

## All rights reserved

## INFORMATION TOALLUSERS

The quality of this reproduction is dependent upon the quality of the copy submitted.
In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.


Published by ProQuest LLC (2017). Copyright of the Dissertation is held by the Author.

All rights reserved
This work is protected against unauthorized copying under Title 17, United States Code Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346

Ann Arbor, MI 48106-1346

# An examination of the control of endurance training, using heart rate and blood lactate analysis. 

## G. M. Joe Dunbar BSc (hons)

Submitted to the Robens Centre for Health Ergonomics, EIHMS, University of Surrey for the degree of Master of Philosophy

April 2000
© G.M. Joe Dunbar


#### Abstract

The purpose of this study was to examine the control of endurance training, using heart rate (HR) and blood lactate (La) analysis. This was done using three experiments covering the sports of endurance running, rowing and triathlon. The first aimed to establish a time efficient, incremental protocol for determining blood lactate profiles. The second examined control of training using running speed or HR in the laboratory and field. The third compared lactate threshold (Tlac) HRs between cycling and running in triathletes.

A continuous 4 minute incremental (4I) protocol was compared with discontinuous 6 and 8 minute protocols. ANOVA with repeated measures revealed that there was no significant difference in HR and La measurements between the protocols. Subsequently, 4 I was compared with a similar 3 minute (3I) version. Despite higher HR and La noted during the later stages of 4 I , the HR - La relationship was unaffected by the protocol used. The 3I is a suitable, time efficient method of assessment.

HR prescribed from 3I was compared with running speed to control training at maximal steady state blood lactate (MSS) in well-trained runners. HR appeared the better means of training control, where increased lactate within training sessions was less frequent than in the speed controlled sessions. The process of HR control during MSS training in the laboratory was also examined in elite junior rowers. In 9 out of 10 sessions MSS was not exceeded. A total of 80 other training sessions were also analysed. Thirty training sessions were performed by trained runners and 50 by trained rowers. Thirty four sessions were aimed at base endurance (BE) and 46 aimed at MSS intensity. In 20\% of cases, athletes exceeded their prescribed HR. In all, $96 \%$ of the sessions were predicted correctly as either steady state or non-steady state on the basis of observed HR. HR from 3I was deemed an acceptable means of intensity control to avoid non-stable La.

HR at Tlac and 2 mmol. $1^{-1}$ of blood La during 3I of both running and cycling exercise was compared in well-trained triathletes. In both cases mean HR was higher during running ( $\mathrm{t}=7.6$, d. $\mathrm{f}=15, \mathrm{p}<0.001$ and $\mathrm{t}=7.6$, d. $\mathrm{f}=15, \mathrm{p}<0.05$, respectively). The mean difference in HR at Tlac was $13.4 \mathrm{~b} \cdot \mathrm{~min}^{-1}$ with a range of 0 to 26 . Separate tests should, therefore, be used for each mode of exercise in triathletes.

In summary, it was concluded that a 3 minute incremental protocol is valid for the determination of blood lactate profiles and the prescription of $H R$ for subsequent training prescription. Also, HR can predict blood lactate conditions during training sessions in well-trained runners and rowers. The HR for set training zones is likely to vary according to the mode of exercise employed.


The Control of training has fascinated me for some time. Yet, at the age of sixteen I was lazily under-performing academically and, in hindsight, mixing with the wrong company. By the age of seventeen I had started running and this was taken seriously from the first day that I trained with a coach at my local athletics track. From that day I stopped smoking cigarettes and trained on a daily basis in commitment to that coach. Within a few years I had competed internationally and run a mile in under four minutes. Yet the influence of that coach went further than my performances on the athletics track. Many car journeys were spent discussing training philosophy, preparation for competition and running in general. The self-discipline instilled in my running also filtered through to other circles. I studied my A-levels, read a sport science degree and ended up working in a sports science department, before working as a consultant in sport.
There is never a "right time" to explain to that coach what a positive influence he had on both my running and my professional work. Perhaps it is for this reason that I dedicate this thesis to George Harrison, with thanks for all he has done for me over the last 15 years.

## AKNOWLEDGEMENTS

The writing of a postgraduate thesis must seem the most selfish thing in the world to the family living with the author. I gratefully thank those living with me for their tolerance and patience during these past years.

My parents supported me through college and after; naturally I offer them thanks and love for this unequivocal support.

Dr. John White supervised my undergraduate thesis and years later agreed to guide me during this one. I cannot remember a day when I could not speak to him on the telephone for expert advice, but always in a calm and reassuring manner. As always, I am indebted to him in helping me get through.

Professor Stubbs, similarly has always been there, probably frustrated with the somewhat intermittent nature of my contact. I am grateful for his continued patience and, of course, his continued support.

Richard Fisher brought me to St. Mary's and gave me the opportunity to develop a career and start this research. I am grateful to him and the Sport Science department at that institute for their role in the early stages.

Naturally none of this would have taken place if athletes were not trying to improve performances. Therefore, I thank not only all of the subjects involved in this study, but also any other athlete I have worked with. It is continued work in the field that helps to enhance my understanding of preparation for competition.

## List of Contents

Page
Abstract ..... 2
Dedication ..... 3
Acknowledgements ..... 4
List of Tables ..... 7
List of Figures ..... 8
Glossary of terms ..... 9
Chapter One: Introduction and overview ..... 10
Chapter Two: Review of Literature ..... 14
Chapter Three: Statement of the Problem and Purpose ..... 42
Null Hypotheses ..... 43
Chapter Four: Experiment One: Establishing a lactate profile ..... 44 for subsequent training prescription.
Chapter Five: Do heart rates in training workouts for Base Endurance ..... 57
and Maximal Steady State, elicit blood lactate values predicted from blood lactate profiles in (i) runners and(ii) rowers, in the laboratory and the field?
Chapter Six: How do blood lactate profiles vary between running and ..... 82 cycling in triathletes?
Chapter Seven: General summary and practical implications ..... 90
Chapter Eight: Further Research ..... 103
References ..... 106
Appendices
Appendix One: Pre-test questionnaire ..... 125
Appendix Two: Form of Informed Consent ..... 126
Appendix Three: Dietary recall sheet ..... 127
Appendix Four: Training Log ..... 128
Appendix Five: Abstract: The effects of different protocols on heart rate and lactate values during graded exercise tests for middle distance runners. (Journal of Sports Sciences (1995) 13:25-26) ..... 130
Appendix Six: Abstract: A comparison of running speed and heart rate to control training at threshold intensity (Journal of Sports Sciences (1997) 15: 47-48) ..... 131
Appendix Seven: Abstract: Training prescription for junior rowers using blood lactate and heart rate (Journal of Sports Sciences (1997) 15: 47) ..... 133

## List of Tables

2.1 The relative contribution (\%) of different energy sources to maximal ..... 14 exercise, utilising large muscle groups over different durations.
2.2 Some one mile running performances and $\stackrel{\vee}{\mathrm{V}}_{2_{\text {max }}}$ values measured ..... 15 this century (adapted from Snell, 1990)
2.3 A description of different methodological protocols ..... 32
4.1 Physical characteristics of subjects in Experiment One ..... 46
4.2 Mean heart rate and blood lactate values obtained in each protocol ..... 49
4.3 Correlation of blood lactate and heart rate values between different ..... 49 protocols
4.4 Mean blood lactate and heart rate values obtained in the 3 and 4 minute ..... 50 protocols
4.5 Mean heart rate at $2 \mathrm{mmol} \mathrm{l}^{-1}$ of blood lactate and lactate threshold in ..... 52 each protocol
5.1 Federation Internationale se Societes d'Aviron (FISA) guidelines for ..... 58 prescription of training intensities (Adapted)
5.2 Physical characteristics of subjects in studies one and two ..... 61
5.3 Physical characteristics of subjects in study three ..... 61
5.4 Mean heart rate and blood lactate values during MSS sessions ( $\mathrm{N}=10$ ) ..... 65
5.5 Mean heart rate, blood lactate and distance rowed through E25 sessions ..... 67 ( $\mathrm{N}=7$ )
6.1 Physical characteristics of subjects ( $\mathrm{N}=16$ ) ..... 83
6.2 Mean heart rate at 2 and lactate threshold during running and cycling ..... 85 tests ( $\mathrm{N}=16$ )

## List of Figures

2.1 IAT as determined by Stegmann et al., (1981) 24
2.2 Calculation of MSS according to La Fontaine et al., (1981) 26
2.3 Lactate Turnpoint as described by Noakes et al., (1991) 27

| 2.4 Change in position and shape of a lactate curve after 4 months of | 35 |
| :--- | :--- |
| endurance training in a junior rower |  |

4.1 Example of a lactate profile, showing HR at 2 mmol. $1^{-1}$ of blood lactate ..... 48
and at lactate threshold.
4.2 Relationships of (a) blood lactate and (b) heart rate values between 3 ..... 50 and 4 minute protocols
4.3 Levels of agreement for (a) blood lactate (b) heart rate values between ..... 51 3 and 4 minute protocols
4.4 Levels of agreement for the heart rate - blood lactate relationship at (a) ..... 52 $2 \mathrm{mmol} . \mathrm{l}^{-1}$ and (b) lactate threshold
5.1 Mean HR and blood lactate values during MSS sessions ( $\mathrm{N}=10$ ) ..... 65
5.2 A comparison of predicted and observed blood lactate response during ..... 66
(a) T 25 sessions and (b) F 25 sessions ( $\mathrm{N}=10$ )
5.3 Mean blood lactate and distance rowed during the E25 sessions ( $\mathrm{N}=7$ ) ..... 68
5.4 A comparison of observed and predicted blood lactate responses during ..... 69 training sessions in runners ( $\mathrm{N}=30$ )
5.5 A comparison of observed and predicted blood lactate responses during ..... 70 training sessions in rowers ( $\mathrm{N}=50$ )
6.1 Relationships between heart rate in running and cycling at (a) 2 ..... 86 mmol. $\mathrm{I}^{-1}$ and (b) Tlac during incremental tests ( $\mathrm{N}=16$ )
6.2 Histogram demonstrating the range of difference in heart rate at Tlac ..... 86

## Glossary of Terms

The field of study of the blood lactate response to exercise is a worldwide phenomenon, with a plethora of protocols, methodologies and terminology. For clarity within this document, the following terminology is adhered to.

Base Endurance (BE). A training intensity commonly employed by endurance athletes. Typically the work is of long duration and low intensity, where the blood lactate concentration remains similar to resting values. The upper limit of such a training zone in this research is determined by the first rise in blood lactate above baseline levels during incremental work (previously referred to as lactate threshold by Weltman (1995).

Blood lactate profile. The plotting of blood lactate values in response to an incremental test. The values may be plotted against intensity and / or heart rate. Values are joined by linear interpolation.

Lactate threshold (Tlac). The point during incremental testing where a sharp increase in blood lactate is witnessed in response to an increase in intensity and / or heart rate. It is suggested that this intensity represents steady state blood lactate during continuous exercise.

Maximal steady state (MSS). A key intensity for endurance training, where blood lactate values are high but stable during continuous exercise (also noted as MLaSS).

Observed steady state lactate. The criteria for stable blood lactate values in this document are where there the increase was no greater than $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$ through a training session or 25 minute trial.

Observed non-steady state lactate. Where blood lactate increased by more than 0.5 mmol. $1^{-1}$ through a training session or 25 minute trial.

### 1.0 Introduction and Overview

### 1.1 Background

World sport becomes more competitive with each and every year. Not only are more nations participating in sport, the contribution from developing nations continues at a pace. This is particularly the case in the world of endurance athletics, where runners from an increasing number of African nations have become more successful at major championships. In the inaugural World Athletics Championships in Helsinki in 1983, it was European athletes who dominated events like the 1500 m . Of the entries for that event only 4 runners had run the distance in under 3:34. In the 1999 Championships in Seville, African athletes won gold and silver and 31 of the 44 entrants had run inside 3:34. Thus the number of competitors achieving sporting excellence is rapidly increasing.

Such improvement in the standard of competition continues in other sports at the same time. In rowing, medals are now won at World Championship level by nations such as Croatia, Slovenia, Ukraine, Belarus and Russia. Such nations did not exist ten years ago; indeed the break up of the former Soviet Union means that many more nations are serious competitors at the top level. The sport of triathlon is still developing both at an elite and mass participant level. It is one of the newest Olympic Sports, first included in the Games in Atlanta 1996. The standard, is thus still improving.

Training for international sport is serious business, most competitors who make World Championship finals train exclusively as full time athletes. The investment in time and the personal sacrifice is great. However, success is not simply a result of training hard. It has been established that athletes can train too hard to attain optimal improvements in physical fitness and competitive performance (Hartmann et al., 1990). This means that training needs some element of control, whether from the advice of a coach, or the intervention of sport science.

Sport science cannot provide all of the answers and there are more aspects to this study than physiology alone. However, the introduction of rapid assay blood analysers has
allowed the determination of blood lactate concentrations during training sessions. Portable heart rate telemetry devices allow the athlete and coach to monitor heart rate during sessions and review the intensity and duration of training sessions on a personal computer with great accuracy straight after the event.
Such information was not available to the likes of Sir Roger Bannister the first man to break 4 minutes for the mile in the 1950's. Indeed it is only in the last 10 to 15 years that this technology has started to influence training methods and regimes. However, despite volumes of research examining concepts such as anaerobic threshold and the lactate response to exercise, there is very little advice available to the athlete and coach as to how to use parameters such as heart rate and blood lactate to improve fitness in an optimal fashion.

### 1.2 Overview

Although the present study cannot provide all the answers to questions about the use of heart rate and blood lactate to positively influence the training process, it is applied in nature and is aimed at improving the understanding of how training can be controlled to help the athlete and coach. In reality, it will merely end by asking more questions, which in turn need further research, but shall establish some small ground rules along the way. The series of studies examines control of training in a range of sports that interest the author. Thus examination in the sports of running, rowing and triathlon are included. Although individually different in nature, the three sports all require the same basic training principles in preparation for competition. They are all predominantly endurance sports, with much of the conditioning aimed at improving aerobic endurance. It is the development of this particular feature, by a variety of means that is the focus of this work.

The principle of controlling training using heart rate and or blood lactate measurements is not new (Janssen 1987, Weltman, 1995). However, where blood lactate kinetics are involved, there is wide controversy, due to a plethora of definitions, terminology and populations used in empirical research. Such controversy is unearthed in the following review of literature.

The series of studies in this present research starts from the beginning, by examining different protocols for determining blood lactate profiles. Such profiles are used for both the monitoring of training (Jacobs, 1986) as well as the prescription of training intensities.

An appropriate incremental protocol was established for the construction of a blood lactate profile in Experiment One. The analysis moved on to assessing different methods of controlling training intensity in Experiment Two examining subject samples from both rowing and running. An in depth analysis of some 80 training sessions using heart rate training zones prescribed from incremental protocols was achieved in the third of a series of studies in this experiment.
Finally, Experiment Three examined differences in training heart rates between running and cycling in triathlon competitors.
The three experiments are reported and discussed independently, with an overall discussion and practical implications completed at the end. This shows, in part, the developmental nature of the work, as both Experiments One and Two contain two and three studies, respectively. Naturally, along the way various factors requiring further research were established and these too are summarised at the end of the thesis.

### 1.5 Delimitations

- All subjects in the study were from a homogenous group: well-trained, of at least county standard, including many internationals.
- The study focussed on entirely physiological aspects of training, although it is accepted that other factors such as technique, biomechanics and psychology all have an important role to play in training and competition.
- Indoor running trials took place on a running machine and indoor rowing trials on an rowing ergometer to accurately standardise the workload.
- In rowing trials individual athletes, rather than boat crews were analysed.
- Only Base Endurance and MSS workouts were analysed, but it is accepted that athletes also use other training intensities in preparation for competition.
- Steady state lactates were the aim in MSS sessions, although it is yet to be unequivocally established that this is indeed optimal for improvement of endurance.
1.6 Limitations
- Although rowing ergometers and running machines do replicate the respective sporting activities, it is noted that technique may vary in the laboratory setting from that in the normal training environment.
- Only young, highly trained, male athletes have been examined in this study, thus the implications from this homogenous sample cannot necessarily be related to a heterogeneous population.
- Blood lactate can be influenced by a number of factors including dietary manipulations and changes in the body's acid base balance prior to exercise. Although diet and training were logged prior to testing, it was not possible to control these directly and the study relies on the honesty and accuracy of the logging by the athletes.


### 2.0 Review of Literature

### 2.10 Energy Systems in relation to sport

Performance of endurance sport requires a great contribution from aerobic metabolism. Indeed, the relative contribution from various energy systems depends on the interplay of both the duration and intensity of exercise. A general overview of how the duration of maximal exercise (presumably the intensity normally associated with the race scenario) has been documented by $\AA$ Åstrand and Rodahl (1986) and is adapted in Table 2.1.

Table 2.1: The relative contribution (\%) of different energy sources to maximal exercise. utilising large muscle groups, over different durations.

| Exercise Time, Maximal Effort |  |  |  |  |  |  |  |  |
| :--- | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Process | 10 sec | 1 min | 2 min | 4 min | 10 min | 30 min | 60 min | 120 min |
|  |  |  |  |  |  |  |  |  |
| Anaerobic | 85 | $65-70$ | 50 | 30 | $10-15$ | 5 | 2 | 1 |
| Aerobic | 15 | $30-35$ | 50 | 70 | $85-90$ | 95 | 98 | 99 |

This shows that the performance of middle to long distance running (one mile and above) or regatta rowing 2000 m ( 6 to 8 minutes depending upon boat type and ability) are fuelled by a predominantly aerobic energy supply. Training performances at a lower intensity over any of the durations noted in Table 2.1 above, require an increased contribution from aerobic processes.

### 2.20 Aerobic Metabolism

The early classic work of Hill and co-workers some $70-80$ years ago represents the start of a huge wave of research into muscle physiology, energetics and biochemistry (Hill, 1913; Hill and Lupton, 1923; Hill, Long and Lupton, 1924). The original findings from their collection of expired air into Douglas Bags, carried on the back of subjects running around a running track at a variety of speeds, established a number of key physiological phenomena (Hill and Lupton, 1923).

### 2.21 Maximum Oxygen Uptake

Not only had they established that oxygen consumption increased in relation to increased muscular work, they also found that there is a point beyond which no increase in oxygen consumption occurs. This maximum level of oxygen consumption ( $\mathrm{VO}_{2 \text { max }}$ ) has been known for years to be a key variable, indeed a predictor of success of, performance in endurance sport. It is perhaps this alone, that has led to the index being used as a gold standard in the assessment of human performance. High values in elite athletes have been seen since the 1930's, where values in excess of $80 \mathrm{ml} . \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}$ have been witnessed in champion athletes (Robinson, Edwards and Dill, 1937). Yet, while performances in easily measurable endurance events, such as distance running, have been seen to improve dramatically in subsequent years (See Table 2.2), there has been little change in the reported values of $\mathrm{VO}_{2 \text { max }}$ in the current athlete.

Table 2.2: Some One Mile Running Performances and $\dot{\mathrm{V}}_{\mathbf{O}_{\text {max }}}$ values measured this century (adapted from Snell, 1990)

| Athlete | Year | Best Mile <br> Time | $\dot{\mathrm{VO}_{2 \text { max }}}$ <br> $\mathrm{ml} . \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}$ | Reference |
| :--- | :---: | :---: | :---: | :--- |
|  |  |  |  |  |
| Archie San |  |  |  |  |
| Romani | 1937 | $4: 07.2$ | 74.2 | Dill et al. (1967) |
| Don Lash | 1937 | $4: 07.5$ | 81.5 | Dill et al. (1967) |
| John Landy | 1954 | $3: 57.9$ | 76.6 | Åstrand (1955) |
| Peter Snell | 1962 | $3: 54.1$ | 72.3 | Carter et al., (1966) |
| Jim Ryun | 1966 | $3: 51.3$ | 81.0 | Daniels (1974) |
| Steve Scott | 1977 | $3: 47.7$ | 80.1 | Conley et al. (1984) |
| Steve Cram | 1985 | $3: 46.36$ | 82 | Personal Communication |

However, the process of measuring $\mathrm{VO}_{2 \text { max }}$ has been a valuable tool in categorising and monitoring the training responses of elite endurance athletes (Dunbar and Faulmann, 1996; Svedenhag and Sjödin, 1984: Svedenhag and Sjödin, 1985).
It has long been established that $\mathrm{VO}_{2 \text { max }}$ values vary with activity, age, gender, genetics and training status. Mode of activity is important, as this represents the musculature
involved in overall activity. Without doubt, whole body exercise using the major muscle groups is responsible for eliciting the highest $\dot{\mathrm{VO}}{ }_{2 \text { max }}$ values, due to both central and peripheral considerations.
The central factors are associated with the supply of blood and, therefore oxygen, to the working musculature. Specifically cardiac output (the product of heart rate and stroke volume) is the key central factor and it is indeed the limitation of this factor that can lead to a plateau effect in many, but not all, $\mathrm{VO}_{2 \text { max }}$ tests (Shephard, 1992; Noakes, 1988).
Peripheral factors such as capillary supply (and more importantly the capillary to muscle fibre ratio), number and size of mitochondria and the level of certain aerobic enzymes (citrate synthase and succinate dehydrogenase) in muscular tissue, all greatly influence the rate of oxygen extraction at tissue level. There is debate as to whether central or peripheral factors are responsible for the limitation to $\dot{\mathrm{V}}_{2_{\text {max }}}$, but Noakes favours the influence of peripheral factors.
A review of $\mathrm{VO}_{2 \text { max }}$ values found in elite competitors of various sports and activities, gives an indication of the influence of mode of exercise. It can be seen that sports such as cross country skiing and middle and long distance running are frequently the sports boasting competitors with the highest values when expressed as a ratio of bodymass. Slightly below this would be rowing and cycling, where the bodymass is supported during the activity. Performers of field games such as hockey and soccer tend to show more modest values. (Åstrand \& Rodahl, 1986; Shephard, 1992). Despite whole body activity in these last examples, the nature of the games do not require a sustained high level of activity as would be seen in racing sports.
Age is another factor that has an influence on $\dot{\mathrm{V}}_{2^{m a x}}$ values and similarly on performance. Typically $\mathrm{VO}_{2 \text { max }}$ values are highest between the ages of $20-30$ years in an untrained population. After these years there is a steady decline in $\stackrel{\vee}{\mathrm{V}}_{2}$ mas with increasing age, so a 65 year old male may typically have a value that is $70 \%$ of that for a 25 year old (Åstrand \& Rodahl, 1986). This can in part be attributed to a decrease in activity with age, but is also explained by a decrease in maximal heart rate (Green and Crouse, 1993), which has detrimental implications for cardiac output.
It is also widely documented that women tend to have lower $\mathrm{VO}_{2 \text { max }}$ values than men. On the face of it, when expressed as a ratio of bodymass, it may appear that this is simply due to the greater level of adipose tissue that women bear and the lower overall mass. However when the bodymass factor is removed, by expressing the $\mathrm{VO}_{2 \text { max }}$ value to the
power $2 / 3$, differences still occur. Most likely, this is accounted for by the lower haemoglobin concentration of women (Åstrand \& Rodahl, 1986), thus reducing the oxygen supply to the tissues.
Although in the untrained person, changes in $\mathrm{VO}_{2 \text { max }}$ can be quite considerable, in the order of $10-20 \%$, (Shephard, 1992), in the highly trained athlete, increases can be rare, despite further improvements in performance (Barbeau et al., 1991; Svedenhag and Sjödin, 1985). Clearly there is a strong genetic component setting an individual ceiling that an individual can achieve with training (Klissouras, 1971). Furthermore, in a heterogeneous population the relationship between $\stackrel{V O}{2}_{2 \text { max }}$ and running performance is that of a strong correlation, whereas when a homogenous group of people is analysed, such as elite athletes, such correlation no longer exists. (Table 2.2).
Despite being an important physiological variable, the implication of the above is that other factors are indeed responsible for success in endurance events. More recent research has attempted to understand the aforementioned improvements in performance despite stable $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ values.

### 2.22 Aerobic Capacity and Running Economy

It had already been established during the work of Hill (1924), that submaximal oxygen consumption varies with running speed or work intensity, yet in the 1970's and 1980's the implications of this had just begun to be fully realised.
It has been noted that the submaximal oxygen cost of running at certain speeds varies amongst subjects. Conley and Krahenbuhl (1980) examined elite athletes in terms of both physiology and performance, in this case $10-\mathrm{km}$ racing. They found that oxygen consumption at set submaximal speeds, also termed running economy, correlated reasonably well with race performance ( $r=0.79-0.83$ ). Similarly in examining marathon runners of a similar standard, Sjödin and Svedenhag (1985) found variation in running economy at submaximal intensities. Being able to utilise a high percentage of $\dot{\mathrm{V}}_{2}{ }_{2 \text { max }}$ for long durations is clearly more advantageous to performance than simply possessing a high $\dot{V}_{O_{2 ~ m a x ~}}$ value (Costill et al., 1973). The physiological adaptations that allows this higher fractional utilisation of oxygen consumption are predominantly confined to the specific musculature involved in work, particularly the capillary network; but the factor that is most responsible for dictating the highest speed to be maintained at a steady state is the anaerobic threshold.

### 2.23 The Anaerobic Threshold

The anaerobic threshold (AT) was first determined from respiratory values (Wasserman et al, 1973), and more recently the blood lactate response to exercise (Jacobs, 1986; Weltman, 1995).
There are few areas in the field of sports physiology that have attracted such controversy and debate as the lactate response to exercise and the associated development of various threshold concepts. This is a result of a variety of factors; difference in terminology for the same or similar physiological events, varied protocols used in assessment, a large variety of samples used in research papers, and a wide range in methodology.
However, there is a wealth of literature to support the fact that AT and the blood lactate response to incremental exercise are far better than $\dot{\mathrm{VO}}_{2 \text { max }}$, or indeed the fractional utilisation of this variable, in predicting endurance performance in running (Duggan and Tebbutt, 1990; Farrell et al., 1979; Fay et al., 1989; Gass, McLellan and Gass, 1991; Jacobs, 1986; Lehmann et al., 1983; Maffulli et al., 1991; Sjödin and Jacobs, 1981; Tanaka and Matsuura, 1984), and rowing (Di Pampero et al., 1971; Doherty et al., 1994; Vermulst et al., 1991; Womack et al., 1992).
The term AT was first introduced by Wasserman and McIlroy (1964), when describing the onset of metabolic acidosis, not in a sporting population, but in a group of patients with heart disease. It is the future variety in sample populations that was to cause some complication through the literature in future years. Volumes of research have been published on the topic, but different samples have been used as subjects. This has, at times led to conclusions being drawn from research on some studies, being applied to completely different sample groups. It is not, therefore, surprising that conflicting opinions have developed.
The early work of Wasserman and McIlroy (1964) linked measurements of expired air (in particular carbon dioxide and respiratory quotient) to arterial blood lactate concentrations. They used the point of a non-linear increase in ventilation, with respect to an increase in oxygen consumption, as the AT. Later the theory was refined to the argument that at some point during incremental exercise, there is insufficient oxygen supplied to active musculature, to cope with the demand aerobically. This imbalance was purported to accelerate the conversion of pyruvate from glycolysis, to lactate. The required increase in bicarbonate buffering was assumed to lead to the production of carbon dioxide, over and above that of normal carbon dioxide production that was
associated with aerobic metabolism. At this point of increased carbon dioxide production, the anaerobic threshold was said to exist (Wasserman et al., 1973), a term that was rapidly adopted internationally. It is pertinent to that note the AT was associated with the level oxygen consumption where there is an insufficient supply of oxygen to the working muscles.
It is understandable why such a concept was so appealing at the time. It must be remembered that during this era, there was no method for rapid determination of blood lactate that gave suitable precision; thus a suitable non-invasive method of detecting changes within the muscle was useful.
In the few years preceding the work of Wasserman and co-workers, modern research had already begun examining the blood lactate response to exercise. In a historical review, Hollman (1985) published details of the work that had been carried out in Germany up to the year of 1966. In fact work had started in this area as early as the late 1950's. This work, too, had noted that during incremental exercise a breakpoint exists, where ventilation rate increases at a greater rate to oxygen consumption. At this stage, it was thought that this shift was related to the increase in blood lactate production. The term used by these authors was the point of optimum ventilatory efficiency, to define the oxygen uptake which could be supplied by exclusively aerobic metabolism.
The work of Wasserman et al. (1973) was supported by further work of Davis et al. (1976), who also found high correlation ( $\mathrm{r}=0.95$ ) between respiratory markers of the AT and blood lactate measurements, in three modes of exercise, namely arm cranking, cycling and treadmill walking / running.
A similar correlation $(r=0.87)$ between AT and lactate threshold was found a few years later by Yoshida et al. (1981), who investigated responses to incremental bicycle exercise.
In the 1980's considerable controversy surrounded the debate as to whether respiratory measures do indeed accurately predict events occurring within the muscle. Heated exchanges took place between Davis (1985a; 1985b) and Brooks (1985a; 1985b). Brooks argued that the AT concept was too simplistic and dismissed the AT concept on the following three grounds.

1. Muscle tissue is not hypoxic during sub-maximal exercise. Brooks (1985a) cited work by Pirnay et al. (1972) who found that during maximal exercise, the femoral PvO 2 did not fall below 10 Torr and that during submaximal exercise of a level of $50 \%$
$\dot{\mathrm{VO}}_{2 \text { max }}, \mathrm{PvO} 2$ was between 20 and 40 Torr. This was evidence that ample oxygen is available at the tissue bed and an anaerobic state does not exist.
2. The theory states that an increase in lactate concentration results in an increase in minute ventilation. However, examination of subjects suffering glycogen depletion show this not to be the case. Segal and Brooks (1979) observed that much lower lactate levels, yet higher minute ventilation values occurred in glycogen depleted subjects. Also adding doubt to the concept was reference to Hagberg's analysis of McArdle's Syndrome patients (Hagberg, 1982). McArdle's Syndrome patients lack the enzyme phosphorylase and thus cannot produce lactate, however, they do produce ATs. This indicates that the two events of lactate threshold and AT are distinct entities and invalidates the argument that it is the buffering of lactate that causes the ventilatory AT.
3. The final argument of Brooks relates to radio isotope studies used to examine lactate production in response to incremental exercise. These have lead to the realisation that blood lactate concentrations are a net result of the processes which add lactate to, and remove it from, circulation. In effect, blood lactate values are simply a result of the balance of the rate of appearance ( Ra ) and rate of disappearance ( Rd ). This had been overlooked by supporters of the AT, who gave the impression that blood lactate concentrations reflected lactate production. Work by Donovan and Brooks (1983) indicated that small increments in blood lactate can belie large increments in lactate production rates. Indeed, it is now understood that stable or minor increases in blood lactate levels in the early stages of an incremental test in the endurance trained athlete, are maintained by a fast rate of clearance.
The concept of lactate being more than the product of oxygen limited metabolism during exercise has been described in some detail by Brooks (1991) when communicating his "Lactate shuttle" hypothesis, which has gained great support from isotope tracer studies, as well as studies based upon arterial-venous lactate concentration differences. Brooks has also shown that the factors controlling the balance of lactate formation, uptake and release are far more complex than simply muscle tissue hypoxia. Tissues such as muscle are capable of simultaneous lactate production and consumption. It is clear that lactate exchange occurs between muscle and blood (Welch and Stainsby, 1967), blood and muscle (Stanley et al., 1986), active and inactive muscles (Ahlborg, 1985; Brooks, 1986), between blood and heart (Gertz et al., 1981) and blood and liver (Davis, Williams and Cherrington, 1984).

In addition to the aforementioned arguments by Brooks, other evidence exists to dispute the link between blood lactate inflections and the AT. Simon et al. (1983) found that AT corresponded to about $52 \%$ of $\mathrm{VO}_{2 \text { mas }}$, whereas lactate threshold was seen at about $63 \%$ of $\mathrm{VO}_{2 \text { max }}$. This, however, was in a limited sample size of 5 subjects of a normal healthy status, who performed incremental work on a cycle ergometer with 30 W increases in workload every 2 minutes.
Other work using the glycogen depleted state also found differences between the AT and lactate threshold. Hughes, Turner and Brooks (1982) found that the lactate threshold occurred at a higher workload and $\% \mathrm{VO}_{2 \text { max }}$ in the
glycogen depleted state, whereas the AT was seen to occur at lower workload and oxygen consumption. The effects of glycogen depletion on the lactate response to exercise, does have practical implications which will be discussed later.
Use of caffeine has also helped to analyse the difference between the AT and blood lactate response to exercise. Berry et al. (1991) found that the ingestion of 7 mg per kg body mass altered the AT without affecting the lactate threshold.
It is, therefore, concluded that there is considerable difference between the traditional AT and the lactate response to exercise. This review shall now concentrate on the lactate response to exercise.

### 2.24 The Lactate Response to Exercise

It has already been stated that blood lactate values are not so much a reflection of lactate production, but a net result of the balance of production and elimination. When plotted against workload or running speed in response to an incremental test, there is typically a curvilinear response (Jacobs, 1986; Weltman, 1995).

A classic belief held was that lactate is produced by muscles as a result of a lack of oxygen in the mitochondria, even in submaximal conditions (Katz and Sahlin, 1990). This in turn led muscles to resort to anaerobic metabolism for their immediate ATP requirements. Such increase in glycolysis was said to increase cytosolic NADH, which shifts the lactate dehydrogenase equilibrium towards an increased production of lactate. However, more recent work would dispute this. Currently, it is thought that lactate production is not solely a result of muscle hypoxia (Stainsby and Brooks, 1990; Spurway, 1992). It is likely that the $\beta$-adrenergic stimulation of skeletal muscle increases the rate
of glycogenolysis. NADH builds up within the mitochondria with a high rate of flux along the electron transport chain, whilst supplying much of the required ATP to fuel continued muscular contraction. As the mitochondrial NADH increases, so does the cytoplasmic NADH pool (Connett et al., 1990). This in turn elevates the rate of reduction of pyruvate, resulting in increased lactate production. This increase in lactate production, therefore, does not have to be a direct result of a low partial pressure of oxygen in the mitochondria. The supply of ATP to the muscle is satisfied predominantly via an aerobic route, even with the production of lactate. Training may reduce the production of lactate at any given work rate, as there is a diminished need for NADH build up in driving the electron transport.

Early German did not always use incremental tests to determine the lactate response to exercise. Early work by Mader et al. (1976), which was later summarised in English by Heck et al. (1985) examined running performed by 16 normal healthy males (four of whom were long distance runners) at a variety of speeds on separate occasions, each for 25 minutes duration. Blood lactate measurements were taken from the earlobe every 5 minutes. If lactates increased by no more than $1 \mathrm{mmol} .1^{-1}$ between minutes 5 and 25 , then a steady state was said to exist. Faster speeds were encountered until an increase in lactate above $1 \mathrm{mmol} .1^{-1}$ was found through the 25 minute run. The highest steady state speed was determined, as was the lactate value associated with this speed (the mean value of the last four lactate measurements on the run). The lactate values at this maximal steady state (MLaSS), similar in essence but determined differently to MSS in this project, for the sixteen subjects ranged between 3.05 and $5.52 \mathrm{mmol} \mathrm{I}^{-1}$, but the overall mean value was $4.02 \pm 0.70 \mathrm{mmol} \mathrm{l}^{-1}$. This, as the title of the paper suggested, was justification of a 4 mmol. $1^{-1}$ Lactate Threshold. However, it should be noted here that the sample of subjects was not highly trained endurance athletes, but sports students. Furthermore, there was a wide range in both the speeds and lactate levels associated with the MLaSS in this subject group. This clearly has implications for the absolute levels of lactate seen at the maximal steady state.

Due to the empirical nature of the establishment of this threshold by Heck and coworkers, many other authors have adopted the use of the lactate value of $4 \mathrm{mmol} . \mathrm{l}^{-1}$ as
both a means of identifying AT and as an optimal aerobic training intensity. Recently, it has been shown that the lactate level at MLaSS could well be related to the mode of exercise (Beneke et al., 1996). In comparing rowing (R), cycling (C) and speed skating (SS), it was found that differences were found in both the workload (R: $316.2 \pm 29.9 \mathrm{~W}$; C: $257.8 \pm 34.6 \mathrm{~W} ; \mathrm{SS}: 300.5 \pm 43.8 \mathrm{~W}$ ) and lactate level (R: $3.1 \pm 0.5 \mathrm{mmol} \mathrm{l}^{-1} ; \mathrm{C} 5.4 \pm$ 1.0 mmol. $1^{-1}$; SS: $6.6 \pm 0.9 \mathrm{mmol} . \mathrm{I}^{-1}$ ) associated with MLaSS. The suggestion was that the difference in sport specific muscle is responsible for the variation in lactate values.

Since the early work of Mader et al. (1976), and Heck et al. (1985), many other researchers around the world have tried to improve on this anaerobic threshold concept. It is here that the variety of definitions has filled the literature, adding confusion, but doing little to address the key issues.
The first development was to try and identify the lactate threshold via just one test, rather than a series of runs on separate days. The appeal is obvious, in terms of saving time and resources. This lead to progressive incremental tests, which are more common place today.
Kindermann et al. (1979) introduced the term of aerobic-anaerobic transition analysing 7 cross country skiers, firstly by means of a progressive incremental protocol and then with supplementary steady state runs. A gradient of $5 \%$ was used throughout the incremental test and the means of control for the steady state runs was either the heart rate associated with $4 \mathrm{mmol} . \mathrm{I}^{-1}$ or the running speed associated with the same physiological marker. This was in accordance of the work of Mader (1976) covered more in detail in the paper by Heck et al., (1985).
It was found if the heart rate was the means of control, the speed had to be reduced through the thirty minute period; whereas when the speed was the means of control, heart rate rose a little through the test and lactate remained high but stable (close to $4 \mathrm{mmol} \mathrm{l}^{-1}$ ). The authors concluded that heart rate could be used as a regulating parameter, when prescribing training from an incremental test on an ergometer. They also found 4 mmol. $1^{-1}$ was a suitable level for training intensity for the development of aerobic capacity.
However, only two years later a more individualised approach was attempted, again in Germany, by Stegmann, Kindermann and Schnabel (1981) in their study examining lactate kinetics and individual anaerobic threshold (IAT). The concept of an individual threshold was also appealing, because the work of Heck et al.(1985), although proposing
a general recommendation of $4 \mathrm{mmol} . \mathrm{l}^{-1}$ as an AT from the results of their group, acknowledged, that individual variation in responses existed.
The incremental test here, relied upon work until exhaustion to enable the construction of a diffusion-elimination model based around the lactate kinetics both during and after the exercise. The calculation of the threshold also uses the tangent of the curve (Figure 2.1).

Figure 2.1: IAT as determined by Stegmann et al. (1981)


Despite being appropriate, in that this process concentrates on establishing the appropriate level for the individual, rather than simply relying on an arbitrary fixed blood lactate concentration, there has been little support for this, or other methods of mathematical models for the prediction of thresholds, such as Keul et al., (1979) and Simon et al., (1983).
Scandinavian researchers such as Sjödin and Jacobs (1981); Svedenhag and Sjödin (1984) and Jacobs (1986) have consistently used the fixed blood lactate marker of 4 mmol. $I^{-1}$ to monitor and prescribe training. Since the early 1980's, linear interpolation of lactate values plotted against running speed in response to an incremental treadmill running test, has been used to identify the speed associated with $4 \mathrm{mmol} . \mathrm{l}^{-1}$. Curiously (Sjödin and Jacobs 1981) used the term onset of blood lactate accumulation (OBLA) in the first paper of a great many on the topic. The term vOBLA is used to define the running speed associated with 4 mmol. $\mathrm{l}^{-1}$, which in essence was exactly the same as the process used by the Germans Heck et al., (1985) when using an incremental protocol. The one benefit of this work was that it came away from using the AT label, which
traditionally was associated with respiratory phenomena. Therefore this approach was perhaps more sound in terminology, if little different in practice.
Work by Tanaka and Masuura (1984) used virtually identical protocols and criteria to Sjödin and Jacobs (1981) in their work, but still preferred the term anaerobic threshold, for running speed or cycling intensity associated with a lactate concentration of 4 mmol. $1^{-1}$. Yet the confusion over nomenclature does not end there, with other terms such as onset of plasma lactate accumulation (OPLA) by (Farrell et al., 1979), lactate threshold (Ivy et al., 1980) maximum steady state (La Fontaine, Londeree and Spath, 1981) and lactate turnpoint (Davis et al., 1983) occurring throughout the international literature, for similar phenomena.

The use of plasma lactate in the OPLA method, was used to correlate performance in a series of submaximal runs, with $\dot{\mathrm{VO}}_{2 \text { max }}$ and distance racing performance in eighteen trained distance runners (Farrell et al., 1979). The running speed at OPLA correlated well with distance running performance ( $\mathrm{r}=.98$ for 42.2 km and $\mathrm{r}=.98$ for 19.3 km and 15 km performances) whilst $\mathrm{V}_{2_{\text {nas }}}$ correlated nearly as well for the same distance run performances ( $\mathrm{r}=.91, .91$ and .89 , respectively). The testing procedure did not use an incremental test, but a series of submaximal runs, each of ten minutes duration. Delta lactate (post minus pre test lactate) was plotted against both running speed and oxygen consumption. The OPLA was determined as the point of rise of delta lactate either by visual inspection or regression analysis (with little difference between the two methods), examining an increase of $1.0 \mathrm{mmol} . \mathrm{l}^{-1}$ above baseline.
Ivy et al. (1981), preferred the term lactate threshold, when examining the effect of substrate availability on the blood lactate concentrations during submaximal exercise. The mode of exercise in this case was cycling on an ergometer and the threshold was deemed as the point just below the onset of blood lactate accumulation, when blood lactate was plotted against $\% \dot{\mathrm{VO}}_{2 \text { max }}$. This in its own right is curious, because $4 \mathrm{mmol} \mathrm{l}^{-1}$ was not used as the OBLA, rather an increase from baseline levels, without specific criteria. It would be easy to confuse their interpretation of OBLA, with that of the aforementioned Scandinavian work.
There has been a variety of definitions of the lactate threshold. Coyle et al., (1983) and Hagberg and Coyle (1983) defined the lactate threshold to be the point where a 1 mmol. $1^{-1}$ increase above baseline levels of lactate occurred. Strangely, however, Hagberg
and colleagues have also defined lactate threshold as the intensity that elicits $2.5 \mathrm{mmol} .1^{-1}$ after 10 minutes of steady state exercise (Hagberg, 1986).
The maximum steady state (MSS) term used by La Fontaine et al. (1981) was quite different to the MLaSS proposed by concurrent German work. In runners, the running velocity on a treadmill associated with a blood lactate concentration of $2.2 \mathrm{mmol} . \mathrm{I}^{-1}$ was used, in response to a protocol of two submaximal 10 minute runs.

Figure 2.2: Calculation of MSS according to La Fontaine, Londeree and Spath (1981)


Lactate turnpoint, first introduced by Davis et al., (1983) is another definition that has been used interchangeably with other terms such as AT. Lactate turnpoint is usually referring to upward shifts of the lactate curve in response to progressive incremental exercise (Noakes, 1991).

Figure 2.3: The Lactate Turnpoint as described by Noakes (1991)


Other investigators have noticed that there can be two shifts in a blood lactate curve, with an increase in intensity. Aunola and Rusko (1986) and Rusko et al. (1986) have entitled the first the aerobic threshold, where blood lactate increases a little above baseline levels, whilst the second is termed AT, where the accumulation of lactate is more rapid. This approach seems logical in all bar the terminology and the fact that the authors associate these points with the fixed blood lactate concentrations of 2 and $4 \mathrm{mmol} . \mathrm{l}^{-1}$. Personal observation of lactate curves of highly trained runners and rowers has shown that athletes, do often - but not always- show these two points during incremental exercise. The first would appear to represent the upper limit for high rates of fat metabolism, whilst the second would be towards the peak clearance rate of lactate (Brooks, 1991).

To summarise the above section

1. The term AT was first determined on the basis of respiratory data.
2. The introduction of blood lactate sampling and more importantly rapid methods for determining the assays has lead to an increase in the popularity of using this method for monitoring and prescribing training.
3. Many different terms do exist to define similar phenomena, either on the basis of a series of continuous runs to determine MLaSS, or via incremental protocols.
4. The incremental protocols appear more favourable in terms of saving time for establishing some type of threshold and predicting steady state.
5. The use of individual profiles is far more preferable than using fixed blood lactate concentrations.
6. The term lactate threshold (Tlac) is the term preferred in this document for the point during incremental testing that represents the Maximal Steady State (MSS) during continuous exercise. It distinguishes itself from threshold concepts based on respiratory data.
7. The Tlac need not rely on fixed blood lactate concentrations, but use individual levels that occur due to differences in muscle morphology.
8. It is acknowledged, however, that the Tlac does not pinpoint a fixed workload or oxygen consumption associated with a sharp increase in lactate production; rather an increase in blood lactate accumulation is seen as a result of the imbalance between the rate of appearance and disappearance.

### 2.30 Methodological Considerations

A number of factors can be seen to effect the Tlac measured in the laboratory. These shall be discussed individually below, but include the mode of assay of the blood sample, site of blood sample, substrate availability, experimental protocol and the mode of exercise.

### 2.31 Mode of Assay

The variety in analytical technique used in determining blood lactate concentrations is perhaps the strongest argument for not using fixed blood lactate concentrations to determine Tlac. It is likely that researchers using different modes of assay in one part of
the world, yet using the criteria of fixed blood lactate values set in another part of the world, have lead to inaccurate assessment of the Tlac and worse still, not given appropriate guidance for exercise prescription.
A letter to the British Journal of Sports Medicine (Forrest, Morton and Lambardarios, 1990) gave a good appreciation of the problem. It noted that those in clinical practice usually collect blood samples in tubes containing a fluoride-oxalate or fluoride EDTA preservative and anti-coagulant. These are normally centrifuged and the supernatant plasma examined for lactate. In the exercise laboratory in the UK, whole blood is normally analysed in rapid assay analysers such as Yellow Springs Instruments or Analox. Although there is little difference in the values at rest, there are clear differences in exercising values, with plasma samples always giving higher values.
Plasma values are often used in the USA (Farrell et al., 1979; La Fontaine et al., 1981) and the clinical procedure noted by Forrest, Morton and Lambardarios (1990) above is most commonly used in Germany. This means that when fixed blood lactate concentrations, such as $4 \mathrm{mmol} . \mathrm{l}^{-1}$ or $2.2 \mathrm{mmol} . \mathrm{l}^{-1}$, are used to assess performance or prescribe training, differences will occur if someone trying to replicate work uses a different mode of assay. Using whole blood values with the criteria of German work, would dictate the prescription of training intensities that are far too high.
The work of Williams, Armstrong and Kirby (1992) examined the difference in exercise lactate concentrations between whole blood (WB), lysed blood (LB) (which enables delayed analysis, due to haemolysis preventing glycolysis taking place and elevating the values) and plasma ( P ) samples. The P samples were significantly higher than the WB and LB ( $4.7 \pm 2.7 \mathrm{WB} ; 5.0 \pm 3.0 \mathrm{LB}$; and $7.0 \pm 3.8 \mathrm{mmol} .1^{-1}$ ).
Bishop et al. (1992a; 1992b) looked at two aspects of the values gained through different methods. The first study compared the results of a rapid assay machine (YSI) with the more conventional techniques (Boehringer automated fluorometric assay). A comparison was also made between lysed and unlysed samples. Results found that the unlysed YSI samples differed from the lysed YSI samples and there were also differences between the machines. This shows caution should be exercised in interpreting values when referring to work from other laboratories. The second study examined differences between WB and $P$ samples, which found although the relationship between the samples was good, there were significant differences.

### 2.32 Site of Sample

It is not only is how the blood sample is analysed that is important, the point of extraction from the body also makes a difference. This issue was also addressed by Williams et al., (1992) who compared lactate values taken from the brachial artery (A), antecubital vein (V) and fingertip capillary (C) during continuous treadmill running. They found good correlation between arterial and venous blood ( $\mathrm{r}=0.858$ ), supporting the work of McLoughlin et al., (1992) who also found a good relationship between the two( $\mathrm{r}=0.97$ ). These values also correlated well with the capillary blood samples ( $\mathrm{r}=0.983$ ), thus giving the recommendation that capillary blood be used due to the ease of this method. However, previously Yoshida, Takeuchi and Suda (1982) had found significant differences between venous and arterial blood lactate values in the exercise mode of cycling. The reason for such a difference is understandable, as there is a time lag between the appearance of lactate in the blood from exercising musculature in this exercise mode and the appearance in venous blood specimens. Lactate formed in the quadriceps muscles during cycling will have been through the capillary beds of the lung and forearm, with the potential for substantial decrease considering the aforementioned lactate shuttle proposed by Brooks (1991).
It appears that there will be differences in the values obtained depending on how the blood is collected and measured. However, in an applied setting, this may not be of significance provided that (i) the results are not interpreted and used in the light of research based on fixed blood lactate concentrations; (ii) there is consistency over time with the methods utilised.
The series of studies in the current research project use earlobe capillary blood. The rationale for the mode is that capillary blood is used for the rapid assay machine of Analox, which gives readings, after 2-3 minutes of mixing, within 45 seconds. The earlobe is the choice of sampling site, because in a laboratory incremental protocol or during field testing, multiple samples are required. With an earlobe sample it is easy to get multiple samples from one puncture, whereas fingertip samples require a separate puncture each time.

### 2.33 Substrate Availability

It has already been mentioned in passing that substrate availability is important to the lactate response to exercise. One of the key papers examining this topic was by Ivy et al. (1981) who examined 9 active male volunteers under three conditions when performing
an incremental cycle test. The three conditions were control, ingesting 75 g of glucose in 300 ml of water, 30 minutes before the task, or with elevated free fatty acids (required to eat a fatty meal 4.0-4.5 hours beforehand). They found that the Tlac could be altered, because when the free fatty acid levels were raised during muscular activity, the fatty acid oxidation rate increased, which gave decreased lactate production. With an increase in blood glucose, the blood lactate was seen to increase above the levels in the control trial at the same workload and $\% \mathrm{VO}_{2 \text { max }}$. However, there was no difference in change in lactate between pre and post exercise across the two trials. The conclusions were that (i) can be altered by substrate availability and (ii) that muscle tissue anoxia is not solely responsible for lactate production during submaximal work.
Another study a little later by Yoshida (1984b) also examined the effect of diet on Tlac and OBLA. A mixed, carbohydrate rich, and low carbohydrate strategy was used each for three days before a progressive cycle test. Although there were no differences between Tlac in any of the cases, at the higher intensity of OBLA, there were. The oxygen consumption at OBLA was significantly lower after a high carbohydrate regime, than after a low carbohydrate meal.
The practical implications of these two studies are that diet can have an effect upon the blood lactate levels elicited in response to exercise. With this being the case, clear guidelines need to be given to athletes before testing, to make sure that glycogen stores are full before testing. This would also have the advantage of maintaining performance, as low glycogen levels have been seen to affect endurance performance (Costill, 1970).

### 2.34 Experimental Protocol

It has already been seen that a variety of protocols exist in assessing the blood lactate response to exercise as Table 2.3 indicates. The protocol used will depend upon the purpose for testing. If simple monitoring is required, it makes little difference what protocol is used, as long as there is consistency on a longitudinal basis.

Table 2.3: A description of different methodological protocols

| Author | Subjects | Mode of exercise | Protocol |
| :--- | :--- | :---: | :--- |
| Aunola \& Rusko (1992) | 17 healthy males | Cycling | 2 min incremental |
| Beaver et al. (1985) | 10 healthy males | Cycling | 1 nin incremental |
| Beneke (1995) | 9 male rowers | Rowing | 3 min incremental |
| Bird and Davison (1997) | (BASES Guidelines) | Running | 4 min incremental |
| Cheng et al. (1992) | 8 male cyclists | Cycling | 5 min incremental |
| Farrell et al. (1979) | 18 male athletes | Running | Series of 10 min nuns |
| Ferry et al. (1988) | 5 healthy males | Cyling | 4 min incremental |
| Foxdall et al. (1996) | 8 male firemen | Running | $4,6,8$ min |
| Lelman et al (1983) | 6 male athletes |  | incremental |
| McLellan (1985) | 11 male athletes | Running | 3 min incremental |
|  | 10 healthy males | Cycling | $1,3,5$ min |
| Sjödin and Jacobs (1981) | 18 healthy males | Running | 4 min incremental |
| Stegmann et al. (1981) | 6 male athletes | Ruuning | 3 min incremental |
| Weltnan (1990b) | 6 female athletes |  | Running |
| Womack et al (1996) | 15 male athletes | 10 male rowers | Rowing incremental |
|  | 3 min incremental |  |  |
| Yoshida (1984a) | 8 healthy males | Cycling | 4 min incremental |

For the prescription of exercise (usually for lactate threshold, deemed to be the optimal aerobic training stimulus (Jacobs, 1986)) the situation is less clear cut. The use of a series of runs may well yield the most reliable results, but is extremely time consuming. Furthermore, the athlete and coach often require prescription of more than one training zone (Janssen, 1987; Foster et al., 1993). Due to these reasons, the incremental protocol is often most practical, especially for the purposes of training prescription.
The specific details of a protocol that can potentially alter the blood lactate values are whether the protocol is continuous or discontinuous, the duration of the stages and the level of increase of running speed or workload between the different stages.
Hagberg (1986) is a major supporter of using 10 minute stages of separate exercise, so as to accurately represent the steady state conditions. This process has also been used
elsewhere (La Fontaine, 1981) although interpreted in a different manner, as mentioned previously. However, more recent research by Weltman et al., (1990b) found little difference between the lactate profiles of sixteen male runners who performed a three minute incremental test and a test consisting of ten minute discontinuous runs. The velocity and oxygen consumption of Tlac and fixed blood lactate concentrations of both 2.5 and $4 \mathrm{mmol} . \mathrm{l}^{-1}$ was seen to be similar in both types of test. The authors summarised by stating that a 3 minute incremental test of horizontal treadmill running gave reliable and valid measurements for determination of Tlac.
A more common stage duration for incremental tests in Britain is four minutes as opposed to three minutes, as stated in the British Association of Sports and Exercise Sciences position statement regarding testing of the elite competitor (Bird and Davison, 1992). This policy probably originated from the previously mentioned Scandinavian work in establishing OBLA, where four minute increments are also used (Sjödin and Jacobs, 1981). The four minute duration differs slightly from the German incremental tests that use three minutes (Heck et al., 1985; Stegmann and Kindermann, 1982). Yoshida (1984a) had compared a 4 minute duration with a shorter incremental protocol, where one minute durations were used in cycling. He found that the shorter version resulted in non-steady state oxygen consumption and a higher workload for a given $\dot{V} O_{z}$. In Germany, for the testing of rowers, Hartmann uses an eight minute protocol, but his personal feeling was that a 6 minute discontinuous protocol is optimal (personal communication). An investigation into the differences between such protocols is the focus of the first experiment in the current study. Foxdall, Sjödin and Sjödin (1996) have examined these protocol options ( $5 \times 4$ minute, $5 \times 6$ minute and $5 \times 8$ minute) in a comparison to 50 minute steady state runs. They found that the $5 \times 4$ minute and $5 \times 6$ minute gave a risk of over-estimating the maximal lactate steady state, but they used OBLA as a reference point. It is clear, however, that when using fixed lactates as a reference point, there is a danger that the individual lactate threshold will be missed (Bird and Davison, 1997). For this reason it is pertinent for this study to examine the different protocol durations without using OBLA as the point of reference.

Jacobs (1986) has also shown that the rate of increase of speed or work will affect the shape of the lactate profile. Particularly in the less well trained, if the increments are too great, the rate of lactate accumulation is far quicker. In this research running speed
increments are usually $1.1 \mathrm{~km} / \mathrm{h}$ and rowing increments about 27 watts. These increases appear suitable for applied work and are currently in line with the procedures used at the British Olympic Medical Centre and this usually enables a range of 4-6 work stages when three minute durations are used.

For the running tests, there is a question of how the running speed on a treadmill relates to that outside. Recently Jones and Doust (1996) demonstrated that a $1 \%$ gradient on a machine reflects the energetic cost of running outdoors, due to the factor of wind resistance. This is in line with the work of Heck et al. (1985), who also examined this with two treadmill models, as well as examining the influence of different outdoor running surfaces on physiological responses. They found that running on grass was equivalent to an angle of about $2 \%$ on a treadmill, as opposed to the $1 \%$ associated with tarmac.

### 2.35 Mode of Exercise

The mode of exercise has important implications for Tlac and MSS. Beneke and von Duvillard (1996) examined MSS in well trained athletes from the sports of rowing, speed skating, cycling and triathlon, during three different exercise modes (rowing, cycling and speed skating). They found that the maximal workload was higher in rowing than in cycling and speed skating and that MSS workload was higher in rowing and speed skating than in cycling. Furthermore, the blood lactate concentration at MSS was higher in speed skating $\left(6.6 \pm 0.9 \mathrm{mmol} . \mathrm{l}^{-1}\right)$ than cycling ( $5.4 \pm 1.0 \mathrm{mmol} . \mathrm{l}^{-1}$ ) and rowing ( $3.1 \pm$ $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$ ). The heart rate representing MSS in each exercise mode was not reported.
In the sport of triathlon, there has been limited research examining Tlac in the modes of running and cycling. It has been demonstrated that Tlac ( $4 \mathrm{mmol} . \mathrm{l}^{-1}$ ) occurs at 72 $88 \% \mathrm{VO}_{2 \text { max }}$ in cycling and $80-85 \% \dot{\mathrm{VO}}_{2 \text { max }}$ in treadmill running (Kohrt et al., 1987; O'Toole, Douglas and Hillier, 1989). The heart rate representing Tlac in these cases was not reported.

### 2.40 Longitudinal Monitoring

The review article by Jacobs (1986) is quite clear in suggesting that the blood lactate response to exercise is the most appropriate way of monitoring training status on a longitudinal basis. It is a far more sensitive marker than $\mathrm{VO}_{2 \text { max }}$ and due to the fact that it is usually submaximal in nature, makes it more appealing as a method of assessment.

In terms of examining the responses to an incremental test, the lactate curve, if plotted against workload, or running speed, would tend to shift to the right in the case of an improvement in aerobic capacity. Similarly, if training status has declined, the curve will shift to the left. Wilmore and Costill (1988) have also shown that the Tlac occurs at a greater percentage of $\mathrm{V}_{2^{\max }}$ after endurance training. The shape of the lactate curve can also change with training, giving a response that is less like a diagonal line, when blood lactate is plotted against speed or intensity, so a flatter curve with a sharper inflection is seen. (Figure 2.4).

Figure 2.4: Change in position and shape of a lactate curve after 4 months of endurance training in a junior rower.


### 2.50 Training Prescription

Despite the wealth of literature about the lactate response to exercise, there is relatively little that has addressed the process of prescribing training intensities, particularly for the serious endurance competitor. It is this area that is under study in the current research. Many training programmes rely on exercise prescription using relative percent techniques, so percentages of maximum heart rate ( $\mathrm{HR}_{\text {max }}$ ), heart rate reserve (HRR) or $\dot{\mathrm{V}}_{2_{2 m a x}}$ are used in accordance with ACSM guidelines (ACSM, 1990). There are, however, problems associated with this type of methodology, in that it was designed more for the development of health related fitness, where the pinpointing, or fine tuning
of specific training zones may not be as critical as in the elite endurance competitor, who wants to train hard, without overtraining. Furthermore, research programmes have shown that such methodology can also be inaccurate. Dwyer and Bybee (1983) showed that the broad training zones recommended by the ACSM, such as $50-85 \% \mathrm{VO}_{2 \text { max }}$ and $70-90 \%$ of $\mathrm{HR}_{\max }$ can lead to enormous differences in metabolic stress, in terms of the ventilatory threshold. The authors did, however, show that heart rate was quite useful for the regulation of training at Tlac; a key point for training prescription.
Weltman et al. (1990a) critically examined the use of ACSM recommended zones, in examining 31 well trained male runners (mean $\mathrm{VO}_{2 \text { max }} 63.5 \pm 6.4$ ). The authors found that 20 of the 31 runners had not reached Tlac by $90 \%$ of maximal heart rate. This would imply an underestimation of training intensity by the ACSM method; which may mean that highly trained athletes require higher percentages of maximal heart rate to attain Tlac. Although there was clearly individual variation within the group of 31 ( 5 runners had blood lactate concentrations above $4 \mathrm{mmol} . \mathrm{l}^{-1}$ at $95 \% \mathrm{HR}_{\max }$ ), this general finding has also been supported by Dunbar and Faulmann (1996) who examined 30 elite middle and long distance runners (mean $\mathrm{VO}_{2 \text { max }} 76.2 \pm 5.2$ ). They found that MLaSS was associated with $93.2 \% \mathrm{HR}_{\text {max }}$ or $83.2 \% \mathrm{VO}_{2 \text { max }}$.

It had previously been shown that there was variation in the association between $\% \mathrm{HR}_{\max }$ and $\% \mathrm{VO}_{2 \text { max }}$ (Swain et al., 1994), who found that fit men (as determined by $\mathrm{VO}_{2 \text { max }}$ values) averaged $2 \%$ higher in percentage of $\mathrm{HR}_{\text {max }}$ than men of a lower fitness level, at any given value of $\% \mathrm{~V}_{2_{\text {nax }}}$.
Furthermore, DiCarlo et al. (1991) found significant differences between the peak and training heart rates in subjects during two different modes of exercise, thus highlighting the need for specificity when it comes to assessment and training prescription. This leaves the option of using blood lactate values to prescribe training intensities for athletes. Weltman (1995) described a number of methods for doing this but stated that there are few references to back up the methodology.
Research has, however, examined various methods of controlling Tlac, but due to a variety of methodologies in establishing the Tlac or AT in the first place; it is difficult to choose the optimal one. However, there is no evidence of strategies to prescribe training for other training zones employed by athletes. For example, Weltman et al (1990b) concluded that the use of heart rates for longer duration training (1 hour or more)
common to endurance runners, has not been examined. This is one gap in the literature that will be addressed by the current study.

This review will now examine other work that has attempted to prescribe training at Tlac or related variables.

Perhaps the classic work in the field is by Sjödin, Jacobs and Svedenhag (1982) who examined changes in OBLA and muscle enzymes after training at OBLA. The purpose of the study was to examine the effect the addition of one session of OBLA training per week in 8 middle and long distance runners. It was unique in not only that it investigated elite athletes in their normal training, unlike most studies that tend to examine the introduction of Tlac training 3-5 times per week in the previously untrained, this also addressed the impact of Tlac training during the normal training week.
The one weakness of the paper from a research methods point of view was that the subjects acted as their own controls, rather than having a separate control group studied concurrently (it is unlikely that this competitive group of runners would have kept to the same training programme throughout the whole study period, as periodisation would have led to change in emphasis towards competition). During the 14 weeks of the study, the subjects added a 20 minute treadmill run to their programme, the speed of which was associated with $4 \mathrm{mmol} \mathrm{l}^{-1}$ (OBLA) during a previously performed incremental test. During each run, blood samples were taken at 5 and 20 minutes, with the treadmill speed being adjusted according to the 5 minute value. Despite this, there was still an increase in blood lactate through the session with a mean increase from $4.1 \pm 0.3 \mathrm{mmol} .^{-1}$ at five minutes to $5.9 \pm 1.0 \mathrm{mmol} \mathrm{I}^{-1}$ at 20 minutes. This in its own right implies that the workload associated with $4 \mathrm{mmol} \mathrm{l}^{-1}$ was too fast to maintain steady state conditions in these well-trained runners. The results of the study showed that the 14 week period gave great increases in the speed at OBLA ( $4.69 \mathrm{~m} \cdot \mathrm{sec}^{-1}$ pre OBLA to $4.89 \mathrm{~m} \cdot \mathrm{sec}^{-1}$ post OBLA training, $\mathrm{p}<0.01$ ), which had not been enjoyed in the 18 week period either before or after the 14 week OBLA training period. There was a significant relationship between the changes in oxygen consumption at $15 \mathrm{~km} / \mathrm{h}$ and changes in the relative activity of H LDH ( $\mathrm{r}<-0.75, \mathrm{p}<0.05$ ), which the authors took to be an indication that the improved running economy was at least partly due to an improved intracellular oxidative capacity. It is this finding that has lead to the much heralded world-wide belief that OBLA training at $4 \mathrm{mmol} \mathrm{I}^{-1}$ is essential for the development of endurance. However, many authors have mistakenly used the $4 \mathrm{mmol} .1^{-1}$ concept only, rather than examine the literature a little
more deeply. It was found that the greatest increase in speed at OBLA, occurred in the subjects who had a smaller increase in blood lactate through the sessions. Thus individual steady state conditions are far more appropriate for endurance conditioning in elite runners, than a pace associated with a blood lactate level of $4 \mathrm{mmol} . \mathrm{l}^{-1}$.
Stegmann and Kindermann (1982) had also found difficulties in establishing steady state conditions when 4 mmol. $\mathrm{l}^{-1}$ was used as the means of intensity control. The study examined 19 rowers who performed two 50 minute workouts in response to a previous incremental test. The rowers were required to work at an intensity either associated with IAT, or 4 mmol. $1^{-1}$. Fifteen of the 19 subjects had stable lactates at IAT, but increased lactate accumulation at the intensity associated with $4 \mathrm{mmol} .^{-1}$. Three of the 19 had coincident IAT and 4 mmol. $1^{-1}$ values and 1 subject had lactates higher at IAT, than 4 mmol. $\mathrm{I}^{-1}$. The authors concluded that the higher the aerobic power, the more the MLaSS would be overestimated if determined by $4 \mathrm{mmol} \mathrm{l}^{-1}$, which implies that an individual approach must be considered for exercise prescription.
Henritze et al (1985) examined training either at, or 69 watts above, Tlac during cycling exercise in previously sedentary subjects. They found that the subjects above Tlac enjoyed an increase in $\mathrm{VO}_{2 \text { max }}$ as well as the oxygen consumption associated with Tlac, changes that were not seen in the Tlac training group. The Tlac in this study was determined by an elevation in blood lactate above resting levels during an incremental test, a level that would represent the upper limit for Base Endurance training in this current study, where the aim of which is not to elevate $\mathrm{VO}_{2 \text { max }}$, rather develop the aerobic capacity of the working musculature.
Another study examining IAT training, but again in untrained subjects, compared 8 weeks of training in a group performing continuous IAT training, with a group which divided the 30 minute period into 7.5 minute blocks both below and above IAT and with a control group (Keith, Jacobs and McLellan 1992). By assessing responses at both 4 and 8 weeks, the authors found increases in $\stackrel{\vee 1}{2}_{2 \text { max }}$ in both training groups, with greater adaptations occurring during the first four weeks. A similar pattern was also noticed for the power associated with Tlac. The fact that there was no difference between improvements in the two previously untrained groups, lead the authors to suggest that the mean intensity during the training session determines the extent of the adaptation regardless of whether the training was performed intermittently or continuously. Whether, this would be the case in trained athletes, who would perform greater volumes of training at higher relative percentages, remains to be resolved.

Work by Coen et al. (1991) focused on whether training recommendations could be made in different ranges of intensity using percentages of the speed associated with IAT. In examining the training of elite runners and triathletes, the study also assessed the impact of climatic conditions and terrain in modifying the recommendations based on laboratory tests.
Endurance running on a flat terrain (5 laps of a 2,200 metre circuit) or graded terrain (5-7 laps of 2060 metres) was analysed with blood lactate samples taken before, after lap 2 and at the end of the workouts. Similarly, interval sessions of $5 \times 1000 \mathrm{~m}$ were assessed in either good, or poor climatic conditions, with blood samples taken during the 4.5 minute recovery period allowed between each repetition. The blood lactate samples were used to examine whether the sessions were performed at the correct intensities. Previous work with marathon runners, by Förenbach Mader and Hollman (1987) had previously recommended that long distance running training be performed at intensities in the range of $85-92 \%$ of IAT to avoid overtraining. The work of Coen et al. (1991) found that the greater the terrain and climatic conditions varied, the less accurate became the attempts to achieve defined blood lactate values on the basis of IAT speed. The authors also found that individuals could vary enormously in the values obtained during the sessions and that a blood lactate level of 3.2 mmol. $1^{-1}$ in two athletes could mean two different things. In one runner, with an IAT lactate of $2.6 \mathrm{mmol} . \mathrm{l}^{-1}$, the work would be hard, whereas another athlete, who's IAT lactate was $3.8 \mathrm{mmol} \mathrm{I}^{-1}$, the session would have been much easier. The paper however, gave training recommendations, in terms of percentage speed of IAT, for different types of workout. The problem here is that it relied on running velocity to be monitored and fed back, which does not allow training in a variety of terrains and venues. The latter point is important, because many athletes use a variety of training routes, which are unlikely to be marked for the purposes of providing running speed feedback.

### 2.60 Heart Rate Monitoring

A more satisfactory means of training control is suggested by Janssen (1987), where training is controlled in the field by heart rate, determined from a lactate profile. This method is appealing, as it uses a biological variable, which will be sensitive to changes in climate and more importantly terrain. If an athlete runs up a hill, the increased metabolic
work will be reflected by an increase in heart rate. The strategy of Janssen does, however, have two problems. Firstly, the principle suggested relies on the prescription of training using fixed lactate values, such a 4 mmol. $1^{-1}$ for Tlac training. A similar, but modified approach will therefore be used in the current work, whereby individual lactate profiles will prescribe the training intensity, rather than arbitrary fixed lactates. Secondly, the efficacy of the process has not been examined empirically, which is another aspect of the current study.
The use of heart rate to control training has gathered momentum, with the increase in availability of portable telemetry devices, which are accurate and give immediate feedback, as well as having the ability to store data for subsequent play back (Gilman, 1996). However, the Gilman review article examining the use of heart rate to monitor the intensity of endurance training, has not examined the potential of using heart rates prescribed from a lactate profile - the focus of the current study.

One element the Gilman review article mentioned is that of cardiovascular drift during endurance exercise, which stated that heart rate will drift upwards in sessions where the duration is longer than 20 minutes. This phenomenon may alter the heart rate - lactate relationship seen in an incremental test, or indeed a subsequent verification run of a short duration. Cardiovascular drift was first noticed and documented by Saltin (1964) and is accompanied by a decrease in stroke volume (Goodman, et al., 1989), as well as factors such as a progressive increase in core temperature and progressive dehydration due to fluid loss from both sweating and ventilatory loss.
Other authors have also noticed similar trends in response to exercise in the heat (Nadel, 1983; Rowell, 1969). The cardiovascular drift is usually greater in treadmill running than running outdoors, due to the fact that there is no convection to aid cooling (Förenbach et al., 1987). This was supported in work by Adams et al., (1992) when cycling for 60 minutes at $56 \% \dot{\mathrm{VO}}_{2_{\text {max }}}$ was performed in varied environmental conditions. Heart rate was significantly higher in a still air condition at $35^{\circ} \mathrm{C}$ as opposed to airflow of $3 \mathrm{~m} / \mathrm{s}$ at this temperature and both airflows at $24^{\circ} \mathrm{C}$.

Work by Cempla et al., (1987) has also noticed that, under laboratory conditions, the heart rate increases during 30 minutes of high intensity running at $81 \% \mathrm{VO}_{2}$ max (close to Tlac) in trained distance runners. They found that rectal temperature increased by $1.95^{\circ} \mathrm{C}$, with progressive increases in both heart rate and minute ventilation.
2.70 Summary

1. Performance of endurance sport requires a great contribution from aerobic metabolism.
2. When blood lactate measurements are plotted against workload or running speed in response to an incremental protocol a curvilinear rise is seen with increasing intensity.
3. Such incremental tests can be used to monitor condition and predict MSS, although the methodology, criteria and terminology can vary considerably between authors worldwide.
4. Attention must, therefore, be given to methodological considerations when determining blood lactate profiles, not least of which the type of protocol used.
5. Although the examination of training aimed at MSS has been popular, little work has previously examined training sessions of different intensities, which are clearly used by athletes preparing for competitive sport.
6. Heart rate has become a popular means of training monitoring and control. Indeed, prescribing heart rate established during an incremental protocol has become a popular means of predicting steady state blood lactate. There is currently little empirical evidence to support such a methodology.

### 3.0Statement of the Problem and Purpose of study

### 3.1 Statement of the problem

Performance in competitive sport is considered responsive to a systematic training regime. To date, quantitative models examining the response to variations in training intensity and duration of training within a programme do not exist. However, it is commonly believed that training intensity is a key factor to optimise training adaptations. Heart rate is one method of prescription and control of training intensity. The present study has sought to examine the use of heart rate to control training intensity in welltrained athletes from the sports of running and rowing.

Previous unpublished work in our laboratory, as well as observation of physiological responses during training, has shown differences in heart rates used for MSS training in different exercise modes. This can cause confusion for athletes in multi-event sports such as triathlon and duathlon, when trying to select the appropriate heart rate to control training in MSS sessions.

### 3.2 Purpose of the study

The purpose of the study was to examine the control of training in well-trained endurance athletes. Firstly, establishing an incremental protocol that was acceptable for the determination of a blood lactate profile, that could be used to establish lactate threshold, a key training intensity where blood lactates are high but stable, giving maximal steady state (MSS). Secondly, to compare heart rate and running speed as methods of training control during MSS sessions in running. Thirdly, examining heart rate as a means of control during MSS sessions rowing. Fourthly, to assess the method of using heart rate prescribed from a blood lactate profile, to control training sessions aimed at MSS and Base Endurance. Finally, to analyse the differences in heart rate at Tlac, a key training
intensity, between running and cycling in well-trained triathletes, in an attempt to identify the magnitude of any difference seen between exercise modes.

### 3.3 Null Hypotheses

1. Heart rate and blood lactate values remain unaltered with varied stage duration, or time period between stages, during incremental treadmill running in trained middle and long distance runners.
2. There is no difference between running speed and heart rate as a means of controlling training sessions aimed at the development of base endurance and MSS in welltrained runners.
3. Lactate values observed during ergometer rowing cannot be predicted from the observation of hart rate in well-trained rowers.
4. Lactate values observed in the training environment cannot be predicted from the observation of training heart rates during BE and MSS training sessions in welltrained runners and rowers.
5. There is no difference in heart rate at fixed blood lactate markers and at Tlac, between the exercise modes of running and cycling in trained triathletes.

### 4.0 Experiment One: Establishing a lactate profile for subsequent training prescription.

### 4.10 Introduction

The blood lactate response to a graded exercise test can be used for a variety of purposes, including screening, the monitoring of training and exercise prescription (Jacobs, 1986). However, there is widespread variation throughout the relevant literature as regards the nature of protocols utilised. Protocols may be either continuous or discontinuous in nature and may have a stage duration ranging between as little as 1 (Yoshida, 1984) and as long as 10 minutes (Weltmann 1990b; Hagberg, 1986). In the case of discontinuous protocols, the length of the pause between stages may also be seen to vary. Sjödin and Jacobs (1981) and Sjödin and Svedenhag (1981) used a classic continuous protocol when examining elite runners, who ran at an initial velocity associated with $45-50 \% \dot{\mathrm{VO}}_{\mathrm{O}_{2}}{ }_{\text {max }}$ for 5 minutes. This was followed by at least 4 stages of 4 minutes in duration, where the subsequent velocity was between 0.5 and $2.0 \mathrm{~km} \mathrm{~h}^{-1}$ faster than the previous level.

Yoshida (1984) examined the difference in blood lactate levels between 1 and 4 minute workloads of cycling in 8 healthy males. He found that the 1 minute incremental protocol resulted in non-steady state conditions for oxygen and a higher workload for a given level of oxygen consumption; this lead to a less accurate determination of lactate threshold (Tlac) and OBLA (the workload associated with 4 mmol. $1^{-1}$ of blood lactate). When examining Anaerobic Threshold (AT), McLellan (1985) found a difference between 1 minute, as opposed to 3 and 5 minute durations when testing 10 male cyclists with a 30 Watt incremental method.

Previously, Hagberg (1986) had suggested that a discontinuous protocol using 10 minute runs, although less time efficient than a shorter continuous method, provided more valid data for the determination of lactate threshold (Tlac) and prediction of performance. Later, Weltmann et al. (1990b) found a continuous incremental protocol with 3 minute stages to be both valid and reliable when compared with a series of 3 ten minute runs and
the associated disadvantage of 2 or 3 extra visits to the laboratory, each of approximately two hours testing time.

In an applied setting, where blood lactate profiles are used for subsequent training prescription, there is a requirement for accurate, but time efficient methodology. The use of a series of runs may well yield the most reliable results, but is extremely time consuming. Furthermore, the athlete and coach often require prescription of more than one training zone (Janssen, 1987; Foster et al., 1993). Due to these reasons, the incremental protocol is often most practical, especially for the purposes of training prescription.

Therefore, the purpose of this study was to examine different types of protocol, which may alter heart rate and blood lactate values obtained during the routine assessment of middle and long distance runners.

### 4.20 Methods

The overall scheme of study was sub-divided into to two distinct sections:

1. Two discontinuous methods were compared with a commonly used 4 minute stage continuous model.
2. The 4 minute protocol was compared with a shorter 3 minute version in an attempt to keep the testing protocol as time efficient as possible.

### 4.21 Subjects

In Study One, the subjects were eight male middle and long distance runners, who were of at least county standard. In Study Two, the subjects were eight male middle and long distance runners of at least county standard. Two subjects participated in both studies. The physical characteristics of both subject groups are displayed in Table 4.1. Each subject was fully informed of the purpose of the study, as well as the potential health risks before completing a medical history screening questionnaire (Appendix One) and
signing a written consent form (Appendix Two). Approval for the study was given by an internal Ethics Committee temporarily in place at St. Mary's College, Strawberry Hill.

### 4.22 Testing Structure

In Study One, subjects were required to perform three graded incremental tests within a three week period. The order of tests was randomised, but testing took place on the same day of each week, so as to fit in consistently with the training schedule. Testing followed either a rest day or day of light running to combat the potential problem of glycogen depletion. Subjects produced both dietary recall sheets (Appendix Three) and training logs (Appendix Four) during the study period. In Study Two, both tests were performed 48 hours apart in a randomised fashion, with subjects restricted to rest or easy running the day before testing.

Table 4.1 Physical Characteristics Mean (S.E.) of Subjects

|  | Age (years) | Mass (Kg) | Height (cm) |
| :--- | :---: | :---: | :---: |
| Study One | $21.0(1.0)$ | $70.3(2.0)$ | $180.7(2.4)$ |
| Study Two | $20.7(.08)$ | $72.5(1.4)$ | $188.5(1.7)$ |

All running took place on a Powerjog M30 running machine (Sport Engineering Ltd, Birmingham) with a level gradient. The running speeds during the testing series were selected for each subject on the basis of current running form and /or from previous exercise tests during routine physiological monitoring. The aim of the speed selection was to reach a significant increase in blood lactate by the end of the last running speed. Typically, the initial speed would be $15.9 \mathrm{~km} . \mathrm{h}^{-1}\left(4.42 \mathrm{~m} . \mathrm{s}^{-1}\right.$ ) with the lowest being 14.8 $\mathrm{km} \cdot \mathrm{h}^{-1}$, but the increase in running speed between stages would always be $1.1 \mathrm{~km} \cdot \mathrm{~h}^{-1}$ $\left(0.31 \mathrm{~m} . \mathrm{s}^{-1}\right)$. All tests were preceded by a controlled warm up of 5 minutes running at $12.6 \mathrm{~km} . \mathrm{h}^{-1}\left(3.5 \mathrm{~m} . \mathrm{s}^{-1}\right)$ and stretching as required. In all cases the laboratory temperature was within a temperature range of $18-22^{\circ} \mathrm{C}$.

### 4.23 Testing Protocols

a) Study One:
i) Protocol One was a modified version of the BASES protocol (Bird and Davison, 1997), consisting of four 4 minute running speeds, interspersed with very short pauses ( $<20 \mathrm{sec}$.) for the collection of blood samples.
ii) Protocol Two was discontinuous in an attempt to isolate the running speeds. Running duration was six minutes on each of the four speeds, separated by 10 minute pauses.
iii) Protocol Three was similar to protocol Two, except 8 minute running durations were used giving a longer total test and running time.
b) Study Two:
i) Protocol One consisted of 5 running speeds in the same format as Protocol One in Study One
ii) Protocol Two consisted of 5 running speeds as in Protocol One, but the running duration was 3 instead of 4 minutes.

### 4.24 Measurements

At the end of each running speed, subjects stood astride of the moving treadmill belt whilst heart rate was recorded via radio telemetry (Polar, Finland). Blood samples were taken from the earlobe for subsequent lactate determination from whole blood (Analox GM7, Hammersmith). The blood analyser was calibrated using both 3 and $8 \mathrm{mmol} .1^{-1}$ standard solutions and also checked with a Quality Control Serum (concentration 2.3-2.5 mmol. $1^{-1}$ ). Standard precautions were taken against blood born viruses in accordance with BASES guidelines (Bird and Davison, 1997).

### 4.25 Data Analysis

For more detailed comparison in study two, individual blood lactate profiles were plotted against both running speed and heart rate, with points joined via linear interpolation. The heart rate associated with $2 \mathrm{mmol} .1^{-1}$ of blood lactate was established, as was the heart rate associated with lactate threshold (Tlac), the point where there was a sharp increase in
blood lactate, in response to an increase in running speed and /or heart rate. This is more easily determined when blood lactate is plotted against heart rate as shown in Figure 4.1 below where the $2 \mathrm{mmol} \mathrm{I}^{-1}$ heart rate is 161 and the Tlac value is 166 .

Figure 4.1: Example of a lactate profile showing the heart rate at $2 \mathrm{mmol} \mathrm{I}^{-1}$ of blood lactate and at Lactate Threshold


### 4.25 Statistical Analysis

To test for differences between the three protocols in study one, ANOVA with repeated measures was used, whilst paired $t$ tests were used in study two. Correlation coefficients were also used to examine the relationship of the variables between protocols. A p value of less or equal to 0.05 was accepted as significant for these analyses. In assessing the level of agreement of the two protocols in study two the graphical and simple calculation methods of Bland and Altman (1986) were used.

Study One: Table 4.2 shows the mean values for blood lactate and heart rate at each running speed. ANOVA revealed that there was no significant difference across the three protocols for either heart rate or blood lactate at any of the four running stages.

Table 4.2: Mean (S.E.) blood lactate and heart rate values obtained in each protocol.

|  | Protocol One (4 min) |  | Protocol Two (6 min) |  | Protocol Three (8 min) |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Stage | $\begin{gathered} \mathrm{La} \\ \mathrm{mmol} . \mathrm{l}^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{HR} \\ \text { b. } \min ^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{La} \\ \mathrm{mmol} \mathrm{l}^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{HR} \\ \text { b. } \mathrm{min}^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{La} \\ \mathrm{mmol.}^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{HR} \\ \text { b. } \mathrm{min}^{-1} \end{gathered}$ |
| One | 1.16 (.16) | 156 (5) | 1.17 (.19) | 153 (4) | 1.16 (.16) | 151 (5) |
| Two | 1.60 (.27) | 165 (4) | 1.54 (.25) | 162 (4) | 1.63 (.28) | 162 (5) |
| Three | 2.16 (.35) | 171 (4) | 2.01 (.32) | 170 (4) | 2.26 (.41) | 171 (4) |
| Four | 3.40 (.55) | 177 (4) | 2.90 (.49) | 177 (4) | 3.13 (.55) | 179 (3) |

There was good correlation between the protocols for heart rate and blood lactate measurements at each stage during the incremental tests, as shown in Table 4.3. In all cases $\mathrm{p}<0.01$.

Table 4.3: Correlation coefficients of lactate and heart rate values between different protocols.

| Protocols | One and Two | One and Three | Two and Three |
| :--- | :---: | :---: | :---: |
| ${\text { Lactate mmol. }{ }^{-1}}^{\text {HR b.min }}$ | $\mathrm{r}=0.95$ | $\mathrm{r}=0.92$ | $\mathrm{r}=0.95$ |
| H | $\mathrm{r}=0.93$ | $\mathrm{r}=0.85$ | $\mathrm{r}=0.91$ |

Study Two: A comparison of both heart rate and blood lactate responses at each speed between the 3 and 4 minute contimuous protocols is displayed in Table 4.4. A paired $t$ test revealed significant differences blood lactate values during stages 3 and 4 as well as
heart rate values during stages 3 and 4. At each running speed, the mean heart rate and blood lactate values were higher during the 4 minute protocol. Such consistent difference was not seen between the three protocols in Study One. The nature of the bias is shown also by the individual cases in Figures 4.2(a) and 4.2(b).

Table 4.4: Mean (S.E.) blood lactate and heart rate values in the 3 and 4 minute protocols.

|  | Protocol One (3 min) |  | Protocol Two (4 min) |  |
| :--- | :---: | :---: | :---: | :---: |
| Stage | $\mathrm{La}\left(\mathrm{mmol.l} \mathrm{l}^{-1}\right)$ | $\mathrm{HR}\left(\mathrm{b} \cdot \mathrm{min}^{-1}\right)$ | $\mathrm{La}\left(\mathrm{mmol} . \mathrm{l}^{-1}\right)$ | $\mathrm{HR}\left(\mathrm{b} . \mathrm{min}^{-1}\right)$ |
| One | $0.85(.09)$ | $146(4.7)$ | $0.94(0.18)$ | $150(5.1)$ |
| Two | $1.06(0.12)$ | $155(4.0)$ | $1.16(0.19)$ | $160(4.2)$ |
| Three | $1.28(0.11)$ | $162(3.7)$ | $1.63(0.21)^{*}$ | $168(4.6)$ |
| Four | $2.08(0.20)$ | $173(3.6)$ | $2.48(0.21)$ | $178(4.3)^{* *}$ |

* Significantly different to $3 \mathrm{~min} \mathrm{p}<0.05$ ** Significantly different to $3 \mathrm{~min} \mathrm{p}<0.005$

Figure 4.2: Relationships of (a) blood lactate and (b) heart rate values between the 3 and 4 minute protocols.
(a) Blood lactate $\mathrm{r}=0.89(\mathrm{p}<0.001)$
(b) Heart Rate $\mathrm{r}=0.90(\mathrm{p}<0.001)$


4 Minute Protocol


Despite the strong correlation between the two protocols for both heart rate and blood lactate responses, the levels of agreement between the two protocols can be seen to be poor when viewed as responses at each speed (Figure 4.3(a) and Figure 4.3(b)). The mean difference between protocols for blood lactate was $-0.23 \mathrm{mmol} . \mathrm{l}^{-1}$ and for heart rate $-5.31 \mathrm{~b} . \mathrm{min}^{-1}$

Figure 4.3: Levels of agreement for (a) blood lactate and (b) heart rate values between the 3 and 4 minute protocols.


When the heart rate - blood lactate relationship is examined, comparison between the two protocols, shows better agreement. Table 4.5 displays the mean heart rate attained at both $2 \mathrm{mmol} . \mathrm{l}^{-1}$ and LT for both protocols. There was no significant difference between the protocols for either of these variables.

Table 4.5: Mean ( $\pm$ SD) heart rate at 2 mmol. $1^{-1}$ blood lactate and LT in each protocol.

| Protocol | HR at $\mathbf{2} \mathbf{~ m m o l . \mathbf { I } ^ { \mathbf { 1 } }}$ | HR at LT |
| :--- | :---: | :---: |
| 3 Minute Stage | $170.9 \pm 9.8$ | $172.8 \pm 9.5$ |
| 4 Minute Stage | $170.7 \pm 9.1$ | $174.3 \pm 12.0$ |

Whether the heart rate at a fixed lactate concentration ( $2 \mathrm{mmol} \mathrm{I}^{-1}$ ) is used, or the heart rate associated with LT, there is better agreement between the two protocols (Figure 4.4(a) and 4.4(b)). The mean difference between protocols for HR at $2 \mathrm{mmol} \mathrm{l}^{-1}$ was 0.19 and LT -1.5 .

Figure 4.4: Levels of agreement for the heart rate - blood lactate relationship at (a) 2 mmol. l $^{-1}$ and (b) Lactate Threshold.
(a) $2 \mathrm{mmol}^{-1}$
(b) Lactate Threshold



### 4.41 Comparing discontinuous with continuous protocols

The good agreement between the protocols in study one, comparing the two discontinuous, longer stage protocols with the shorter 4 minute incremental protocol is consistent with the findings of Weltman et al (1990b), who compared a 3 minute incremental protocol with 10 minute stages and found no differences. Although minor differences existed between the mean heart rate and blood lactate values at each running speed, there was no significant difference at any stage. Furthermore, there was no consistent difference to indicate any bias towards higher readings in any one protocol. The implication of this finding is that as there is no difference between the methodologies and that the shorter incremental protocol can be accepted as suitable in representing the heart rate and blood lactate values associated with any particular running speed. For the sake of time and efficiency, the incremental protocol would be chosen as there is a total test time of about 15 minutes with a five stage test, as opposed to 70 minutes in the six minute version and 80 minutes in the 8 minute version.
The findings do disagree with Foxdall, Sjödin and Sjödin (1996) who found that 8 minute stages in a continuous protocol better represented the heart rate and blood lactate values seen during 50 minute steady state runs in firemen and marathon runners. However, the criterion measurement in that work was OBLA, which does not necessarily relate to steady state lactate in long duration activity. In individuals whose Tlac is at an absolute blood lactate level below $4 \mathrm{mmol} . \mathrm{l}^{-1}$, it is expected that blood lactate will continue to rise through steady state exercise at this high intensity. Indeed, the classic work by Sjödin, Jacobs and Svedenhag (1982) found that well-trained middle and long distance runners, who trained at a running speed associated with OBLA for 20 minutes suffered increased lactates throughout the run. The mean blood lactate after 5 minutes was 4.1 mmol. $1^{-1}$, but this had risen to a mean of $5.9 \mathrm{mmol} . \mathrm{I}^{-1}$ by the end of the 20 minute run.
Furthermore, the protocols involved in the Foxdall, Sjödin and Sjödin (1996) study also compared three continuous protocols, admittedly with the same running duration as the three in this current study, but may have suffered a cumulative effect. That is to say that
the work in a previous stage could cause an increase in the blood lactate at a higher running speed, particularly when running at speeds close to or above LT. In the current study, the longer stages were more isolated, to avoid such a cumulative effect.

### 4.42 Comparing continuous protocols

It was more surprising to see the greater difference in heart rate and blood lactate response between the two continuous protocols of 3 and 4 minute stage duration. In this respect, the current finding is more in line with the findings of Foxdall et al (1996). When both the heart rate and blood lactate response at different speeds was compared between the two protocols it can be seen that the 4 minute version often gave higher readings than the shorter 3 minute version (Figure 4.2). For blood lactate 24 of the 32 ( $75 \%$ ) of the readings were on the left hand side of the line $\mathrm{Y}=\mathrm{X}$, whilst 4 were actually on this line and 4 to the left. For heart rate, the situation was similar with $24(75 \%)$ to the right of $Y=X, 5(16 \%)$ on the line and $3(9 \%)$ to the right. Not only was the general trend for the 4 minute protocol to give higher lactates than the 3 minute version, it appeared to be more the case at the higher intensity. It could well be that this is further evidence of the aforementioned cumulative effect of preceding running stages, possibly a result of cardiovascular drift (Rowell, 1969; Nadel, 1983).

### 4.43 Implications for longitudinal monitoring

Such difference in the blood lactate and heart rate measurements at each speed between protocols does have important implications if the established blood lactate profile is used for longitudinal monitoring. It is clear that exactly the same stage duration should be used in a longitudinal manner, due to the lack of agreement shown by the two methods, better graphically displayed in Figure 4.3 a and b. Indeed, not all values were within 2 standard deviations of the mean difference between the two methods. Therefore, for purposes of longitudinal monitoring, the 3 and 4 minute stage durations cannot be used interchangeably.

### 4.44 Implications for training prescription

When considering the use of the lactate profile for training prescription, in particular by use of heart rate, it can be seen that the difference between the two protocols is considerably less. The heart rate - blood lactate relationship appears more robust between the two methods, whether determined by the heart rate at a fixed blood lactate concentration (of a level in the realms of normal endurance training) of $2 \mathrm{mmol} \mathrm{l}^{-1}$, or indeed at Tlac. Table 4.5 shows that the mean heart rate at either marker is similar in both protocols. Whereas the mean difference in heart rate at each running speed between the two protocols was just over 5 beats, the difference in heart rate for the lactate related variables was 0.2 for 2 mmol. $1^{-1}$ and 1.5 for Tlac. Figure 4.4 shows that all cases fitted comfortably within 2 standard deviations of the mean difference, indicating that there is good agreement between the two methods for these two heart rate - blood lactate variables.

### 4.50 Conclusions

It is clear that the protocol used does have some bearing on the blood lactate and heart rate measurements attained in constructing an individual blood lactate profile. There appears little difference between the heart rate and blood lactate values obtained when comparing an incremental protocol with 4 minute running stages to discontinuous protocols with 6 or 8 minute stages. As the shorter incremental protocol is much shorter in overall duration, it is recommended that such a protocol be utilised. Although the comparison of heart rate and blood lactate response to 3 and 4 minute incremental protocols shows higher readings in the latter, there appears little difference in the heart rate blood lactate relationship between the two. Therefore, it is recommended that either option could be used for training prescription when using heart rate as a means of control and once again, it is likely that the shorter option is to be preferred on the grounds of time efficiency. When considering longitudinal monitoring of athletic condition by means of a
blood lactate profile, it is important that the same protocol is used on each occasion, as a different duration of running speed can affect the physiological response measured.

# 5.0 Experiment Two: Do heart rates in training workouts for Base Endurance and Maximal Steady State, elicit blood lactate values predicted from blood lactate profiles in (i) runners and (ii) rowers, in both the laboratory and the field? 

5.10 Introduction

Performance of endurance sport is determined by three key physiological variables: maximal aerobic power, lactate threshold and economy (Pate and Branch, 1992). As training adaptations are specific to the training process utilised, it makes sense that improvement of endurance performance requires attention to these three physiological variables. Unlike health related fitness, where one general intensity may be used to develop and maintain cardio-respiratory fitness (ACSM 1978; 1990), endurance athletes use a variety of training elements in an overall programme to address these three characteristics. The use of training sessions designed to elicit high but stable blood lactate levels are increasingly popular in endurance sport (Snyder, 1994). However the concept of using just one type of workout for the preparation for competition is questionable. It is more common for a range of training intensities to be used to form a balanced programme (Hartmann, Mader and Hollman, 1990; Janssen, 1987; Jones 1996a). Although the general principles of the range of training sessions within a programme are similar in different modes of exercise, there is variation in both nomenclature and classification from sport to sport.

Three general classifications are (i) long duration low / moderate intensity (ii) moderate duration, high intensity training (iii) and short duration, high intensity training (Pate and Branch, 1992). An example of a sport specific classification in the sport of rowing is categorised in Table 5.1.

Table 5.1: Federation Internationale des Societes d'Aviron_(FISA) guidelines for prescription of training intensities (adapted)

| Session | Example | Approx. \% of HR |
| :--- | :--- | :---: |
| Uax |  |  |
| Utilisation II | $60-120$ min continuous | $65-75$ |
| Anaerobic Threshold | $60-90$ min continuous | $75-85$ |
| Transportation | $2-3 \times 15$ minutes | $85-90$ |
| Anaerobic / Intensive Interval | $6 \times 1$ minute | $90-95$ |

The classification to be used in this paper is based on the input of coaches from a variety of sports who required assistance with the control of training intensity in some of the classifications. It also takes into consideration observations from peer reviewed manuscripts (Doherty, 1992; Pate and Branch, 1992; Sjödin \& Svedenhag, 1985; Shephard and Åstrand, 1992).

1. Base Endurance (BE). This is the training zone used for long duration low intensity workouts. Duration of such workouts would typically be $30-120$ minutes and the intensity is low to allow such duration. The training adaptations enjoyed from such workouts would be increased fat metabolism capability and increased oxidative capacity of skeletal muscle (Gollnick, 1988) as well as increased tolerance to heat stress (Nadel et al., 1974). It is normal to have blood lactate concentrations that are similar to resting values, so the upper limit for this zone may be determined by the first rise in blood lactate above baseline levels during incremental work, a point that has previously been referred to as lactate threshold (Weltman, 1995). In practical terms, this has shown for lactates to be below $2 \mathrm{mmol} \mathrm{l}^{-1}$ for plasma samples (Hartmann, Mader and Hollman, 1990), the equivalent of $1.5 \mathrm{mmol} \mathrm{l}^{-1}$ using the methodology employed in the present study, where whole blood lactates give lower values than plasma samples.
2. Aerobic Maintenance (AM). This is a training zone used for long duration moderate intensity workouts. Due to the slightly higher intensity, the duration of workouts is shorter, typically $30-60$ minutes on the grounds of available substrate (Hartmann, 1990). This and Base endurance workouts would comprise the major portion of the athlete's training load, without a high level of stress to either musculo-skeletal or physiologic systems (Pate and Branch, 1992).
3. Maximum Steady State (MSS). Although the definitions of this training zone are wide ranging, the principle of sustained high intensity aerobic exercise for the development of endurance is very much in vogue (Janssen, 1987). The principle involves high, but stable, blood lactates for a duration of 20-30 minutes in either continuous or intermittent activity. The physiological benefits associated with this form of exercise is increasing the speed associated with the maximum steady state and is deemed particularly important in well-trained endurance athletes whose maximum oxygen consumption may have already reached its genetically determined ceiling (Pate and Branch, 1992).
4. Speed Endurance. This training zone is that above the maximum steady state and requires a greater contribution from anaerobic metabolism (Spurway, 1992). This higher quality work is responsible for increasing maximum oxygen consumption and is classically performed in interval training to enable a greater volume of higher intensity work than could normally be maintained in constant load performance (Wenger and Bell, 1986). Also known as lactate tolerance, it is by definition an intensity above MSS, where blood lactate continues to rise, despite constant work at this high intensity.

The purpose of the following series of examinations was to see if a lactate profile can be used to prescribe training intensities for the aforementioned training zones. Two obvious means of control of training can be used. Firstly, the running speed or workload associated with given physiological conditions is one option, alternatively the heart rate associated with those same physiological conditions is another. Given that four broad training categories have been highlighted above, control of base endurance (BE) and
maximum steady state (MSS) are the aim, because the other two training zones dovetail around these i.e. aerobic maintenance fits between BE and MSS, whilst speed endurance is above the MSS. Such training prescription from lactate profiles has been shown to be possible in the past (Janssen, 1987), where training heart rates for a variety of training zones were prescribed; however, that work used fixed blood lactate concentrations to set the training intensities. The current work differs in that it uses the shape of the individual blood lactate profile to set the training intensity, rather than fixed blood lactate markers. Although accepted practice in the applied setting (Jones, 1996b), such practice has yet to be examined.
5.20 Methods

The overall experiment was sub-divided into three distinct sections:

Study 1: A comparison of heart rate and running speed to control training intensity in well-trained middle distance runners.

Study 2: An examination of heart rate controlled MSS sessions on a rowing ergometer in international junior rowers.

Study 3: A review of both BE and MSS training sessions performed by highly trained runners and rowers in their usual training environment.

### 5.21 Subjects

Study One involved ten male middle distance runners of at least county standard. Study Two examined 7 highly trained male junior rowers from the national squad. Study Three investigated training responses in 47 subjects: 11 Runners ( 5 middle distance, 4 long distance and 2 tri-athletes) and 36 rowers ( 21 junior national squad and 15 elite seniors) who performed a total of 80 monitored training sessions. The physical characteristics of the subjects are presented in Tables 5.2 and 5.3. Before any testing commenced, each
subject was fully informed of the purpose of the study, as well as the potential health risks before completing a medical history screening questionnaire approved by the tester (Appendix One) and signing a written consent form (Appendix Two).

Table 5.2 Physical characteristics of subjects in Studies One and Two (Mean $\pm$ S.E.)

| Study | Sample | Age (yrs) | Mass (Kg) |
| :--- | :---: | :---: | :---: |
| One | County standard middle distance runners (N=10) | $20.4 \pm 2.1$ | $70.6 \pm 4.6$ |
| Two | National squad junior rowers (N=7) | $17.4 \pm 0.6$ | $76.7 \pm 4.8$ |

Table 5.2: Physical Characteristics of subjects in Study Three (Mean $\pm$ SE)

| Group | N | Age (yrs) | Mass (Kg) |
| :--- | :--- | :---: | :---: |
| Runners | 11 | $25.7 \pm 1.3$ | $69.3 \pm 1.8$ |
| Junior Rowers | 21 | $17.7 \pm 0.1$ | $79.8 \pm 1.1$ |
| Senior Rowers | 15 | $26.3 \pm 1.3$ | $76.2 \pm 1.3$ |

### 5.22 Testing Structure

Study One: The runners reported to the laboratory on three occasions. The first was an incremental run on the treadmill to determine an individual blood lactate profile. From this, the heart rate and running speed associated with Tlac were determined in the same manner as in Experiment One (Chapter 4). On two subsequent occasions the runners performed a 25 minute "Threshold Session" aiming for maximal steady state (MSS) blood lactate, in a randomised order. Either a treadmill session (T25) was performed at a constant running speed associated with Tlac, or an outdoor trial (F25) was performed at the heart rate associated with Tlac. In the F25 sessions, athletes wore heart rate monitors with alarm limits to indicate the suggested training zone and data recall facility (Accurex Plus, Polar, Finland). Blood lactate was deemed stable if there was an increase in blood
lactate throughout the session of no greater than $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$. The order of tests was randomised, but testing took place on the same day of each week at the same time of day for each subject, so as to fit in consistently with the training schedule. Testing followed either a rest day or day of light running to combat the potential problem of glycogen depletion. Subjects produced both dietary recall sheets (Appendix Three) and training logs (Appendix Four) during the study period. These were used to check that hard training was not performed the previous day and that the carbohydrate and calorific consumption was sufficient for the recent energy expenditure. Checks were also made to ensure that there was consistency in both training and diet within individuals throughout the study period.

The incremental protocol was the same 3 minute continuous incremental used in Experiment One (Chapter 4, Study 2). The continuous MSS runs have been used as validation trials in normal practice when testing and monitoring athletes in this laboratory. Both runs followed a 5 minute period of jogging to warm up.

Study Two: The rowers reported to the laboratory on two occasions. The first test involved a 3 minute incremental protocol, as in Experiment One, but work was performed on an air braked rowing ergometer (Concept II, Nottingham) with power output being calculated from the deceleration of the flywheel and displayed on an LCD screen. Work rate increased by $28.4 \pm 8.4$ Watts between each stage from a starting workload of 175 Watts. In a similar fashion to the running tests in previous experiments, it was normal for each athlete to complete five work stages, but could in fact vary between four and six, depending on individual ability and training status. The incremental test was concluded once a sharp increase in blood lactate had been noticed, but did not necessarily continue to exhaustion.

The second visit (exactly one week later to fit in the same place within the training schedule) involved a 25 minute steady state MSS session on the same ergometer (E25) using the heart rate associated with Tlac from the incremental tests as the means of intensity control. Heart rate and blood lactate were measured in the same fashion as above every five minutes during brief pauses throughout the 25 min MSS session. Blood
lactates were deemed stable provided there was a rise of no greater than $0.5 \mathrm{mmol} \mathrm{l}^{-1}$ between minutes 5 and 25 .

Study Three:
Incremental testing was first performed during running or rowing and individual blood lactate profiles constructed as above. Training heart rates for MSS sessions were determined as in Study Two. Training heart rates for Base Endurance sessions were also determined by establishing the heart rate associated with the first rise in blood lactate above baseline levels during incremental exercise. This has previously been referred to as LT (Weltman, 1995).

A total of 80 subsequent training sessions performed in the athlete's normal training environment were monitored by means of heart rate and blood lactate measurements. In most cases sessions took place within one week of the incremental test, but never further than three weeks. The exact duration and sampling intervals during the sessions varied a little according to geographical and logistical factors. For runners, it was normal to analyse blood lactate at the end of a training loop within a run. The MSS sessions would last between 20 and 30 minutes and at least two blood samples would be taken to examine whether the blood lactate was stable, thus the loop would typically be between 6 and 8 minutes of running. Once again, a rise of no more than $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$ throughout the session would deem the session to be stable in terms of the lactate response.

During base endurance workouts the duration would typically be of a 60 minute duration, with measurements taken at either 30 and 60 minutes, or 20,40 and 60 minutes, depending on the distance of the training circuit. A rise in blood lactate of no more than 0.5 during the session and an absolute blood lactate of less than $1.5 \mathrm{mmol} \mathrm{I}^{-1}$ was considered appropriate to satisfy the demands of the session. This was based on both consultation with coaches and according to the plasma lactate of $2 \mathrm{mmol} \mathrm{l}^{-1}$ cited by Hartmann (1990) for utilisation / base endurance work.

Rowing sessions were all performed on a purpose built lake at the National Water Sports Centre at Nottingham. The distance of the man made lake was 2000 m . During MSS sessions the boat would pull into a landing stage within 60 seconds of completion of a repetition. A typical MSS session would be $3 \times 2000 \mathrm{~m}$ with a blood sample taken after
the first and third repetition. Base Endurance workouts would last between 60 and 90 minutes. Measurements would be taken every 4 or 8 km .

A total of 30 sessions by the 11 runners were analysed; 11 were base endurance workouts and 19 were MSS workouts. A total of 50 sessions were performed by the 36 rowers, of which 27 were MSS workouts and 23 base endurance workouts. It is clear, therefore, that some athletes performed more than one training session within the project.

In all cases athletes wore heart rate monitors for intensity control and data recall in the same fashion as the F25 trial in Study One. In all laboratory trials, the temperature was between $18-22^{\circ} \mathrm{C}$.

### 5.23 Data and Statistical Analysis

Descriptive statistics were used to describe the physical characteristics in all three studies. Repeated Measures ANOVA was used in studies one and two to analyse the change in heart rate and blood lactate response through the duration of the 25 minute MSS sessions. In study two, the percentage decline in metres rowed during the E25 sessions were calculated for each subject and a mean value for the group percentage decline was also determined. In study three, comparison was made of the blood lactate response observed during the training sessions compared to that predicted from the incremental tests.
5.30 Results

Study One: The mean heart rate and blood lactate values at 5 and 25 minutes during the T25 and F25 MSS sessions are shown in Table 5.4, where it can be seen that the two methods of intensity control resulted in significantly different trends in blood lactate. This is further illustrated in detail in Figure 5.1a and 5.1b. In all cases during the F25 session, athletes adhered to their prescribed heart rates, whilst during T25 runs, there was a significant rise in heart rate between 5 and 25 minutes ( $\mathrm{P}<0.05$ ). It was noted that the
mean absolute lactate at 5 minutes was significantly higher than the mean lactate value recorded at Tlac during the incremental test: $4.1 \pm 1.4 \mathrm{mmol} .1^{-1}$, as compared with $2.3 \pm$ 0.51 mmol. $\mathrm{l}^{-1}$. However, the mean blood lactate was seen to drop and then stabilise through the 25 minute run with the stable heart rate. During the treadmill run, the increase in mean heart rate gave rise to an increase in the mean blood lactate throughout the session.

Table 5.4 Mean $\pm$ SE Heart Rate and Blood Lactate values during MSS sessions ( $\mathrm{N}=10$ ).

| Time (min) | F25 HR <br> b.min | F25 La <br> mmol. $^{-1}$ | T25 HR <br> b.min $^{-1}$ | T25 La <br> Mmol.I $^{-1}$ |
| :---: | :---: | :---: | :---: | :---: |
| $\mathbf{5}$ | $174 \pm 9$ | $4.1 \pm 1.4^{*}$ | $170 \pm 9$ | $1.9 \pm 0.4$ |
| $\mathbf{2 5}$ | $177 \pm 7$ | $3.7 \pm 1.2$ | $182 \pm 10^{* *}$ | $2.4 \pm 0.8$ |

* significantly greater than incremental Tlac $\mathrm{P}<0.05$. ** significantly different than $5 \mathrm{~min} \mathrm{P}<0.05$

Figure 5.1: Mean Heart Rate and Blood Lactate values during MSS sessions (a) on the treadmill and (b) in the field ( $\mathrm{N}=10$ ).
(a) T 25
(b) F25



When individual cases were analysed blood lactate was noted to rise in 6 out of 10 cases during the treadmill runs. This is shown in Figure 5.2(a) where non-steady cases are shown in the lower half of the grid. Four of these six non-steady sessions in the T25 sample, were predicted from the observed heart rate during the sessions and are plotted on the left side of the grid. The 2 cases where predicted blood lactate was stable from the heart rate response are plotted on the right hand side of the grid in the lower section. Figure 5.2 (b) shows that in 9 out of 10 cases the predicted steady state lactates from the controlled heart rate response actually gave observed steady state blood lactate. These nine are plotted in the upper right hand zone of the quadrant, whereas the one case that gave non steady state blood lactate through the session is plotted in the lower half of the grid.

Figure 5.2: A comparison of predicted and observed blood lactate responses during the (a) T25 and (b) F25 MSS sessions.
(a) Treadmill MSS sessions (T25)
(b) Field MSS sessions (F25)


Study Two: During the E25 MSS sessions, all rowers were able to maintain the heart rate prescribed from Tlac during their incremental tests, thus heart rate was stable. The mean blood lactate throughout the E25 session was higher than the mean blood lactate value of $3.7 \pm 1.0$ mmol. $1^{-1}$ at Tlac during the incremental tests, but was stable. A trend of a slight drop in mean blood lactate was noticed through the E25 session, but this was not statistically significant (Table 5.5 and Figure 5.3). Furthermore, to keep the heart rate at a stable level throughout the E25 sessions, the mean work intensity (shown by the metres rowed in each 5 minute section) declined significantly between 5 and 25 minutes. The mean decline in metres rowed in percentage terms was $5.4 \%$. In only one out of the seven E25 sessions did blood lactate rise by more than $0.5 \mathrm{mmol} \mathrm{I}^{-1}$ between 5 and 25 minutes. In this case the blood lactate rose from 5.3 to 6.2 between 5 and 25 minutes. In one other case, blood lactate dropped significantly through the session, starting at 6.0 and declining steadily to 3.0 . The percentage decline in metres rowed in this subject was $11.4 \%$, much greater than the mean decline for the group of $5.4 \%$. To put this further into context the range in percentage decline for the other six subjects in the group was $3.1 \%$ to $5.1 \%$.

Table 5.5: Mean ( $\pm$ SE) HR La and distance rowed through the E25 sessions ( $\mathrm{N}=7$ )

| Time (min) | HR $\left(\mathbf{b} \cdot \mathbf{m i n}^{\mathbf{- 1}}\right)$ | La $\left(\mathbf{m m o l} \mathbf{.}^{\mathbf{- 1}}\right)$ | Distance $(\mathbf{m})$ |
| :---: | :---: | :---: | :---: |
| $\mathbf{5}$ | $174 \pm 5.4$ | $5.3 \pm 0.7$ | $1343 \pm 20$ |
| $\mathbf{1 0}$ | $178 \pm 4.7$ | $5.6 \pm 1.3$ | $1327 \pm 31$ |
| $\mathbf{1 5}$ | $177 \pm 5.1$ | $5.2 \pm 1.9$ | $1284 \pm 46$ |
| $\mathbf{2 0}$ | $177 \pm 5.9$ | $4.8 \pm 1.9$ | $1279 \pm 45$ |
| $\mathbf{2 5}$ | $177 \pm 5.5$ | $4.6 \pm 2.0$ | $1265 \pm 41^{*}$ |

[^0]Figure 5.3: Mean ( $\pm$ SE) Blood lactate and distance rowed during each 5 minutes of the E25 sessions ( $\mathrm{N}=7$ ).


Study Three: Unlike in the laboratory sessions in the previous two studies, not all cases during this analysis saw the target heart rates achieved during monitored training sessions. It was seen that a total of 16 training sessions ( $20 \%$ ) were performed above the target heart rates set from the incremental tests. Only 3 training sessions out of the total of $80(4 \%)$ did not elicit blood lactates that would have been predicted; therefore $96 \%$ of the sessions were correctly predicted as steady state or non-steady state. The prediction failure to achieve the required level of blood lactate came in the rowing MSS sessions (2) and the running MSS session. In no case of the BE training sessions did the observed blood lactate exceed that predicted from the incremental test.
A summary of the findings in running and rowing sessions can be seen in Figures 5.4 and 5.5 respectively.

Figure 5.4: A comparison of the observed and predicted blood lactate responses during training sessions in runners ( $\mathrm{N}=3 \mathrm{O}$ ).
(a) Base Endurance Sessions ( $\mathrm{n}=11$ )
(b) MSS Sessions ( $\mathrm{n}=19$ )


It can be seen that during base endurance (5.4 (a)) only one of the 11 training sessions saw blood lactate increase significantly through the session. Indeed, this was predicted from the heart rate response; in effect, the athlete had been working above the target heart rate. This case is plotted in the bottom left hand part of the grid, where the observed nonsteady state lactate was predicted from the monitored heart rate response. In the MSS sessions (Figure 5.2 (b)), 12 training sessions produced steady state blood lactate values which were predicted from the heart rate response. One case showed steady state blood lactates where the subject exceeded the target heart rate. Six sessions showed non-steady state blood lactate through the session, but this was again predicted to be the case with the monitored heart rate response.

Figure 5.5: A comparison of the observed and predicted blood lactate responses during training sessions in rowers ( $\mathrm{N}=50$ ).
(a) Base Endurance sessions ( $\mathrm{n}=23$ )

(b) MSS Sessions ( $\mathrm{n}=27$ )


In the rowing training, 23 base endurance sessions were monitored and 21 showed lactates that gave stable blood lactates that were predicted from the heart rates response (Figure 5.5a). Two sessions gave lactates that increased through the session and were higher than those normally expected in base endurance workouts, but this was predicted from the fact that the athletes were working above their prescribed training heart rates. In the MSS sessions (Figure 5.4 (b)), 25 sessions gave blood lactate levels that were predicted from the heart rate response. Four of these gave non-steady blood lactate levels, where the athletes had exceed their prescribed heart rate zone. Two subjects displayed stable blood lactates, despite working at a heart rate level above their prescribed training zone.

### 5.40 Discussion

5.41 Differences in blood lactate between incremental tests and constant load.

It was observed during the MSS sessions in the first two studies that subjects were always able to maintain the heart rates or work intensities prescribed from their incremental tests. Although this has previously been shown to be the case with Individual Anaerobic Thresholds, it is not always the case when fixed blood lactates, from incremental protocols are used to control constant load performance (Stegmann and Kindermann, 1982). The use of fixed blood lactates concentrations to prescribe training intensities was favoured by earlier German research (Heck et al., 1985) but is questionable, particularly in well trained endurance competitors, where the absolute blood lactate level at MSS is seen to be low.
In this current research, the absolute blood lactate level at Tlac has been seen to be lower than 4 mmol. ${ }^{-1}$ at $2.3 \mathrm{mmol} . \mathrm{l}^{-1}$ in the runners in study one and 3.6 in the junior rowers. The fact that the junior rowers have a higher mean blood lactate at Tlac than the older runners, is not surprising. It would seem that greater levels of endurance training tends to lower the absolute concentration of blood lactate during training, due to the increased oxidative capacity as a result of training and the associated greater level of lactate removal at tissue level (Donovan and Brooks, 1983; MacRae et al, 1992).
The fact that the mean absolute blood lactate at Tlac is lower than during the sessions at the same constant running speed is not surprising either. Urhausen et al. (1993) demonstrated that blood lactates during constant load performance produced higher lactates than at the same level during incremental work. In well-trained runners the mean blood lactate at their IAT during an incremental test was $2.44 \mathrm{mmol} . \mathrm{I}^{-1}$, similar to the level of 2.3 mmol. $1^{-1}$ at Tlac in the runners in this study. During continued running at the same speed, blood lactate was 3.05 and $3.69 \mathrm{mmol} .1^{-1}$ at 15 and 45 minutes of running respectively in the Urhausen et al. (1993) study. In well-trained cyclists they found a mean value of $3.46 \mathrm{mmol} \mathrm{I}^{-1}$ at IAT during the incremental test, but 4.20 and 4.16
mmol. $1^{-1}$ at 15 and 45 minutes of cycling respectively. Similar findings had been documented by Orok et al. (1989) in investigating moderately trained cyclists.
5.42 Comparing heart rate and workload or running speed as a means of training control In the current work, during the treadmill T 25 sessions the mean blood lactate started lower than, and increased through the session to above, the blood lactate level associated with Tlac in the incremental tests. At the same time, the heart rate also increased significantly. In the outdoor F25 session, where heart rate was controlled, the mean blood lactate started high and decreased throughout the 25 minutes. A similar pattern was observed during the E25 sessions performed by the rowers where, controlled by heart rate, the workload and blood lactate declined as the session progressed. Such a phenomenon has also been observed in cross country skiers who also suffered a decline in workload during heart rate controlled sessions on a treadmill (Kindermann, 1979). One likely causative factor suggested is that of cardiovascular drift; a rise in core temperature and drop in plasma volume seen in continuous exercise (Nadel, 1977, Rowell, 1969).

### 5.43 Pacing strategy in MSS sessions

The high blood lactate levels seen in the initial phases of heart rate controlled sessions in the runners (F25) and rowers (E25) is possibly a cause of the athletes working at a level above Tlac in the early stages to raise heart rate initially. Although the running speed was not measured in the F25 trial, such an explanation seems plausible in light of the high workload seen in the early stages of the E25 rowing trial and the subsequent decline. The mean intensity of the first 5 minute portion of the E25 session was equivalent to 4.48 $\mathrm{m} . \mathrm{sec}^{-1}$, which is certainly above the work intensity associated with Tlac in the incremental tests. During the last 5 minute portion the mean intensity declined to 4.21 $\mathrm{m} . \sec ^{-1}$ which is just below that workload associated with Tlac in the incremental tests. It would be of interest to see if a more circumspect pacing strategy during the early stages of the heart rate controlled workouts, would give lower initial lactates and such values
more in line with the lactate level seen in the incremental test. This is an aspect that would warrant further research.

It was noted that during the treadmill sessions, the target heart rate was generally not achieved until after the first blood sample, so it may take a longer than 5 minutes to reach a steady state plateau in heart rate and blood lactate during constant running velocity. In the Kindermann (1979) study, peak lactates were seen after 10 minutes of the 30 minute heart rate controlled workouts, which is the same as in the present E25 study. It is interesting to note that not only did blood lactate drop through time in the Kindermann (1979) controlled study, there was also a drop in oxygen consumption, but this was not statistically significant. Guidance to athletes therefore, may be to take longer in reaching the target heart rate. This may keep the training intensity slightly lower at the start, rather than to start off at an intensity above that which produces steady state lactate in a quest to quickly reach the target heart rate.

### 5.44 Warm up before MSS sessions

The athletes in the present study were allowed warm up before the MSS sessions, so it is not possible that a lack of warm up is a causative factor. However, the intensity and duration of warm up is another factor that may warrant further investigation.

### 5.45 Blood lactate throughout MSS sessions

The results of the mean blood lactate levels during the intensity controlled sessions (T25) compared to the heart rate controlled sessions (F25) and (E25) may suggest that the intensity method is best at controlling MSS lactates. The mean absolute lactates are lower in the T25 and closer to the level at Tlac in the incremental sessions. In the heart rate controlled sessions during both exercise modes, the blood lactate initiated at a higher level but is then seen to drop. It may well be that the work intensity needs to drop to enable this to happen.

The mean blood lactate was observed to increase through the T25 session and just fits the criteria for MSS, as the mean increase is $0.5 \mathrm{mmol} \mathrm{I}^{-1}$. In the heart rate controlled sessions, the mean blood lactate was stable or even falling. This point is of relevance,
because if the blood lactate is increasing, the implication is that the athlete is not in steady state. The classic work by Sjödin, Jacobs and Svedenhag (1982) examined a subject group of elite middle and long distance runners, assessing longitudinal change in aerobic fitness in response to the addition of treadmill training at OBLA. The authors found that those athletes who had the greater increase in blood lactate during these sessions enjoyed the least longitudinal improvement in performance. Thus it is possible to over-train using this method of conditioning and some element of control is of use. This has also been supported by the work of Noack cited in Heck et al. (1985) who trained middle distance runners using lactate controlled treadmill sessions. Only after reducing the intensity of these workouts, with no change in volume, could an abrupt beneficial longitudinal change in their anaerobic threshold and competitive performance be observed.

### 5.46 Analysing individual cases in Treadmill MSS sessions

Examination of individual MSS cases in the present study with runners is more revealing than observation of group means. Of the 10 sessions controlled by intensity (T25 sessions), only 4 gave predicted and observed steady state blood lactates. The other 6 cases produced non-steady state lactates through the session, although 4 of these were predicted by analysing the associated heart rate response (Figure 5.2a). This, however, may be little compensation to the athlete and coach, striving to achieve steady state lactate in the training environment.

Furthermore, the other 2 cases produced non-steady state lactates that could not be predicted on the basis of observed heart rate. When the MSS sessions controlled by heart rate were examined, 9 of the 10 sessions produced observed and predicted steady state lactates (Figure 5.2b). The one subject who did not was an athlete that had the onset of an upper respiratory tract infection, which developed fully several days later. However, this subject was not excluded from the study, because it seemed reasonable that the heart rate would be elevated and would dictate lower training intensity and still maintain steady state blood lactate. The area of the heart rate blood lactate relationship under extreme conditions, such as temperature, altitude and infection, warrants further investigation.

### 5.47 Analysing individual cases in rowing ergometer MSS sessions

In the rowers in the E25 sessions, only one subject demonstrated a blood lactate rise by more than $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$, the criteria set for steady state. Once again, therefore, when individual subjects were analysed, using heart rate from an incremental test seems a valid means of controlling steady state conditions in the training sessions. Not only does heart rate seem a more valid means of intensity control from the evidence above, consideration should also be given to the practicalities. It is unlikely that athletes are going to perform all training sessions on a treadmill or rowing ergometer under laboratory conditions. This leaves the option of controlling the running or rowing speed in the training environment, which is difficult.

### 5.48 Practicalities of pace judgement

It is unlikely that runners are going to adhere to running on a track, or even a few training courses with accurately measured distances. In such a field situation, good pacing would be a pre-requisite for controlled training sessions. This is something that cannot always be guaranteed, because running level pace is a skill that varies between athletes. Furthermore, constant feedback would have to be provided regarding the pacing during each training event, which may not always be possible. Even if this was the case, other problems arise. It has clearly been demonstrated that other factors alter the physiological response to running. Heck et al. (1985) have shown quite clearly that the running surface alters the blood lactate response to running at set speeds. More recently Coen et al. (1991) have shown that well trained middle and long distance runners demonstrated different lactate response to training sessions in good as opposed to poor environmental conditions. This would be the same in rowers, where a change in wind and water conditions, would make a dramatic difference to the relative work intensity required to maintain a given boat speed.

Such problems can be reduced using heart rate as a means of control, as this method like blood lactate monitoring - gives an indication of the relative intensity a given athlete is working at during a given workload or training speed. As mentioned above, it is clear
that further work is required to see how robust this heart rate - blood lactate relationship is under a variety of given conditions. However, evidence suggests that heart rate will reflect changes in environmental conditions and also cater for cardiovascular drift (Snyder et al., 1994), neither of which would be reflected when intensity or running speed is the controlling factor.

### 5.49 Decreasing blood lactate throughout training sessions

It is not clear whether the drop in mean blood lactates is desirable or not in these sessions. In the one subject where blood lactate dropped significantly through the E25 session, the decline in workload through the session was far greater than the average for the group. The drop in blood lactate from $6 \mathrm{mmol} . \mathrm{l}^{-1}$ to $3 \mathrm{mmol} \mathrm{l}^{-1}$ was by far the greatest drop in blood lactate of any subject in all of the studies here. The blood lactate at Tlac in the incremental test was $2.8 \mathrm{mmol} .1^{-1}$, so the session finished with blood lactate close to this level. The heart rate was virtually unchanged from 5 minutes to the end of the session at 173. Despite the large decline in workload through the session, it is clear that the athlete did not start abnormally fast. The average speed during the first 5 minutes was 4.56 $\mathrm{m} . \mathrm{sec}^{-1}$, which was remarkably similar to the intensity seen at Tlac in the subject's incremental test ( $4.58 \mathrm{~m} . \mathrm{sec}^{-1}$ ).
The wider issue of whether declining blood lactates during heart rate controlled sessions can be optimal, still needs further investigation. Clearly, the fact that there is no increase in blood lactate is desirable, but the question is then left as to whether the session is performed at a truly maximal steady state. It is possible that in some cases a truly maximal state did not exist, in which case the session could have perhaps been performed at a higher intensity. However, because subsequent runs were not performed at slightly higher intensities, it is impossible to judge. The study by Urhausen et al. (1993) examined a series of continuous runs in response to incremental work, using their IAT. The runs were performed at $85,95,100$ and $105 \%$ of IAT intensity. They found that despite stable lactates at $100 \%$ IAT, most subjects could not complete the work at $105 \%$. They felt that, for methodological reasons, it is hardly possible to delineate the range of MSS more precisely than steps of $5 \%$.

In 3 of the 4 subjects in the present study where both predicted and observed non-steady state conditions were noted during the T25 sessions, the heart rate was either 2 or 3 beats above the target range set for the F25 sessions. This small increase in heart rate above the target zone, did give rise to non-steady blood lactate conditions. When the heart rate was controlled just these 2 or 3 beats per minute lower, stable blood lactate was observed. In these subjects, there was little scope for greater intensity with stable lactates. However, this cannot be guaranteed to always be the case.

### 5.410 Summary of studies one and two

What is clear from the first two studies, is that when individual cases were analysed, by evaluating the change in blood lactate through the sessions (Figures 5.2a and 5.2b), heart rate was the better means of controlling steady state blood lactates than running speed. It may be that the methodology errs on the side of caution, but given that the avoidance of an increase in blood lactate is the aim, heart rate is the better means of control.

### 5.411 Adherence to target heart rates in training

When examining the 80 training sessions outside of laboratory conditions in study three, it can be seen that the process is successful in predicting the presence or absence of steady state blood lactate conditions on the basis of heart rate during the sessions. The first point of note, however, is that unlike the laboratory sessions in studies one and two, where all athletes performed training at the prescribed workload, speed or heart rate, 16 training sessions (20\%) were observed to be above the individuals' recommended target heart rates. Although the athletes were educated to the benefit of such control by both physiologist and coach, it is clear that some athletes still have the tendency to do work above the suggested heart rate range.

### 5.412 Predicting training lactates from observed heart rate

There was a high rate of success (96\%) in predicting the presence or absence of steady state blood lactates on the grounds of heart rate noted and reference to the incremental test. Interestingly, this is a similar success rate (in terms of percentage) to that seen by

Urhausen et al. (1993), who used different methodology in their testing procedure (establishing IAT). All their continuous tests at $100 \%$ of IAT were performed on the treadmill, controlled by speed, rather than using heart rate as a means of control in the field. The authors did find that when $105 \%$ of IAT was used for the continuous workouts, more than half of the subjects showed progressive acidosis and a premature break off in the sessions. Their $96 \%$ success rate should be put in the context that only 14 sessions examined in that study. Their findings did however concur with previous research by Schnabel et al. (1982) and Jacobs and Mcllelan (1988), which both used IAT from incremental tests to set the intensity for continuous work.

When heart rate has been used to control the intensity to achieve steady state blood lactates in the field, other methods have not been as successful. In an examination of elite speed skaters Foster et al., (1995) compared predicted and observed responses during steady state training, on the basis of a direct blood lactate profile (in a similar fashion to the current study), using relative velocity and \%HRmax. The authors found an $81 \%$ success rate of prediction in the direct blood lactate profile method, as opposed to $78 \%$ and $68 \%$ in the other two methods respectively. The $81 \%$ accuracy in their methods also included, in the author's terms, "conservative" mistakes. This is where blood lactate was steady state, but predicted non-steady. This was deemed more favourable than predicting steady state blood lactate when a non-steady state exists. In the \%HRmax method, the authors found the mistakes were not only greater (32\%), they were also of a more random nature.

Previously, Snyder et al. (1994) had examined the ability to use \%HRmax in well-trained runners and cyclists, although not as highly trained as the subjects in the present study. They found $76 \%$ and $81 \%$ accuracy in the runners and cyclists respectively and concluded that the simple heart rate models may predict MSS with sufficient accuracy. They also stated that this was successful, given the relative simplicity for athletes to define their maximal heart rate. However, this process is brought into question by the work of Ingjer (1991), who investigated a series of methods and warm up strategies used by well-trained cross-country skiers in establishing maximal heart rate. The finding was that the maximal heart rate recorded could vary significantly depending upon the protocol
used to establish such an index. Maximal testing also requires great motivation, which is difficult to measure in athletes, so there is always the question as to whether the heart rate achieved, is a genuine maximal value.

### 5.413 Analysing Base Endurance workouts

The present study differs from previous work in that it has analysed sessions other than those attempting to achieve MSS. The BE workouts often occupy the greatest training volume in the athletes year (Hartmannn, Mader and Hollman, 1990; Pate and Branch, 1992), yet have received little attention in the published literature. There are two possible reasons for this. Firstly, MSS sessions have been the focus of most attention because it is believed that these are optimal in enhancing aerobic fitness (Jacobs, 1986). Even if this is not the case, these sessions should at least represent the most time effective way of integrating volume and intensity (Snyder et al., 1994). The second reason that BE sessions may not have received much attention is the difficulty in setting the criteria for both the prescription from the incremental test and the evaluation of the workouts themselves.

The first rise in blood lactate from baseline levels during an incremental test is less controversial than establishing Tlac and has indeed previously been referred to as Lactate Threshold in some work (Weltman, 1995). If the blood lactate is not increasing above baseline levels, then fat metabolism is predominantly responsible for the energy supply (Spurway, 1992). This is important, because if greater intensity is used, there is restriction in the volume of training that can be performed in a training week, due to the limited amount of supply of carbohydrate (Hartmann, Mader and Hollmann 1990). Judging whether the blood lactates seen in the field are appropriate is a little more difficult. It makes sense that there should be little increase in blood lactate through the session. Using the increase of more than $0.5 \mathrm{mmol} .1^{-1}$ as in the MSS sessions may be too generous, but to lower the allowable increase in blood lactate through the session would bring the level too close to the limits of measurement error of the analyser. To rigidly use fixed blood lactate levels is also questionable, as it is clear that a given blood lactate concentration can represent quite different relative intensity in two different athletes.

This could be due to genetic factors such as muscle fibre composition and training status (Ivy et al., 1980), or glycogen status within the muscle (Davis, Williams and Cherrington, 1984; Hughes, Turner and Brooks, 1982).

### 5.414 Criteria for MSS sessions

The criteria for steady state lactate in the MSS sessions in the current work could also be questioned. An increase of no more than $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$ through the session has been set. The work by Urhausen et al. (1993) used an increase of 1 mmol. $1^{-1}$ between minutes 10 and 30 , which cited German research that has used both 0.2 and $1 \mathrm{mmol} .1^{-1}$ through the session. To have the criteria set to $0.2 \mathrm{mmol} . \mathrm{l}^{-1}$ would require incredible precision in measurement and leave little room for fluctuations that often appear during the 100 or so sessions here with steady state conditions. The use of $1 \mathrm{mmol} .1^{-1}$ offers a wide range when put in the context of the low lactate levels often seen both during training and exhaustion in highly trained endurance athletes. Unpublished findings from the subject pool used in the present study have seen a blood lactate level of 2 at Tlac and as low at 4.6 mmol. $1^{-1}$ at exhaustion. There is thus a small gap between Tlac / MSS blood lactate and exhaustion if a rise of $0.9 \mathrm{mmol} .1^{-1}$ through a MSS session is acceptable. It may well be that in less trained athletes, or sports people not of an endurance nature, that $1 \mathrm{mmol} . \mathrm{l}^{-}$ ${ }^{1}$ is a more acceptable limit.
5.415 The volume, duration and balance of training in the schedule Another factor open to question is what balance of the types of training session is optimal for improvement in athletic performance. Also the optimal length of session, be it BE or MSS, or others not addressed in this scheme of study, warrants further examination. It is normal for the athletes in this study to perform MSS sessions for about 25 minutes, or in repetitions totalling about the same. BE workouts tend to last a minimum of 1 hour and can last double this time. Clearly the latter depends upon the demands of the event being trained for (a marathon runner will typically do more and longer BE workouts than a middle distance runner). As yet, the duration of the workouts has been coach driven and is yet to be examined empirically.

The problem, as with many of the questions raised above, is that it is difficult to do the depth of analysis required to make definitive statements about the correct way to train. Elite athletes are rarely prepared to alter their training for the purpose of research. This leaves two options: either observational research, as seen in study three, or examining populations more willing to adapt their training for the sake of research. The problem with the latter is that findings from one population cannot always be transferred to another. Indeed this is one of the factors that has caused much controversy and contradiction in the literature related to the concept of anaerobic threshold.

### 5.50 Conclusions

In summary, it has been noted that heart rate may be a better means of maintaining steady state lactates during training for MSS, than running speed - particularly when individual sessions are analysed and plotted in grids (Figures 5.2a and 5.2b). The methodology of prescribing heart rates from a blood lactate profile has been demonstrated to be an accurate means of controlling steady state blood lactate in MSS workouts and a valid means of controlling blood lactate in BE workouts. Such control may be conservative in nature, but given that avoidance of an increase in blood lactate was the aim, the methodology was considered appropriate in this instance.

### 6.0 Experiment Three: How do blood lactate profiles vary between running and cycling in Tri-athletes?

### 6.10 Introduction

It is clear that energy demands vary between different exercise modes (ACSM, 1991). Athletes who compete in activities which require two exercise modes (duathlon: cycling and running) or three exercise modes (triathlon: swimming, cycling and running) may well require training prescription specific to each exercise mode. For some time, it has been established that peak values for oxygen consumption are higher during running than cycling (Diaz et al. 1978), as a result of higher cardiac output at both submaximal and maximal levels in running (Hermanssen, Ekblom and Saltin, (1970). More recently it has been shown that cycle ergometry in triathletes produced $\dot{\mathrm{V}}_{2^{\max }}$ values $3-6 \%$ lower than those seen on a treadmill (Kohrt et al., 1987; O'Toole et al., 1987). This is a smaller difference than reported for single sport athletes, where the difference is between $9-11 \%$ (Åstrand and Rodahl, 1986).
Specificity of training is important for optimal development of triathletes (Daniels, 1992) and that training should be aimed at improving lactate threshold in both running and cycling (Kohrt et al., 1989). It is useful to establish whether there is a consistent difference in the heart rate blood - lactate relationship across these two exercise modes. If a consistent difference in heart rate for various training zones exists, it may be that only one incremental test is necessary to predict the training heart rate for both exercise modes. The purpose of this study, therefore, was to examine the heart rate and blood lactate response during incremental tests of running and cycling in athletes involved in multi-event competition.

### 6.21 Subjects

Sixteen male athletes were examined in this study during routine physiological assessment. All were competitive athletes in triathlon ( $n=14$ ) or duathlon ( $n=2$ ) with between 2 and 8 years of competition experience. A summary of the physical characteristics of the subjects is provided in Table 6.1. Each subject was informed of the fact that data would be used in this study, with their right of privacy retained. Each subject also completed a medical history questionnaire (Appendix One), recent training $\log$ (Appendix Four), dietary recall sheets (Appendix Three) and a written consent form (Appendix Two).

Table 6.1: Phvsical Characteristics of subiects ( $\mathrm{N}=16$ ).

|  | Age (years) | Height (m) | Mass (Kg) | Years of experience |
| :---: | :---: | :---: | :---: | :---: |
| Mean | 30.30 | 1.79 | 76.43 | 5.36 |
| S.E. | 1.22 | 0.01 | 1.29 | 0.58 |

### 6.22 Testing Structure

Subjects performed both a cycling and running incremental test in a randomised order. The tests were performed one week apart, on the same day of each week at the same time appointment, so as to fit consistently within training programmes. On each occasion, testing followed either a rest day of a day of light training, in each case confirmed by written training logs.

### 6.23 Testing Protocols

Running Test: A three minute continuous incremental protocol was used on a running machine as in previous experiments (Chapter 4, study two).

Cycling Test: A continuous incremental protocol was used with athletes riding their own bicycle on a Kingcycle (EDS Portaprompt, High Wycombe, UK) rig interfaced to a PC with workload in watts calculated by the deceleration of the flywheel. A controlled warm up of five minutes was given. A series of three minute workloads followed with 20 Watt increments. As with other tests in the present series of investigations, athletes performed at least four stages, but as many as seven, depending upon individual ability and current level conditioning. The level of the initial workload was based either upon previous testing experience or recent training / racing performances.

### 6.24 Measurements

Running Test: At the end of each running speed, subjects stood astride of the moving treadmill belt whilst heart rate was recorded via radio telemetry (Polar, Finland). Blood samples were taken from the earlobe for subsequent lactate determination from whole blood (Analox GM7, Hammersmith). The pause for sampling was usually 20 seconds, but in no case did this exceed 26 seconds. The blood analyser was calibrated using both 3 and 8 mmol. $1^{-1}$ standard solutions and also checked with a Quality Control Serum (concentration 2.3-2.5 mmol. $1^{-1}$ ).
Cycling Test: During the last ten seconds of each workload, blood samples were taken and analysed in the same manner as the running test and heart rate was recorded in the same fashion. Once the blood was collected the athletes moved straight to the next workload, by either increasing cadence or selecting an alternative gear.

### 6.25 Data and Statistical Analysis

Individual blood lactate profiles were plotted against both running speed, or cycling workload, and heart rate, with points joined via linear interpolation. The heart rate associated with $2 \mathrm{mmol} . \mathrm{l}^{-1}$ of blood lactate was established, as was the heart rate associated with lactate threshold (Tlac) as in experiment one. Mean values and standard deviations were calculated for the heart rate at $2 \mathrm{mmol} . \mathrm{l}^{-1}$ and Tlac for each mode of exercise, as was the mean difference between those modes. For statistical comparisons, a paired t-test was used using SPSS with a level of significance of $\mathrm{p}<0.05$.

The mean heart rate at a blood lactate level of 2 mmol. $1^{-1}$ and at Tlac for both exercise modes are shown it Table 6.2. In both cases the mean heart rate was significantly higher during running than cycling. At $2 \mathrm{mmol} .1^{-1}$ the mean difference was $19.1 \pm 12.0$, with a range of -3 to 44 beats. At Tlac the mean difference was $13.4 \pm 7.0$ and a range of 0 to 26 beats.

Table 6.2: Mean $\pm$ (SE) heart rate at 2 mmol. $\mathrm{I}^{-1}$ and Tlac during running and cveling incremental tests ( $\mathrm{N}=16$ ).

| Heart Rate | Cycling (b.min ${ }^{-1}$ ) | Running (b.min ${ }^{-1}$ ) |
| :--- | :---: | :---: |
| 2 mmol. $\mathbf{I}^{-1}$ | $141.4(3.3)$ | $160.5(2.5)^{*}$ |
| Tlac | $152.6(2.8)$ | $165.9(2.0)^{* *}$ |
| 6.4, d.f $=15, \mathrm{p}<0.05$ | $* * \mathrm{t}=7.6$, d. $\mathrm{f}=15, \mathrm{p}<0.001$ |  |

At both 2 mmol. $1^{-1}$ and Tlac, the relationship between the cycling and running demonstrated moderate correlations of $\mathrm{r}=0.50$ and $\mathrm{r}=0.78$, respectively. The relationship between the heart rate at each of these markers in the two exercise modes is illustrated in Figures 6.1 a and b . Furthermore, the range in the difference in heart rate between the two exercise modes at Tlac is illustrated in Figure 6.2.

Figure 6.1: Relationships between heart rate (beats. $\mathrm{min}^{-1}$ ) in running and cycling at (a) 2 mmol. $\mathrm{I}^{-1}$ and (b) Tlac during incremental tests.

(a)

Running
Figure 6.2: Histogram demonstrating the range of difference in heart rate at Tlac.


Mean difference in HR between exercise modes (beats. $\mathrm{min}^{-1}$ )

### 6.40 Discussion

6.41 Differences in heart rate between cycling and running

The higher mean heart rate during running at Tlac and $2 \mathrm{mmol} . \mathrm{l}^{-1}$ was expected in light of the previously published research comparing the difference in maximal heart rate on cycling and running in triathletes. O'Toole et al., (1989) found that maximal heart rates in cycling are $96 \pm 5 \%$ of treadmill maximal heart rate. However, there has been little other work previously published examining differences in submaximal or training heart rates between the two exercise modes in triathlon.

Although there has been investigation into differences in the workload and blood lactate concentrations at maximal lactate steady state in various exercise modes (Beneke, 1996), this did not address the disciplines in triathlon. For triathletes, Schneider, La Croix and Atkinson (1990) found ventilatory threshold (VT) to be $61-81 \% \dot{V}_{2_{2 \max }}$ in cycling and $70-72 \% \mathrm{VO}_{2 \text { max }}$ in running, whilst Khort et al. (1987) found lactate threshold to be between $72-88 \% \dot{\mathrm{~V}}_{2 \text { max }}$ in cycling and $80-85 \% \dot{\mathrm{~V}}_{2 \text { max }}$ in treadmill running.

The change in lactate threshold and maximal oxygen consumption during both exercise modes in response to either running or cycle training has also been examined (Pierce et al. 1990). It was seen that run training boosted both running and cycling lactate threshold, whilst cycle training boosted cycle lactate threshold but left the same marker in running unchanged. As maximum oxygen consumption was increased as a result of training in either exercise mode, the specificity of the training response is emphasised.

The lower heart rate during cycling in the present study would reflect the lower oxygen consumption and cardiac output during this exercise mode. This would concur with the findings of Jacobs and Sjödin (1985), who found that a blood lactate level of $4 \mathrm{mmol} \mathrm{I}^{-1}$ (OBLA) occurs at a higher steady state oxygen consumption during treadmill running than cycling. This would be due to the smaller demand by the working musculature in the cycling mode as opposed to running. The clear difference in heart rates for the two physiological markers of $2 \mathrm{mmol} . \mathrm{l}^{-1}$ and Tlac in the present study, indicate that for the same physiological effect, training heart rates can be different. However, despite the
large difference in mean heart rate for the group, Figure 6.2 shows a wide range in the extent of the difference in heart rate between the two exercise modes at Tlac.

### 6.42 An exceptional case

In one particular athlete, the heart rate at Tlac was the same in both exercise modes. This athlete was the highest trained of the group; a former world champion triathlete, whose strongest discipline is cycling. This subject's training diary revealed nearly $60 \%$ of his training volume taken by cycling, which is higher than normal for triathletes, who typically split training volumes to $50 \%$ cycling, $32 \%$ running and $18 \%$ swimming (Zhou et al., 1989). It has been shown that in highly trained cyclists, maximum oxygen consumption and anaerobic threshold can actually be higher in the cycling mode than in running (Withers et al., 1981).
6.43 The relationship between heart rate during running and cycling

The lack of a consistent difference in heart rate, at either fixed blood lactates, or at Tlac, (Figure 6.1) and the associated moderate correlation between the two modes, leads to the recommendation that if guidance to specific training heart rates in each exercise mode is required, then incremental testing in both exercise modes is necessary. It is clear that, in general, maximal and training heart rates will be lower during cycling than running. Such a difference (mean 13 beats per minute) has been shown to be statistically significant in this group of well-trained triathletes. However, giving a general guideline of a difference in heart rate of about 15 beats per minute could lead to gross over or under estimation of the appropriate training heart rate. Indeed, only 9 of the 16 subjects had a difference in heart rate between the two modes at Tlac of between $10-15$ beats, thus a generalised zone is also unacceptable. Furthermore, the work of Pierce et al. (1990) has already shown that for longitudinal monitoring, testing in each mode of exercise is necessary.

In summary it has been shown that there is clear difference in training heart rates between running and cycling in well-trained triathletes. Running gives higher heart rates, but there is no consistency in the magnitude of such difference across a range of individuals in a homogenous group of trained athletes. It is, therefore, recommended that if triathletes seek physiological monitoring, they do so in all exercise modes.

### 7.0 General Summary and Practical Implications

### 7.1 Incremental Protocols to determine blood lactate profiles

Hardly innovative in its nature, the present research has found that an incremental protocol, with relatively short duration work stages, is acceptable for determining blood lactate profiles. The detailed discussion of the present findings in light of previous work has already taken place in section 4.40. It is clear that the nature of the protocol used does have considerable influence upon the physiological responses noted. Although not examined here, other aspects of the protocol, such as interval between exercise stages and the size of the increment (Jacobs, 1986), can affect the results obtained.

The early stage of the present study did not try to reinvent the wheel, it tried to find the most time efficient method for getting the necessary information that the coach, athlete and physiologist require. This is usually a picture of the current fitness level, which where appropriate, can be set in context with previous results for the same individual. Also prescription of training heart rates are required to give some guidance as to appropriate intensities for a range of training workouts usually employed to further develop fitness.

The starting point was the BASES protocol, an incremental protocol with a stage duration of four minutes. Personal communication with Ulrich Hartmann of the German Sports University in Cologne, revealed that it was normal for the German system to submaximally test athletes using a discontinuous method of 8 minute stage duration. It was Hartmann's belief, however, that this was unnecessary and that a stage duration of 6 minutes was appropriate to glean the required information. Nonetheless, Hartmann felt unable to change to the shorter method, as so many athletes had previously been tested with 8 minute version and to change to a different method might lose confidence the of the athletes.

It was the discussion with Hartmann that motivated the first study in Experiment One in the present project. The findings here showed little difference in heart rate and blood lactate response between the BASES method and the method of Hartmann, or indeed Hartmann's hunch of the shorter stage duration.

The second study in Experiment One was an effort to fine-tune this continuous incremental protocol. The present author was aware that practitioners at the British Olympic Medical Centre (BOMC) were using a stage duration of 3 minutes when assessing international rowers. This method, although only saving some 5 minutes per test in contrast to the BASES method, could create a significant reduction of testing time in the squad assessment situation. Thus a comparison of these two methods was employed in study two.
It was surprising that there appeared more difference in the measurements between the two continuous methods, than between the continuous and longer stage discontinuous methods. However, when examining the heart rate - blood lactate relationship, the factor used for training prescription, it was seen that little difference appeared between the two continuous protocols.

The practical implications are clear and twofold. Firstly, the shorter incremental protocol is acceptable for determining the heart rate blood lactate relationship. This would be further supported by the high rate of success in predicting the presence or absence of steady state blood lactates during training sessions, purely on the basis of observed heart rates during these training sessions and with reference to data from the 3 minute incremental test. Secondly, the protocols can not be used interchangeably when considering longitudinal monitoring. Adjustments to the stage duration do affect the heart rate and blood lactate at any given running speed or workload, thus a direct comparison of the condition of the athlete cannot be made unless the protocol is replicated identically.

Because it is the heart rate that is subsequently used to control training intensity and not the running speed, the issue of treadmill gradient has not been an issue in the present study. It has been shown that unless environmental conditions (e.g. running surface) during training match those from the test situation i.e. running surface, then techniques that predict blood lactate conditions using training velocity are subject to error (Coen et al., 1991, Heck et al., 1985).

What has become clear is that if treadmill speed is to match the energetics of outdoor running, a $1 \%$ gradient should be administered (Jones and Doust, 1996). The MSS sessions in Experiment Two, study one, controlled by running speed were also performed on the treadmill, so the gradient was irrelevant in this instance. Although, the argument for using a $1 \%$ gradient during incremental testing is strong, the current author is in the same trap as Hartmann above. All athletes previously tested have used a flat treadmill, so if a $1 \%$ gradient is added, longitudinal monitoring would be affected.
In light of the fact that other environmental factors (such as climate) have also been seen to alter the ability to predict the outcome of training sessions, in terms of the blood lactate response (Coen et al., 1991; Heck et al., 1985), it remains open to question as to how this might affect the heart rate blood - lactate relationship. It is clear that the workload - heart rate and workload - blood lactate relationship can be altered (Coen et al., 1991; Heck et al., 1985).

In summary, a 3 minute stage incremental protocol is valid for the determination of blood lactate profiles. This methodology may be used both for longitudinal monitoring (provided the method is repeated exactly) and the prescription of heart rates for subsequent training sessions.

### 7.2 Heart rate $v$ intensity to control training sessions.

It has been shown in Experiment Two that heart rate may well be a better means of controlling training sessions, than using the intensity. This did not appear obvious at first in the first study of Experiment Two, where MSS running sessions on the treadmill saw the mean blood lactate increase by $0.5 \mathrm{mmol} \mathrm{l}^{-1}$ between minutes 5 and 25. However, examination of the individual cases showed that $60 \%$ of the subjects (Figure 5.2 a ) noted increased blood lactate through the session over and above the $0.5 \mathrm{mmol} . \mathrm{l}^{-1}$ criteria set in this study. This illustrates that mean values do not tell the whole story, individual cases follow different patterns. The elite athlete is little concerned about the general trend expected when adopting means of training control; they are concerned merely with
exercising at an intensity that is most likely to lead to personal optimal gains in performance.

The heart rate controlled MSS sessions generally produced stable lactates provided athletes adhered to their target heart rate zones. This was the case in all three studies in Experiment Two. Aside of this important finding within the framework of Experiment Two, one has to consider the practical aspect. Even if an intensity controlled regime worked perfectly in the field, other limitations would still be great. In running, athletes would have to use accurately pre-measured routes, with little change to terrain or surface, as it is clear that variation in these factors can cause prediction errors for the lactate response (Coen et al., 1991; Heck et al., 1985). In the real world, this would be unacceptable to many runners, whose enjoyment is often running in varied parkland. Many runners do train on road or asphalt, but the injury risk of continued running on such surfaces as opposed to grass is seen to rise (Noakes, 1991).

In rowing, training venues tend to be less varied and are either man-made lakes, or stretches of river. In this case the use of measured sections is more viable and varied terrain is not a factor, but two other considerations come into place. Firstly, weather conditions alter the workload. This is seen in racing situations, where finish times are strongly influenced by windspeed and direction. Secondly, rowing is not always an individual sport, thus a sculler can be with a partner (double) or three other crew members (quad), whilst a sweep oarsman will either row in a pair, a four, or an eight. In such crew boats, the boat-speed is also influenced by other crew members, thus boatspeed is not a valid means of control for individual training intensity.
Once again, for practical reasons, heart rate offers a more viable means of training control, as this takes into account the relative intensity at which any given individual is working. If a runner goes up a hill, the heart rate is elevated, so in order to maintain the same relative intensity, the running speed must drop. Similarly, if boat speed is slow, individual oarsmen may not necessarily be below their desired training intensity according to their heart rate. However, if the oarsman is below the desired heart rate, he can always pull harder to increase the relative intensity.

In summary, therefore, the present study proposes that heart rate may be a better means of training intensity control, than running speed or intensity in rowing. This is upon the grounds of both the findings in Experiment Two and the logistical / practical reasons mentioned above.

### 7.3 The use of heart rate to control training

The concept of using heart rate, prescribed from incremental protocols, is not new. Kindermann, Simon and Keul (1979) used this principle when controlling training sessions of cross-country skiers on a treadmill in the laboratory. They observed that stable heart rates during training gave rise to a drop in workload, a factor he attributed to cardiovascular drift. However, the present work differs in that individual blood lactate profiles were used to set the training heart rates, rather than an arbitrary fixed blood lactate marker. Furthermore, the present work has gone considerably further, by analysing a large number of training sessions in the field.

In the laboratory controlled sessions in the present work, as well as the work by Kindermann, Simon and Keul (1979), subjects adhered to the target heart rates. The present study analysed training sessions in the field and noted that $20 \%$ of the subject sessions were performed above the prescribed heart rates. This was more understandable in rowers who, in crew boats, often comment that they need to keep the boat speed up, despite the fact that they exceed their target heart rate. However, opinions' of the coaches to these athletes appears to differ. It was the belief of the Chief Coach of the rowers, that athletes should concentrate on keeping to the prescribed heart rate during training sessions and worry less about the absolute boat speed. Whether, this is indeed in the best interest of the athlete on a long-term basis is yet to be empirically established and was beyond the scope of the present study.

The present study has provided evidence that heart rate can predict the presence or absence of steady state blood lactates during training sessions, in the light of heart rate from a blood lactate profile. The $96 \%$ success rate in Experiment Two, study three, is high compared to simply using \%HRmax or a bloodless lactate profile (Foster et al.
1995). The use of percentage of HRmax is likely to be problematic considering the difficulty in attaining a genuine maximal value (Ingjer, 1991).

The present study has not addressed factors that affect heart rate. A leading review article of heart rate and endurance training (Gilman, 1996) has highlighted several of these factors. Cardiovascular drift, often mentioned in the discussion of the present study, is one such factor. It is clear that heart rate does indeed rise, despite stable workload or running speed, once the duration of exercise exceeds 20 minutes. Not only has this been shown by the aforementioned work of Kindermann, Simon and Keul (1979); it has also been demonstrated elsewhere. Mognoni et al. (1990) and Scheen et al., (1981) have shown heart rate to drift by up to 20 beats per minute between 20 and 60 minutes of constant load exercise.
Heart rate is also excessively elevated for a given work intensity when environmental heat stress is elevated (Astrand and Rodahl, 1986; Claremont et al., 1975). It is clear that both acclimatisation and increased fitness, minimise the affect of increased ambient heat upon the heart rate response (Gilman, 1996). One of the major adaptations to an increase in endurance fitness, is better heat tolerance ( $\AA$ strand and Rodahl, 1986).
It has also been established that Tlac occurs at higher workloads when performed in colder environments than normal. Therminarias et al., (1989) found clear differences in the physiological responses between a cold environment $\left(-2^{\circ} \mathrm{C}\right)$ and a normal environment $\left(24^{\circ} \mathrm{C}\right)$. Although Tlac occurred at a workload some 30 watts greater in the cold environment, the mean heart rate at Tlac was not significantly different ( 147 v 152 ). Thus, whilst it is clear that temperature affects the heart rate at any given workload, it has yet to be empirically established how this affects the heart rate - blood lactate relationship. It may be that this relationship is unaffected by temperature, in the same way that changes in the incremental protocol had little effect in the second study of Experiment One in the current research.

The issue of the influence of temperature and indeed other environmental conditions upon the heart rate - blood lactate relationship is the logical extension of the present research, which due to time and resources was beyond the current scope. It is very much an avenue of future research for this author, because if factors such as temperature and
humidity inhibit the ability to predict the lactate response during training sessions, the validity of the current methodology as well as many others (Janssen, 1987; Weltman, 1995) is brought into question.

Altitude is another factor that clearly affects heart rate for a given workload. Currently athletes use heart rates prescribed from incremental tests at sea-level, in an attempt to safely control training at altitude. The validity of such methodology is open to question in light of the dearth of research in this area.

Despite the doubt about the validity of heart rate accurately representing metabolic activity in a range of environmental conditions, it still remains a popular means of monitoring training in athletes (Gilman, 1996). Other options of such monitoring are based on the athletes perception of intensity (Hopkins, 1991) or volume of training (Noakes, 1991). Although coaches traditionally favour these two monitoring methods, Gilman (1996) questions whether these can indeed accurately assess the metabolic stress experienced by the athlete even in normal conditions.
With the ability to download heart rate information from portable telemetry devices to a personal computer, it is possible to build a training diary of heart rate information to quantify the work achieved by an athlete in any given training cycle. This is a considerable advancement to the logging and assessment of training, the benefits of which have yet to be evaluated.

In summary, when used in conjunction with an incremental test, heart rate has been shown to predict blood lactate conditions during training sessions. Although other research has shown that the heart rate associated with a given workload can vary with environmental conditions, it remains unclear as to how environmental conditions affect the heart rate - blood lactate relationship and the use of heart rate as a valid means of controlling training intensity.

### 7.4 The use of blood lactate to control training

Blood lactate values are not an indication of the production rate of lactate, rather a net balance of the rate of production and elimination (Donovan and Brooks, 1983). Such
values are widely accepted as a means of training prescription in athletes (Coen et al., 1991; Forenbach, Mader and Hollman, 1987; Heck et al., 1985; Jacobs, 1986; Kinderemann, Simon and Keul, 1979; McLellan and Skinner, 1981; Sjödin, Jacobs and Svedenhag, 1982). The consensus of opinion is that during, what are currently in vogue, "threshold" or MSS sessions, the blood lactate should be high but stable for optimal development of aerobic endurance. It has previously been shown that in healthy males it does not matter whether such sessions are continuous or intermittent in nature, provided that the average power output is equivalent to the individual Tlac (Keith, Jacobs and McLellan, 1992). However, it is yet to be determined whether this is also the case in well-trained athletes.
The difference is that in non-athletes, training frequency is often lower, in the realms of ACSM guidelines for the development and maintenance of aerobic fitness (3 times per week), thus the training week may consist of exclusively MSS training. In athletes preparing for competition, a variety of training intensity and duration may form the training week, with MSS training contributing no more than $30 \%$ (Forensbach et al., 1987; Janssen, 1987).
Despite the general world-wide acceptance of the requirement for stable blood lactates during training sessions for optimal longitudinal improvements in fitness, the justification is limited. Most authors refer to the work of Sjödin, Jacobs and Svedenhag (1982) who examined changes in aerobic fitness in elite athletes, in response to the introduction of OBLA training. It should be mentioned that the study was not well controlled, as subjects acted as their own controls. The findings were that fitness improved in response to an 8 week period of OBLA training and such improvements were lost after reverting to the previous training regime.
The Sjödin, Jacobs and Svedenahg (1982) work demonstrated that the more blood lactate increased through the OBLA sessions, the less longitudinal improvement in fitness occurred. It is because of this that the aim of the MSS sessions in the present study was for stable, not increasing blood lactates. However, it is yet to be more clearly identified how damaging increasing lactates are to an athlete's fitness.

In some of the heart rate controlled sessions during the present study, blood lactate was seen to drop. It seems likely that training was not optimal in these cases, because the overall intensity could presumably have been higher had the lactates remained stable. Such a drop in lactate is possibly to be a result of one or more of four factors.

Firstly, the pacing strategy may have been incorrect. The athlete may have started too fast to raise the heart rate to the target zone initially and subsequently had to slow in the remaining part of the session. Such was the case in some of the cross-country skiers in the study by Kindermann et al., (1979).
Secondly, related to the above, warm up may have been insufficient. It is possible that a more thorough warm-up could have given lower initial blood lactates and thus given more stable lactate through the session.
Thirdly, environmental factors could have varied, giving rise to an abnormally high heart rate for the given work intensity. This could have lead to a reduction in workload and blood lactate to keep within the confines of the target heart rate. Although ambient temperature in the outdoor training sessions was generally between $16-22^{\circ} \mathrm{C}$, there may have been some minor fluctuations. Once again, this strengthens the demand for further research into this area.
Finally, it could be that the training intensity set from the incremental test underestimated the heart rate required for high but stable blood lactate through the training sessions. This could only have been confirmed had a series of training sessions either side of the target heart rate been performed.

The prescription of training for base endurance (BE) training in study three of Experiment Two, is the most innovative aspect of the present study. Despite that fact that athletes perform the bulk of their training in this zone, little research has been in this direction. Clearly, it is easier to assess MSS sessions, where high but stable blood lactates are the aim. The purpose of these BE sessions, also commonly known as Utilisation II (UTII) training in rowing, is to work using predominantly fat metabolism for basic endurance training and economisation (Altenburg, 1992). Because training volumes in this zone are high, the intensity has to be low, due to the limited glycogen
stores within the muscles and liver (Hartmann, 1990). Indeed when measurements of blood lactate were taken in the field during training sessions performed at intensities selected by athletes (using subjective reasons), it was seen that values were lower than 2 mmol. $\mathrm{I}^{-1}$ of plasma lactate, lower than those desired by coaches.

It has been shown that there does not necessarily have to be elevation in blood lactate for an endurance training effect to take place (Casaburi et al., 1995). However, the subject pool in that study was 27 sedentary men performing cycle exercise 5 times per week. The impact of BE training and the desired minimum and maximal intensity for athletes is yet to be established.

Although not perfect, the present study has attempted to make a start in analysing BE training. It is viewed as a point from which further research can enhance understanding of such training and refine knowledge as regards both the desired intensity and duration of such workouts for optimal longitudinal development. The difficulty, of course, lies in actually assessing the impact of such sessions in the highly trained athlete. It seems the best way to assess such sessions is to manipulate them in some way, with two or more groups performing slightly different versions. Yet highly trained athletes are reluctant to alter their own training for the purposes of research, despite the fact that there is currently no empirical evidence to support the notion that what they are currently doing, is the optimal way to prepare for competition. Greater focus on Base Endurance training is clearly an aspect that warrants further research.
The criteria used in this study for the existence of stable lactates in MSS, or BE sessions is perhaps the most controversial. An increase in blood lactate of $0.5 \mathrm{mmol} .1^{-1}$ appears little in light of the $1 \mathrm{mmol} .1^{-1}$ used initially by Heck et al., (1985) and adopted by others (Coen et al., 1991; Forenbach et al., 1987; Scnabel et al., 1982). The rationale for the criteria in Experiment Two has been argued in its discussion (Section 5.414). With whole blood lactates an increase as much as $1 \mathrm{mmol} .^{-1}$ can be substantial in well-trained endurance athletes, whose peak lactates can be below 6 mmol. $\mathrm{l}^{-1}$. As the whole process of evaluating BE sessions is previously unresearched in highly trained athletes, it is likely that criteria for these sessions may be refined with future analysis in years to come.

In summary, it appears that much of the popularity surrounding MSS training and the requirement of high but stable blood lactates stems from the work of Sjödin, Jacobs and Svedenhag (1982). The present study has shown that blood lactate can actually decrease in such sessions and four potential causes have be postulated. The long term impact of such decline through training sessions has yet to be determined. Blood lactate has also been used to determine training intensities for BE. Research into this area is new, which is surprising in that this type of work forms the bulk of most training programmes.

### 7.6 Balance of the training

The present study has examined just two types of training session commonly used by the endurance athlete. In the introduction to Experiment Two, one example of a range of intensities used by athletes is displayed (Table 5.1). Governing bodies of different sports have similar training guidelines, using their own definitions or containing features pertinent to particular sports. It must be remembered that the purpose of such tables is usually for coach education, but such literature does illustrate the variety of methods used to bring the athlete to peak condition. There is no one correct way to condition that athlete. It has long been established that if two athletes are given the same training programme, they may well improve fitness at different rates (Saltin, 1968). In essence, training is a combination of art and science. Science should underpin the basic principles used, yet the art usually comes from the coach who tries to blend a variety of sessions in a fashion that is appropriate to the physical and physiological demands of the competition event, as well as to enable peaking at the most important time.
The appropriate fraction of any given intensity within the overall training programme remains an unanswered question. Once again, because well-trained athletes are reluctant to participate in research at the expense of their preferred training philosophy, the issue is not likely to be resolved for some considerable time.
A comprehensive review of studies by Wenger and Bell (1986) established guidelines for the improvement of maximum oxygen consumption. They suggested that training intensity should be between 50 and $100 \% \dot{\mathrm{VO}}_{2 \text { max }}$. They further suggest that an intensity
between $90-100 \% \mathrm{VO}_{2 \max }$ gives most improvement in this endurance index. However, not all of the research cited related to well-trained athletes and gave little consideration to mixed intensity training regimes.

Clearly intensity is a key factor to the improvement of endurance fitness, but it has been established that too much training can lead to overtraining (Kuipers and Keizer, 1988; Hartmann, 1990, Noakes, 1986). Hollman et al., (1981) demonstrated that training at OBLA for 30 minutes 5 times per week in low trained subjects gave improvement in endurance, whilst training at a higher intensity of $95 \% \dot{\mathrm{~V}}_{2_{2} \max }$ with a similar duration and frequency resulted in no change in fitness. The Sjödin, Jacobs and Svedenhag (1982) research showed that the introduction of MSS (in their terms OBLA training) to the normal programmes of elite athletes gave greater improvement in aerobic endurance. The athletes with the smallest increase in blood lactate through the MSS sessions improved fitness the most. The authors did not state what the normal training programme was, but it is inconceivable that it did not contain at least some training above MSS. Another interesting finding of this study was that the two 800 m runners included in the study, benefited less from the MSS training than the endurance athletes. Once again there is clearly an individual element as to how much increase in training intensity can be tolerated by different athletes.
Sleamaker (1989) recommends that the bulk of training (over 80\%) for athletes be at an easy intensity, which would be the equivalent of BE sessions in the present study. He further suggests that high intensity training should increase to $30 \%$ in the competitive season. Hartmann (1990) has demonstrated that BE or UTII training should form 90$94 \%$ of the volume in the winter period for rowers and $70-77 \%$ of volume in the competition period. Thus there is approximate agreement between the two authors here. It seems reasonable that although an increase in training volume can boost endurance fitness (Foster, Daniels and Yarborough, 1977; Hagan, Smith and Gettman, 1981), such volumes should not be too excessive. This is because it has been shown that this too can lead to overtraining syndrome (Fry, Morton and Keast, 1992; Lehmann, Foster and Keul, 1993).

In summary, it has been shown elsewhere that during MSS sessions, controlled training is of benefit for optimal improvements in fitness. Furthermore, controlled intensity may be important for the avoidance of overtraining. The exact nature of the balance of sessions within a training programme for optimal improvement in endurance fitness remains unclear, but is likely to vary among individuals.

### 7.7 Multiple event sport

Given that there are often constraints upon time and resources, it would have been useful if the training heart rate prescription from one mode of exercise, could also be used to predict training heart rates for different exercise modes. Physiological monitoring is not cheap and for those involved in sports such as triathlon, where three exercise modes are utilised, three laboratory visits would be required for accurate training information. This requires greater interruption to the training process, because not only is a day of training lost as a result of the laboratory visit, the day before the test also demands low intensity work or rest. In light of the increasing incidence of overtraining in elite sport, it could be argued that such training reduction for the purpose of physiological monitoring could be advantageous; however it is unlikely to be viewed this way by the motivated athlete, rarely keen to miss hard training days!

Clear differences in training heart rates have been demonstrated between exercise modes in Experiment Three. Unfortunately, although the mean difference in heart rate at Tlac between cycling and running is 13 beats per minute, there is a wide range in this difference in the group of well-trained triathletes. This means that it is unsafe to give a generalised guidance of a range of $10-15$ beats per minute difference between modes when athletes have had just one test. This would be similar to athletes using the 220 -age formula for estimating maximal heart rate; it may be accurate for some subjects but is inaccurate for most.

In summary, if triathletes require guidance as to appropriate training heart rates in more than one mode of exercise, separate mode specific lactate profiles are required.

### 8.0 Further Research

The course of discussion through the present research has brought to light a number of issues that require future research to further our understanding of training control, in order to give the coach and athlete better guidance in a quest for optimal performance. These are summarised below.
8.1 The effect of environmental conditions on the heart rate-blood lactate relationship.

It remains unclear as to what effect different environmental conditions such as temperature, humidity and altitude have upon the heart rate - blood lactate relationship. It is clear that these environmental factors alter the heart rate and blood lactate measurements noted for any given workload or running speed. Such research is the logical progression of the current work and would have been addressed if the scope of this study, time and resources allowed.
It is suggested firstly that incremental protocols are performed by subjects in a range of temperatures $\left(0,18-22\right.$ and $\left.>30^{\circ} \mathrm{C}\right)$. Not only will it be seen that the workload of Tlac is likely to alter, investigation should analyse how the heart rate -blood lactate relationship is affected by different temperatures. Secondly the effect of humidity should be analysed in the same manner. Thirdly, sessions training in the different environments should be monitored in a similar fashion to that in Experiment Two, study three in the current research. This will evaluate how athletes may need to adjust their prescribed training heart rates when experiencing varied environmental conditions.
Finally, the assessment of training sessions similar to that in the current research should be employed at altitude. Further investigation may be warranted to produce training zone correction factors when training in such an environment.

### 8.2 Pacing Strategy during heart rate controlled MSS sessions

It is recommended that a range of pacing strategies be used during MSS sessions, to evaluate whether the manner in which the target heart rate is attained affects the blood lactate response through the session. It is suggested that a more gentle approach to the initial stages of the session be assessed in comparison to the naturally adopted method employed by athletes. Such a gentle approach may involve either increasing heart rate to $80 \%$ of the target heart rate for the first $5-8$ minutes and then stepping up, or by increasing heart rate in a ramped procedure, say by 5 beats per minute every minute. The profile of blood lactate through the session, along with the associated speed or workload should be monitored and compared across method employed.

### 8.3 Warm-up before sessions

In a similar manner to the assessment of pace judgement for MSS sessions, evaluation of warm-up procedure is also recommended. Both the intensity and duration of warm up should be in light of the behaviour of blood lactate through controlled MSS sessions. In this instance, the MSS sessions should be controlled by workload / running speed under laboratory conditions, rather than heart rate controlled in the field.

### 8.4 Different terrain used in MSS sessions

As it is unlikely that runners will train only on flat training routes, a comparison of hilly and flat terrain during heart rate controlled training sessions is warranted. Naturally heart rate will rise on the uphill part of a training route given a particular running speed. In normal circumstances a runner will slow down in order to remain within the target heart rate zone. The implication of such variation in pace and gradient should be assessed in light of the blood lactate behaviour through such sessions.

### 8.5 Base Endurance Training

Given that this is the training zone that forms the bulk of training for the endurance competitor, much research into this type of training is pertinent. Assessment of the optimal duration and intensity of such training is required to give better guidance and feedback to the athlete and coach. The difficulty is in controlling other training variables, such as the other training sessions that take place within a training programme. If providing information for the athlete is the key, there is little point in analysing these sessions in isolation, as is so often done for "threshold sessions" or in non-athlete populations, where single intensity training is often employed for the development and maintenance of general cardio-respiratory fitness.

## REFERENCES

Adams, W.C., Mack G.W., Langhans G.W., and Nadel E.R. (1992) "Effects of varied air velocity on sweating and evaporative rates during exercise." J.Appl. Physiol. 73 (6): 2668-2674

Ahlborg G. (1985) "Mechanisms for glycogenolysis in non exercising human muscle during and after exercise." Am. J.Physiol. 248: E540-E545

Altenburg D. (1992) 'Planning and organizing general fitness training for rowers." FISA Coach 3(1): 1-4

American College of Sports Medicine (1978) "Position statement on the recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults." Med. Sci. Sports 10

American College of Sports Medicine (1990) "Position stand on the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults." Med. Sci. Sports Exerc. 22: 265-274

Armstrong L.E., and Pandolf K.B. (1988) "Physical training, cardiorespiratory physical fitness and exercise heat tolerance." in Pandolf K.B., Sawka M.N., and Gonzalez R.R (eds) Human Performance Phvsiology and Environmental Medicine at Terrestrial Extremes: 199-226 Benchmark Press, Indianapolis.

Åstrand P.O. (1955) "New records in human power." Nature 176: 922-923

Åstrand P.O., and Rodahl K (1986) Textbook of work physiology: Physiological Bases of exercise. (3rd Ed), Singapore, McGraw-Hill.

Aunola S., and Rusko H. (1986) "Aerobic and anaerobic thresholds determined from venous lactate or from ventilation and gas exchange in relation to muscle fibre composition." Int. J. Sports Med. 7: 161-166

Barbeau P., Serresse O., Giroux M., and Boulay M.R. (1991) "The usefulness of in charting the progress of elite and non-elite cyclists during a training and competition season." Med. Sci. Sports Exerc. 23 (4): S9

Beaver W.L., Wasserman K., and Whipp B.J. (1985) 'Improved detection of lactate threshold during exercise using a log-log transformation." J.Appl.Physiol. 59(6): 19361940

Beneke R. (1995) "Anaerobic threshold, individual anaerobic threshold, and maximal lactate steady state in rowing." Medicine and Science in Sports and Exercise 27(6): 863867

Beneke R. and von Duvillard S.P. (1996) "Determination of maximal lactate steady state response in selected sports events." Medicine and Science in Sports and Exercise 28 (2): 241-246

Berry M.J., Stoneman J.V., Weyrich A.S., and Burney B (1991) "Dissociation of the ventilatory and lactate thresholds following caffeine ingestion." Med. Sci. Sports Exerc. 23: 463-469

Bilzon J., and George K.P. (1996) "Lactate and ventilatory thresholds are coincident irrespective of changes in ambient temperature." J.Sports Sci. 14 (1):72

Bird S. and Davison R. (1997) BASES Phvsiological testing guidelines ( $3^{\text {rd }}$ Edition) Leeds. BASES

Bishop P.A., Smith F.A., Klime J.C., Mayo J.M., and Tin Y.H. (1992a) "Comparison of a manual and an automated enzymatic technique for determining blood lactate concentrations." Int. J.Sports Med. 13(1): 36-39

Bishop P.A., May M., Smith J.F., Klime J., Mayo J., and Murphy M. (1992b) 'Influence of blood handling techniques on lactic acid concentrations." Int. J. Sports Med. 13(1): 5659

Bland J.M. and Altman D.G. (1986) "Statistical methods for assessing agreement between two methods of clinical measurement." Lancet 307-310

British Standards Institution (1979) Precision of test methods 1: Guide for the determination and reproducibility of a standard test method (BS 5497, Part 1). London:BSI

Brooks G.A (1985a) "Anaerobic threshold: review of the concept and directions for future research." Med. Sci. Sports Exerc. 17: 22-31

Brooks G.A (1985b) "Response to Davis' manuscript." Med. Sci. Sports Exerc. 17: 19-21

Brooks G.A. (1986) "The lactate shuttle during exercise and recovery." Med. Sci. Sports Exerc. 18: 360-368

Brooks G.A. (1991) "Current concepts in lactate exchange." Med. Sci. Sports Exerc. 23 (8): 895-906

Buskirk E.R., Iampietro P.F., and Bass D.E. (1958) "Work performance after dehydration: effects of physical conditioning and heat acclimation. J.Appl.Physiol. 12: 189-194.

Carter J.E.L., Katch F.W., Bowyer J.L., Phillips W.H., Ross W.D., and Susec A. (1966) "Structural and functional assessments of a champion runner - Peter Snell." Research Quarterlv 38: 355-365

Casaburi R., Storer T.W., Sullivan C.S. and Wasserman C. (1995) "Elevation of blood lactate as an intensity criterion for exercise training." Med. Sci. Sports Exerc. 27 (6) 852862

Cempla J., Blachura L., and Zoladz J. (1987) "Physiological responses in distance runners during 30 minutes high intensity running. Biology of Sport. 4: 5-14

Cheng B., Kuipers H., Snyder A.C., Keizer A., Jeukendrup A., and Hesselink M. (1992) "A new approach for the determination of ventilatory and lactate thresholds." Int.J. Sports Med.

Claremont A.D., Nadel F., and Reddan W.D. (1975) "Comparosin of metabolic temperature, heart rate and ventilatory responses to exercise at extreme ambient temperature." Med. Sci.Sports 7(2): 150-154

Coen B., Schwarz L., Urhausen A., Kindermann W. (1991) "Control of training in middle- and long- distance running by means of the individual anaerobic threshold." Int.J. Sports Med. 12: 519-524

Conley D.L., and Krahenbuhl G.S. (1980) "Running economy and distance running performance in highly trained athletes." Med. Sci. Sports Exerc. 12: 357-360

Conley D.L., Krahenbuhl G.S., Burkett L.N., and Millar A.L. (1984) Following Steve Scott: Physiological changes accompanying training. Physician and Sportsmedicine 12: 103-106

Connett R.J., Honig C.R., Gayeski T.E.J., and Brooks G.A. (1990) "Defining hypoxia: a systems view of VO2, glycolysis, energetics and intracellular PO2." J.Appl. Physiol. 68: 833-842

Costill D.L. (1967) "The relationship between selected physiological variables and distance running performance." J. Sports Med. 7: 61-66

Costill D.L. (1970) "Metabolic responses during distance running performance." J. Appl. Physiol. 28: 251-255

Costill D.L., Thomason H., and Roberts E. (1973) "Fractional utilisation of aerobic capacity during distance running. Med. Sci. Sports. 5:248-252

Costill D.L. (1979) A scientific approach to distance running. Tafnews, USA

Coyle E.F., Martin W.H., Ehsani A.A., Hagberg J.M., Bloomfield S.A., Sinacore D.R., Holloszy J.O. (1983) "Blood lactate threshold in some well-trained ischemic heart disease patients." J.Appl.Physiol. 54: 18-23

Craig F.N., and Cummings E.G. (1966) "Dehydration and muscular work." J.Appl.Physiol. 21: 670-674

Daniels J.T. (1974) "Running with Jim Ryun: a five year study." Physician and Sportsmedicine 2: 62-67

Daniels J.T. (1992) "Triatholon training and competition." in Shephard R.J and Åstrand, P.O. (eds) (1992) Endurance in Sport. Oxford; Blackwell Scientific Publications. 597-601

Davis J.A., Vodak P., Wilmore J.H., Vodak J., and Kurtz. P. (1976) "Anaerobic threshold and maximal aerobic power for three modes of exercise." J.Appl. Physiol. 41: 544-550

Davis J.A., Frank M.H., Whipp B.J. and Wasserman K. (1979) "Anaerobic Threshold alterations caused by endurance training in middle aged men." J. Appl. Physiol. 46: 10391046.

Davis J.A., Caiozzo V.J., Lamarra N., Ellis J.F., Vandagriff R., Prietto C.A., McMaster W.C. (1983) 'Does the gas exchange anaerobic threshold occur at a fixed blood lactate concentration of 2 or 4 mM ?" Int.J. Sports Med. 4: 89-93

Davis J.A. (1985a) "Anaerobic threshold: review of the concept and future directions for research." Med. Sci. Sports Exerc. 17: 6-18

Davis J.A. (1985b)"Response to Brook's manuscript." Med. Sci. Sports Exerc. 17: 32-34

Davis M.A. Williams P.E., and Cherrington A.D. (1984) "Effects of a mixed meal on hepatic lactate and glucogenic precursor metabolism in dogs." Am.J.Phvsiol. 247: E362E369

Diaz F.J., Hagan R.D., Wright J.E., and Horvarth S.M. (1978) "Maximal and submaximal exercise in different positions." Med.Sci.Sports 10(3):214-217

DiCarlo L.J., Sparling P.B., Millard-Stafford M.L., and Rupp J.C. (1991) "Peak heart rates during maximal running and swimming: implications for exercise prescription." Int.J.Sports Med. 12(3): 309-312

Dill D.B., Robinson S., and Ross J.C. (1967) "A longitudinal study of 16 champion runners." J. Sports Med. Phys. Fit. 7: 4-27

Di Pampero P.E., Cortili G., Celentano F., and Cerretelli (1971) "Physiological aspects of rowing." J.Appl. Physiol. 31 (6): 853-857

Doherty M., (1992) "Practical applications of lactate testing - rowing." Coaching Focus 21: 7-9

Doherty M., Warrington G.D., Hughes M., Graham S.A., Godfrey R., and Fullerton F.M. (1994) "Selected physiological measures and rowing ergometer performance in the 1993 men's Great Britain boat crews." J.Sports Sci. 12 (2): 160

Donovan C.M., and Brooks G.A. (1983) "Enhanced efficiency of lactate removal after endurance training." Am.J.Endocrinol. Metab. 244 (7): E83-E92

Duggan A., and Tebbutt S.D. (1990) "Blood lactate at $12 \mathrm{~km} / \mathrm{h}$ and vOBLA as predictors of run performance in non-endurance athletes." Int. J. Sports Med. 11: 111-115

Dunbar G.M.J., Heagarty, R., Warrington, G. and White, J.A. (1995) "The effects of different protocols on heart rates and blood lactate values during graded exercise tests for middle distance runners. J. Sports Sci. 13: 25-26

Dunbar G.M.J and Faulmann L (1996) "An analysis of physiological responses to submaximal and maximal exercise in elite middle- and long- distance runners." J.Sports Sci. 14 (1): 77

Dwyer J. and Bybee R. (1983) "Heart rate indices of the anaerobic threshold." Med.Sci. Sports Exerc. 15: 72-76

Farrell P.A., Wilmore J.H., Coyle E. F., Billing J.E. and Costill D.L. (1979) "Plasma lactate accumulation and distance running performance." Med. Sci. Sports. 11 (4) 338344

Fay L., Londeree B.R., LaFontaine T.P., and Volek M.R. (1989) "Physiological parameters related to distance running performance in female athletes." Med. Sci. Sports Exerc. 21: 319-324

Ferry A., Duvallet A., and Rieu M. (1988) " The effect of experimental protocol on the relationship between blood lactate and workload." J. Sports Med. 28:341-347

Flore P., Therminarias A., Oddou-Chirpaz M.F., and Quirnon A. (1992) "Influence of moderate cold exposure on blood lactate during incremental exercise." Eur.J.Appl. Physiol. 64: 213-217

Förenbach R., Mader A., and Hollman W. (1987) "Determination of endurance capacity and prediction of exercise intensities for training and competition in marathon runners." Int.J.Sports Med. 8: 11-18

Forrest A.R.W., Morton S., and Lambadarios C. (1990) "Blood or plasma lactate?" Br.J.Sports Med. 24 (2): 132

Foster C., Daniels J., and Yarborough R. (1977) "Physiological and training correlates of marathon running performance." Aus.J.Sports Med. 9: 58-61

Foster C., Cohen J., Donovan. K., Gastrau P., Killian P.J., Schrager M., Snyder A.C. (1993) "Fixed Time Versus Fixed Distance Protocols for the Blood Lactate Profile in Athletes." Int. J. Sports Med. 14 (5): 264-268

Foster C., Crowe M.P., Holum D., Sandvig S., Schrager M., Snyder A.C., and Zajakowski S. (1995) "The bloodless lactate profile." Med.Sci.Sports Exerc. 27(6): 927933

Foxdall P., Sjödin A., and Sjödin B. (1996) "Comparison of blood lactate concentrations obtained during incremental and constant intensity exercise." Int. J. Sports Med 17(5): 360-365

Fry R., Morton A.R., and Keast D (1991) "Overtraining in athletes: an update." Sports Med. 12: 32-65

Gass G.C., McLellan T.M., and Gass E.M. (1991) "Effects of prolonged exercise at a similar percentage of maximal oxygen consumption in trained and untrained subjects." Eur.J.Appl. Physiol. 63: 430-435

Gertz E.W., Wisneski J.A., Neese R., Bristow J.D., Searle GF., and Hanlon J.T. (1981) "Myocardial lactate metabolism: evidence of lactate release during net chemical extraction." Circulation 1273-1279

Gilman M.B. (1996) "The use of heart rate to monitor the intensity of endurance training." Sports Med. 21 (2): 73-79

Gollnick P.D. (1988) "Energy metabolism and prolonged performance." Perspect. Exerc. Sci. Sports Med. 1: 1-42

Goodman J., Lapsade A., and Lui P. (1989) "Effects of exercise on left ventricular function." Can.J. Sport Sci. 14: 113

Green J.S., and Crouse S.F. (1993) "Endurance training, cardiovascular function and the aged." Sports Med. 16(5): 331-341

Gullstrand L. "Swimming as an endurance sport" in Shephard R.J and Astrand, P.O. (eds) (1992) Endurance in Sport. Oxford; Blackwell Scientific Publications. 531-541.

Hagan R.D., Smith M.G., and Gettman L.R. (1981) "Marathon performance in relation to maximal aerobic power and training indices." Med. Sci.Sports Excer. 13: 185-189

Hagberg J., Coyle E.F., Carroll J.E., Miller J.M., Martin W.H., and Brooke M.H. (1982) "Exercise hyperventilation in patients with McArdle's disease." J.Appl. Phyiol. 52: 991994

Hagberg J. and Coyle E.F.. (1983) "Physiological determinants of endurance performance as studied in competitive racewalkers." Med. Sci. Sports Exerc. 15:287-289

Hagberg J.M. (1986) "Physiological implications of the lactate threshold." Int.J.Sports Med. 5 (Suppl.): 106-109

Hagerman F.C., Connors M.C., Gault J.A., Hagerman G.R., and Polinski W.J. (1978) "Energy expenditure during simulated rowing." J.Appl. Physiol. 45: 87-93

Hale T., Armstrong N., Hardmann A., Jakeman P., Sharpe C., Winter E. (1988) Position Statement on the Physiological Testing of the Elite Competitor. Leeds. BASS.

Hartmann U., Mader A., and Hollman W. (1990) 'Heart Rate and Lactate during endurance training programmes in rowing and its relation to the duration of exercise by top elite rowers." FISA Coach 1(1): 1-4.

Heck H., Mader A., Hess G., Mucke B., Muller R., Hollmann W. (1985) "Justification of the $4 \mathrm{mmol} / \mathrm{l}$ lactate threshold." Int. J. Sports Med. 6: 117-130.

Henritze J., Weltman A., Schuner., and Barlow K. (1985) "Effects of training at and above the lactate threshold on the lactate threshold and maximum oxygen uptake." Eur. J. Appl. Physiol 54: 84-88

Hermanssen L., Ekblom B., and Saltin B. (1970) "Cardiac output during submaximal and maximal treadmill and bicycle exercise." J.Appl. Physiol. 32:467-473

Hill A.V. (1913) "The energy degraded in recovery processes of stimulated muscles." J.Phvsiol. 46: 28-80

Hill A.V., and Lupton H. (1923) "Muscular exercise, lactic acid and the supply and utilization of oxygen." Ouarterly Journal of Medicine. 16: 135-171

Hill A.V., Long C.N.H., and Lupton H. (1924) "Muscular exercise, lactic acid and the supply and utilisation of oxygen. I-III" Proc. Rov. Lon. Ser. B. 96: 438-475

Hollman W., Rost R., Liesen H., Dufaux B., and Heck B. (1981) "Assessment of different forms of physical activity with respect to preventative and rehabilitative cardiology." Int.J. Sports Med. 2: 67-80

Hollman W. (1985) "Historical remarks on the development of the aerobic-anaerobic threshold up to 1966." Int.J. Sports Med. 6: 109-116

Hopkins W.G. (1991) "Quantification of training in competitive sports, methods and application." Sportsmedicine 12(3): 161-183

Hughes E.F., Turner S.C., and Brooks G.A. (1982) "Effects of glycogen depletion and peddling speed on "anaerobic threshold"." J.Appl Physiol. 52:1598-1607

Ingjer F. (1991) "Factors influencing the assessment of maximal heart rate." Scand.J.Med.Sci.Sports 1:134-140

Ivy J.L., Withers R.T., Van Handel P.J.,Elger D.H., and Costill D.L. (1980) 'Muscle respiratory capacity and fiber type as determinants of the lactate threshold." J.Appl Phvsiol. 48 (3): 523-527

Ivy J.L., Costill D.L., Van Handel P.J., Essig D.A., and Lower R.W. (1981) "Alteration in the lactate threshold with changes in substrate availability." Int.J.Sports Med. 2 (3): 139-142

Jacobs I. (1986) "Blood Lactate: Implications for Training and Sports Performance." Sports Medicine. 3: 10-25

Jacobs I., and Mcllelan T., (1988) "Validity of individual anaerobic threshold (IAT)." Can.J.Sport Sci. 13:60

Jacobs I., and Sjödin B. (1985) 'Relationship of ergometer-specific VO2max and muscle enzymes to blood lactate during submaximal exercise." Br.J.Sports Med. 19:77-80

Janssen P.G.J.M. (1987) Training, Lactate. Pulse rate. Oulu Finland, Polar Electro Oy.

Jones A.M. (1996a) "The use of heart rate monitoring in distance running." Coaching Focus 33: 20

Jones A.M. (1996b) "Heart rate, lactate threshold and endurance training." Coaching Focus. 33: 12-13

Jones A.M., and Doust J.H. (1996) "A 1\% treadmill gradient most accurately reflects the energetic cost of running outdoors." J.Sports Sci. 14: 321-327

Katz A., and Sahlin K. (1990) "Role of oxygen regulation of glycolysis and lactate production in human skeletal muscle." In Pandolf Exercise and sport science reviews. Baltimore: Williams and Wilkins: 1-28

Keith S.P., Jacobs I., and McLellan T.M. (1992) "Adaptations to training at the individual anaerobic threshold." Eur.J.Appl. Phvsiol. 65: 316-323

Keul J., Simon G., and Berg A (1979) 'Bestimmung der individuellen anaerobn Schwelle zur Leistungsbewertung und Trainingsgestaltung. Deutsch Z SportMed 30: 212-218

Kindermann W., Simon G. and Keul J. (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

Klissouras V. (1971) "Heritability of adaptive variation." J.Appl. Phvsiol. 31:338-344

Kohrt W.M., Morgan D.W., Bates B., and Skinner J.S. (1987) 'Physiological responses of triathletes to maximal swimming, cycling and running." Med Sci Sports Exerc. 19(1):51-55

Kohrt W.M., O'Connor J.S., and Skinnner J.S. (1989) 'Longitudinal assessment of responses by triathletes to swimming, cycling and running." Med.Sci. Sports Exerc. 21(5)569-575.

Kruk B., Pekkarinen H., Manninen K., and Haenninen O. (1991) "Comparison of men in physiological responses to exercise of increasing intensity at low and moderate ambient temperatures." Eur.J.Appl. Physiol. 62 (5): 353-357

Kuipers H., and Keizer H.A. (1988) "Overtraining in elite athletes, review and directions for the future." Sports Med. 6: 79-92

La Fontaine T.P., Londeree B.R., Spath W.K. (1981) "The maximal steady state versus selected running events. ${ }^{\text {" Med. Sci. Sports Exerc. 13: 190-192 }}$

Lehmann M., Berg A., Kapp R., Wessinghage T., and Keul J. (1983) "Correlations between laboratory testing and distance running performance in marathon runners of similar performance ability." Int. J. Sports Med. 4: 226-230

Lehmann M., Foster C., and Keul J. (1993) "Overtraining in endurance athletes: a brief review." Med. Sci. Sports Exerc 25: 854-862

Maffulli N., Testa V., Lancia A., Capasso G., and Lombardi S. (1991) "Indices of sustained aerobic power in young middle distance runners." Med. Sci. Sports Exerc. 23: 1090-1096

MacDougall J.D., Reddan W.G., Layton C.R., and Dempsey J.A. (1974) "Effects of metabolic hyperthermia on performance during heavy prolonged exercise." J.Appl. Physiol. 36: 538-544

MacRae H.S., Dennis S., Bosch A.N., and Noakes T.D. (1992) "Effects of training on lactate production and removal during progressive exercise in humans." J.Appl. Physiol. 72: 1649-1656

Mader A., Liesen H., Heck H., Philippi H, Schürch P.M., and Hollman W. (1976) Zur Beurteilung der sportartspezifischen Ausdauerlei-stungsfähigkeit. Sportarzt Sportmed. 25: 4 80-88

McLellan T.M. (1985) "Ventilatory and plama lactate response with different experimental protocols: A comparison of methods." Int.J.Sports Med. 6(1): 30-35

McLoughlin P., Popham P., Linton R.A.F., Bruce R.C.H., and Band D.M. (1992) "Use of arterialised venous blood sampling during incremental exercise." J.Appl.Phvsiol. 73(3): 937-940

Mognoni P., Sirtori M.D., and Lorenzelli F. (1990) 'Physiological responses during prolonged exercise at the power output corresponding to the blood lactate threshold." Eur.J.Appl.Physiol. 60: 239-243

Nadel E.R., Pandolf K.B., Roberts M.F., and Stolwuk J.A.J. (1974) 'Mechanisms of thermal acclimation to exercise and heat." J.Appl. Phvsiol. 37: 515-520

Nadel E.R., (1983) "Effects of temperature on muscle metabolism." in Knuttgen H.G., Vogel A., Poortmans J. (eds) Biochemistry of Exercise. International Series on Sport Sciences, Vol. 13, Champaign, Illinois: Human Kinetics.

Noakes T.D. (1988) "Implications of exercise testing for prediction of athletic performance: A contemporary perspective." Med. Sci. Sports Exerc. 20:319-330

Noakes T.D., (1991) Lore of Running $3^{\text {rd }}$ Edition. Leisure Press, Chamaign, IL

Orok C.J., Hughson R.L., Green H.J., Thomson J.A. (1989) "Blood lactate responses in incremental exercise as predictors of constant load performance." Eur. J. Appl. Physiol. 59: 262-267

O'Toole M.L., Hillier W.D.B., Crosby L.O., and Douglas P.S. (1987) "The ultraendurance triathlete: a physiological profile." Med. Sci. Sports Exerc. 19:45-50

O'Toole M.L., Douglas P.S., Hillier W.D..B. (1989) "Lactate, oxygen uptake and cycling performance in triathletes." Int.J. Sports Med. 10:413-418

Pate R.R., and Branch D.J. (1992) "Training for endurance sport." Med. Sci. Sports Exerc. 24:S340-S343

Pierce E.F., Weltman A., Seip R.L.., and Snead D. (1990) 'Effects of specificity of trainingon the lactate threshold and $\mathrm{VO}_{2}$ peak." Int.J. Sports Med. 11:267-272

Pirnay F.M., Lamy M., Dujardin J., Deroanne R., and Petit J.M. (1972) "Analysis of femoral venous blood during maximal muscular exercise." J.Appl. Physiol. 33:289-292

Robinson S., Edwards H.T., and Dill D.B. (1937) "New records in human power" Science 83: 409-410

Rowell L.B. (1969) "Medicine and science in sports." Circulation. 1: 15

Rusko H., Luhtanen P., Rahkila P., Viitasalo J., Rehunen S., Harkonen M. (1986) "Muscle metabolism, blood lactate and oxygen uptake in steady state exercise at aerobic and anaerobic thresholds." Eur.J.Appl. Physiol. 55: 181-186

Saltin B. (1964) "Aerobic work capacity and circulation at exercise in man. With special reference to the effect of prolonged exercise and/or heat exposure. Acta Physiol. Scand. 62 (Suppl. 230): 1-52

Saltin B., Blomqvist B., Mitchell J.H., Johnson R.L., Wildenthal K., and Chapman C.B. (1968) 'Response to submaximal and maximal exercise after bed rest and training." Circulation 38 (supple 7)

Sawka M.N. and Pandolf K.B. (1990) "Effects of body water loss on exercise performance and physiological functions." In Gisolfi C.V., and Lamb D.R. (eds) Perspectives in exercise science and sports medicine: Fluid homeostasis during exercise. Vol. 3 : 1-38. Benchmark Press, Indianapolis.

Scheen A., Juchmes J., and Cession-Foisson A. (1981) "Critical analysis of the 'anaerobic threshold' during exercise at constant workloads." Eur.J.Appl.Physiol. 46: 367-377

Schnabel A., Kindermann W., Smith W.M., Biro G., and Stegmann H. (1982) 'Hormonal and metabolic consequences of prolonges running at individual anaerobic threshold and fixed blood lactate of $4 \mathrm{mmol} / \mathrm{l}$." Int.J. Sports Med. 3: 163-168

Schrager M., Cohen J., Donovan K., Gastrau P., Snyder A.C. (1993) "Time versus distance protocols for the blood lactate profile." Med. Sci. Sports Exerc. 25 (5): S65 (suppl)

Schneider D.A., La Croix K.A., and Atkinson G.R. (1990) "Ventilatory threshold and maximal oxygen uptake during cycling and running in triathletes." Med. Sci. Sports Exerc. 22:257-264

Segal S.S., and Brooks G.A. (1979) "Effects of glycogen depletion and workload on postexercise oxygen consumption and blood lactate." J.Appl. Phvsiol. 47: 512-514

Shephard R.J., and Åstrand P.O. (1992) Endurance in Sport. Oxford; Blackwell Scientific Publications.

Shephard R.J. "Maximal Oxygen Intake" in Shephard R.J., and Åstrand P.O. (eds) (1992) Endurance in Sport. Oxford; Blackwell Scientific Publications. 192-200

Simon J., Young J.L., Gutin B., Blood D.K., and Case R.B. (1983) "Lactate accumulation relative the anaerobic and respiratory compensation thresholds." J.Appl. Phvsiol. 60: 777781

Sjödin B. and Jacobs I. (1981) "Onset of blood lactate accumulation and marathon running performance." Int. J. Sports Med. 2: 23-26

Sjödin B., Jacobs I., Svedenhag J. (1982) "Changes in onset of blood lactate accumulation (OBLA) and muscle enzymes after training at OBLA." Eur. J. Appl. Physiol. 49: 45-57

Sjödin B. and Svedenhag J. (1985) "Applied Physiology of marathon running." Sports Medicine. 2: 83-89

Sleamaker R. (1989) Serious training for serious athletes. Chapaign IL: Leisure Press 180-182

Snell P. (1990) "Middle distance running" in Reilley T., Secher N., Snell P., and Williams C. (eds) Physiology of Sports. E. \& F.N. Spon: London: 101-120

Snyder A.C., Woulfe T., Welsh R., Foster C. (1994) "A simplified approach to estimating the maximal Lactate steady state." Int. J. Sports Med. 15 (1): 27-31

Spurway N.C., (1992) "Aerobic exercise, anaerobic exercise and the lactate threshold." British Medical Bulletin. 48(3): 569-591

Stainsby W.N. and Brooks G.A. (1990) "Control of lactic acid metabolism in contracting muscles and during exercise." In Pandolf K.B Exercise and sport sciences reviews Baltimore: Williams and Wilkins: 29-63

Stanley W.C., Gertz E.W., Wisneski J.A., Morrison D.L., Neese R., and Brooks G.A. (1986) "Lactate metabolism in exercising human skeletal muscle: evidence for lactate extraction during net lactate release." J.Appl. Physiol. 60: 1116-1120

Stegmann H., Kinderemann W., and Schnabel A. (1981) 'Lactate kinetics and individual anaerobic threshold." Int. J. Sports Med. 2: 160-165

Stegmann H., Kindermann W., (1982) "Comparison of prolonged exercise tests at the individual anaerobic threshold and fixed anaerobic threshold of $4 \mathrm{mmol} / / \mathrm{lactate} . "$ Int. J. Sports Med. 3: 105-110

Svedenhag J., and Sjödin B. (1984) Maximal and sub-maximal oxygen uptakes and blood lactate levels in elite male middle and long distance runners. Int. J. Sports Med. 5: 255261

Svedenhag J., and Sjödin B. (1985) "Physiological characteristics of elite male runners in and off-season." Can. J. Appl. Sports Sci. 10: 127-133

Svedenhag J. "Endurance Conditioning" in Shephard R.J and Åstrand P.O., (eds) (1992) Endurance in Sport. Oxford; Blackwell Scientific Publications. 290-296

Swain D.P., Abernathy K.S., Smith C.S., Lee S.J., and Bunn S.A. (1994) "Target heart rates for the development of cardiorespiratory fitness." Med.Sci.Sports Exerc. 26 (1): 112-116

Tanaka K., and Matsuura Y. (1984) "Marathon performance, anaerobic threshold, and onset of blood lactate accumulation." J.Appl. Phvsiol. 57: 640-643

Therminarias A., Flore P., Chirpaz M.F., and Quirion A. (1989) "Influence of cold exposure on blood lactate response to exercise." Eur.J.Appl.Physiol. 58: 411-418

Urhausen A., Coen B., Weiler B., and Kindermann W. (1993) "Individual anaerobic threshold and maximum lactate steady state." Int.J. Sports Med. 14(3): 134-139

Vermulst L.J.M., Vervoorn C., Boelens-Quist A.M., Koppeschaar W.B.M., Thijssen J.H.H., and de Vries W.R. (1991) "Analysis of seasonal training volume and working capacity in elite female rowers." Int.J.Sports Med. 12 (6): 567-572

Wasserman K. and McIlroy M.B. (1964) "Detecting the threshold of anaerobic metabolism." Am.J.Cardiol. 14:844-852

Wasserman K., Whipp B.J., Koyal S.N., and Beaver W.L. (1973) "Anaerobic threshold and respiratory gas exchange during exercise." J.Appl. Phvsiol 35: 236-243

Welch H.G., and Stainsby W.N. (1967) "Oxygen debt in contracting dog skeletal muscle." Resp. Physiol. 3: 229-242

Weltman A., Nead D., Seip R., Schurrer R., Weltman J., Rut R., Rogal A. (1990a) "Percentages of maximal heart rate, heart rate reserve, and $\mathrm{VO}_{2 \text { max }}$ for determining training intensity in male runners." Int. J. Sports Med 11: 218-222

Weltman A., Snead D., Stein P., Seip R., Schurrer R., Rutt R., Weltman J. (1990b) "Reliability and validity of a continuous treadmill protocol for the determination of lactate threshold, fixed blood lactate concentrations and $\mathrm{VO}_{2 \text { max. }}$ Int. J. Sports Med. 11: 26-32

Weltman A (1995) The blood Lactate Response to Exercise. Champaign, Illinois, Human Kinetics.

Wenger H.A., and Bell G.J. (1986) "The intereactions of intensity, frequency and duration of exercise in altering cardiorespiratory fitness." Sports Medicine 3:346-356

Williams J.R., Armstrong N., and Kirby B.J. (1992) "The influence of sampling site and assay medium upon the measurement and interpretation of blood lactate responses to exercise." J.Sports Sci. 10: 95-107

Wilmore J.H., and Costill D.L. (1988) The physiological basis of the conditioning process. Champaign, Illinois: Human Kinetics

Withers R.T., Sherman W.M., Miller J.M., and Costill D.L. (1981) "Specificity of the anaerobic threshold in endurance trained cyclists and runners." Eur.J.Appl.Physiol. 47:93-104

Womack C.J., Davis S.E., Wood C.M., Alvarez J., and Sauer K. (1992) "The blood lactate response during rowing ergometry as a predictor of rowing performance." Med. Sci. Sports Exerc. 24 (2): S122

Womack C.J., Davis S.E., Wood C.E., Sauer K., Alvarez J., Weltman A., and Gaesser G.A. (1996) "Effects of rowing training on physiological correlates of rowing ergometry performance." Journal of Strength and Conditioning Research. 10(4): 234-238.

Yoshida T., Nagata A., Muro M., Takeuchi N., and Suda Y. (1981) "The validity of anaerobic threshold determination by a Douglas bag method compared with arterial blood lactate." Eur.J.Appl. Phyiol. 46: 423-430

Yoshida T., Takeuchi N., and Suda Y. (1982) "Arterial versus venous blood lactate in the forearm during incremental bicycle exercise." Eur.J.Appl. Phyiol. 50: 87-93

Yoshida T. (1984a) "Effect of exercise duration during incremental exercise on the determination of anaerobic threshold and onset of blood lactate accumulation." Eur. J.Appl. Phvsiol. 53:196-199

Yoshida T (1984b) "Effect of dietary modifications on lactate threshold and onset of blood lactate accumulation during incremental exercise." Eur.J.Appl. Physiol. 53: 200205

Zhou S., Robinson S.J., King M.J., and Davie A.J. (1990) "Correlations between short course triathlon performance and physiological variables determined in laboratory cycle and treadmill tests." Med. Sci. Sports Exerc. 21(5):S200-204

## Appendix One

## PRE-TEST OUESTIONNAIRE

NAME $\qquad$ DATE $\qquad$
D.O.B. $\qquad$ AGE: $\qquad$

As you are going to perform test an incremental test, we would be grateful if you complete the following questionnaire. It is important for our purposes and that of safety. Please answer as accurately as possible. All information is CONFDENTIAL.

1. How do you rate your present fitness level?

Very unfit / moderately fit / trained / highly trained.
2. How do you rate your present body weight? Underweight / ideal / slightly overweight / very overweight
3. Do you, or have you in the past been a smoker? Yes / No If Yes, regular / occasional / ex-smoker
4. Have you had to consult your doctor in the last six months? Yes / No If yes, please detail here or overleaf.
5. Do you currently take any form of medication? Yes / No If yes please give details
6. Please circle any conditions you have suffered:

Asthma Diabetes Epilepsy Bronchitis
7. Do you, or have you, suffered a heart complaint? Yes / No
8. Do you currently have any muscle or joint injury? Yes / No
9. Have you had to halt your training in the last two weeks? Yes / No
10. Do you feel well rested? Yes / No
11. Time of your last meal? $\qquad$ Please give details.
12. Is there any other reason, not mentioned, that should prevent you comfortably performing this exercise test? Yes / No

Signature of subject
Signature of test supervisor $\qquad$

## Appendix Two

## INFORMED CONSENT FOR

## A BLOOD LACTATE PROFLLE

I am being asked to participate in a test where I will be performing a progressive incremental test on a ergometer to determine a lactate profile. The test will begin at an intensity that is very easy to accomplish. The intensity will then increase every three minutes and will terminate on the instruction of the test administrator, or when ever I choose. During the test small blood samples will be taken from a tiny puncture in my earlobe. These samples will be subsequently analysed. The purpose of the test is to assess and monitor my present level of aerobic fitness. The test will last approximately 15 minutes.

I understand that potential risks do exist during the performance of this test, such as loss of balance on the ergometer, disorders of heart beats, abnormal blood pressure, fainting and in extremely rare instances heart attack, or death. I may experience dizziness or light headedness associated with whole body exercise, particularly if I am not well conditioned.

Every effort will be made by the test administrator to minimise such risks and discomforts, through the provision of complete instructions and by observations of signs and symptoms throughout the test. All health and safety precautions will be taken during blood sampling to avoid chances of contamination.

The benefits of the test include:
(i) An assessment and comparison of my present state of fitness with those of other people.
(ii) The monitoring of training adaptations and thereby evaluating the conditioning programme effectiveness.
(iii) The provision of an educational setting where I may be better able to understand the relationship between fitness and health.

The information obtained is treated as privileged and confidential and will not be released to unauthorised personnel, without my expressed permission. The information may, however, be used for statistical purposes, with my right of privacy retained.

Further details of the test have been discussed with the test administrator and any questions I have, have been answered to my satisfaction. Permission for this test is voluntary and I understand that I am able to withdraw from the test at any time I should so desire, whatever the reason, with no penalty.

I have read and fully understand this form and hereby give my consent to participate in the aforementioned incremental test.
$\qquad$

[^1]Appendix Three
Athlete daily food log
Name.......................... Day ............... Date................

| Composition | Quantity |
| :--- | :--- |
| Meal One |  |
| Meal Two |  |
| Meal Three |  |
| Meal Four |  |
| Meal Five |  |
| Snacks intake |  |

## Appendix Four

## Training Log

Name ............................ Week ending

| Monday | a.m. |
| :---: | :---: |
|  | p.m. |
| Tuesday | a.m. |
|  | p.m. |
| Wednesday | a.m. |
|  | p.m. |
| Thursday | a.m. |
|  | p.m. |
| Friday | a.m. |
|  | p.m. |
| Saturday | a.m. |
|  | p.m. |
| Sunday | a.m. |
|  | p.m. |

## Appendix Five

(Published in Journal of Sports Sciences (1995) 13: 25-26

# THE EFFECTS OF DIFFERENT PROTOCOLS ON HEART RATE AND LACTATE VALUES DURING GRADED EXERCISE TESTS FOR MIDDLE DISTANCE RUNNERS 

G.M.J.Dunbar ${ }^{1}$, R. Heagarty ${ }^{1}$, G. Warrington ${ }^{2}$ and J.A.White ${ }^{3}$

1 Human Performance Laboratory, St Mary's College, Strawberry Hill, Twickenham TW1 4SX 2 NCTC, University of Limerick, Limerick, Eire and
3 Department of Public Health Medicine and Epidemiology, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH

Blood lactate measurements are frequently used both to monitor the condition of, and prescribe training intensities for, endurance athletes. The current BASES guidelines suggest a four-stage, 4 min continuous incremental protocol for such assessment (Hale et al., 1988, Position Statement on the Physiological Testing of the Elite Competitor. Leeds: BASS). Not all laboratories do adhere to these guidelines; rather, the protocol is modified to specific needs. The purpose of this study, therefore, was to examine different types of protocol which may alter lactate values obtained during the assessment of middle- and long- distance runners.
Eight county standard male middle and long distance runners (height $1.83 \pm 0.05 \mathrm{~m}$, body mass $72.5 \pm 4.1 \mathrm{~kg}$ ) performed three graded exercise tests in a random order. All testing took place within a 20 day period and each test was performed on the same day of each week, in consecutive weeks, in order to fit consistently within individual training programmes. The three protocols consisted of four submaximal exercise stages, with the same individual running speeds on each occasion. The three protocols were as follows:
(1) 4 min stages with no more than 20 s in between for blood collection, giving a nearly continuous method; (2) 6 min stages separated by a 10 min recovery period, in order to isolate the running speeds; (3) 8 min stages separated by a 10 min recovery period. At the end of each running speed, heart rate was recorded and blood samples were taken from an earlobe to determine lactate concentration (Analox GM6 blood analyser).
An ANOVA with repeated measures revealed that there was no significant difference $(\mathrm{P}<0.05)$ between the mean heart rates and blood lactates values recorded with each protocol (Table 1).
Table 1 Mean ( $\pm$ SE) blood lactate and heart rate values obtained in each protocol

| Stage | Protocol One (4 min) |  | Protocol Two ( 6 min ) |  | Protocol Three (8 min) |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\begin{gathered} \mathrm{La} \\ \mathrm{mmol.}^{-1} \end{gathered}$ | HR b. $\min ^{-1}$ | $\begin{gathered} \mathrm{La} \\ \text { mmol. } \mathrm{l}^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{HR} \\ \text { b. } \min ^{-1} \end{gathered}$ | $\begin{gathered} \mathrm{La} \\ \mathrm{mmol} . \mathrm{l}^{-1} \end{gathered}$ | HR b. $\min ^{-1}$ |
| One | 1.16 (.16) | 156 (5) | 1.17 (.19) | 153 (4) | 1.16 (.16) | 151 (5) |
| Two | 1.60 (.27) | 165 (4) | 1.54 (.25) | 162 (4) | 1.63 (.28) | 162 (5) |
| Three | 2.16 (.35) | 171 (4) | 2.01 (.32) | 170 (4) | 2.26 (.41) | 171 (4) |
| Four | 3.40 (.55) | 177 (4) | 2.90 (.49) | 177 (4) | 3.13 (.55) | 179 (3) |

Further there was good correlation between the values obtained (Table 2).

Table 2: Correlation of lactate and heart rate values between different protocols.

| Protocols | One and Two | One and Three | Two and Three |
| :--- | :---: | :---: | :---: |
| ${\text { Lactate mmol. } \mathbf{l}^{-1}}^{\text {HR b.min }}{ }^{-1}$ | 0.95 | 0.92 | 0.95 |

It was concluded that there is no difference between the 4 min protocol with very short pauses and the 6 and 8 min protocols with 10 min pauses between the running speeds. It is therefore recommended that 6 min running will give the same data as 8 min during a discontinuous protocol and, if time is a constraint, the continuous 4 min protocol is an acceptable alternative.

## Appendix Six

(Published in Journal of Sports Sciences (1997) 15: 47-48)

## A COMPARISON OF SPEED AND HEART RATE TO CONTROL RUNNING AT THRESHOLD INTENSITY.

G.M.J Dunbar ${ }^{1}$, A. Renfree ${ }^{2}$ and J.A.White ${ }^{3}$

1 Human Performance, Unit 1 Brands Hatch Park, Fawkham, Kent DA3 8PU, 2 St. Mary's University College, Strawberry Hill, Twickenham, Middx. TW1 4SX and 3 Department of Public Health Medicine \& Epidemiology, Queen's Medical Centre, Nottingham, NG7 2UH

The purpose of this study was to examine two methods of controlling training at a threshold intensity in well-trained middle distance runners. Ten male middle distance runners of at least county standard (age $20.4 \pm 2.1$ years and mass $70.6 \pm 4.6 \mathrm{~kg}$ ) performed a continuous 3 min incremental protocol as described by Dunbar et al. (1995, Journal of Sports Sciences, 13, 25-26) on a level motor driven treadmill (Powerjog M30, Sports Engineering Ltd, Birmingham). Heart rate (HR) was monitored continuously throughout the test (Baumann and Haldi BHL 6000, Switzerland) and capillarised earlobe blood samples were taken at the end of each speed for the determination of blood lactate (La) concentration by means of an Analox GM6 blood analyser (Analox Instruments, Hammersmith). Visual observation by two independent reviewers established the running speed and heart rate at the lactate threshold (Tlac), for the purpose of subsequent exercise prescription. The mean absolute lactate at Tlac was $2.3 \pm 0.51 \mathrm{mM}$.
On two subsequent visits to the laboratory runners performed 25 min threshold sessions, aiming for Maximal Lactate Steady State (MLaSS), in a randomised order. A treadmill session (T25) was performed at the running speed associated with Tlac; whilst an outdoor trial (F25) was performed at the HR associated with Tlac. Both HR and La were recorded every 5 min during these trials and the values at 5 and 25 min are listed in Table 1.

Table 1 Mean $\pm$ SE HR (beats. $\mathrm{min}^{-1}$ ) and $\mathrm{La}(\mathrm{mM}$ ) values during threshold sessions.

| Time (min) | F25 HR | F25 La | T25 La | T25 HR |
| :---: | :---: | :---: | :---: | :---: |
| 5 | $174 \pm 9$ | $4.1 \pm 1.4^{*}$ | $1.9 \pm 0.4$ | $170 \pm 9$ |
| 25 | $177 \pm 7$ | $3.3 \pm 1.2$ | $2.4 \pm 0.8$ | $182 \pm 10^{* *}$ |

* significantly greater than La at Tlac $\mathrm{P}<0.05$. ${ }^{* *}$ significantly greater than $5 \min \mathrm{P}<0.05$

It was seen that after 5 min in F 25 , La was higher than La at Tlac during the incremental test, However La declined during the session and HR remained stable. In this case, where HR was the means of control, 9 of the 10 sessions saw no rise in La. During T25, HR rose throughout the session, possibly through increase in body temperature and
reduction in plasma volume. Furthermore, only 4 of the 10 sessions demonstrated steady state La.
When individual cases were analysed, HR was a more successful than running speed as a means of controlling steady state conditions for threshold (MLaSS) training in middle distance runners. It is proposed that HR is a more sensitive means of training control, as it better reflects La conditions than prescribed running speed.

## Appendix Seven

(Published in Journal of Sports Sciences (1997) 15: 47)

## TRAINING PRESCRIPTION FOR JUNIOR ROWERS USING BLOOD LACTATE AND HEART RATES.

G.M.J. Dunbar ${ }^{1}$, G.D Warrington ${ }^{2}$ and J.A.White ${ }^{3}$

1 Human Performance, Unit 1 Brands Hatch Park, Fawkham, Kent DA3 8PU, UK
2 National Coaching \& Training Centre, University of Limerick, Limerick, Ireland 3 Department of Public Health Medicine \& Epidemiology, Queen's Medical Centre, Nottingham, NG7 2UH, UK

The purpose of this study was to examine the efficacy of using heart rates prescribed from a blood lactate profile, to control the training intensity required to attain Maximal Lactate Steady State (MLaSS) in subsequent training sessions. Seven well-trained junior rowers (mean $\pm \mathrm{SD}$ ) age $17.4 \pm 0.6$ years and mass $76.7 \pm 4.8 \mathrm{~kg}$ visited the laboratory on two occasions. During the first, a progressive incremental protocol was performed on a rowing ergometer (Concept II, Nottingham), whereby the work rate increased every 3 $\min$ by $28.4 \pm 8.4 \mathrm{~W}$. Heart rate (HR) was monitored continuously throughout the test (Baumann and Haldi BHI 6000, Switzerland) and capillarised earlobe blood samples were taken at the end of each work rate for the determination of blood lactate (La) concentration by means of an Analox GM6 blood analyser (Analox Instruments, Hammersmith). Visual observation by two independent reviewers established the work intensity and heart rate at the lactate threshold (Tlac), for the purpose of subsequent exercise prescription.
A second test involved constant steady state exercise on the same ergometer for 25 min at the intensity associated with the HR at Tlac. This was used as a verification trial for the prescribed threshold session from the incremental test. Both HR and La were recorded every 5 min , as was the distance rowed (m).
Table 1 shows HR was stable throughout the 25 min session corresponded to the prescribed level for each subject. During the session La tended to decline, but this was not significant; and the mean lactate throughout the sessions remained above 4 mM . The distance rowed in each 5 min portion of the session reduced as the session progressed. It was concluded that such methodology for the prescription of training HR prevented La rising through the session, but dictated lower work rates. The decline in work rate through the session, despite stable HR is likely to be a function of cardiovascular drift, due to a rise in core temperature and reduction in plasma volume.

Table 1: Mean ( $\pm$ S.E) HR, La and distance rowed through the 25 min session.

| Time (min) | HR (beats. min $\left.^{\mathbf{- 1}}\right)$ | La (mM) | Distance (m) |
| :---: | :---: | :---: | :---: |
| 5 | $174 \pm 5$ | $5.3 \pm 0.7$ | $1343 \pm 20$ |
| 10 | $178 \pm 5$ | $5.6 \pm 1.3$ | $1327 \pm 31$ |
| 15 | $177 \pm 5$ | $5.2 \pm 1.9$ | $1284 \pm 46$ |
| 20 | $177 \pm 6$ | $4.8 \pm 1.9$ | $1279 \pm 45$ |
| 25 | $177 \pm 6$ | $4.6 \pm 2.0$ | $1265 \pm 41^{*}$ |

[^2]
[^0]:    *significantly lower than 5 minutes ( $\mathrm{p}<0.01$ )

[^1]:    (subject)
    (test administrator)

[^2]:    *significantly lower than $5 \mathrm{~min}(\mathrm{P}<0.01)$

