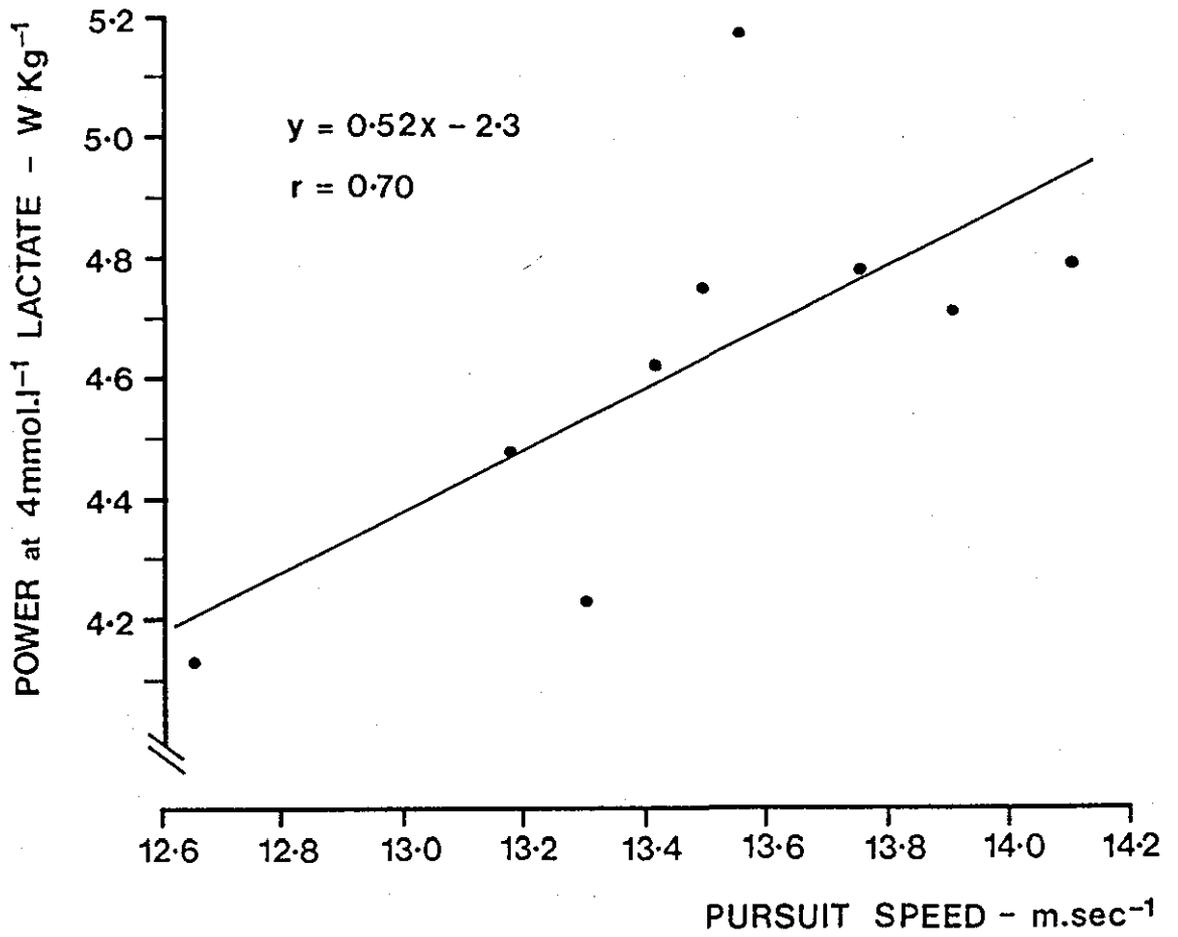
**TABLE 8.** Pursuit race speed ( $\text{m}\cdot\text{s}^{-1}$ ) and  $\dot{V}\text{O}_{2\text{max}}$  data expressed in various units.

SUBJECT	RACE SPEED	----- $\dot{V}\text{O}_{2\text{max}}$ -----			
		$\text{l}\cdot\text{min}^{-1}$	$\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	$\text{ml}\cdot\text{kg}^{-0.667}\cdot\text{min}^{-1}$	$\text{l}\cdot\text{m}^{-2}$
1	12.65	5.32	72.9	305	2.80
2	13.17	5.37	79.8	322	2.99
3	13.30	6.23	77.6	335	2.97
4	13.41	5.30	69.1	293	2.67
5	13.49	5.45	74.0	313	2.84
6	13.55	5.55	83.8	337	3.09
7	13.75	6.01	75.6	324	2.99
8	13.90	5.97	76.1	325	3.04
9	14.08	6.75	81.8	356	3.27
$\bar{X}$	13.47	5.78	76.7	323	2.96
S.D.	0.42	0.5	4.6	17	0.2

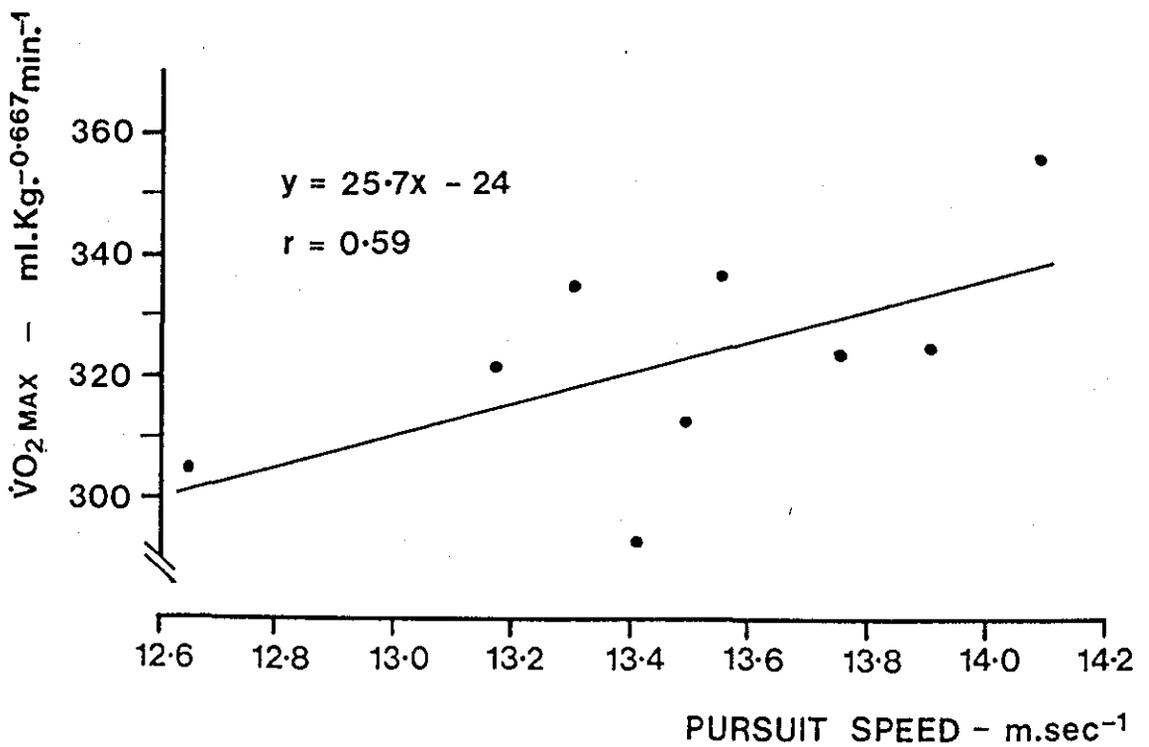
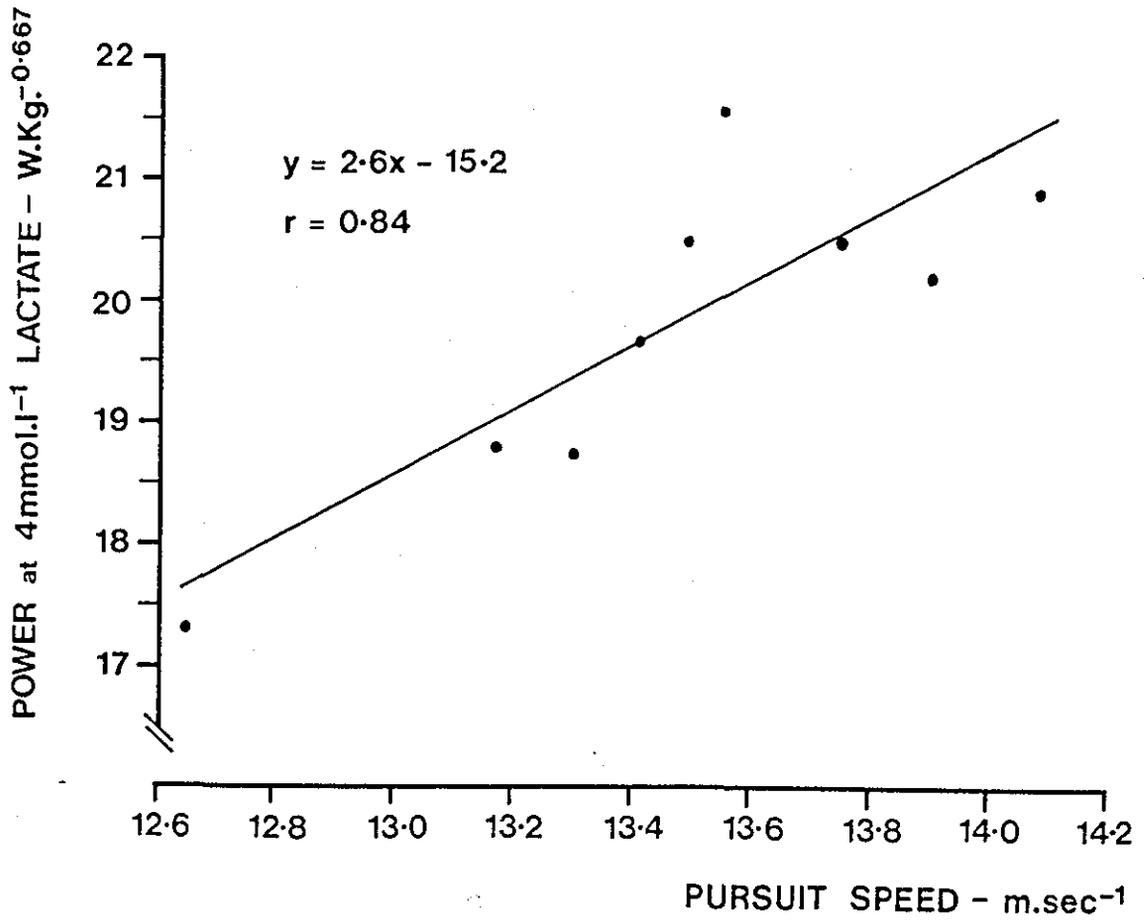
**FIGURE 5.** Relationships between pursuit speed and absolute measures of Power 4 mmol.l<sup>-1</sup> and VO<sub>2</sub>max.



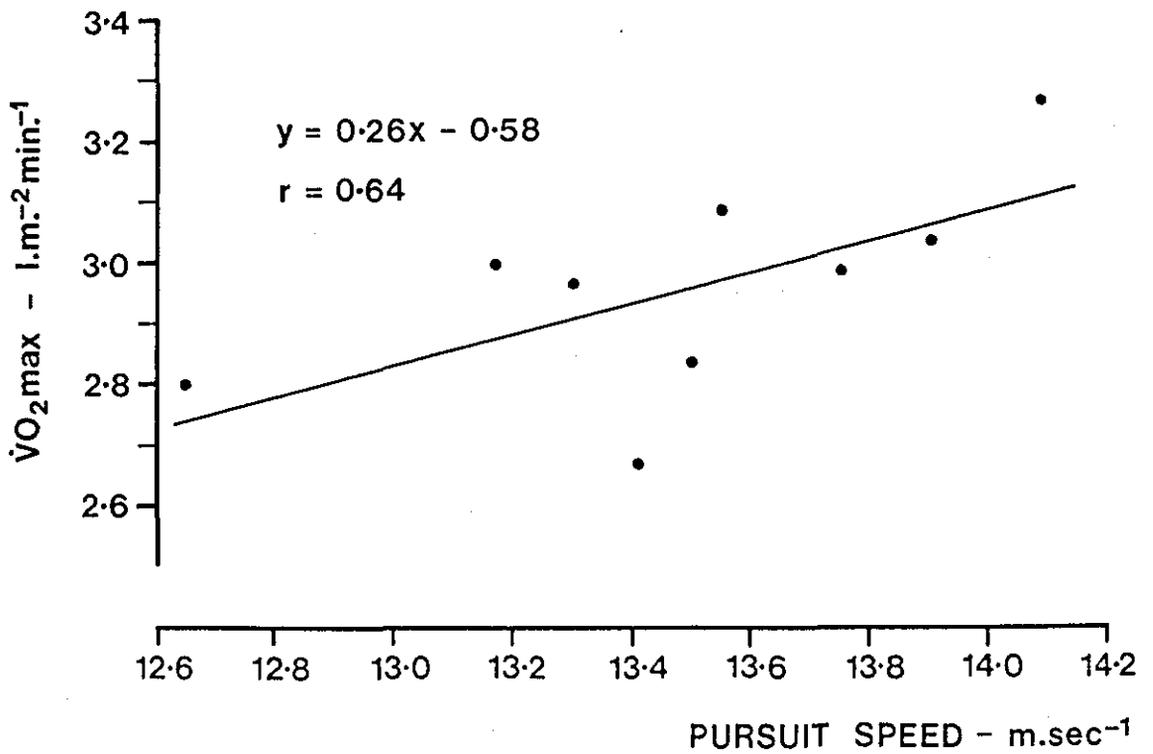
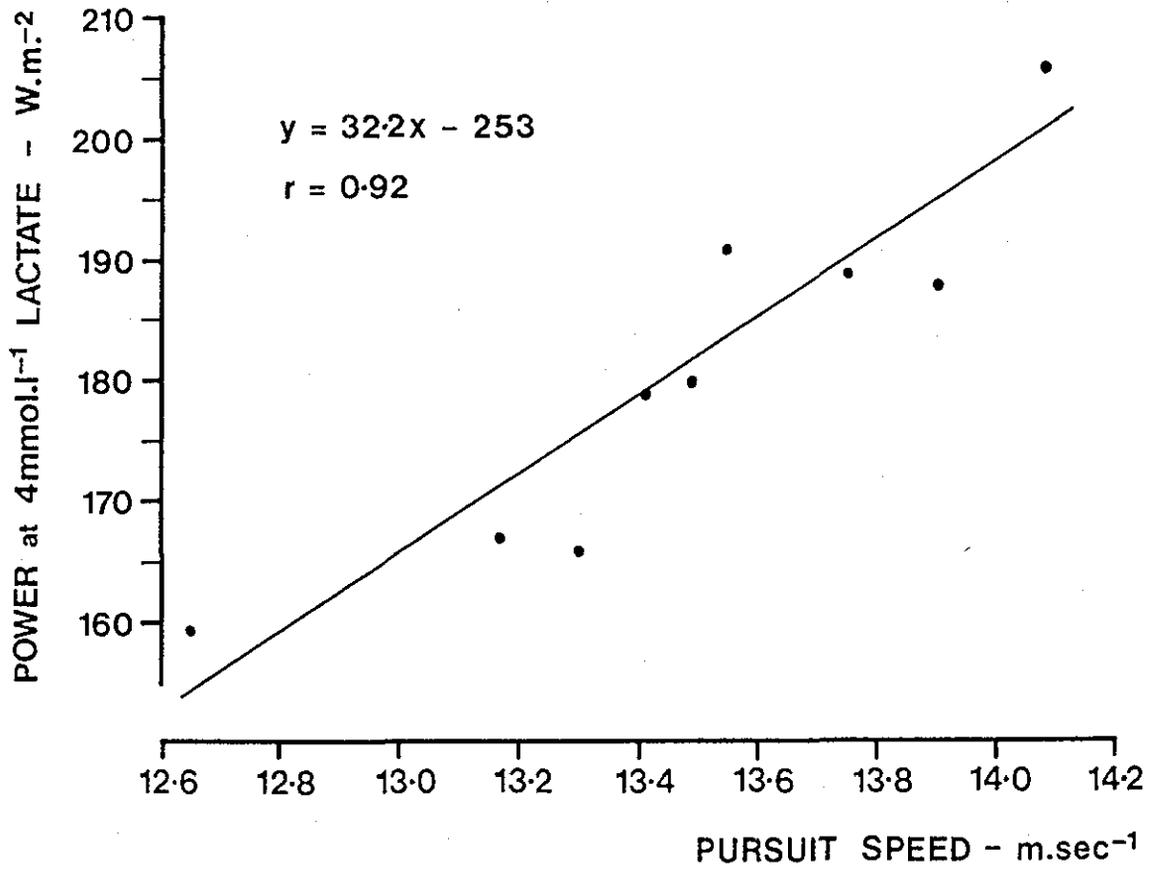
**FIGURE 6.** Relationships between pursuit speed and body mass related measures of Power 4 mmol.l<sup>-1</sup> and  $\dot{V}O_{2max}$  and pursuit speed.



**FIGURE 7.** Relationships between pursuit speed and body mass<sup>-0.667</sup> related measures of Power 4 mmol.l<sup>-1</sup> and  $\dot{V}O_{2max}$ .



**FIGURE 8.** Relationships between pursuit speed and body surface area related measures of Power  $4 \text{ mmol.l}^{-1}$  and  $\dot{V}O_{2\text{max}}$ .



## DISCUSSION

Unlike the pilot study that preceded this thesis (Keen et al., 1985) significant correlations were found between a number of the laboratory measures and pursuit performance. Although it is possible that these results occurred by chance due to the small number of subjects, this potential error is offset by the homogenous nature of the group. It can be seen from table 8 that all the subjects possessed a  $\dot{V}O_{2max}$  in excess of  $5.3 \text{ l}\cdot\text{min}^{-1}$  and the difference in race speed between the fastest and slowest rider was only  $1.43 \text{ m}\cdot\text{s}^{-1}$ . A characteristic of many studies that report high correlations between laboratory measures and endurance performance is the use of subjects with widely differing exercise capacities (e.g. Sjodin and Jacobs, 1981). Under such circumstances it is not surprising that clear relationships emerge. The fact that the tests used in this investigation appear capable of predicting performance within such a homogenous athletic sample suggests that they must reflect to a reasonable degree the physiological mechanisms underlying successful pursuit performance.

The central role of aerobic metabolism in determining pursuit performance suggested by the review of literature was confirmed by the strong correlations with both the maximal and sub-maximal measures of aerobic capacity. However, although a significant correlation was found between pursuit speed and absolute  $\dot{V}O_{2max}$  (0.69) the highest  $r$  value (0.93) was found for the sub-maximal Power 4  $\text{mmol}\cdot\text{l}^{-1}$  index (see figure 5). A similar finding was made by Jakeman (1986), who found a higher correlation between running velocity at 4  $\text{mmol}\cdot\text{l}^{-1}$  and 1500 M run time than  $\dot{V}O_{2max}$  in 18 male middle distance runners ( $R = -0.66$  and  $-0.39$  respectively). This is somewhat surprising as it has already been established that the pursuit race demands a rate of energy expenditure in excess of the  $\dot{V}O_{2max}$  of elite cyclists. Intuitively one might therefore expect a parameter that is dependent on a subject exercising to exhaustion to predict performance requiring a similar effort with greater accuracy than one based on steady state sub-maximal responses.

An answer to this paradox may lie in the role of acidosis in limiting pursuit performance. It was concluded from the review of literature that the 2 major requirements for successful pursuit performance were the capacity to generate a high volume of mechanical energy via the aerobic combustion of glycogen and the ability to sustain a near maximal metabolic rate without a rapid decay in cellular homeostasis. If, as appears likely, the anaerobic threshold reflects a work rate at which the exercising musculature is no longer capable of generating ATP without a disturbance to the intracellular environment, it could be argued that this measure provides a more accurate picture of the performance potential of a middle distance athlete. This is because a high AT power output not only requires a high oxygen uptake but the ability to maintain intracellular redox balance and hence regulate lactate/proton production as well. Thus, although two individuals may possess equal  $\dot{V}O_{2max}$  values it is conceivable that differences in the relative exercise intensity at the anaerobic threshold could result in widely differing rates of net proton production during exercise at an intensity approximating  $\dot{V}O_{2max}$ .

The lack of a satisfactory laboratory test of anaerobic capacity ruled out any direct attempt at evaluating the importance of this energy system in pursuing. Peak blood lactate levels, although only indicative of the degree of anaerobic stress, do however offer a means of estimating the degree to which this metabolic pathway was taxed (Jacobs, 1986). The results of the blood lactate levels recorded after the pursuit are therefore of considerable interest. The poor correlation between this measure and pursuit performance ( $r = 0.13$ ) would appear to prompt the conclusion that tolerance of a high blood lactate, and by inference a severe metabolic acidosis, does not determine pursuit performance. This interpretation may, however, be too simplistic.

The belief that intracellular acidosis is a primary cause of fatigue in short term, maximal exercise has lead to the concept of a critical intracellular pH level at which either a variety of feedback mechanisms inhibit metabolic activity to such an extent that further pH reductions are not possible, or the contractile process itself is

directly impaired (Karlsson, 1979). The frequent reporting of muscle pH levels of approximately 6.4 at exhaustion lend support to this view (Hermansen and Osnes, 1972; Hultman and Sahlin, 1980; Sahlin, 1978; Sharp et al., 1986). A common deduction arising from this line of thought is that acidosis can only be limiting exercise performance if maximal acid-base changes are observed. Whether such maximal changes occur in pursuing cannot be directly determined due to the limitations of the procedures currently available for measuring muscle metabolites. If, however, it could be established that the riders had achieved maximal blood lactate levels this may indicate that a maximal acidosis had occurred.

The low standard deviation in post race blood lactate of  $0.9 \text{ mmol.l}^{-1}$  demonstrates little variability within the group and suggests that a similar degree of acidosis was experienced by all the pursuiter. The mean value of  $14.1 \text{ mmol.l}^{-1}$  compares favorably with the  $15.2 (\pm 1.8) \text{ mmol.l}^{-1}$  measured 5 minutes post race on competitors in the USA pursuit championships (Burke, 1981), the small difference possibly being the result of a longer sampling delay in the latter study. It is interesting to note that Burke also failed to find a significant correlation between pursuit performance and blood lactate concentration, ( $r = 0.44$ ;  $p > 0.05$ ;  $n = 12$ ).

Further evidence that riders attained near maximal blood lactate levels was gathered by chance when the 1st and 2nd placed senior pursuit riders competed in the 1Km time trial championship. This is an event lasting around 70 seconds, a duration that has been shown to yield the greatest changes in blood and muscle pH and lactate (Hermansen and Osnes, 1972; Kindermann and Keul, 1977; Osnes and Hermansen, 1972). The post 1 Km race blood lactate values for these riders were  $14.5$  and  $15.0 \text{ mmol.l}^{-1}$ , which compare closely to the  $13.5$  and  $15.3 \text{ mmol.l}^{-1}$  they achieved in the pursuit. The riders finished 2nd and 5th respectively.

The fact that considerably higher blood lactate levels than these have been reported in the literature would appear to cast doubt on the claim that they represent a performance limiting acidosis in the pursuit riders. Osnes and Hermansen (1972) have reported blood

lactate concentrations above  $30 \text{ mmol.l}^{-1}$  following exhaustive running for 40-60 seconds, but serious doubts about the validity of the assay technique employed in this study have been raised by Kindermann and Keul (1977). The latter authors have recorded post competition blood lactate levels from athletes competing in a variety of international events. The highest value,  $24.9 \text{ mmol.l}^{-1}$ , was found for a 400m runner and they concluded that this level approximates "the maximal extent of lactate acidosis in man".

A similar value of  $24.2 \text{ mmol.l}^{-1}$  was recorded in the process of this research for an elite sprint cyclist following the 1 Km time trial championship. The individual concerned slowed dramatically in the last lap of the 3 lap race to finish in 8th place and behind the 2 pursuit riders. This perhaps indicates the importance of aerobic power even in an event as short as 70 seconds.

There are a number of possible explanations for the lower blood lactate levels of the pursuit cyclists compared with the above data. A lower proportion of total muscle mass is working maximally in cycling than in running so total lactate production may be smaller (although this cannot explain the value recorded in the sprint cyclist). Furthermore, the less active muscles of the upper body may actually remove lactate from circulation during the ride and will certainly increase the lactate distribution volume. It is also possible that a marked intracellular acidosis occurred through only moderate rates of lactate production due to a relative lack of muscle buffering capacity. It has been shown that elite endurance athletes have a markedly lower buffering capacity than elite sprint athletes (McKenzie et al., 1983), probably as a result of variations in fibre type (Burton, 1978). The physiological profile of the pursuiter is very much that of an endurance athlete and they may therefore suffer a greater reduction in pH for a given rate of proton production.

A third mechanism that may affect the relationship between muscle pH changes and lactate accumulation is a failure to adequately remove the  $\text{CO}_2$  produced by the combustion of glycogen. It was pointed out in the review of literature that respiratory acidosis is

not thought to occur in healthy untrained humans during maximal exercise, a fact demonstrated by the marked drop in arterial CO<sub>2</sub> concentration at high work rates that results from a respiratory compensation for metabolic acidosis (Wasserman et al., 1986). Recently however, Dempsey (1986) has questioned the assumption that the lung is fully capable of meeting all the demands for gas exchange during severe exercise in individuals with exceptionally high  $\dot{V}O_{2\max}$  values. His argument stems from the fact that arterial hypoxaemia has been observed during intense exercise in athletes with exceptionally high  $\dot{V}O_{2\max}$  values. A linear relationship between the degree of arterial haemoglobin de-saturation during sustained heavy exercise and  $\dot{V}O_{2\max}$  (l.min<sup>-1</sup>) has been reported (Williams et al., 1986).

Dempsey proposes two mechanisms to be responsible for this phenomenon - a diffusion limitation arising from a failure to preserve alveolar PO<sub>2</sub> through sufficient hyperventilation and a reduction in the transit time of erythrocytes through the pulmonary capillary bed. The latter is suspected to occur when increases in cardiac output can no longer be accommodated by an expansion in pulmonary capillary blood volume, the result being a rise in flow rate through the now fully expanded capillary bed.

The relationship between hyperventilation, alveolar PO<sub>2</sub> and arterial PO<sub>2</sub> during intense exercise in elite runners was investigated by Dempsey et al., (1984). They found that the greatest reductions in arterial PO<sub>2</sub> were associated with little or no hyperventilation, reduced alveolar PO<sub>2</sub> and increased arterial PCO<sub>2</sub>. Conversely, the runners who demonstrated a marked hyperventilation suffered only minor reductions in arterial PO<sub>2</sub> and successfully reduced arterial PCO<sub>2</sub> levels. A strong negative correlation was found between arterial PO<sub>2</sub> and PCO<sub>2</sub> levels. When a low density normoxic-helium inspirate gas was administered to the athletes who normally failed to hyperventilate, a marked hyperventilation was observed coupled with improvements in arterial gas concentrations, although it is not clear from the data whether the increase in ventilation was achieved through a greater tidal volume or higher respiratory frequency. Dempsey argues this implicates mechanical constraint as

the cause of relative hypoventilation in individuals with exceptional aerobic power, i.e. an excessive load on the ventilatory apparatus is avoided at the expense of arterial O<sub>2</sub> saturation and optimal compensation for metabolic acidosis. Furthermore, he calculated that the ventilatory rate required to maintain adequate alveolar PO<sub>2</sub> and arterial PCO<sub>2</sub> levels in an athlete with a  $\dot{V}CO_2$  of 5-6 l.min<sup>-1</sup> is in excess of 200 l.min<sup>-1</sup> (Dempsey, 1986). Such a flow rate may well approach the structural limitations of the lung.

If Dempsey's hypothesis is correct then significant arterial hypoxaemia and poor respiratory compensation for metabolic acidosis is to be expected in elite pursuit cyclists as a high  $\dot{V}O_{2max}$  appears to be a pre-requisite for high level performance.

An alternative explanation for arterial hypoxaemia in elite athletes is that alveolar gas mixing efficiency may fall as a result of the very high respiratory frequency, and hence shorter diffusion time, that characterizes maximal ventilation in elite athletes (Hale, 1987). This might explain the relationship between de-saturation and  $\dot{V}O_{2max}$  found by Williams et al. as individuals with a higher aerobic power would be expected to demonstrate a greater minute volume. However, Dempsey's observation that hyperventilation actually decreased arterial hypoxaemia appears to be in direct conflict with the notion of a reduced gas mixing efficiency.

A second phenomenon that may impair pulmonary gas exchange during a pursuit is a transient interstitial lung oedema. Rasmussen et al., (1988) reported a significant reduction in lung diffusing capacity in 6 elite oarsmen following a 4 minute, exhausting all-out effort on a rowing ergometer. They concluded that this was a result of a sub-clinical oedema, possibly resulting from the very high lung capillary blood flow that such athletes will, out of necessity, attain. Evidence of ventilatory muscle fatigue was also observed in this study as peak expiratory flow rate was significantly reduced following the test. This supports Dempsey's view that the mechanical work required to adequately ventilate the lungs may become excessive in large aerobic athletes. The post exercise symptoms of severe coughing with expectoration and

dyspnoea described by the above authors are commonly observed in pursuit cyclists following competition and often persist for many hours. Rasmussen et al. also found that lung diffusing capacity was still reduced 2 days after the single exercise test, a fact that may have implications for performance over the duration of a pursuit competition.

Dodd et al., (1989) have recently investigated the role of respiratory muscle fatigue in constant load exercise tests to exhaustion on a cycle ergometer lasting around 7 minutes. The experimental condition was preceded by 10 minutes of volitional isocapnic hyperventilation at approximately 85% of maximal minute ventilation ( $\dot{V}_E$ ). No changes in lung function, blood gases, arterial saturation or time to fatigue compared to the control condition were found. Although these data indicate that respiratory muscle fatigue failed to occur this is to be expected in non-athletic subjects;  $\dot{V}O_{2max}$  averaged  $50.7 \text{ ml.kg.}^{-1}\text{min}^{-1}$  and maximal  $\dot{V}_E$   $121 \text{ l.min}^{-1}$ . Such levels of ventilation are not thought to threaten the functional capacity of the lung (Dempsey, 1986).

If, as suggested by the above evidence, the demand for pulmonary gas exchange in a pursuit race is such that arterial  $\text{PCO}_2$  is prevented from falling in the expected manner, the relationship between lactate accumulation and cellular pH may be altered. A relative retention of  $\text{CO}_2$ , in comparison to humans with less aerobic power, would affect both the rate of lactate and proton efflux from the muscle by reducing blood pH, and the intracellular  $\text{PCO}_2$  concentration. The latter would result in a drop in cell pH in addition to that caused by metabolic proton production. Both effects would lead to a relatively low level of post race blood lactate even though a marked intracellular acidosis may be present. A degree of caution may therefore be necessary in interpreting peak lactate levels in large elite endurance athletes.

A final observation on the degree of acidosis suffered during the pursuit race is that a low pH may have detrimental effects on the riders style and control of the bicycle. Kindermann and Keul (1977) claim that acidosis results in a deterioration of neuromuscular

function and co-ordination and suggest that the sub-maximal acid-base changes observed in events demanding both skill and high energy production are the result of a compromise between the two factors. Pursuit racing demands a high level of co-ordination due to pedal frequencies used, and considerable bike handling precision - it is advantageous to ride as close to the inside of the track as possible without hitting the regulatory distance markers. Furthermore, the competition cycles currently favoured sacrifice ease of handling for lightness and aerodynamic efficiency. Pursuit riders who experience marked fatigue during a race often weave on the track and any movement up the steep banking of a velodrome is extremely wasteful of energy. It may therefore be impossible to tolerate extreme acid-base changes in a pursuit race without incurring costly decreases in cycling technique.

Another important finding in this experiment was the effect of various units of expression on the correlations between physiological parameters and performance. It can be seen from figures 5-8 that for both the  $\dot{V}O_{2max}$  and Power 4mmol.l<sup>-1</sup> measures the best performance relationship was found using the absolute values for the parameters. Likewise, the weakest index of both parameters was the body mass related expression. The implication of this finding is best illustrated by the following hypothetical example.

Two cyclists, A and B, have identical relative  $\dot{V}O_{2max}$  values of 80 ml.kg.<sup>-1</sup>min<sup>-1</sup>. A has a mass of 80 kg and therefore an absolute  $\dot{V}O_{2max}$  of 6.4 l.min<sup>-1</sup> whereas B weighs only 70 kg and thus possesses a  $\dot{V}O_{2max}$  of 5.6 l.min<sup>-1</sup>. Both have a 4 mmol.l<sup>-1</sup> lactate power output equating to 85% of their  $\dot{V}O_{2max}$  and an identical  $\dot{V}O_2$  - Power relationship. Rider A will therefore have a  $\dot{V}O_2$  0.68 l.min<sup>-1</sup> higher than B at Power 4 mmol.l<sup>-1</sup> (5.44 vs 4.76 l.min<sup>-1</sup>). Based on the typical  $\dot{V}O_2$  - Power relationship observed in this research their corresponding Power 4 mmol.l<sup>-1</sup> values would be 390 and 338 W. When substituted into the regression equation for pursuit speed vs Power 4 mmol.l<sup>-1</sup> ( $y = 68.7x - 577$ ) the following 4000 M pursuit times are predicted: A 284s, B 300s. Clearly then, in

a group of highly trained cyclists the larger individuals appear to be at a distinct advantage in pursuing due to their greater absolute power output.

The most probable reason for this is that the increase in air resistance suffered by a large rider is more than compensated for by the additional absolute power output resulting from a greater muscle mass and cardiovascular system. A similar conclusion was reached by Swain et al. (1987) who found that the oxygen cost of cycling per unit body mass or per unit frontal area was significantly lower in a group of heavy (>80 kg) racing cyclists compared to lightweight riders (<60 kg). The fact that the parameters expressed per M<sup>3</sup> BSA yielded correlations of the same order as the absolute values would appear to support this notion.

The primary role of absolute power in pursuing was given further support by the result of the womens championship. The gold and silver medalists had Power 4 mmol.l<sup>-1</sup> figures of 242 and 224 W respectively, but when expressed related to bodyweight the figures become 4.1 and 4.5 W. kg<sup>-1</sup>. The superior power to weight ratio of the second placed rider was clearly no advantage in this discipline.

A similar conclusion regarding rowing was made by Secher (1983) who also found a significant correlation between performance and absolute  $\dot{V}O_{2max}$ . Furthermore, in a comparison of international oarsmen he found that the unsuccessful individuals were only inferior to the more successful counterparts in terms of weight (84 to 93 kg), absolute  $\dot{V}O_{2max}$  (5.58 to 5.89 l.min<sup>-1</sup>) and maximal ventilation (173 to 200 l.min<sup>-1</sup>). The last is interesting in the context of the earlier discussion regarding the role of lung function in large elite athletes performing maximal aerobic work. Secher also points out that a larger body mass in a highly trained rower is advantageous in terms of a greater total volume of stored phosphagens, myoglobin, glycogen and lactate distribution space.

The mean  $\dot{V}O_{2max}$  of 5.78 l.min<sup>-1</sup> recorded in this study is higher than the value reported for other groups of elite cyclists in the literature reviewed (Hahn et al., 1986; Hermansen, 1973; Sjogaard,

1984; Stromme et al., 1977). One explanation for this is that no data specifically relating to elite pursuit cyclists could be located. Virtually all the studies on aerobic power in cyclists have examined road racers who are typically lighter than their track racing counterparts (Sjogaard et al., 1982). It is therefore possible that the high absolute value for this group simply reflects the mass of the riders, a point emphasized by the less exceptional mean body mass related  $\dot{V}O_{2\max}$  of  $76.7 \text{ ml.kg.}^{-1}\text{min}^{-1}$ .

Another possible explanation for the apparently high absolute values found in this study is the relative lack of information on elite cyclists from the leading nations in international cycle sport. A common feature of the sports science literature is that most data on "elite" performers is collected from leading competitors in the countries most active in the publication of sports related research. Amateur cycling is currently dominated by Eastern Bloc countries, from where performance data on elite sports people rarely emerges. In this context, it is worth noting that one of the subjects in this study was twice world professional pursuit champion (1980 and 1986) and holds the current world's best time over 5000 M. A second subject finished 6th in the 1987 world amateur pursuit championship and 4th in the 1988 Olympic pursuit. These individuals, at least, would appear to merit the title of "elite".

A final observation on the validity of "elite" data in the literature is the timing of data collection relative to the competitive season. Although desirable, it is difficult to assess elite performers when they are in peak condition as they often travel between competitions and are sometimes unwilling to undergo testing so close to major events. This study, however, benefited from the close co-operation and interest of the cyclists who participated in it, to the extent that they were prepared to undergo testing at the peak of the competitive season. The data presented here is probably unusual in this respect.

## Chapter Seven

### EXPERIMENT III: A LONGITUDINAL ANALYSIS OF MAXIMAL AND SUB-MAXIMAL RESPONSES TO INCREMENTAL EXERCISE IN PURSUIT CYCLISTS.

#### INTRODUCTION

A second aim of this thesis was to evaluate, in a group of pursuit cyclists, the effects of long term training patterns on the two indices of endurance capacity focussed on in the previous chapters. However, during the 4 years in which data was collected for this research the significance of individual adaptations and responses to training became increasingly clear.

The use of group data is a justifiable, and sometimes essential feature of sports science research that explores poorly understood or hypothetical relationships, but when the primary objective of a study is to provide a greater understanding of elite level performance valuable information is too often concealed by statistical analysis. The quantitative study of elite performers is also complicated by the practical difficulties of regularly monitoring a group of athletes spread over a wide geographical area. Furthermore, the frequency of illness and injury, and the demands of international competition on elite athletes are simply too great to permit the degree of control required to ensure valid or comparable data.

For these reasons it was decided that the most appropriate method of presenting some of the longitudinal data collected over the duration of this thesis was through a series of individual case studies of pursuit cyclists.

#### METHODS

Longitudinal maximal and sub-maximal exercise data were examined from 3 male pursuit cyclists, selected from more than 30 riders undergoing regular exercise testing during this research programme.

These individuals were chosen because their competitive levels differed greatly and their data therefore reflects a wide perspective of the performance spectrum. All three had undergone a large number of tests over an extended period. The exercise protocols used to measure Power 4  $\text{mmol.l}^{-1}$  lactate and  $\dot{V}O_{2\text{max}}$  were identical to those described in detail in chapter 5.

Heart rate at a fixed sub-maximal power output was also examined on the ground that this variable should be sensitive to specific changes in cardiac/circulatory performance. As cardiac output is not thought to vary at a given sub-maximal exercise intensity (Rowell, 1969) a reduction in heart rate can be taken to indicate an increase  $V_s$  (Astrand and Rodahl, 1986). A reference power output was selected that ensured every datapoint lay on the linear portion of the power-heart rate relationship recorded in the sub-maximal exercise test. The Differences in reference power thus reflect the varying abilities of the cyclists studied.

## CASE I

Subject 1 was a highly accomplished male professional cyclist who had won 2 world professional pursuit championships, finished 2nd on three other occasions and is the current holder of the worlds best time over 5000m (5 min. 40.34 sec). His was 28 years old at the start of the project, and 1.85 m in height. Body mass averaged 83.7 kg and ranged from 81.5 to 86.2 kg.

This subject attended the laboratory on 21 separate occasions between October 1985 and July 1988 and underwent the determination of Power 4 mmol.l<sup>-1</sup>.  $\dot{V}O_{2max}$  was also measured but only on 11 of these occasions. The latter parameter was assessed less frequently due to the short time intervals between visits to the laboratory and the physically demanding nature of the test.

Although the longitudinal physiological changes in subject 1 are the main topic of this case study the mean magnitude of the major parameters measured for this individual during the course of the study are particularly interesting and worthy of some discussion.

$\dot{V}O_{2max}$  averaged 6.91 l.min<sup>-1</sup>, a value considerably higher than those found in the literature concerning elite cyclists. The previously highest individual value reported was 6.5 l.min<sup>-1</sup> recorded on another world professional pursuit champion (Sjogaard et al., 1982) The mean body mass related value of 82.5 ml.kg.<sup>-1</sup>min<sup>-1</sup> for subject 1 is slightly lower than the 85.4 ml.kg.<sup>-1</sup>min<sup>-1</sup> reported for a world professional road race champion by Veicsteinas et al., (1984) but on 3 occasions a value above 85 ml.kg.<sup>-1</sup>min<sup>-1</sup> was measured. Towards the end of the monitoring period changes in body mass related  $\dot{V}O_{2max}$  appeared to be solely due to variations in body mass.

The highest  $\dot{V}O_{2max}$  values, both absolute and body mass related, are widely believed to occur in elite cross-country skiers (Astrand and Rodahl, 1986), a fact usually attributed to the extensive range of muscle groups recruited in this form of exercise (Hermansen, 1973). Bergh (1987) has presented  $\dot{V}O_{2max}$  data on 5 elite male

cross-country skiers, all of whom were recent Olympic and/or World Champions. The mean absolute value ( $\pm$  S.D.) was 6.54 (0.48)  $\text{l}\cdot\text{min}^{-1}$ , and a body mass related 83.8 (6.4)  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , both of which are similar to those found for subject 1. In view of these data it seems reasonable to conclude that this cyclist possessed an aerobic capacity approaching the highest levels recorded in human subjects.

Data concerning Power 4  $\text{mmol}\cdot\text{l}^{-1}$  lactate in elite cyclists or other athletes rarely appears in the literature. Buchanan and Weltman (1985) found a mean of 291 W in category 1 American road cyclists, a value similar to the 309 W recorded for 7 professional road cyclists by Hardman and Williams (1985). However, both figures do not appear exceptional in comparison to data already reported in this thesis for non-elite cyclists. Roth et al. (1981) has measured the 4  $\text{mmol}\cdot\text{l}^{-1}$  parameter in East German cyclists and reported a mean of 390 W for a group of 7 individuals of unclassified performance status, and 454 W for an individual described as elite. It is interesting to note, in the context of the relationship between Power 4  $\text{mmol}\cdot\text{l}^{-1}$  and body mass, that the mean mass of the East German cyclists was 84.5 kg, a value close to the upper limit of body mass observed in cycle sport. (Of the 403 male cyclists competing at the 1988 Olympic games only 20 had a mass in excess of 84Kg and the mean mass was  $71.7 \pm 7.5$  kg).

The last Power 4  $\text{mmol}\cdot\text{l}^{-1}$  values discussed appear more reasonable as "elite" data, particularly when the following information is considered. The 100 Km 4 man team time trial is an international discipline dominated in recent years by East Germany, whose teams have occasionally completed the course in under 2 hours, i.e. a race speed in excess of  $13.9 \text{ m}\cdot\text{s}^{-1}$ . In the review of literature the power output required at this speed was conservatively estimated to be of the order of 520 W. Such a high power output can only be maintained by a team of cyclists continuously sharing the pace (drafting). Using wind tunnel tests Kyle (1986) calculated that the power output for riders sheltering behind a leader of similar build is reduced by around 30%, which at this speed equates to 360 W. Each rider would therefore be required to produce an average

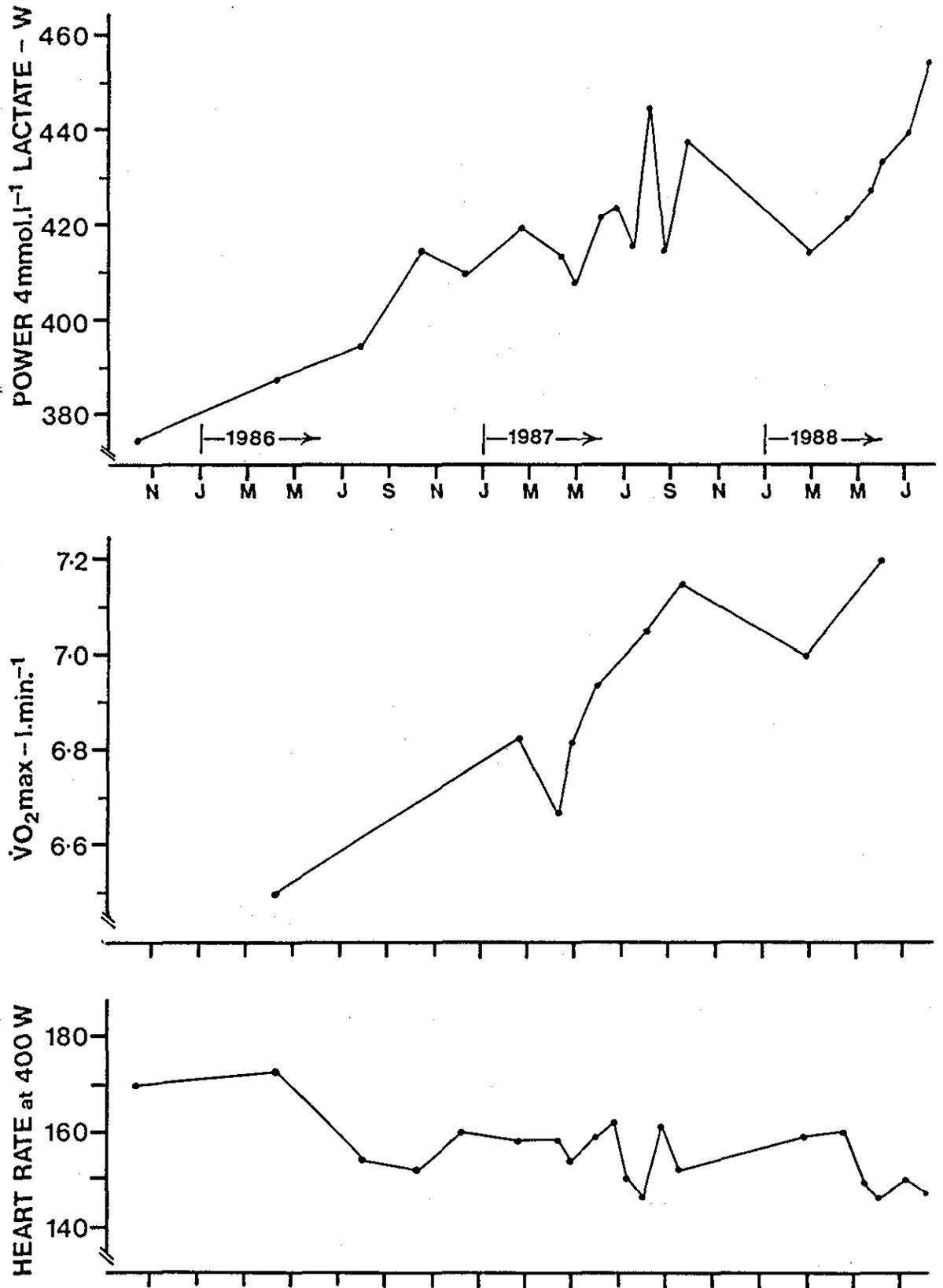
power approximating 403 W for 2 hours, assuming the pace setting was equally divided. As experiment I revealed, exercise of this duration cannot be performed above the 4 mmol.l<sup>-1</sup> exercise intensity, so a Power 4 mmol.l<sup>-1</sup> approaching 400 W is to be expected in elite cyclists.

Power 4 mmol.l<sup>-1</sup> in Subject 1 averaged 419 W and the highest value measured was 455 W. Thus, although comparative data for this parameter is scarce it seems reasonable to describe these data as representative of the upper limit of functional capacity in cycling athletes. A final indication of the extraordinary level of conditioning present in subject 1 is the fractional utilization of  $\dot{V}O_{2max}$  at the Power 4 mmol.l<sup>-1</sup> exercise level. Svedenhag and Sjodin (1984) have reported a mean of 87.6%  $\dot{V}O_{2max}$  at the running speed corresponding to 4 mmol.l<sup>-1</sup> lactate in elite marathon runners, and a similar figure of 89% was found by Kindermann et al. (1979), again in elite marathoners. This parameter (%  $\dot{V}O_{2max}$  at Power 4 mmol.l<sup>-1</sup>) averaged 89.5%  $\dot{V}O_{2max}$  in subject 1, indicating a near optimal utilization of an exceptionally high  $\dot{V}O_{2max}$ .

The longitudinal changes in both  $\dot{V}O_{2max}$  and Power 4 mmol.l<sup>-1</sup> lactate, together with heart rate at a power output of 400 W are shown graphically in figure 9, and raw data are listed in appendix 4. All parameters indicate a general progressive improvement in endurance capacity across the period of monitoring. Of particular interest is the 11% increase observed in  $\dot{V}O_{2max}$  from 6.50 to 7.2 l.min<sup>-1</sup>. This demonstrates that marked improvements in maximum oxygen uptake are still possible in a mature elite athlete who has been training and competing for 15 years. These results cannot be explained simply in terms of variations in seasonal training volume or intensity as subject 1 races in 6-day indoor track competitions throughout the winter months, the period in which cyclists are typically least active.

Astrand and Rodahl (1986) state that increases of  $\dot{V}O_{2max}$  in highly trained individuals do not result from an increased Ca-Cvo<sub>2</sub> and suggest that adjustment of cardiac output is the primary factor responsible for increases in aerobic capacity. As the maximal heart

**FIGURE 9.** Longitudinal variations in Power 4 mmol.l<sup>-1</sup>,  $\dot{V}O_{2max}$  and Heart Rate at 400 W for subject 1. The X-axis is graduated in alternate months commencing November 1985.



rate of subject 1 varied little throughout the 3 years (See appendix 4) any increase in cardiac output must have resulted from a greater stroke volume ( $V_s$ ). Evidence of a marked increase in  $V_s$  is provided by the changes observed in heart rate at 400 W, which varied between 173 and 146  $\text{b}\cdot\text{min}^{-1}$  during the study. A significant correlation between this variable and  $\dot{V}O_{2\text{max}}$  was found ( $r = 0.83$ ;  $p < 0.01$ ;  $n = 10$ ), which supports the view that  $V_s$  and  $\dot{V}O_{2\text{max}}$  are closely coupled.

Blomqvist and Saltin (1983) propose that increases in  $V_s$  could result from a greater left ventricular end-diastolic volume, increases in myocardial contractility, blood volume or central venous pressure (pre-load), or a reduced systemic resistance to blood flow (after-load). Although there is evidence that changes in cardiac dimensions mirror adjustments to  $\dot{V}O_{2\text{max}}$  (Peronnet et al., 1981), this adaptive relationship has only been documented in individuals undergoing relatively short training programmes. It seems unlikely, however, that the apparently large increase in stroke volume seen in subject 1 could be attributed to changes in cardiac dimensions alone. The individual concerned had been a professional athlete for 10 years so the likelihood of significant morphological adaptations occurring in the latter 3 years would seem remote. A more plausible explanation for the improvement in cardiac performance may lie in the poorly understood mechanisms responsible for controlling local blood flow and therefore systemic peripheral resistance, i.e. a regulatory rather than structural adaptation. This view is in agreement with Blomqvist and Saltin (1983) who claim that "a release of (localized muscular) vasoconstrictor activity is a crucial training-induced adaptation". It is interesting to note that during the 3 years of monitoring there was a progressive change in the training strategy of subject 1 from a programme based on very high volume - low intensity workouts to one in which duration was compromised in favour of intensity. Such changes may well have provided the stimulus for the changes in cardiac performance observed.

A 17.6% increase in Power 4  $\text{mmol}\cdot\text{l}^{-1}$  lactate occurred during the monitoring period, which was somewhat higher than the 11% rise in

$\dot{V}O_{2max}$ . Nevertheless, a strong correlation was found between both parameters ( $r = 0.87$ ;  $p < 0.01$ ;  $n = 10$ ) suggesting a large degree of inter-dependence. This seems perfectly reasonable as it was pointed out in the literature review that the oxygen uptake - power output relationship in cycling varies little between individuals, so a high oxygen uptake is a pre-requisite for a high threshold high power output. Further evidence of the close link between cardiovascular performance and the AT parameter is given by the strong relationship between Power 4  $\text{mmol.l}^{-1}$  and heart rate at 400 W ( $r = -0.77$ ;  $p < 0.01$ ;  $n = 20$ ).

The fractional utilization of  $\dot{V}O_{2max}$  at Power 4  $\text{mmol.l}^{-1}$  showed only minor variations throughout the monitoring period (see appendix 4). There was a tendency towards a lower fractional utilization near the end of the monitoring period but this can be explained by the small decrease in the oxygen cost of work which also occurred (see data on  $\dot{V}O_2$  at 300 W in appendix 4).

A notable reduction in Power 4  $\text{mmol.l}^{-1}$  occurred in August 1987. At the time of this test subject 1 was recovering from a mild upper respiratory tract infection he contracted during a period of intense training for the world championships. Although his power output recovered to just below the level recorded prior to the illness within 4 weeks, the temporary loss of form resulted in a poorer than expected performance in the world championships. The Power 4  $\text{mmol.l}^{-1}$  parameter may therefore be sensitive to the effects of viral infection, a problem that plagues many top sports-people as they approach peak fitness.

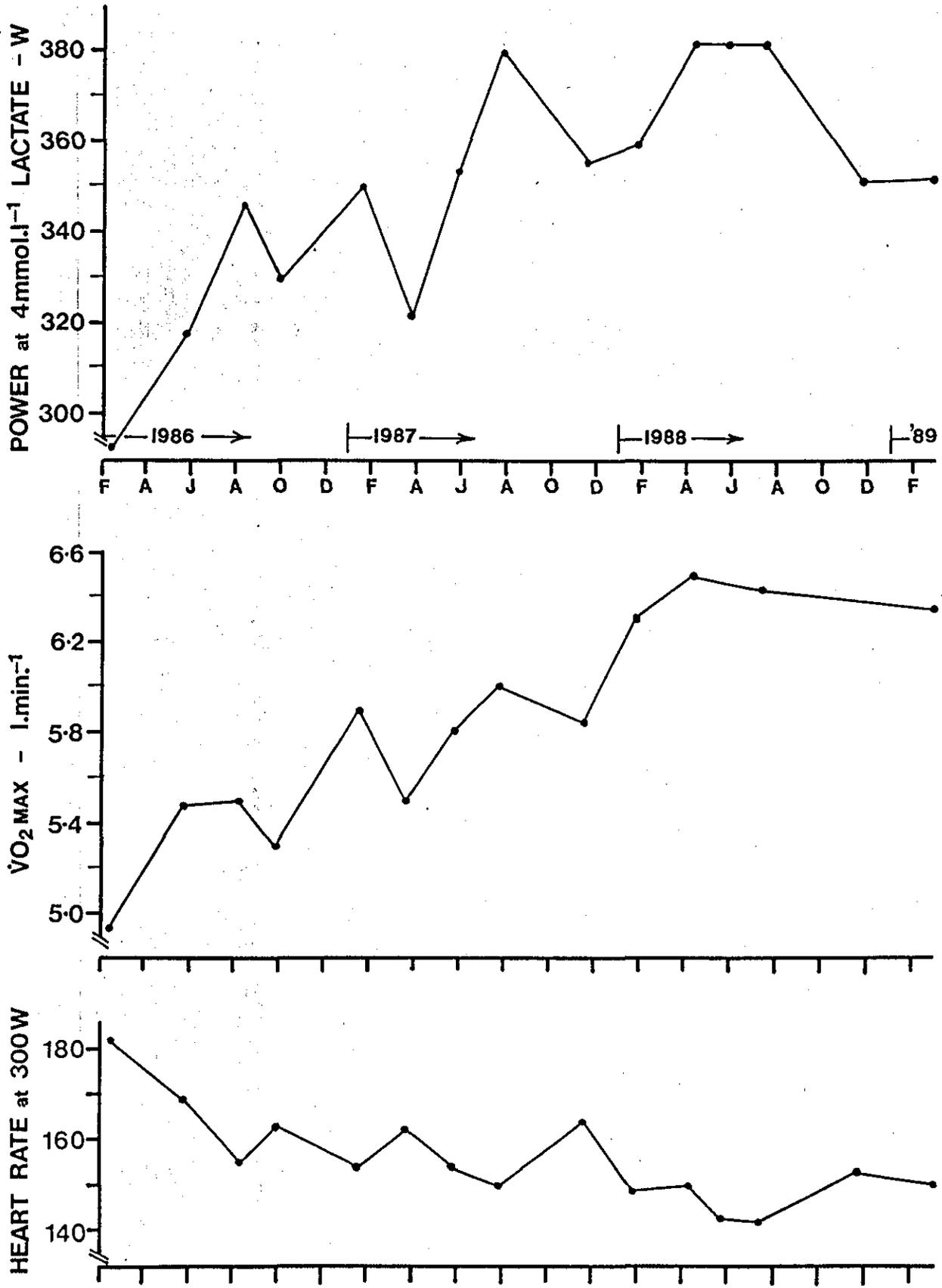
## CASE II

Subject 2 was an 18 year old national level junior pursuit cyclist when monitoring commenced in February 1986. He subsequently spent 2 seasons competing as an international amateur and one season as a professional. His height remained stable at 1.82 m and body mass varied between 79.2 and 83.5 kg, averaging 81.4 kg.  $\dot{V}O_{2max}$  and Power  $4mmol.l^{-1}$  were measured on 15 occasions in the 3 year period up to February 1989. Figure 10 illustrates the variations in both parameters, together with heart rate at 300W, the latter again taken as an indication of cardiac performance. Raw data are given in appendix 5.

$\dot{V}O_{2max}$  demonstrated a 24% increase from  $4.94 l.min^{-1}$  to a peak of  $6.50 l.min^{-1}$  approximately 2 years later. Such a rise in aerobic power appears extraordinary, bearing in mind subject 2 was already a well established athlete before monitoring commenced. This change cannot be explained in terms of a large increase in body mass as this was identical (82.5 kg) on the occasions when the highest and lowest  $\dot{V}O_{2max}$  values were measured. Changes in body composition, although not assessed, are also unlikely to be the cause of the large changes observed as a considerable increase in lean body mass would have to have taken place.

In a major review of the effects of endurance training on  $\dot{V}O_{2max}$  Pollock (1973) observed that the largest gains in aerobic power (up to +30%) occur in healthy individuals with the lowest levels of prior conditioning. Furthermore, he concluded that subjects already endowed with considerable cardio-respiratory fitness can be expected to demonstrate only minor increases in  $\dot{V}O_{2max}$  (<10%) following months of training. The current results appear to conflict with this view and highlight the danger of making assumptions about the likely improvement to an individuals  $\dot{V}O_{2max}$ , particularly when most of information on this topic is derived from group studies involving non-athletic or highly trained subjects (Pollock, 1973).

**FIGURE 10.** Longitudinal variations in Power  $4 \text{ mmol.l}^{-1}$ ,  $\dot{V}O_{2\text{max}}$  and Heart Rate at 300 W for subject 2. The X-axis is graduated in alternate months commencing February 1986.



No major change in maximal heart rate was apparent but heart rate at 300 W varied from 182 to 142 b.min<sup>-1</sup>, and a significant correlation was observed between this variable and  $\dot{V}O_{2max}$  ( $r = 0.91$ ;  $p < 0.01$ ;  $n = 13$ ). This strongly suggests an adaptive relationship between cardiac performance and  $\dot{V}O_{2max}$ .

Power 4 mmol.l<sup>-1</sup> also increased by 24% from 292 W to a peak level of 382 W, and variations in this parameter were closely coupled with  $\dot{V}O_{2max}$  ( $r = 0.89$ ;  $p < 0.01$ ;  $n = 13$ ) and heart rate at 300 W ( $r = -0.91$ ;  $p < 0.01$ ;  $n = 15$ ).

It can be seen from figure 10 that seasonal peaks in all parameters coincided with the July-August period when major national and international pursuit championships take place. An 8% reduction in both  $\dot{V}O_{2max}$  and Power 4 mmol.l<sup>-1</sup> occurred between January and March 1987, a pre-season phase in which improvements in aerobic power would be expected. It later emerged that subject 2 was performing low repetition-high intensity strength training in conjunction with his endurance training programme during this period. This suggests that intense strength training may exert a negative influence on endurance. A potential mechanism for such an effect was found by MacDougall et al. (1979) who reported a 26% reduction in mitochondrial volume density and a 25% drop in mitochondrial to myofibrillar volume ratio in humans following heavy resistance training. The corresponding hypertrophy of both ST and FT fibres led this group to conclude that a simple dilution of the pre-training level of mitochondria had taken place and that strength training may consequently reduce endurance.

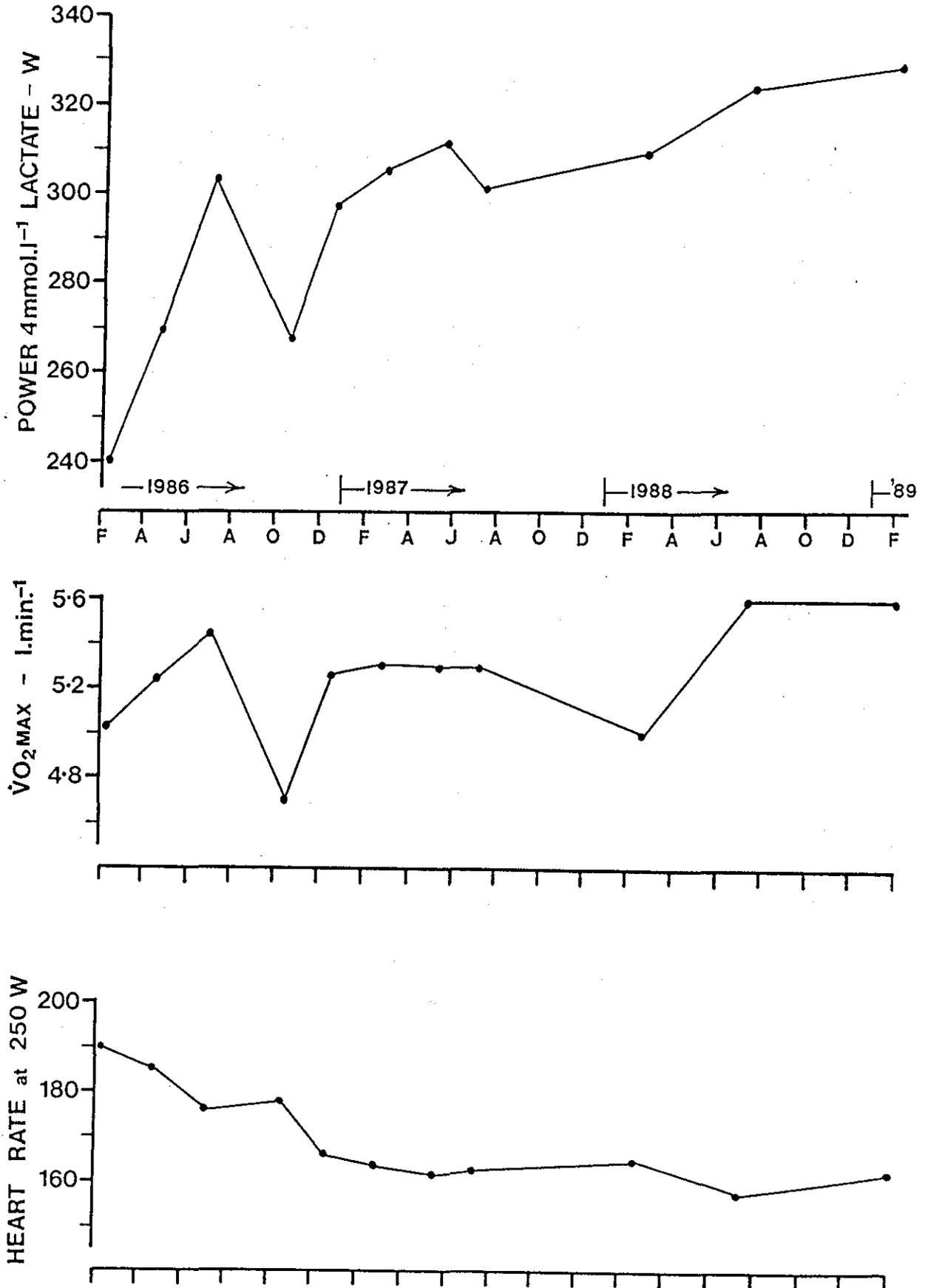
### CASE III

Subject 3 was a regular competitor in national level competitions and during the monitoring period (February 1986-89) he reached the last 16 in the national senior pursuit championships on two occasions. His age and height were 18 years and 1.76 m at the start of the project, the latter remaining unchanged during the three year period. Body mass averaged 73.2 kg and ranged from 71.1 to 75.1 kg.

A total of 11 separate tests were performed by subject 3, and the pertinent data from these are shown graphically in figure 11. Sub-maximal heart rate was measured at a reference power of 250 W.  $\dot{V}O_{2max}$  remained fairly stable in the region of 5.3 l.min<sup>-1</sup> except on one occasion in October 1986 when a 14.8% reduction from the previous value was recorded. This anomaly could not have been the result of the subject failing to exert himself fully during the test as his final heart rate of 206 b.min<sup>-1</sup> was within 2 beats of the highest value recorded (see appendix 6). A parallel reduction of 11.8% in Power 4 mmol.l<sup>-1</sup> confirmed that a considerable reduction in fitness had occurred. It transpired that subject 3 had been suffering from a viral infection for a considerable period prior to this particular test. Ignoring this datapoint, a maximal improvement in  $\dot{V}O_{2max}$  of 10.7% was observed.

Unlike the previous two case studies, only a poor relationship was found between  $\dot{V}O_{2max}$  and reference heart rate @ 250 W for subject 3 ( $r = -0.49$ ;  $p > 0.05$ ;  $n = 11$ ). The reason for this is unclear, but the relatively small variations in  $\dot{V}O_{2max}$  perhaps demonstrate a lack of sensitivity in the sub-maximal heart rate parameter. Maximal heart rate remained unmodified although small variations were noted. A somewhat larger increase in Power 4 mmol.l<sup>-1</sup> of 27.3% was recorded, which was achieved in spite of the much smaller rise in  $\dot{V}O_{2max}$ . As a consequence, the fractional utilization of  $\dot{V}O_{2max}$  at Power 4 mmol.l<sup>-1</sup> increased from 73 to 86%. Changes in Power 4 mmol.l<sup>-1</sup> correlated strongly with heart rate at 250 W ( $r = 0.91$ ;  $p < 0.01$ ;  $n = 11$ ).

FIGURE 11. Longitudinal variations in Power  $4 \text{ mmol.l}^{-1}$ ,  $\dot{V}O_{2\text{max}}$  and Heart Rate at 250 W for subject 3. The X-axis is graduated in alternate months commencing February 1986.



## GENERAL OBSERVATIONS ON LONG TERM TRAINING RESPONSES.

Probably the simplest, and hardly the most original conclusion that arises from these case studies is that training responses in individual athletes cannot be predicted with any degree of certainty. Subject 1, demonstrated progressive improvements in both  $\dot{V}O_{2max}$  and Power 4  $mmol.l^{-1}$  despite having been an elite competitor for many years. Subject 2, although regarded as well trained before monitoring commenced, increased his endurance capacity far beyond the level expected on the basis of previous research.

The above data demonstrate the lack of information on longitudinal adaptations in highly trained athletes. The literature concerning training responses may provide perfectly valid information where normal populations are under investigation, but the application of this information to elite athletic groups seems at best questionable and at worst entirely inappropriate.

The specificity of individual training responses was highlighted by the data from Subject 3. Unlike Subjects 1 and 2, this cyclist improved his Power 4  $mmol.l^{-1}$  to a much greater degree than  $\dot{V}O_{2max}$ . If the latter parameter had been the only measure of aerobic power employed in this study the marked improvement in Subject 1's potential for pursuit performance as indicated by Power 4  $mmol.l^{-1}$  would have remained undetected.

Both measures of endurance conditioning appeared to be sensitive to the effects of viral infection, a finding which perhaps highlights the reason why even the common cold is so often the downfall of an athlete approaching the peak of fitness. On a practical note, the use of a  $\dot{V}O_{2max}$  test to confirm a suspicion that a lack of form due to ill-health is obviously an undesirable procedure. The determination of Power 4  $mmol.l^{-1}$ , on the other hand, requires much less stress to be placed on the athlete and may therefore be a more appropriate tool in such circumstances.

It was pointed out in the review of literature that the primary determinant of  $\dot{V}O_{2max}$  is widely thought to be  $V_s$ . Conversely, a wealth of evidence supports the notion that the anaerobic threshold is principally governed by peripheral factors such as muscle fibre type (Tesch et al., 1981), enzymatic profiles (Sjodin et al., 1981, 1982) substrate availability (Ivy et al., 1981) and muscle recruitment patterns (Jacobs and Sjodin, 1985; Withers et al., 1981). It was therefore surprising that higher correlations were found between sub-maximal heart rate responses and Power 4  $mmol.l^{-1}$  than  $\dot{V}O_{2max}$ , bearing in mind that adjustments to steady state heart rate theoretically result from changes in  $V_s$ . This suggests that  $V_s$  and peripheral adaptations are tightly coupled.

Support for such a view is provided by the results of a study by Saltin et al., (1976) in which humans performed endurance training with one leg only. This group reported a reduction in sub-maximal heart rate when exercising the trained leg, but no reciprocal change was observed in the untrained leg. Thus, although  $V_s$  appeared to have increased the mechanism responsible for the adaptation was confined to the trained leg as no cross transfer occurred. Blomqvist and Saltin (1983) argue that a reduction in local vasoconstriction in response to the metabolic state of the exercising musculature was the cause of the increase in  $V_s$ .

An increase in vasodilation would explain the close relationship between sub-maximal heart rate and Power 4  $mmol.l^{-1}$  observed here, but only if lactate production during sub-maximal exercise is heavily  $O_2$  dependent. A greater perfusion of the muscle bed arising from a reduction in vasoconstriction would result in increased  $O_2$  delivery to the mitochondria thereby reducing lactate production. The changes in sub-maximal heart rate thus appear to support the recent proposal by Katz and Sahlin (1988) that lactate production is indeed dependent on mitochondrial  $O_2$  tension and not simply the result of an overstimulation of glycolysis.

The heavy demand for aerobic energy production in pursuing was reflected in the training and racing patterns of the subjects. The vast majority of training consisted of steady state endurance riding

often over periods of several hours, and all three riders regularly competed in long distance road races of between 100 and 180 Km. In the 4 to 6 weeks before major pursuit events, all three subjects introduced intensive interval training into their programmes. This would seem a logical action as it has been established that the pursuit demands the toleration of high levels of acidosis, and interval training programmes have been shown to increase peak blood lactate levels and muscle buffering capacity (Sharp et al., 1986). However, Roberts et al., (1982) have reported significant increases in glycolytic enzymes following a five week intense interval training programme, suggesting an enhancement of the glycolytic pathway. Such an adaptation could theoretically compromise endurance capacity as the exercise intensity at 4 mmol.l<sup>-1</sup> lactate has been shown to negatively correlate with both the concentration of glycolytic enzymes and the ratio of glycolytic to oxidative enzymes (Sjodin et al., 1981). Furthermore, a number of authors are of the opinion that increases in endurance capacity, as measured by changes in blood lactate profiles, are the direct result of a reduction in glycolytic flux (Brooks and Fahey, 1984; Holloszy and Coyle, 1984; Mader and Heck, 1986). However, such potentially negative effects could be detected in these subjects who typically reached their highest  $\dot{V}O_{2max}$  and Power 4 mmol.l<sup>-1</sup> levels between June and August, the period in the season when training intensity reached a peak. Thus it appears that a short period of high intensity training does not necessarily conflict with endurance capacity.

PART THREE

GENERAL SUMMARY

AND

CONCLUDING REMARKS

## Chapter Eight

### PHYSIOLOGICAL LIMITATIONS TO PURSUIT PERFORMANCE.

In the introduction to this thesis it was stated that our comprehension of the factors underlying athletic performance in many sporting events is far from adequate. The individual pursuit cycle race is one such discipline. It not only presents a harsh physical challenge to the competitor, but also an intellectual challenge to the Sport. The simple arithmetic of energy demand and potential sources of supply clearly demonstrates that a high aerobic power is essential for elite pursuit performance. However, the estimated energy demand of elite pursuit racing considerably exceeds the highest rates of aerobic energy production measured in man. There must therefore be a considerable anaerobic energy yield, and by inference, a requirement to tolerate the metabolic acidosis accompanying this. The event therefore appears to demand of the pursuit cyclist the physiological attributes of both a sprint and an endurance athlete.

The cause of fatigue in pursuing was a subject discussed at length in the review of literature in an attempt to build a conceptual base for this research. Although the reduction of intramuscular pH emerged as the leading candidate, it must be acknowledged that despite a mass of circumstantial evidence linking proton accumulation and diminished muscular performance, the exact mechanisms by which acidosis suppresses the contractile process remain a mystery.

Technical and practical limitations prevent changes in intramuscular pH during the pursuit race from being measured, so the degree of acidosis experienced by pursuiterers remains the subject of conjecture. The information on post race blood lactate levels provided indirect support for the view that pursuit cyclists experience a relatively severe metabolic acidosis, but the mean value of  $14.1 \text{ mmol.l}^{-1}$  recorded is considerably lower than the highest levels recorded in humans (Kindermann and Keul, 1977). This does not, however, exclude acidosis as the cause of fatigue. It

is possible that the lactate concentrations observed represent the extent to which acidosis can be tolerated without a marked impairment of co-ordination and concentration, factors that are crucial in an event that requires the precision handling of a highly responsive bicycle travelling in excess of  $14 \text{ m.s}^{-1}$  on a steeply banked track. The small standard deviation of  $0.9 \text{ mmol.l}^{-1}$  in the post race lactate values would appear to support this notion.

The observation that elite endurance trained athletes usually attain blood lactate levels below  $10 \text{ mmol.l}^{-1}$  following maximal exercise is common in the literature, a fact often attributed a reduced glycolytic capacity in such athletes (Mader and Heck, 1986; Roth et al., 1981). The peak blood lactate for the pursuiter of  $14.1 \text{ mmol.l}^{-1}$  is therefore unusually high for athletes possessing elite levels of aerobic power and is suggestive of an adaptation in the response to acid-base disturbance. Such an adaptation has been linked to changes in muscle buffering capacity ( $\beta$ ). Parkhouse et al., (1983) have reported a 27% greater  $\beta$  in oarsmen compared to marathoners with identical  $\dot{V}O_{2\text{max}}$  values ( $60 \text{ ml.kg}^{-1}\text{min}^{-1}$ ). As the physiological demand of rowing closely resembles that of pursuing in terms of aerobic and anaerobic energy demand (Secher, 1983), it seems reasonable to propose that pursuit cyclists may also possess an elevated  $\beta$ , possibly as a result of the intense interval training that precedes major competitions. This view is supported by Sharp et al., (1986) who found significant increases in peak muscle and blood lactate but an unchanged muscle pH at  $\dot{V}O_{2\text{max}}$  following 8 weeks of interval training, a result they attributed to an increase in  $\beta$ .  $\dot{V}O_{2\text{max}}$  also increased significantly, which re-enforces the belief that sprint training is not necessarily detrimental to endurance capacity.

A central role for aerobic metabolism in determining pursuit performance was proposed on the basis that the energy requirement of the event was such that only a minor percentage of this demand could be derived from anaerobic sources. This hypothesis was confirmed by the significant correlations observed in experiment II between pursuit speed and both the maximal and sub-maximal indices of aerobic conditioning,  $\dot{V}O_{2\text{max}}$  and Power  $4 \text{ mmol.l}^{-1}$ .

Thus, although the exact mechanism responsible for fatigue in pursuing remains elusive, the principle factor limiting performance is almost certainly the rate at which molecular oxygen can be consumed by the active mitochondria in the locomotor muscles of the pursuit cyclist without a serious disturbance of cell homeostasis.

The introduction to this thesis was critical of the pre-occupation in exercise physiology with factors limiting  $\dot{V}O_{2max}$ , on the grounds that the debate was often not relevant to sporting performance. Although a number of reservations concerning the applicability of the  $\dot{V}O_{2max}$  parameter will be expressed in the next section, it has become clear that the discussion of limitations to oxygen transport and consumption in conceptual terms is of primary importance here, particularly as the sub-maximal lactate response now appears to be heavily dependent on mitochondrial  $O_2$  tension (Katz and Sahlén, 1988).

Wagner (1988) has recently introduced a refreshing approach to the question of determinants of oxygen consumption which does not attempt to pin point a single limiting step but instead describes the interaction between the convective and diffusive transport of  $O_2$  to the mitochondria. An important assumption made is that  $O_2$  uptake is not limited by biochemical constraints within the muscle cell such as oxidative enzyme concentration. This view is supported by a wealth of evidence, some of which was discussed in the review of literature.

Wagner's basic premise is that the primary limitation to  $O_2$  consumption is diffusional transport between the red cells and the mitochondria. This is suggested to be a function of capillary  $PO_2$ , which represents the diffusion gradient as mitochondrial  $PO_2$  is thought to be negligible at maximal work rates, and the "tissue diffusing capacity". The latter term represents the ease with which  $O_2$  can move from haemoglobin (Hb) to the mitochondria at a given  $PO_2$ , and will be influenced by factors such as capillary density and the cellular location of the mitochondria with respect to these vessels. Because diffusional transport is dependent on capillary  $PO_2$

a secondary limitation resulting from convective O<sub>2</sub> delivery is implicated, as the volume of O<sub>2</sub> supplied to the muscle will influence capillary PO<sub>2</sub> and hence the diffusion gradient. Convective O<sub>2</sub> delivery will in turn will be governed by local blood flow regulation, cardiac performance, Hb concentration, pulmonary gas exchange and inspired PO<sub>2</sub>. Thus, any modification in capillary PO<sub>2</sub> resulting from changes in the variables just listed will affect O<sub>2</sub> consumption, but for any given maximal rate of convective O<sub>2</sub> delivery the ultrastructural properties that determine tissue diffusing capacity will be the limiting factor.

This scenario is attractive as it appears to offer grounds for explaining most of the experimental data concerning variations in  $\dot{V}O_{2max}$ . The increases in capillary and mitochondrial density that result from endurance training would raise tissue diffusing capacity by reducing the diffusion distance for O<sub>2</sub>, and the cardiovascular adaptations already discussed at length in this thesis would serve to elevate capillary PO<sub>2</sub> at a given workload. Furthermore, the results of experimentally manipulating arterial PO<sub>2</sub> through changes in inspired PO<sub>2</sub> or blood status can be accounted for by the effect these procedures have on O<sub>2</sub> delivery and therefore the diffusion gradient between capillary and mitochondria.

Such an integrated view of O<sub>2</sub> transport does not encourage the notion of a single rate limiting step in the oxygen uptake pathway. On the contrary, inter-individual differences in the relationship between the convective and diffusive processes should be expected, and variations of these factors in a single person following an extended period of training are also conceivable. Clearly then, a simple conclusion regarding the limitation to oxygen transport in pursuit cyclists cannot be made. There is however, one potential limiting factor that may be of increased significance in pursuited.

It was clear from the correlation analysis in experiment II that a high absolute aerobic power is an advantage in the pursuit. By inference, the best pursuit cyclists must therefore possess a very high cardiac output ( $\dot{Q}$ ). The  $\dot{V}O_{2max}$  of 7.2 l.min<sup>-1</sup> recorded on one occasion for the elite professional cyclist in this study would

require a  $\dot{Q}$  in excess of 40 l.min<sup>-1</sup>, assuming an exceptionally high Ca-C $\bar{V}O_2$  of 180 ml.l<sup>-1</sup>. According to Dempsey (1986) a cardiac output of this order will result in a marked reduction in red cell transit time through the pulmonary capillaries because such flow rates cannot be accommodated simply by increasing pulmonary blood volume. An increased venous-arterial shunt is also a possibility under such circumstances. These factors, combined with the low mixed venous PO<sub>2</sub> found in athletes will result in the need for a high alveolar PO<sub>2</sub> in order to prevent hypoxaemia arising from a failure to diffuse sufficient O<sub>2</sub> to the red cells.

The correlation between arterial de-saturation and absolute  $\dot{V}O_{2max}$  reported by Williams et al. (1986) suggests that elite endurance athletes cannot elevate alveolar PO<sub>2</sub> sufficiently to ensure successful O<sub>2</sub> diffusion across the alveolar-capillary junction, and more importantly, the greater the absolute  $\dot{V}O_{2max}$  of the athlete the larger the likely reduction in arterial PO<sub>2</sub>. Dempsey et al., (1984) have presented data indicating that insufficient hyperventilation arising from the mechanical limitations of the lung leads to inadequate alveolar PO<sub>2</sub> in such athletes, and is thus the cause of the hypoxaemia observed. An equally plausible possibility proposed by Hale (1987) is that a diffusive limitation between the atmosphere and the alveoli due to the structural nature of the lung and the diffusion time per breath is to blame. Both explanations however, imply that lung function may represent the ultimate limitation to aerobic capacity in humans, a view which has recently been expressed by a number of authors (Blomqvist and Saltin, 1983; Lindstedt et al., 1988).

With the above discussion in mind, it is worth noting two physiological characteristics of the most successful pursuiter in this study that distinguished him from the other cyclists. Hb concentration averaged 177 ( $\pm$  8) g.l<sup>-1</sup> throughout the monitoring period, a value considerably higher than the 140 to 160 g.l<sup>-1</sup> typically recorded for the other cyclists in this study. As blood viscosity in the physiologic range has been shown to have no measurable effect on  $\dot{V}O_{2max}$  (Celsing et al., 1987) this polycythaemic condition almost certainly resulted in an increased

convective O<sub>2</sub> delivery. Secondly, maximal minute volume occasionally exceeded 230 l.min<sup>-1</sup> (BTPS) for this subject during maximal exercise (see appendix 4). This ventilatory rate was approximately 20% higher than that found in the 2 pursuiter of nearest ability, and must be seen as an advantage in view of the finding by Dempsey et al. that arterial hypoxaemia is negatively related to the magnitude of the hyperventilatory response. It is tempting to speculate that this individual possessed exceptional lung function characteristics which enabled him to attain the extreme ventilatory flow rates recorded.

In summary, the physiological limitations to pursuit performance can be stated thus: muscle contractile performance appears to be constrained by the disturbance of intracellular homeostasis that results from the accumulation of protons generated by anaerobic ATP production. The requirement for the latter is the result of the failure to supply sufficient molecular O<sub>2</sub> to the mitochondria. The primary limiting factor would therefore seem to be the rate at which O<sub>2</sub> can diffuse from capillary to mitochondria. Whilst both central and peripheral adaptations will profoundly influence this diffusion rate, the upper limit to pursuit performance is likely to be set by the least plastic components of the O<sub>2</sub> pathway, namely blood composition and lung morphology.

A secondary limitation may be the extent to which the protons generated can be buffered in both the intra- and extra-cellular compartments. This is, however, entirely speculative as little data on this subject is available.

## Chapter Nine

### LABORATORY MEASURES OF AEROBIC PERFORMANCE CAPACITY.

The research strategy of this thesis was principally constructed around an examination of the relationship between competitive pursuit performance and physiological measures of O<sub>2</sub> transport. During the process of selecting the most appropriate measures the validity of  $\dot{V}O_{2max}$  as the best index of aerobic power was challenged. Although this parameter has been the cornerstone of physiological assessment programmes in the past, it appeared that the anaerobic threshold concept might offer a more accurate tool with which to examine the physiological attributes of pursuit cyclists. Parameters based on this concept purport to measure the ability to delay the onset of metabolic acidosis as well as the capacity to achieve a high rate of cellular respiration.

Experiment I investigated both the rationale behind the anaerobic threshold and the most appropriate method of detecting the exercise intensity corresponding to the AT in racing cyclists. A progressive metabolic acidosis during prolonged sub-maximal exercise above a certain intensity was observed in all 11 subjects, thus confirming the existence of a threshold. Furthermore, the power output at which this onset of acidosis was observed corresponded closely to a blood lactate level of 4 mmol.l<sup>-1</sup> recorded during a continuous 4 minute 35 W increment worktest. The determination of Power 4 mmol.l<sup>-1</sup> was therefore adopted as the method for identifying the AT of the cyclists in this research.

The increasing interest in the anaerobic threshold concept has led to a re-examination of the relationship between  $\dot{V}O_{2max}$ , training responses and endurance performance. Many researchers now believe that AT parameters more closely reflect the actual mechanisms that determine the rate of energy expenditure in endurance performance, a view that is supported by the findings of this thesis. As a result,  $\dot{V}O_{2max}$  is increasingly being viewed as determining the upper limit of an individuals performance potential, hence the popularity of

expressing the fraction of an individual's  $\dot{V}O_{2max}$  at which the AT occurs. Coupled to this is the idea that the two indices are limited by different factors, namely  $\dot{V}O_{2max}$  is determined by central circulatory mechanisms whereas AT reflects adaptations at the cellular level (Barlow et al., 1985; Hurley et al., 1984). This paradigm is attractive as it could offer a way of specifically targeting training on the mechanism appearing to limit an athlete's progress. However, a number of observations were made during the course of this work that raise doubts about the validity of this division of AT and  $\dot{V}O_{2max}$  into separate conceptual compartments.

The belief that AT is simply a function of the intramuscular milieu stems from experimental evidence purported to prove that sub-maximal lactate production is not  $O_2$  dependent (Brooks, 1986). This view has recently been challenged in a major review by Katz and Sahlin (1988) who reaffirm the classical notion that lactate production stems from insufficient  $O_2$  delivery. Their hypothesis, in short, is this. As exercise intensity increases, mitochondrial  $PO_2$  progressively declines. Although cellular respiration is not thought to be impaired until mitochondrial  $PO_2$  is virtually zero (i.e. at  $\dot{V}O_{2max}$ ), the same is not true for aerobic metabolism which becomes disturbed at a much higher  $PO_2$ . When the latter occurs an impairment of respiration is avoided by an increase in mitochondrial NADH and cytosolic ADP and  $P_i$ , all of which are claimed to stimulate mitochondrial respiration. Respiratory rate can therefore continue to rise but the resulting elevations of cytosolic NADH, ADP and  $P_i$  stimulate glycolysis and thus lactate production. Sub-maximal lactate production is therefore seen as a regulatory phenomenon primarily arising from a disturbance of cellular metabolism due to insufficient mitochondrial  $O_2$ . The degree of glycolytic stimulation will be influenced by factors such as carbohydrate status, individual glycolytic and oxidative enzyme profiles, fibre type/recruitment pattern and sympathetic drive, so individual responses to disturbed cell metabolism are to be expected.

Katz and Sahlin present considerable experimental support for this

model, the most important being the observation that increases in cell NADH parallel increases in lactate production during intense sub-maximal exercise. The fact that  $\dot{V}O_2$  at a given sub-maximal workload remains unchanged when lactate production is reduced is dismissed as evidence that lactate production is not  $O_2$  dependent on the grounds that the fractional anaerobic ATP contribution in sub-maximal work is below the resolution of  $\dot{V}O_2$  measurement procedures.

It thus seems that the AT is fundamentally linked to the capacity to supply  $O_2$  to the mitochondria, a view that Wasserman and co-workers have been expressing for many years (e.g. Wasserman et al., 1986). Hence, on theoretical grounds the diffusional and convective transport of  $O_2$  not only appears to determine  $\dot{V}O_{2max}$  but the AT as well. The idea that  $\dot{V}O_{2max}$  and AT are independently limited therefore looks to be flawed.

This integrated view of  $\dot{V}O_{2max}$  and AT is supported by the finding that changes in Power 4  $mmol.l^{-1}$  and sub-maximal heart rate were highly correlated in all the longitudinal case studies. Furthermore, in 2 of the 3 individuals monitored changes in  $\dot{V}O_{2max}$  and Power 4  $mmol.l^{-1}$  were closely coupled, and no progressive improvements in fractional utilization were seen.

The important question arising from this discussion is whether there is a need to measure both the  $\dot{V}O_{2max}$  and the AT of an endurance athlete in order to monitor training progression and performance potential? Jacobs et al., (1985) have recently argued that sub-maximal blood lactate measures could replace  $\dot{V}O_{2max}$  as an endurance measure without any loss of information relevant to athletic performance. This findings of this thesis suggest that there are grounds for such a view.

Firstly, Power 4  $mmol.l^{-1}$  predicted pursuit performance with greater accuracy than  $\dot{V}O_{2max}$ , regardless of the units of expression. This re-enforces the notion that AT measures reflect more closely the performance limiting factors in endurance

disciplines. Furthermore, the case studies revealed the AT to be more sensitive to training adaptations than  $\dot{V}O_{2max}$ , a finding that has been previously reported (Davis et al., 1979).

Secondly, the AT exercise protocol can provide important information to the coach. There is now a large volume of research that suggests that training intensity should be set with respect to the AT, rather than at an arbitrary speed or heart rate in order to maximize overload (Dwyer and Bypsee, 1983; Fohrenbach et al., 1987, Heck et al., 1985; Katch et al., 1978; Kindermann et al., 1979; Parkhouse et al., 1982; Tanaka et al., 1986). The case studies also revealed the sensitivity of this measure to viral infections, an experience that has been described elsewhere (Conconi et al., 1982).

Perhaps the major advantage of AT measures such as Power 4  $mmol.l^{-1}$  over  $\dot{V}O_{2max}$  is, however, the objectivity of the test. A recent review by Noakes (1988) has strongly criticized the  $\dot{V}O_{2max}$  procedure on the grounds that it is often not possible to establish that an individual has reached a true  $\dot{V}O_{2max}$  because of the absence of a clear plateau in  $O_2$  consumption with increasing work. When the  $\dot{V}O_{2max}$  data from this research was analyzed it was found that a clear plateau ( $<0.15$  l increase in  $\dot{V}O_2$ ) was only detectable in 82 of the 158 tests, even though the subjects, who were all highly trained cyclists, were strongly encouraged to exercise to exhaustion. The absence of a plateau raises the possibility that local muscle fatigue prevented some subjects reaching a work level at which the  $O_2$  transport system was fully taxed. Under such conditions any future increase in  $\dot{V}O_{2max}$  could not, with certainty, be ascribed to an increase in  $O_2$  transport. A greater tolerance of local fatigue or increased motivation may have enabled a higher workload to be reached and therefore a greater  $\dot{V}O_2$ .  $\dot{V}O_{2max}$  may therefore be a concept rather than a reality in many cases.

An AT measure such as Power 4  $mmol.l^{-1}$  does not require maximal exertion and therefore does not suffer from the problems associated with subject motivation. Furthermore, where fixed blood lactate

levels are used, the determination of the exercise intensity at AT is a simple, objective procedure. Other advantages include the ease with which lactate data can be collected in the field, the lower cost of the apparatus required in comparison to that necessary for measuring respiratory gas exchange variables, and the frequency with which a sub-maximal test can be used. If careful monitoring of an elite athlete is required, then frequent exposure to exhaustive protocols is undesirable, particularly during a period of intense training or close to a major competition. Finally, practical experience with individuals tested on a regular basis has shown that markedly reduced blood lactate responses can occur during a severe racing or training programme, a phenomenon almost certainly due to glycogen depletion (Hughes et al., 1982). It might therefore be possible, with experience, to identify poor carbohydrate status in an athlete undergoing regular laboratory assessment.

Thus from a purely practical perspective there appears to be little justification for the continued use of  $\dot{V}O_{2max}$  as a means of monitoring the physiological responses of racing cyclists. Furthermore, there are grounds for arguing that the pre-eminence of this parameter as a research tool should also be challenged.

## Chapter Ten

### PRACTICAL IMPLICATIONS OF THE RESEARCH FINDINGS.

#### Athlete suitability and monitoring procedures.

The clear relationship between aerobic conditioning and success in pursuing found in Experiment II indicates that potential champion pursuiterers are to be found amongst individuals endowed with a high  $O_2$  transport potential. Furthermore, because absolute power was found to be of overriding importance, absolute measures of functional capacity should be employed when assessing pursuit cyclists.

The primary implication for pursuit competition of the highest calibre is that cyclists of low body mass are likely to be at a disadvantage compared to their heavier counterparts because of their inferior absolute power. The only condition when bodyweight may become less advantageous is in pursuit competitions held on small diameter, tightly banked tracks (250 m or less). On such tracks the gravitational force that determines rolling resistance is compounded by the centripetal force generated when the cyclist negotiates the banking. As the latter force will be mass and speed dependent it seems reasonable to suppose that a heavier rider will suffer greater energy losses at the tyre-track interface through the bankings, which in turn occur more frequently on a small track for a given race distance. A pursuiter's speed on exiting a tight banking is therefore likely to be fractionally reduced and must be corrected by acceleration down the straights, the energy cost of which is inertia (mass) dependent. Thus, the advantage a heavy elite pursuiter may have over a lighter opponent may be compromised to a certain extent by the geometry of the track.

The previous section recommended the measurement of Power  $4$   $\text{mmol.l}^{-1}$  in preference to  $\dot{V}O_{2\text{max}}$  as the most suitable protocol for assessing pursuit performance capability and training responses. Although the latter procedure has been criticized for being

motivation dependent, a controlled maximal work bout such as that employed for measuring  $\dot{V}O_{2max}$  in thesis may provide useful data in the evaluation of pursuit conditioning. The sampling of peak blood lactate following such an effort might, for example, provide an index of the buffering capacity of an individual, a property one would seek to enhance when approaching major competitions. Further research however, is clearly needed into the whole area of training responses and tolerance of acidosis, particularly in endurance athletes.

The importance of the oxygen transport pathway highlighted in chapter 8 suggests that routine monitoring of haematological variables would be a desirable feature of a monitoring programme for pursuited. Whilst little can be done to increase the Hb concentration of a healthy individual without resorting to dangerous or proscribed methods, poor nutrition or excessive training may have a detrimental effect on this parameter (Clement and Asmussen, 1982).

The strong relationship between changes in Power  $4 \text{ mmol.l}^{-1}$  and sub-maximal heart rate reported in chapter 7 indicate that the monitoring of heart rate response to a standardized load could provide coaches with useful information regarding the effectiveness of endurance training programmes. The availability of cheap, reliable pulsemeters today makes such a procedure a viable proposition.

### Training Strategy

From a physiological standpoint there seems little doubt that the primary aim of a pursuit training programme should be to increase the volume of  $O_2$  that can be transported to the mitochondria. Because of the apparent interdependence of both central and peripheral factors in determining the latter, a variety of training stimuli may be needed to ensure that adaptations take place across the whole of the  $O_2$  pathway. The very long duration, steady state

low intensity riding much favoured by cyclists may well induce changes to cardiac function but is unlikely to greatly stress the metabolic capacity of the exercising musculature. The intensity at which such training is performed is typically below that at which changes in blood lactate can be detected (unpublished observations), and there can therefore be no disturbance of mitochondrial redox balance. This is only likely to be achieved when training at an intensity that results in permanently elevated lactate levels. As the duration of the training stimulus is thought to be of considerable importance (Pollock, 1973), prolonged exercise at or just below the AT is likely to provide an optimal overload on muscle metabolism, as exercising above this level rapidly results in fatigue (see experiment I).

High intensity interval training is thought to be an effective method of increasing aerobic power (Astrand and Rodahl, 1986) but the pursuit cyclist stands to benefit from such training in other ways. Although information on the subject is scarce, the enhanced muscle buffering capacity thought to result from repeated bursts of heavily anaerobic exercise may be of considerable importance to pursuit cyclists who have to suffer a marked acidosis during competition. Secondly, because the speed and power output attained in interval training sessions should at least match those experienced in pursuit competition, the appropriate force generation and fibre recruitment patterns for this event will be developed.

### Racing Strategy

The speed profile of an elite pursuit race shown in figure 1 (page 14) is typical of that found at all competition levels and is characterized by a rapid acceleration in the first 30 seconds to a peak speed which then diminishes gradually for the rest of the race. Wilberg and Pratt (1988) have analyzed the lap speed profiles of over 200 pursuit competitors and concluded that riders were most successful when they achieved a lower initial peak speed and subsequently maintained a consistent lap time for the remainder of the race. Nevertheless, such a race strategy is infrequently seen in

current major competitions where there is a strong emphasis on maximal starting efforts which inevitably result in the decaying speed profile first described.

From a physiological perspective maximal power production in the first minute of the race would seem to be undesirable. Although information relating to the oxygen uptake kinetics at the start of intense exercise in elite athletes could not be found, available data indicate that the inertia of the O<sub>2</sub> transport system is such that maximal rates of O<sub>2</sub> consumption will not occur until at least 60 s into the race (Hagberg et al., 1980; Hughson and Morrissey, 1982; Keen et al., 1985). The myoglobin O<sub>2</sub> store only is thought to be approximately 0.5 l (Astrand et al., 1986) so there must be a heavy dependence on anaerobic ATP production in at least the first 30 s. As the acidosis that results from this energy pathway is a possible cause of fatigue in pursuing, it unlikely that an advantage is gained by reaching the highest rate of energy production when O<sub>2</sub> supply is poor. One potentially positive effect of a marked acidosis at the start of the event might be a greater increase in muscle blood flow through a pH mediated reduction in vasoconstriction, but such a biochemical trade-off for increased O<sub>2</sub> delivery is purely speculative as no data to support this view could be found.

From a mechanical perspective, any acceleration above the desired race speed will be costly in terms of energy production. The curvilinear relationship between power and cycling speed dictates that even a small increase in speed above that required to achieve an even pace will necessitate a large increase in power output. Furthermore, the work done overcoming inertia during acceleration will demand a large energy release which cannot be completely recouped during a period of deceleration as mechanical energy losses are unavoidable.

Thus, there appear to be considerable grounds for recommending that pursuit cyclists should not accelerate excessively during the first minute of the race, but instead aim to maintain a constant speed for the majority of the event. Further acceleration should

only occur when the rider can be certain that a fatigue induced reduction in speed will compromise performance. The analysis of lap or half-lap times would considerably assist the coach in developing the racing skills needed to ensure this.

A long warm-up, usually performed on stationary cycle rollers, is a classic feature of a pursuit rider's preparation for competition. Unfortunately, there is a notable lack of data in the literature concerning the physiological effects of warm-up, so this subject can only be discussed in broad terms. On the basis of the above discussion an advantage might be gained if the pursuit cyclist could start the event with a  $\dot{V}O_2$  above a resting value. This is unlikely to occur as the delay between finishing a warm-up and starting a race is rarely less than 5 minutes. Hagberg et al., (1980) have studied  $\dot{V}O_2$  decay rates in trained subjects following exercise at 70%  $\dot{V}O_{2max}$ . They found that  $\dot{V}O_2$  had returned to within 5% of the resting value after only 74 seconds of rest. In one of the few specific studies on warm-up procedures Martin et al., (1975) also found  $\dot{V}O_2$  had returned to resting levels 3 minutes after the completion of a warm-up, but in the subsequent maximal running exercise a faster acceleration in  $\dot{V}O_2$  coupled with a reduction in lactate accumulation was observed compared to the no warm-up condition. These authors concluded that the major benefits of warming up are the increases in both muscle temperature and muscle perfusion that occur.

Perhaps the most constructive comment that can be made regarding warm-up procedure concerns the optimal exercise intensity. The negative effects of raised blood lactate levels on subsequent maximal aerobic performance demonstrated by Hogan and Welch, (1984) suggests that warm-up intensity should not exceed the level at which blood lactate begins to rise, i.e. the lactate threshold. Knowledge of the heart rate at which this occurs would enable a pursuiter to achieve maximal increases in muscle temperature and blood flow during a warm-up routine, without running the risk of accumulating lactate. The optimal duration of a warm-up is much less clear and is a subject that requires investigation.

PART FOUR

REFERENCES

## REFERENCES

ALLEN, K. W., D. R. SEALS, B. F. HURLEY, A. A. AHSANI and J. M. HAGBERG (1985). *Lactate threshold and distance running performance in young and older endurance runners.*  
J. Appl. Physiol. 58(4): 1281-1284

ARMSTRONG, L. E., D. L. COSTILL and W. J. FINK (1985). *Influence of diuretic-induced dehydration on competitive running performance.*  
Med. Sci. Sports 17(4): 456-461

ASMUSSEN, E. (1979). *Muscle Fatigue.*  
Med. Sci. Sports 11(4): 313-321

ASTRAND, P-O. (1984). *Principles of ergometry and their implications in sports practice.*  
Sports Med. 1: 1-5

ASTRAND, P-O., E. HULTMAN, A. JUHLIN-DANNFELT and G. REYNOLDS (1986). *Disposal of lactate during and after strenuous exercise in humans.*  
J. Appl. Physiol. 61(1):338-343

ASTRAND, P- O. and K. RODAHL (1986). *Textbook of Work Physiology: Physiological Bases of Exercise (3rd Edition).*  
New York: McGraw-Hill

BAGSHAW, C. R. (1982). *Muscle contraction.*  
London: Chapman and Hall.

BAR-OR, O. (1987). *The wingate anaerobic test. An update on methodology, reliability and validity.*  
Sports Medicine 4: 381-394

BARLOW, K., A. WELTMAN, R. SCHURRER and J. HENRITZE (1985). *Prediction of maximal effort bicycle ergometer endurance performance.*  
Int. J. Sports Med. 6(4): 190-196

BEAVER, W. L., K. WASSERMANN and B. J. WHIPP (1985). *Improved detection of lactate threshold during exercise using a log-log transformation.*  
J. Appl. Physiol. 59(6): 1936-1940

BERGH, U. (1987). *The influence of body mass in cross-country skiing.*  
Med. Sci. Sports 19(4): 324-331

BERGSTROM, J. and E. HULTMAN (1967). *A study of the glycogen metabolism during exercise of man.*  
Scand. J. Clin. Lab. Invest. 19:218-228

BERGSTROM, J. R. C., HARRIS, E. HULTMAN and L-O. NORDESJO (1971). *Energy rich phosphagens in dynamic and static work*. In: PERNOW, B and B. SALTIN (Eds.), *Muscle metabolism during exercise*. New York, Plenum Press: 341-355

BEYERHOLM, J. (1987). *The hour record*.  
Le Monde Cycliste 4: 6-11  
Milan: Union Cycliste Internationale - January 87

BIGLAND-RITCHIE, B. (1981). *EMG and fatigue of human voluntary and stimulated contractions*.  
In: PORTER R. and J. WHELEN (Eds.), *Human muscle fatigue: physiological mechanisms*. Ciba Foundation Symposium 82.  
London, Pitman Medical: 120-129

BLOMQUIST, C. G., and B. SALTIN (1983). *Cardiovascular adaptations to physical exercise*.  
Ann. Rev. Physiol. 45: 169-189

BONING, D., Y. GONEN and N. MAASSEN (1984). *Relationship between work load, pedal frequency and physical fitness*.  
Int. J. Sports Med. 5(2): 92-97

BROOKS, G. A. (1986) *The lactate shuttle during exercise and recovery*.  
Med. Sci. Sports 18(3): 360-368

BROOKS, G. A. and T. D. FAHEY (1984). *Exercise Physiology: Human Bioenergetics and its applications*.  
New York: John Wiley

BROWN, N. J., L. A. STEPHENSON, G. LISTER and E. R. NADEL (1982). *Relative anaerobiosis during heavy exercise in the heat*.  
Fed. Proc. 41: 1677

BUCHANAN, M. and A. WELTMAN (1985). *Effects of pedal frequency on  $\dot{V}O_2$  and work output at lactate threshold (LT), fixed blood lactate concentrations of 2mM and 4mM and max in competitive cyclists*.  
Int. J. Sports Med. 6(3): 163-168

BUNC, V., J. HELLER, J. LESO, S. SPRYNAROVA and R. ZDANOWICZ (1987). *Ventilatory threshold in various groups of highly trained athletes*.  
Int. J. Sports Med. 8: 275-280

BUONO, M. J. and F. B. ROBY (1982). *Acid-base, metabolic, and ventilatory responses to repeated bouts of exercise*.  
J. Appl. Physiol. 53(2): 436-439

BURKE, E. R., S. FLECK and T. DICKSON (1981). *Post-competition blood lactate concentrations in competitive track cyclists*.  
Brit. J. Sports Med. 15(4): 242-245

BURTON, R. F. (1978). *Intracellular buffering*.  
Resp. Physiol. 33: 51-58

CAVANAGH, P. and D. J. SANDERSON (1986). *The biomechanics of cycling: studies of the pedaling mechanics of elite pursuit riders.*  
In: BURKE, E. (Ed.), *Science of Cycling*. Champaign, IL. Human Kinetics: 91-122

CELSING, F., J. SVEDENHAG, P. PIHLSTEDT and B. EKBLOM (1987). *Effects of anemia and stepwise-induced polycythaemia on maximal aerobic power in individuals with high and low haemoglobin concentrations.*  
*Acta Physiol. Scand.* 129: 47-54

CITTERIO, G. and E. AGOSTONI (1984). *Selective activation of quadriceps muscle fibres according to bicycle rate.*  
*J. Appl. Physiol.* 57: 371-379

CLEMENT, D. B. and R. C. ASMUNDSON (1982). *Nutritional intake and haematological parameters in endurance runners.*  
*Physician Sports Med.* 10(3): 37-43

COAST, J. R. and H. G. WELCH (1985). *Linear increases in optimal pedal rate with increased power output in cycle ergometry.*  
*Eur. J. Appl. Physiol.* 53: 339-342

COHEN, J. L. and K. R. SEGAL (1985). *Left ventricular hypertrophy in athletes: an exercise echocardiographic study.*  
*Med. Sci. Sports* 17(6): 695-700

COLEMAN, S. G. S., T. HALE and E. J. HAMLEY (1986). *Correct power measurement in the wingate test.*  
In: REILLY, T., J. WATKINS, J. BORMS (Eds.), *Proceedings of the VIII Commonwealth Conference on Sport (Kinanthropometry III)*. London, E & F.N. Spon: 308-309

CONCONI, F., M. FERRARI, P. G. ZIGLIO, P. DROGHETTI and L. CODECA (1982). *Determination of the anaerobic threshold by a non-invasive field test in runners.*  
*J Appl. Physiol.* 52(4): 869-873

CONLEE, R. K. (1987). *Muscle glycogen and exercise endurance: a twenty-year perspective.*  
In: PANDOLF, K. B. (Ed.), *Exercise and Sports Science Reviews* Vol. 15. New York, MacMillan: 1-29

COSTILL, D. L., J. DANIELS, W. EVANS, W. FINK, G. KRAHENBUHL and B. SALTIN (1976). *Skeletal muscle enzymes and fibre composition in male and female track athletes.*  
*J. Appl. Physiol.* 40: 149-154

DAVIS, A., M. H. FRANK, B. J. WHIPP and K. WASSERMAN (1979). *Anaerobic threshold alterations caused by endurance training in middle aged men.*  
*J. Appl. Physiol.* 46(6): 1039-1046

DAWSON, M. J. (1983). *Phosphorus metabolites and the control of glycolysis studied by nuclear magnetic resonance.*  
In: KNUTTGEN, H., J. VOGEL and J. POORTMANS, (Eds.),  
International series on Sport Sciences 13: Biochemistry of Exercise.  
Champaign, Il. Human Kinetics: 116-125

DEMPSEY, J. A. (1986). *Is the lung built for exercise?*  
Med. Sci. Sports 18(2): 143-155

DEMPSEY, J. A., P. HANSON and K. HENDERSON (1984).  
*Exercise-induced alveolar hypoxemia in healthy human subjects at sea-level.*  
J. Physiol. (London) 355: 161-175

DENIS, C., R. FOUQUET, P. POTY, A. GEYSSANT and J. R. LAKIER  
(1982). *Effect of 40 weeks of endurance training on the anaerobic threshold.*  
Int. J. Sports Med. 3(4): 208-214

DI PRAMPARO, P. E., G. CORTILLI, P. MOGNONI and F. SAIBENE  
(1979). *Equation of motion of a cyclist.*  
J. Appl. Physiol. 47(1): 201-206

DI PRAMPARO, P.E., C.T.M. DAVIES, P. CERETELLI and R. MARGARIA  
(1970). *An analysis of oxygen debt contracted in submaximal exercise.*  
J. Appl. Physiol. 29: 547-551

DILL, D. B. and F. N. MARZULLI (1954). *Energy expenditure in bicycle riding.*  
J. Appl. Physiol. 7: 320-324

DODD, S. L., S. K. POWERS, D. THOMSON, G. LANDRY and J. LAWLER  
(1989). *Exercise performance following intense, short-term ventilatory work.*  
Int. J. Sports Med. 10(1): 48-52

DONALDSON, S. K. B. (1983). *Effect of acidosis on maximum force generation in peeled mammalian skeletal muscle fibers.*  
In: KNUTTGEN, H., J. VOGEL and J. POORTMANS, (Eds.),  
International series on Sport Sciences 13: Biochemistry of Exercise.  
Champaign, Il. Human Kinetics: 126-13

DONALDSON, S. K. B. and L. HERMANSEN (1978). *Differential, direct effects of H<sup>+</sup> and Ca<sup>++</sup> activated free skinned fibres from the soleus, cardiac and adductor magnus muscle of rabbits.*  
Pflugers Arch. 376: 55-6

DU BOIS, D., and E. F. DU BOIS (1916). *A formula to estimate the approximate surface area if height and weight be known.*  
Arch. Int. Med. 17: 863-871

DWYER, J. and R. BYPEE (1983). *Heart rate indices of the anaerobic threshold.*  
Med. Sci. Sports 15(1): 72-76

EDWARDS, R. H. T. (1983). *Biochemical bases of fatigue in exercise performance: catastrophe theory of muscular fatigue.*

In: KNUTTGEN, H., J. VOGEL and J. POORTMANS, (Eds.), International series on Sport Sciences 13: Biochemistry of Exercise. Champaign, Il. Human Kinetics: 3-28

EDWARDS, R. H. T. (1981). *Human muscle function and fatigue.*

In: PORTER R. and J. WHELAN (Eds.), Human muscle fatigue: Physiological mechanisms. Ciba Foundation Symposium, No. 82. London, Pitman medical: 1-18

EKBLOM, B., R. HUOT, E. M. STEIN and A. T. THORSTENSSON (1975). *Effect of changes in arterial oxygen content on circulation and physical performance.*

J. Appl. Physiol. 39(1): 71-75

FAVIER, R. J., S. H. CONSTABLE, M. CHEN and J. O. HOLLOSZY (1986). *Endurance training reduces lactate production.*

J. Appl. Physiol. 61(3): 885-889

FOHRENBACH, R., A. MADER and W. HOLLMANN (1987). *Determination of endurance capacity and prediction of exercise intensities for training and competition in marathon runners.*

Int. J. Sports Med. 8(1): 11-18

FORSTER, H. V., J. A. DEMPSEY, J. THOMSON, E. VIDRUK and G. A. DOPICO (1972). *Estimation of arterial PO<sub>2</sub>, PCO<sub>2</sub>, pH and lactate from arterialized venous blood.*

J. Appl. Physiol. 32: 134-138

FOSTER, C. (1983).  *$\dot{V}O_{2max}$  and training indices as determinants of competitive running performance.*

J. Sports Sciences 1: 13-22

FROESE, E. A. and M. E. HOUSTON (1987). *Performance during the wingate anaerobic test and muscle morphology in males and females.*

Int. J. Sports Med. 8(1): 35-39

GLEDHILL, N. (1982). *Blood doping and related issues: a brief review.*

Med. Sci. Sports 14(3): 183-189

GOLLNICK, P. and B. SALTIN (1982). *Significance of skeletal muscle oxidative enzyme enhancement with endurance training.*

Clinical Physiology 2: 1-12

GOLLNICK, P. D., W. M. BAYLY and D. R. HODGSON (1986). *Exercise intensity, training, diet, and lactate concentration in muscle and blood.*

Med. Sci. Sports 18(3): 334-340

GOLLNICK, P.D. and L. HERMANSEN (1973). *Biochemical adaptations to exercise: Anaerobic Metabolism.*

In: WILMORE, J. (Ed.), Exercise and Sports Science Reviews Vol. 1 New York, Academic Press: 1-43

GRIMBY, G. (1969). *Respiration during exercise*.  
Med. Sci. Sports 1(1): 9-14

HAGBERG, J. (1984). *Physiological implications of the lactate Threshold*.  
Int. J. Sports Med. 5: 106-109

HAGBERG, J. M., J. P. MULLIN, M. D. GEISE and E. SPITZNAGEL (1981). *Effect of pedaling rate on submaximal exercise responses of competitive cyclists*.  
J. Appl. Physiol. 51(2): 447-451

HAGBERG, J. M., M. D. GIESE and R. B. SCHNEIDER (1978). *Comparison of the three procedures for measuring  $VO_{2max}$  in competitive cyclists*.  
Eur. J. Appl. Physiol. 39: 47-52

HAGBERG, J. M., R. C. HICKSON, A. A. EHSANI and J. O. HOLLOSZY (1980). *Faster adjustment to and recovery from submaximal exercise in the trained state*.  
J. Appl. Physiol. 48(2): 218-224

HAHN, A. G., D. A. TUMILTY, R. D. TELFORD and B. T. WAKEFIELD (1986). *Effect of test duration on maximum oxygen uptake in elite road cyclists*.  
In: WATKINS J., T. REILLY, L. BURWITZ (Eds.), *Proceedings of the VIII Commonwealth Conference on Sport (Sports Science)*. London, E & F.N. Spon: 36-42

HALE, T. (1987). *Lung gas mixing efficiency in exercise*.  
D.Phil. Thesis. University of Surrey

HAMLEY, E. J. and V. THOMAS (1967). *Physiological and postural factors in the calibration of the bicycle ergometer*.  
J. Physiol. 191: 55-56 P

HARDMAN, A. E. and C. WILLIAMS (1985). *Blood lactate concentration during submaximal cycling in elite male road racing cyclists*.  
In: HALE, T. A. MORRIS, N. SMITH and S. COLEMAN (Eds.), *Proceedings of the Annual Sport and Science Conference*

HECK, H., A. MADER, G. HESS, S. MUCKE, R. MULLER and W. HOLLMAN (1985). *Justification of the  $4\text{mmol.l}^{-1}$  lactate threshold*.  
Int. J. Sports Med. 6: 117-130

HELAL, J. N., C. Y. GUEZENNEC and F. GOUBEL (1987). *The aerobic-anaerobic transition: re-examination of the threshold concept including an electromyographic approach*.  
Eur. J. Appl. Physiol. 56: 643-649

HERMANSEN, L. (1971). *Lactate production during exercise*.  
In: PERNOW, B and B. SALTIN (Eds.), *Muscle metabolism during exercise*.  
New York, Plenum Press: 401-408

HERMANSEN, L. (1973). *Oxygen transport during exercise in human subjects.*

Acta Physiol. Scand. (suppl) 399

HERMANSEN, L. and B. SALTIN (1969). *Oxygen uptake during maximal treadmill and bicycle exercise.*

J. Appl. Physiol. 26(1): 31-37

HERMANSEN, L. and I. STENSVOLD (1972). *Production and removal of lactate during exercise in man.*

Acta. Physiol. Scand. 86: 191-201

HERMANSEN, L. and J-B. OSNES (1972). *Blood and muscle pH after maximal exercise in man.*

J. Appl. Physiol. 32: 304-308

HERMANSEN, L. and O. VAAGE (1977). *Lactate disappearance and glycogen synthesis in human muscle after maximal exercise.*

AM. J. Physiol. 233(5): E422-E429

HESSER, C. M., D. LINNARSSON and L. FAGRAEUS (1981). *Pulmonary mechanics and work of breathing at maximal ventilation and raised air pressure.*

J. Appl. Physiol. 50: 747-753

HILL, A. V. and H. LUPTON (1923). *Muscular exercise, lactic acid, and the supply and utilization of oxygen.*

Q. J. Med. 16: 131-171

HIRCHE, H., B. HANBACK, H. D. LANGOHR, U. WACKER and J. BUSS (1975). *Lactic acid permeation rate in working gastrocnemii of dogs during metabolic alkalosis and acidosis.*

Pflugers Archiv. 356: 209-222

HOCHACHKA, P. W., and T. P. MOMMSEN (1983). *Protons and anaerobiosis.*

Science 219: 1391-1397

HOGAN, M. C. and H. G. WELCH (1984). *Effect of varied lactate levels on bicycle ergometer performance.*

J. Appl. Physiol. 57(2): 507-513

HOGAN, M. C., R. H. COX and H. G. WELCH (1983). *Lactate accumulation during incremental exercise with varied inspired oxygen fractions.*

J. Appl. Physiol. 55(4): 1134-1140

HOLLOSZY, J. O. and E. F. COYLE (1984) *Adaptations of skeletal muscle to endurance exercise and their metabolic consequences.*

J. Appl. Physiol. 56: 831-838

HOPPELER, H., H. HAWOLD, K. CONLEY, S. LINDSTEDT, H. CLAASEN, P. VOCK and E. WEIBEL (1985). *Endurance capacity in humans: aerobic capacity and structure of skeletal muscle.*

J. Appl. Physiol. 59(2): 320-327

HUGHES, E. F., S. C. TURNER and G. A. BROOKS (1982). *Effects of glycogen depletion and pedaling speed on anaerobic threshold.* J. Appl. Physiol. 52(6): 1598-1607

HUGHSON, R.L. and M. MORRISSEY (1982). *Delayed kinetics of respiratory gas exchange in the transition from prior exercise.* J. Appl. Physiol. 52(4): 921-929

HULTMAN, E. and K. SAHLIN (1980). *Acid-base balance during exercise.*

In: HUTTON, R. and D. MILLAR (Eds.), *Exercise and Sports Science Reviews* Vol. 8. Philadelphia, Franklin Institute Press: 41-128

HURLEY, B. F., J. M. HAGBERG, W. K. ALLEN, D. R. SEALS, J. C. YOUNG, R. W. CUDDIHEE and J. O. HOLLOSZY (1984). *Effect of training on blood lactate levels during submaximal exercise.* J. Appl. Physiol. 56(5): 1260-1264

INBAR, O., A. ROTSTEIN, I. JACOBS, P. KAISER, R. DLIN and R. DOTAN (1983). *The effects of alkaline treatment on short-term maximal exercise.* J. Sports Sciences 1: 95-104

IVY, J. L., D. L. COSTILL and B. D. MAXWELL (1980). *Skeletal muscle determinants of maximum aerobic power in man.* Eur. J. Appl. Physiol. 44: 1-8

IVY, J. L., D. L. COSTILL, P. J. VAN HANDEL, D. A. ESSIG and R. W. LOWER (1981). *Alterations in the lactate threshold with changes in substrate availability.* Int. J. Sports med. 2(3): 139-142

IVY, J. L., R. T. WITHERS, P. J. VAN HANDEL, D. H. ELGER and D. L. COSTILL (1980). *Muscle respiratory capacity and fibre type as determinants of the lactate threshold.* J. Appl. Physiol. 48(3): 523-527

JACOBS, I. (1981). *Lactate, muscle glycogen and exercise performance in man.* Acta Physiol. Scand. (suppl) 495

JACOBS, I. (1986). *Blood lactate implications for training and sports performance.* Sports Medicine 3: 10-25

JACOBS, I. and B. SJODIN (1985). *Relationship of ergometer-specific  $\dot{V}O_{2max}$  and muscle enzymes to blood lactate during submaximal exercise.* Brit. J. Sports Med. 19: 77-80

JACOBS, I., O. BAR-OR, J. KARLSSON, R. DOTAN, P. TESCH, P. KAISER and O. INBAR (1982). *Changes in muscle metabolites in females with 30-s exhaustive exercise.* Int. J. Sports Med. 14(6): 457-460

JACOBS, I., R. SCHELE and B. SJODIN (1985). *Blood lactate vs exhaustive exercise to evaluate aerobic fitness.*  
Eur. J. Appl. Physiol. 54: 151-155

JAKEMAN, P. M. (1986). *Physiological correlates of middle distance running performance in trained athletes.*

In: WATKINS, J. T. REILLY, L. BURWITZ (Eds.), *Proceedings of the VIII Commonwealth Conference on Sport (Sports Science)* London, E & F.N. Spon: 53-59

JAKEMAN, P. M. and B. DAVIES (1978). *The characteristics of a low resistance breathing valve designed for the measurement of high aerobic power.*

Brit. J. Sports Med. 11: 81-83

JONES, N. L., J. R. SUTTON, R. TAYLOR and C. J. TOEWS (1977). *Effect of pH on cardiorespiratory and metabolic responses to exercise.*

J. Appl. Physiol. 43(6): 959-964

JORDAN, L. and E. G. MERRILL (1979). *Relative efficiency as a function of pedalling rate for racing cyclists.*

J. Physiol. 296: 49-50P

JORFELDT, L., A. JUHLIN-DANFELT and J. KARLSSON (1978). *Lactate release in relation to tissue lactate in human skeletal muscle during exercise.*

J. Appl. Physiol. 44(3): 350-352

KAIJSER, L. (1970). *Limiting factors for aerobic muscle performance.*

Acta. Physiol. Scand. suppl. 346: 1-96

KARLSSON, J. (1971). *Muscle ATP, CP, and lactate in submaximal and maximal exercise.*

In: PERNOW, B. and B. SALTIN (Eds.), *Muscle metabolism during exercise.*

New York, Plenum Press: 383-393

KARLSSON, J. (1979). *Localized muscle fatigue: role of muscle metabolism and substrate depletion.*

In: HUTTON, R. and D. MILLAR (Eds.), *Exercise and Sports Science Reviews* Vol. 7

Philadelphia, Franklin Institute Press: 1-42

KARLSSON, J. and I. JACOBS (1982). *Onset of blood lactate accumulation during muscular exercise as a threshold concept.*

Int. J. Sports Med. 3(2):190-201

KARLSSON, J., L. NORDESJO, L. JORFELDT and B. SALTIN (1972). *Muscle lactate, ATP and CP levels during exercise after physical training in man.*

J. Appl. Physiol. 33(2): 199-203

- KATCH, V. (1974). *Body weight, leg volume, leg weight and leg density as determiners of short duration work performance on the bicycle ergometer.*  
Med. Sci. Sports 6(4): 267-270
- KATCH, V., A. WELTMAN, S. SADY and P. FREEDSON (1978). *Validity of the relative percent concept for equating training intensity.*  
Eur. J. Appl. Physiol. 39: 219-227
- KATZ, A. and K. SAHLIN (1988). *Regulation of lactic acid production during exercise.*  
J. Appl. Physiol. 65(2): 509-518
- KEEN, P. S., S. COLEMAN and T. HALE (1985). *The physiological demands of 4000m cycle pursuit racing. (abstract)*  
J. Sports Sciences. 3: 212-21
- KINDERMANN, W. and J. KEUL (1977). *Lactate acidosis with different forms of sports activities.*  
Can. J. Appl. Sports Sci. 2: 177-182
- KINDERMANN, W., G. SIMON and J. KEUL (1979). *The significance of the aerobic - anaerobic transition for the determination of work load intensities during endurance training.*  
Eur. J. Appl. Physiol. 42: 25-34
- KREBS, P. S., S. ZINKGRAF and S. J. VIRGILIO (1986). *Predicting competitive bicycle performance with training and physiological variables.*  
J. Sports Med. Phys. Fit. 26(4): 323-330
- KYLE, C. (1986) *Mechanical factors affecting the speed of a cycle.*  
In: BURKE, E. (Ed.), *Science of Cycling.*  
Champaign, IL: Human Kinetics: 123-136
- LA FONTAINE, T. P., B. R. LONDEREE and W. K. SPATH (1981). *The maximum steady state versus selected running events.*  
Med. Sci. Sports 13(3): 190-192
- LEGER, L. A., G. SELIGER, and L. BRASSARD (1980). *Backward extrapolation of  $\dot{V}O_{2max}$  from oxygen recovery curve in humans.*  
Med. Sci. Sports 12: 24-27
- LEMHANN, M., J. KEUL, G. HUBER and M. DA PRADA (1981). *Plasma catecholamines in trained and untrained volunteers during graduated exercise.*  
Int. J. Sports Med. 2(3): 143-147
- LINDSTEDT, S. L., D. J. WELLS, J. W. JONES, H. HOPPELER and H. A. THRONSON JR. (1988). *Limitations to aerobic performance in mammals: interaction of structure and demand.*  
Int. J. Sports Med. 9(3): 210-217

LOLLGEN, H., T. GRAHAM and G. SJOGAARD (1980). *Muscle metabolites, force, and perceived exertion bicycling at varying rates.*

Med. Sci. Sports 12(5): 345-351

MACDOUGALL, J. D., D. SALE, J. MOROZ, G. ELDER, J. SUTTON and H. HOWALD (1979). *Mitochondrial volume density in human skeletal muscle following heavy resistance training.*

Med. Sci. Sports 11(2): 164-167

MACLAREN, D. (1986). *Alkalinizers, hydrogen ions accumulation and muscle fatigue: a brief review.*

In: WATKINS, J., T. REILLY, L. BURWITZ (Eds.), *Proceedings of the VIII Commonwealth Conference on Sport (Sports Science).* London, E & F.N. Spon: 104-109

MADER, A. and H. HECK (1986). *A theory of the metabolic origin of anaerobic threshold.*

Int. J. Sports Med. 7:45-65

MADER, A., H. LIESEN, H. HECK, H. PHILIPPI, R. ROST, P. SCHURCH and W. HOLLMANN (1976). *Zur beurteilung der sportartspezifischen ausdauerleistungsfähigkeit im labor.*

Sportarzt Sportmed. 27: 80-88

MARION, G. A. and L. A. LEGER (1988). *Energetics of indoor track cycling in trained competitors.*

Int. J. Sports Med. 9(3): 234-239

MARTIN, B. J., S. ROBINSON, D. WEIGMAN and L. H. AULICK (1975). *Effect of warm-up on metabolic responses to strenuous exercise.*

Med. Sci. Sports 7: 146-149

MASSAGRANDE, A. (1982). *Agonistic Cycling. (Official coaching text of F.I.A.C)*

Milan, Landoni.

MCCARTNEY, N., G. HEIGENHAUSER and N. L. JONES (1983). *Effects of pH on maximal power output and fatigue during short-term dynamic exercise.*

J. Appl. Physiol. 55(1): 225-229

McENROY, J. D. S. and N. L. JONES (1975). *Arterialised capillary blood gases in exercise studies.*

Med. Sci. Sports 7: 312-315

McKENZIE, D.C., W. PARKHOUSE, E. RHODES, P. W. HOCHOCHKA, W. OVALLE, T. MOMMSEN and S. L. SHINN (1983). *Skeletal muscle buffering capacity in elite athletes.*

In: KNUTTGEN, H., J. VOGEL, J. POORTMANS (Eds.), *International series on Sport Sciences 13: Biochemistry of Exercise.*

Champaign Il. Human Kinetics: 584-489

McLELLAN, T. M. and J. S. SKINNER (1982). *Blood lactate removal during active recovery related to the aerobic threshold.*

Int. J. Sports Med. 3: 224-229

- McLELLAN., T. M. (1985). *Ventilatory and plasma lactate response with different exercise protocols: a comparison of methods.*  
Int. J. Sports Med. 6: 30-35
- MERTON, P. A., D. K. HILL and H. B. MORTON (1981). *Indirect and direct stimulation of fatigued human muscle.*  
In: PORTER, R. and J. WHELEN (Eds.), *Human muscle fatigue: physiological mechanisms.* Ciba Foundation Symposium 82  
London, Pitman Medical: 120-129
- NEWSHOLME, E. A. (1986). *Application of principles of metabolic control to the problem of metabolic limitations in sprinting, middle distance and marathon running.*  
Int. J. Sports Med. 7: 66-70 (Supplement)
- NOAKES, T. D. (1988). *Implications of exercise testing for prediction of athletic performance: a contemporary perspective.*  
Med. Sci. Sports 20(4): 319-330
- NONWEILER, T. (1957). *Power output of racing cyclists - wind tunnel tests at Cranfield (abstract).*  
Engineering 183: 586
- OSNES, J-B., and L. HERMANSEN (1972). *Acid-base balance after maximal exercise of short duration.*  
J. Appl. Physiol. 32(1): 59-63
- PARKHOUSE, W. S. and D. C. MCKENZIE (1984) *Possible contribution of skeletal muscle buffers to enhanced anaerobic performance: a brief review.*  
Med. Sci. Sports 16: 328-338
- PARKHOUSE, W.S., D. C. MCKENZIE, E. C. RHODES, D. DUNNING and P. WILEY (1982). *Cardiac frequency and anaerobic threshold. Implication for prescriptive training programs.*  
Eur. J. Appl. Physiol. 50: 117-123
- PARKHOUSE, W.S., D. C. MCKENZIE, and P. W. HOCHACHKA (1983). *The relationship between carnosine levels, buffering capacity, fibre types and anaerobic capacity in elite athletes.*  
In: KNUTTGEN. H., J. VOGEL and J. POORTMANS, (Eds.), *International series on Sport Sciences 13: Biochemistry of Exercise.* Champaign, Il. Human Kinetics: 275-278
- PERONNET, F., R. J. FERGUSON, H. PERRAULT, G. RICCI and D. LAJOIE (1981). *Echocardiography and the athletes heart.*  
Phys. Sports med. 9: 102-112
- PETTE, D. (1984). *Activity-induced fast to slow transition in mammalian muscle.*  
Med. Sci. Sports 16: 517-528
- POLLOCK, M. L. (1973). *The quantification of endurance training programmes.*  
In: WILMORE, J. (Ed.), *Exercise and Sports Science Reviews Vol. 1*  
New York, Academic Press: 155-188

POOLE, D. C. and G. A. GAESSER (1985). *Response of ventilatory and lactate thresholds to continuous and interval training.*  
J. Appl. Physiol. 58(4): 1115-1121

POORTMANS, J. R., J. DELESCAILLE-VANDEN BOSSCHE and R. LECLERCQ (1978). *Lactate uptake by inactive forearm during progressive leg exercise.*  
J. Appl. Physiol. 45(6): 835-839

PUGH, L. G. C. E. (1974). *The relation of oxygen intake and speed in competition cycling and comparative observations on the bicycle ergometer.*  
J. Physiol. 241: 795-808

RASMUSSEN, B. S., P. ELKJAER and B. JUHL (1988). *Impaired pulmonary and cardiac function after maximal exercise.*  
J. Sports Science 6: 219-228

ROBERTS, A. D., R. BILLETTER and H. HOWARD (1982). *Anaerobic muscle enzyme changes after interval training.*  
Int. J. Sports Med. 3(1): 18-21

ROTH, V., B. PANSFOLD, E. HASART, J. ZINNER, and B. GABRIEL (1981). *Zum informationsgehalt leistungs - diagnostischer parameter in abhlangigkeit von der zunahme der leistungsfahigkeit bei sportlern.*  
Medicin und Sport 21: 326-336

ROWELL, L. B. (1969). *Circulation.*  
Med. Sci. Sports 1(1): 15-22

SAHLIN, K. (1983). *Effect of acidosis on energy metabolism and force generation in skeletal muscle.*  
In: KNUTTGEN, H., J. VOGEL and J. POORTMANS, (Eds.), International series on Sport Sciences 13: Biochemistry of Exercise. Champaign, Il. Human Kinetics: 151-160

SAHLIN, K. (1978). *Intracellular pH and energy metabolism in skeletal muscle of man with special reference to exercise.*  
Acta Physiol. Scand. (suppl) 455

SAHLIN, K. and J. HENRIKSSON (1984). *Buffer capacity and lactate accumulation in skeletal muscle of trained and untrained men.*  
Acta Physiol. Scand. 122: 331-339

SAIKI, H. R. MARGARIA and F. CUTTICA (1967). *Lactic acid production in submaximal exercise.*  
Int. Z. Angew. Physiol. 24: 57-61

SALTIN, B. (1969). *Physiological effects of physical conditioning.*  
Med. Sci. Sports 1: 50-56

SALTIN, B. and P-O. ASTRAND (1967). *Maximal oxygen uptake in athletes.*  
J. Appl. Physiol. 23: 353-358

- SALTIN, B. and J. KARLSSON (1971a). *Muscle glycogen usage during work of different intensities.*  
 In: PERNOW, B and B. SALTIN (Eds.), *Muscle metabolism during exercise.*  
 New York, Plenum Press: 289-299
- SALTIN, B. and J. KARLSSON (1971b). *Muscle ATP, CP, and lactate during and after physical conditioning.*  
 In: PERNOW, B and B. SALTIN (Eds.), *Muscle metabolism during exercise.*  
 New York, Plenum Press: 395-399
- SALTIN, B., K. NAZAR, D. L. COSTILL, E. STEIN, E. JANSSON, B. ESSEN and P. D. GOLLNICK (1976). *The nature of the training response; peripheral and central adaptations to one-legged exercise.*  
*Acta Physiol. Scand.* 96: 289-305
- SCHEEN, A., J. JUCHMES and A. CESSION-FOSSION (1981). *Critical analysis of the anaerobic threshold during exercise at constant workloads.*  
*Eur. J. Appl. Physiol.* 46: 367-377
- SECHER, N. (1983). *The physiology of rowing.*  
*J. Sports Sciences* 1: 23-53
- SHARP, R. L., D. L. COSTILL, W. J. FINK and D. S. KING (1986). *Effects of eight weeks of bicycle ergometer sprint training on human muscle buffer capacity.*  
*Int. J. Sports med.* 7(1): 13-17
- SIESJO, B. K. and MESSETER (1971). *factors determining intracellular pH.*  
 In: SIESJO, B. K. and S. C. SORENSON (Eds.), *Ion homeostasis in the brain.*  
 New York, Academic Press: 244-262
- SJODIN, B. and I. JACOBS (1981). *Onset of blood lactate accumulation and marathon running performance.*  
*Int. J. Sports Med.* 2(1): 23-26
- SJODIN, B., I. JACOBS and J. KARLSSON (1981). *Onset of blood lactate accumulation and enzyme activities in M. vastus lateralis in man.*  
*Int. J. Sports med.* 2(3): 166-170
- SJODIN, B., I. JACOBS and J. SVEDENHAG (1982). *Changes in onset of blood lactate accumulation (OBLA) and muscle enzymes after training at OBLA.*  
*Eur. J. Appl. Physiol.* 49: 45-57
- SJOGAARD, G. (1984). *Muscle morphology and metabolic potential in elite road cyclists during a season.*  
*Int. J. Sports Med.* 5(5): 250-254
- SJOGAARD, G., B. NIELSEN, F. MIKKELSEN, B. SALTIN and E. R. BURKE (1982). *Physiology in bicycling.*  
 New York: Mouvement Publications.

- SOMERVILLE, K. A. and H. A. QUINNEY (1987). *A modified cycle ergometer.*  
Can. J. Sports Sci. 12(4): 225-228
- STAINSBY, W. N. (1986). *Biochemical and physiological bases for lactate production.*  
Med. Sci. Sports 18(3): 341-343
- STEGMANN H., W. KINDERMANN and A. SCHNABEL (1981). *Lactate kinetics and individual anaerobic threshold.*  
Int. J. Sports Med. 2: 160-165
- STEGMANN, H. and W. KINDERMANN (1982). *Comparison of prolonged exercise tests at the individual anaerobic threshold and the fixed anaerobic threshold of 4mmol.l<sup>-1</sup> lactate.*  
Int. J. Sports Med. 3(2): 105-110
- STROMME, S. B., F. INGJER and H. D. MEEN (1977). *Assessment of maximal aerobic power in specifically trained athletes.*  
J. Appl. Physiol. 42(6): 833-837
- SUZUKI, Y. (1979). *Mechanical efficiency of fast- and slow-twitch muscle fibres in man during cycling.*  
J. Appl. Physiol. 47(2): 263-267
- SVEDENHAG, J. and B. SJODIN (1984). *Maximal and submaximal oxygen uptakes and blood lactate levels in elite male middle- and long-distance runners.*  
Int. J. Sports Med. 5: 255-261
- SWAIN, D. P., R. J. COAST, P. S. CLIFFORD, M. C. MILLIKEN and J. STRAY-GUNDERSON (1987). *Influence of body size on oxygen consumption during bicycling.*  
J. Appl. Physiol. 62(2): 668-672
- TANAKA, K. and Y. MATSUURA (1984a). *Marathon performance, anaerobic threshold, and onset of blood lactate accumulation.*  
J. Appl. Physiol. 57(3): 640-643
- TANAKA, K., Y. MATSUURA, A. MATSUZAKA, K. HIRAKOBA, S. KUMAGAI, S. SUN and K. ASANO. (1984b). *A longitudinal assessment of anaerobic threshold and distance-running performance.*  
Med. Sci. Sports 16(3): 278-282
- TANAKA, K., T. YOSHIMURA, S. SUMIDA, R. MITSUZONO, S. TANAKA, Y. KONISHI, H. WATANABE, T. YAMADA and K. MAEDA (1986). *Transient responses in cardiac function below, at, and above anaerobic threshold.*  
Eur. J. Appl. Physiol 55: 356-261
- TESCH, P. (1980). *Muscle fatigue in man with special reference to lactate accumulation during short term intense exercise.*  
Acta Physiol. Scand. (suppl) 480

TESCH, P. A., D. S. SHARP and W. L. DANIELS (1981). *Influence of fibre type composition and capillary density on onset of blood lactate accumulation.*

Int. J. Sports Med. 2(4): 252-255

TRIVIDI, B. and W. H. DANFORTH (1966). *Effect of pH on the kinetics of frog muscle phosphofructokinase.*

J. Biol. Chem. 241: 4110-4114

VAN BAAK, M. A. and R. A. BINKHORST (1981). *Oxygen consumption during outdoor recreational cycling.*

Ergonomics 24: 725-733

VEICSTEINAS, A., M. SAMAJA, M. GUSSONI and P. CERRETELLI (1984). *Blood O<sub>2</sub> affinity and maximal O<sub>2</sub> consumption in elite bicycle racers.*

J. Appl. Physiol. 57(1): 52-58

WAGNER, P. D. (1988). *An integrated view of the determinants of maximum oxygen uptake.*

In: FODDE, F. R. and A. C. GONZALEZ (Eds.), *Oxygen transfer from atmosphere to tissues.*

Plenum, New York: 245-256

WASSERMAN, K., B. J. WHIPP, S. N. KOYAL and W. L. BEAVER (1973). *Anaerobic threshold and respiratory gas exchange.*

J. Appl. Physiol. 35(2): 236-243

WASSERMAN, K., W. L. BEAVER, J. A. DAVIS, J. PU, D. HEBER and B. J. WHIPP (1985). *Lactate, pyruvate, and lactate to pyruvate ratio during exercise and recovery.*

J. Appl. Physiol. 59(3): 935-940

WASSERMAN, K., W. L. BEAVER and B. J. WHIPP (1986). *Mechanisms and patterns of blood lactate increase during exercise in man.*

Med. Sci. Sports 18(3): 344-252

WELCH, H. G. and P. K. PEDERSON (1981). *Measurement of metabolic rate in hyperoxia.*

J. Appl. Physiol. 51: 725-731

WHITT, F. R. (1971). *A note on the estimation of the energy expenditure of sporting cyclists.*

Ergonomics 14(3): 419-424

WILBERG, R. B. and J. PRATT (1988). *A survey of the race profiles of cyclists in the pursuit and kilo track events.*

Can. J. Sports Sci. 13(4): 208-213

WILKIE, D. (1981). *Shortage of chemical energy as a cause of fatigue: studies by nuclear magnetic resonance and bicycle ergometry.*

In: PORTER, R. and J. WHELAN (Eds.), *Human muscle fatigue: Physiological mechanisms.* Ciba Foundation Symposium, No. 82. London, Pitman medical: 102-119

WILLIAMS, J. H., S. K. POWERS and M. K. STUART (1986).  
*Haemoglobin desaturation in highly trained athletes during heavy  
exercise.*  
Med. Sci. Sports 18(2): 168-173

WITHERS, R. T., W. M. SHERMAN, J. M. MILLER and D. L.  
COSTILL (1981). *Specificity of the anaerobic threshold in endurance  
trained cyclists and runners.*  
Eur. J. Appl. Physiol. 47: 93-104

YEH, M., R. M. GARDNER, E. D. ADAMS, F. G. YANOWITZ and R.  
O.CRAPO (1983). *Anaerobic threshold: problems of determination  
and validation.*  
J. Appl. Physiol. 55(4): 1178-1186

PART FIVE

APPENDICES

APPENDIX 1. GENERAL ANTHROPOMETRIC DATA FROM EXPERIMENT I

SUBJECT	AGE (years)	HEIGHT (m)	WEIGHT (kg)	$\dot{V}O_{2\max}$ ( $l \cdot \text{min}^{-1}$ )
1	28	1.85	83.5	6.75
2	21	1.92	75.4	4.45
3	20	1.93	76.6	5.93
4	50	1.82	77.6	----
5	20	1.77	73.1	5.33
6	24	1.73	62.7	4.05
7	22	1.84	76.2	5.95
8	23	1.91	74.3	4.48
9	18	1.89	72.4	5.22
10	21	1.79	69.4	4.52
11	19	1.83	82.8	5.24
$\bar{X}$	24.2	1.84	74.9	5.24
S.D	8.9	6.5	5.8	0.9

APPENDIX 2 BLOOD LACTATE LEVELS MEASURED AT 5 MINUTE INTERVALS DURING SUSTAINED EXERCISE AT THE POWER OUTPUT CORRESPONDING TO MSSL.

SUBJECT	MSSL POWER (watts)	BLOOD LACTATE CONCENTRATION (mmol.l <sup>-1</sup> )						mean
		-----time of sampling (minutes)-----						
		5	10	15	20	25	30	
1	375	2.18	2.23	2.48	2.30	2.90	2.63	2.47
2	235	4.70	5.00	5.26	5.20	5.21	4.57	4.88
3	327	3.35	3.59	3.74	3.65	3.34	3.46	3.45
4	203	3.65	3.10	2.91	2.84	2.63	2.51	2.80
5	309	3.08	3.23	2.91	----	2.74	2.95	2.98
6	195	3.61	3.83	4.21	4.16	4.15	4.35	4.02
7	297	2.57	2.87	2.85	2.92	2.63	2.76	2.77
8	245	4.80	5.75	6.09	5.66	5.65	5.58	5.58
9	297	3.91	4.16	4.00	3.86	4.19	4.50	4.10
10	265	3.99	4.09	----	4.13	4.01	3.97	4.02
11	318	3.38	3.52	3.79	4.20	4.24	4.23	3.85
$\bar{X}$	279	3.65	3.76	3.82	3.89	3.79	3.77	3.75
S.D.	55	0.7	1.0	1.1	1.0	1.0	1.0	0.9

BLOOD LACTATE LEVELS MEASURED AT 5 MINUTE  
INTERVALS DURING SUSTAINED EXERCISE AT ONE WORK  
LOAD ABOVE THE POWER OUTPUT CORRESPONDING TO MSSL.

SUBJECT	POWER (watts)	BLOOD LACTATE CONCENTRATION (mmol.l <sup>-1</sup> )					
		-----time of sampling (minutes)-----					
		5	10	15	20	25	30
1	390	2.80	4.71	5.60*			
2	250	5.00	6.75	8.32	8.95*		
3	336	3.76	4.81	5.95	7.20*		
4	203	4.08	4.48	5.19	5.24	5.70*	
5	318	3.58	4.27	4.98	5.65	6.32*	
6	210	3.36	4.23	4.57	4.47	5.32	5.75
7	308	3.87	4.78	5.02	5.25	5.46	6.00
8	260	5.50	7.72	8.77	9.65*		
9	310	4.17	4.80	5.60	7.45*		
10	280	3.55	4.10	4.95	6.06	-----	7.20
11	336	3.83	5.25	5.45	6.82	6.90*	
$\bar{X}$	291	3.96	5.08	5.85			
S.D.	57	0.7	1.1	1.4			

\* denotes that subject failed to complete the 30 minute test.

APPENDIX 3 GENERAL ANTHROPOMETRIC DATA FROM EXPERIMENT II

SUBJECT	AGE (years)	HEIGHT (m)	WEIGHT (kg)
1	21	1.76	73.1
2	18	1.70	67.8
3	20	1.93	80.4
4	18	1.82	76.6
5	20	1.80	73.7
6	18	1.74	66.7
7	20	1.83	79.5
8	19	1.79	78.5
9	29	1.85	82.7
$\bar{X}$	20.2	1.80	75.4
S.D.	3.5	0.07	5.7

APPENDIX 4      LONGITUDINAL EXERCISE DATA FOR SUBJECT 1.

-----Maximal data-----      -----4 mmol.l<sup>-1</sup> Lactate data-----

TEST DATE	WEIGHT Kg	VO <sub>2</sub> max l.min <sup>-1</sup>	HEART RATE	Ve l.min <sup>-1</sup>	POWER watts	HEART RATE	% VO <sub>2</sub> max	HR @ 400W	VO <sub>2</sub> @ 300W l.min <sup>-1</sup>
08.10.85	83.4	----	182	---	375	163	--	170	----
10.04.86	83.5	6.50	179	198	388	169	90	173	4.50
23.07.86	84.0	----	---	---	395	153	--	154	4.45
10.10.86	84.5	----	---	---	415	155	--	152	4.35
09.12.86	85.8	----	---	---	410	155	--	160	4.55
19.02.87	86.2	6.83	176	192	420	164	91	158	4.40
08.04.87	85.3	6.66	175	211	414	162	91	158	4.45
28.04.87	83.5	6.82	176	224	408	156	89	154	4.50
26.05.87	82.7	6.94	178	211	422	159	89	159	4.45
29.05.87	83.1	6.98	176	206	---	---	--	---	----
16.06.87	81.5	----	---	---	424	169	--	162	4.40
08.07.87	82.2	----	---	---	416	154	--	150	4.45
28.07.87	82.6	7.05	175	227	445	156	92	146	4.35
13.08.87	82.3	----	---	---	416	164	--	161	4.45
15.09.87	83.1	7.16	178	235	438	162	87	152	4.30
29.02.88	85.4	7.00	185	235	415	163	87	159	4.30
19.04.88	84.8	----	---	---	422	165	--	160	4.45
12.05.88	83.7	----	---	---	428	156	--	149	4.25
26.05.88	83.8	7.20	180	227	434	155	87	146	4.25
07.07.88	83.1	----	---	---	440	158	--	150	4.30
25.07.88	83.5	----	---	---	455	158	--	147	4.35
X	83.7	6.93	178	217	419	160	89	156	4.39
S.D.	1.2	0.2	3	15	19	4.9	1.9	7.4	0.08

APPENDIX 5    LONGITUDINAL EXERCISE DATA FOR SUBJECT 2.

TEST DATE	WEIGHT Kg	-----Maximal data-----			-----4 mmol.l <sup>-1</sup> Lactate data----				
		VO <sub>2</sub> max l.min <sup>-1</sup>	HEART RATE	Ve l.min <sup>-1</sup>	POWER watts	HEART RATE	% VO <sub>2</sub> max	HR @ 300W	VO <sub>2</sub> @ 300W l.min <sup>-1</sup>
12.02.86	82.5	4.94	200	162	292	182	85	182	4.35
21.05.86	82.8	5.48	198	181	318	169	79	164	4.30
11.08.86	81.7	5.50	192	186	346	168	86	155	4.20
29.09.86	82.8	5.30	192	165	330	173	89	163	4.30
18.01.87	80.0	5.90	197	160	350	170	83	154	4.20
25.03.87	79.2	5.50	197	155	322	170	82	162	4.15
22.07.87	79.5	6.01	197	180	380	171	86	150	4.20
15.11.87	82.0	5.85	208	183	356	181	84	164	4.20
26.01.88	82.7	6.32	202	194	360	171	78	149	4.20
12.04.88	82.5	6.50	195	186	382	171	82	150	4.25
24.05.88	83.5	----	---	---	382	165	--	143	4.15
11.07.88	81.0	6.44	196	186	382	166	80	142	4.20
21.11.88	80.6	----	---	---	352	169	--	153	----
07.03.89	80.5	6.32	198	192	352	165	77	150	4.15
X	81.5	5.84	198	177	350	171	83	156	4.22
S.D.	1.4	0.5	4.5	13	27	5	3.6	10	0.06

APPENDIX 6    LONGITUDINAL EXERCISE DATA FOR SUBJECT 3.

TEST DATE	WEIGHT Kg	-----Maximal data-----			-----4 mmol.l <sup>-1</sup> Lactate data-----				
		VO <sub>2</sub> max l.min <sup>-1</sup>	HEART RATE	Ve l.min <sup>-1</sup>	POWER watts	HEART RATE	% VO <sub>2</sub> max	HR @ 250W	VO <sub>2</sub> @ 300W l.min <sup>-1</sup>
07.02.86	73.6	5.03	208	162	240	182	73	190	4.35
18.04.86	73.2	5.25	208	180	270	192	78	185	4.40
28.06.86	72.1	5.45	208	187	304	194	79	176	4.25
15.10.86	75.1	4.64	206	168	268	182	86	178	4.35
12.12.86	74.9	5.27	206	182	298	180	83	166	4.40
20.02.87	74.5	5.31	203	182	306	180	85	164	4.40
12.05.87	72.9	5.30	202	185	312	181	87	162	4.45
02.07.87	73.0	5.30	205	190	302	180	82	163	4.45
18.02.88	73.4	5.00	205	168	310	183	88	165	4.30
08.07.88	71.1	5.60	205	182	325	180	84	158	4.35
23.02.89	72.1	5.60	208	184	330	185	86	162	4.35
X	73.2	5.25	206	179	297	183	83	170	4.36
S.D.	1.2	0.3	2.2	9.0	27	5	4.5	11	0.07

