TRAINING THRESHOLDS IN RUNNERS

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DECLARATION

I Elias Zacharogiannis hereby certify that this thesis has been composed by myself, that it is a record of my own work, and that it has not been accepted in partial or complete fulfilment of any other degree or professional qualification.

Signed .... Date 20-9-91

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DEDICATION

This project is dedicated to my wife Vasilia and my daughter Ageliki-Violeta who have kindly tolerated my absence as well as self-indulgence in research and running, two enjoyable ways to pass the day.

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I would like to extend the greatest thanks to Dr Martin Farrally, Director of the Physical Education at the University of St Andrews who was always available for consultation, suggestions and constant stimulation of thought about the project. His sincere interest in the study was evident and helped me in striving to meet the goals. I would like to extend my appreciation to Dr Konstantin Pavlou, Director of the Exercise Physiology laboratory at the Hellenic Sports Research Institute, for his guidance and help during the data collection period.

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LIST OF ABBREVIATIONS

bpm	beats per minute
CWLVd	constant workload at heart rate deflection point
CWLVT	constant workload at ventilatory threshold
$FECO_2$	fraction of expired carbon dioxide
FEO_2	fraction of expired oxygen
F_H	heart rate frequency
IAT	individual anaerobic threshold
km/ h	kilometers per hour
Kg	kilogramme
LT	lactate threshold
FT	fast twitch
m	meter
min	minute
ml	millilitre
mmol	millimole
µl	microlitre
MSS	maximal steady state
OBLA	onset of blood lactate accumulation
OMA	onset of metabolic acidosis
OPLA	onset of plasma lactate accumulation
Pco_2	partial pressure of carbon dioxide
Po_2	partial pressure of oxygen
T_c	core temperature
QO_2	muscle respiratory capacity
R	respiratory exchange ratio
RCT	respiratory compensation threshold
RPE	rate of perceived exertion

RQ	respiratory quotient
SD	standard deviation
sec	second
ST	slow twitch
TV	treadmill velocity
$\dot{V}CO_2$	volume of carbon dioxide
$\dot{V}E$	volume of expired air
$\dot{V}E/\dot{V}CO_2$	ventilatory equivalent for carbon dioxide
$\dot{V}E/\dot{V}O_2$	ventilatory equivalent for oxygen
$\dot{V}I$	volume of inspired air
VF	ventilatory frequency
$\dot{V}O_2$	oxygen uptake
$\dot{V}O_2max$	maximal oxygen uptake
Vd	heart rate deflection point
VT	ventilatory threshold
$\%CO_2$	percent of carbon dioxide
$\%O_2$	percent of oxygen

ABSTRACT

Ventilatory threshold (VT) and heart rate deflection point (Vd) have recently been used interchangeably as a means to regulate continuous running training intensities. There is also evidence to suggest that these variables may occur at different exercise intensities suggesting that the physiological strain imposed by these exercise intensities may not be the same. Therefore, the purpose of this study was to compare: 1) the oxygen uptake ($\dot{V}O_2$), %$\dot{V}O_2$max, heart rate (HR), %HRmax, and treadmill velocity (TV) at which VT and Vd occur, 2) the validity and reliability of VT and Vd, by test-retest comparison, and 3) the lactate, HR, and gas exchange responses to constant work load exercise (CWL) at a speed which corresponds either with the VT (CWLVT) or Vd (CWLVd).

Twenty male well trained runners were tested on four different occasions: a) an incremental treadmill test to exhaustion was carried out to determine VT, Vd, and peak $\dot{V}O_2$, b) 48 hours following the peak $\dot{V}O_2$ test VT and Vd reproducibility was tested by employing a submaximal test (i.e. 95%$\dot{V}O_2$max), c) constant work load measures were performed on two consecutive days, in a random order, immediately after the submaximal test. The VT was determined as the work load before systematic increase in FEO_2 (fractional expired oxygen) without a concomitant increase in $FECO_2$ (fractional expired carbon dioxide). The work load at which HR departed from linearity was considered as Vd.

A paired t test showed that VT was significantly lower than Vd when expressed as $\dot{V}O_2$, HR, and TV ($p<0.05$).

Direct correlations yielded significant relationships ($p<0.05$) between VT and Vd for all measures apart from %HRmax and %$\dot{V}O_2$max. The highest correlation was found for TV ($r=0.87$). The treadmill velocity also provided the highest test-retest correlation for expressing VT ($r=0.95$) followed by HR ($r=0.94$), $\dot{V}O_2$ and %HRmax

(r=0.86), and %$\dot{V}O_2$max (r=0.75). Vd variables were not equally reproducible being HR (r=0.75), TV (r=0.69), $\dot{V}O_2$ (r=0.52), %HRmax (r=0.29) and %$\dot{V}O_2$max (r=0.07). VT was noted in all tests, three subjects failed to demonstrate Vd in at least one of the evaluations while one subject did not present Vd in both tests.

Blood lactate concentration during CWL trials after initial increase stabilised at different levels without any substantial trend in the individual lactate curves, except for the CWLVd where lactate values at the10th minute of exercise were all greater than the resting value. Analysis of variance showed that the lactate values at CWLVT were significantly lower for all data points, except the resting value, compared with the corresponding CWLVd values.

Volume of expired air ($\dot{V}E$), ventilatory frequency (VF), respiratory exchange ratio (R), $\dot{V}O_2$, ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$) and ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$) values were significantly higher at CWLVd compared with the respective CWLVT values at all measured data points.

It is concluded that in well trained runners:

I) deflection in heart rate does not always occur and when it does it correlates with ventilatory threshold but does not coincide.

II) athletes can maintain a running speed equivalent to CWLVd for extended periods of time. This results in greater physiological strain than the corresponding exercise at VT.

CHAPTER 1: INTRODUCTION

1.01: INTRODUCTION

CHARTER 1: INTRODUCTION

1.01: INTRODUCTION

Cardiorespiratory endurance has traditionally been quantified by measuring maximal oxygen uptake (referred to either as $\dot{V}O_2$max or peak $\dot{V}O_2$) [McConnell, 1988; Costill et al., 1970; Farrell et al., 1979]. However, in spite of the physiological significance of peak $\dot{V}O_2$, a wide variation in the relationship between peak $\dot{V}O_2$ and running performance (r=0.08-0.90) has been demonstrated (Astrand and Rodahl, 1986). Indeed, individuals with similar peak $\dot{V}O_2$ values often perform differently in endurance events and may improve endurance performance without improving peak $\dot{V}O_2$ (Costill et al., 1974; Conley and Krahenbuhl, 1980). There is also evidence to support the contention that peak $\dot{V}O_2$ is an insensitive indicator of the training induced improvements in endurance capacity of trained male and female runners (Williams and Nute, 1982). Additionally in recent years evidence has indicated that as much as 50-93% of the variability in this measurement may be explained by genetic potential (Klissouras, 1973; Buchard and Lortie, 1984; Fagard, 1991). The low correlation presented by this measure with performance in highly trained runners (i.e. for those who posses equal peak $\dot{V}O_2$ values) and the differences in the physiological stress encountered by the use of the same fractional utilisation of the peak $\dot{V}O_2$ to regulate training and racing pace forced Sports Scientists to search for other variables to explain greater variance in the running performance.

Another factor, "anaerobic threshold" (AT) has been suggested to be a better measure of cardiorespiratory endurance capacity than peak $\dot{V}O_2$ (Wasserman et al., 1973; Davis, 1985). Some investigators believe that the AT provides a more

complete description of the circulatory and metabolic adaptations to endurance exercise than the peak $\dot{V}O_2$ (Wasserman and McIlroy, 1964; Jones and Ehrsam, 1982).

The AT is broadly defined as the $\dot{V}O_2$ or workload just below the onset of metabolic acidosis (OMA) [Wasserman et al., 1973; Weltman et al., 1978] and has received much criticism with regard to the concept itself and the methods measuring it. Since the AT was first defined it has become a very popular topic, yet one which continues to stimulate much debate. Several investigators have questioned the very term "anaerobic" and whether it indeed is due to anaerobiosis (Chirtel et al., 1984; Davis and Gass, 1981; Holloszy, 1973; Hughes et al., 1982). Due to these doubts about decrements of oxygen to the tissues, the terms lactate threshold (LT), ventilatory threshold (VT), and heart rate deflection point (Vd) have been invoked to describe the phenomenon with respect to the various methods used for measurements.

Hill et al., (1924) and later Wasserman and McIlroy, (1964) felt that the increase in blood lactate was inextricably linked to the onset of local muscle hypoxia at a certain work rate. Wasserman used the word "anaerobic" to indicate that O_2 supply was insufficient for the working muscle's energy need. Thereafter blood lactate kinetics have been used to identify AT. Different terminology has been used in the literature to describe lactate kinetics during either incremental exercise or submaximal constant workload tests. The aerobic threshold (Kindermann et al., 1979), anaerobic threshold (Wasserman et al., 1973), aerobic-anaerobic threshold (Mader et al., 1976), onset of blood lactate accumulation (OBLA) [Sjodin and Jacobs, 1981], onset of plasma lactate accumulation (Farrell et al., 1979), lactate threshold (Ivy et al., 1980), lactate turning point (Davis et al., 1983), maximal steady state (Lafontaine et al., 1981), the individual anaerobic threshold (IAT) [Stegmann et al., 1981], excess lactate (Williams et al., 1967), and aerobic capacity (Davis et al.,

1970) are examples. Some of these terms refer to the exercise intensity at which a given lactate accumulation occurs, others refer to the intensity which elicits a lactate concentration above resting value. These intensities can be expressed in absolute terms as treadmill velocity (TV), cycle ergometer power outputs, heart rate (HR), or oxygen uptake ($\dot{V}O_2$). They can also be expressed as the $\%\dot{V}O_2max$ or %HRmax at which they occur. The most often used technique is the subjective determination of the systematic increase above resting value in blood lactate this is termed as the Lactate Threshold.

At the heart of the controversy surrounding these measurements are the mechanisms by which these changes in blood lactate concentration, gas exchange, and HR occur during incremental exercise. One theory suggests that these changes are due to an increased reliance on the glycolytic pathway as a result of an increased need for energy that cannot be provided by the TCA cycle alone (Wasserman and McIlroy, 1964). An alternative theory states that the increase in lactate is due to the decreased clearance of lactate by the liver and muscles (Donovan and Brooks, 1983). A third theory suggests that the increase in blood lactate is due to the recruitment of fast twitch (glycolytic) muscle fibers at high workloads (Clausen, 1976).

The VT has been measured as the $\dot{V}O_2$, $\%\dot{V}O_2max$, HR, %HRmax or workload above which there is a non-linear increase in the volume of expired air per minute ($\dot{V}$Eatps/minute), the volume of expired carbon dioxide per minute ($\dot{V}CO_2$/minute), or an abrupt increase in the fraction of expired oxygen (FEO_2) [Wasserman et al., 1973]. To avoid the increase in these variables due to other reasons such as anxiety, pain, hypoxemia, and volitional ventilation, recently a sensitive and more reliable measure of the VT has been proposed by Davis, (1985). This was based on the concept of "isocapnic buffering" described by Wasserman and McIlroy, (1964). These investigators have shown that for "rapid" incremental exercise tests, $\dot{V}E$ and

$\dot{V}CO_2$ increase at the same rate for a few work rates beyond OMA. This is evident by the fact that $\dot{V}E/\dot{V}CO_2$ does not increase at the OMA. Thus the workload or $\dot{V}O_2$ at which there is an abrupt, continuous rise in $\dot{V}E/\dot{V}O_2$ without a concomitant increase in $\dot{V}E/\dot{V}CO_2$ is the most specific gas exchange method for detection of VT (Caiozzo et al., 1982; Davis, 1985).

The velocity at which the linearity of the work rate-heart rate relationship is lost or the gradient changes has been called deflection velocity (Vd) [Conconi et al., 1982]. Although the physiological rational behind this HR response, which occurs at near maximal effort during incremental exercise test, is not clear (Wyndham et al., 1959; Davis, 1968), Conconi and colleagues, (1982) suggest that this point coincides with OMA. In a study with 10 runners they showed that Vd was almost perfectly correlated (r=0.99) and coincident with the LT. Besides running, similar results have been obtained in cycling, rowing, cross-country skiing, roller skating, walking, and even horse racing (Conconi et al., 1984; Droghetti et al., 1985; Cellini et al., 1986; Borsetto et al., 1989; Bunc et al., 1988). Furthermore, this method has also been used as a research tool. For instance, it was selected to evaluate the anabolic effects of stanozolol in runners (Ballarin et al., 1986) and it was also used to determine the proportion of speed obtained from anaerobic glycolysis in runners (Borsseto et al., 1989).

The term "threshold" has also been debated. Some investigators believe that there is no single point where these changes occur and that there is simply a continuous curvilinear rise in the variables measured without a real threshold being apparent (Chirtel et al., 1984; Davis and Gass, 1981; Yeh et al., 1983).

The VT is based on the concept that an increase in blood lactate concentration during incremental exercise leads to an increased buffering of H^+ (hydrogen proton), carried by lactate, (therefore, lactic acid) by the bicarbonate system. This results in an increase in blood CO_2 which is expelled by the lungs, thus

increasing $\dot{V}CO_2$ and $\dot{V}E$ above OMA (Wasserman et al., 1973). Several studies have shown that VT and LT occur at similar exercise intensities (Caiozzo et al., 1982; Davis et al., 1976; Reinhard et al, 1979; Yoshida et al., 1981) while others have shown that these two measures are dissociated (Green et al., 1983; Hughes et al., 1982; Simon et al., 1983; Yeh et al., 1983). ATP hydrolysis, as a major source of H^+ (Hochacha and Momsen, 1983) is successively related to ATP formation, glycolysis, muscle lactate formation and blood lactate concentration. Changes in ventilation are linked to H^+ and $P\bar{v}CO_2$ (Blood partial pressure of carbon dioxide) while changes in $P\bar{v}CO_2$ are related to H^+ via the bicarbonate buffer system. Thus, not surprisingly, many studies have shown a correlation between blood lactate concentration and ventilation. Since this is not a causal relationship (Walsh and Banister, 1988) it explains the numerous experimental manipulations that dissociate LT from VT.

The physiological rational link behind Vd, as its proponents believe, is that above a certain exercise intensity which corresponds with OMA, oxygen demand does not rely mainly upon increasing cardiac frequency. A small amount of extra oxygen, near maximal effort, is extracted by the muscles from the circulatory blood by widening the A-V O_2 difference. Indeed there is evidence in the literature to support this contention (Wade et al., 1962; Donald et al., 1954) but there are no direct indications to justify that this exercise intensity corresponds with the OMA. Several studies have shown that Vd coincides with either the LT or VT (Kispert et al., 1988; Brettoni et al., 1989; Bunc et al., 1988) whereas other studies failed to do so (Goodman et al., 1986; Francis et al., 1989). Doubts arise also about the reproducibility and the validity of Vd as a consistent physiological phenomenon. Several studies reported excellent reproducibility (Conconi et al., 1982; Maffulli et al., 1987) while other studies either failed to validate the measure or found it to correlate, in fact to coincide, with the 4 mmol LT (Ribeiro et al., 1985; Tokmakides

and Leger, 1987; Kuipers et al., 1988).

Many of the problems in unifying these theories are associated with measurements of LT, VT, and Vd. Different methodological approaches pertinent to the estimation of LT which have been used as the criterion measures for the validation of VT and Vd, together with inconsistent methods of blood sampling, have contributed to the problem of comparing studies. Studies using blood samples for lactate have included venous, arterial, and so-called arterialized venous samples (Caiozzo et al., 1982; Davis et al., 1976; Ivy et al., 1980). The measurement of VT has been inconsistent because different researchers have used different parameters to estimate it- e g. $\dot{V}E$, $\dot{V}CO_2$, FEO_2, and /or $\dot{V}E/\dot{V}O_2$.

Another problem associated with the determination of VT, LT, and Vd is that these are usually determined by subjective visual selection of breaking points on a graph. Studies have shown that a large inter-investigator variability exists in selecting these points which decreases objectivity and precision (Davis et al., 1976; Yeh et al., 1983). A truly objective determination of the LT, VT, and Vd has not yet been accepted.

Despite the problems and controversies involving LT, VT, and Vd these measures have some attractive features. Studies have found high correlations between VT, LT, and Vd and middle and long distance running performance (Farrell et al., 1979; Kumagai et al., 1982; Powers et al., 1983; Tanaka et al., 1984; Maffuli et al., 1987; Iwaoka et al., 1988; Cunningham, 1991). It has also been shown that VT and Vd increased significantly in response to endurance training in highly trained runners (Tanaka et al., 1984; Conconi et al., 1982; Olsen et al; 1988). VT has been shown to have high test-retest correlations (Aunola and Rusko, 1984; Davies et al., 1979). Available evidence also suggest that the regular middle and long distance runner's training and racing pace can be monitored in relation to the individual running speed either at VT or Vd (Sjodin and Svedenhag, 1985; MacDougal and

Sale, 1981; Conconi et al., 1983).

These findings suggest that VT and Vd may be highly representative of cardiorespiratory endurance performance in trained runners and therefore worthy of inclusion in the physiological profile of runners.

A great deal of research has been done studying the responses of VT and Vd during incremental exercise tests while relatively few studies have examined these responses to constant workload exercise (CWL).

CWL tests have been conducted either at various percentages of $\dot{V}O_2$max or at VT and LT (Scheem et al., 1981; Ribeiro et al., 1986). Lactate profiles from these tests have shown that lactate levels plateau after the first minutes of exercise at low workloads, but during higher exercise intensities, lactate continues to rise throughout the entire test. The highest exercise intensity which keeps in balance lactate accumulation and removal from the blood (i.e. there is no continuous increase in blood lactate levels after the initial rise) during prolonged exercise has been shown to correspond with the 4 mmol LT or the second $\dot{V}E$ and /or $\dot{V}CO_2$ breaking point (VT_2), as determined from an incremental exercise test (Stegmann and Kinderman, 1982; Ribeiro et al., 1986). Furthermore, there are no studies to examine lactate and ventilatory responses to CWL exercise relative to Vd.

Investigation of the lactate and gas exchange responses during prolonged CWL exercise at VT and Vd, compared to the responses during incremental exercise, may help to provide a better understanding of what is commonly referred to as OMA. The study of gas exchange and lactate kinetics during CWL at VT and Vd may also contribute to a better understanding of the cardiovascular and metabolic adaptations to endurance exercise. Due to the problems of subjective determination of OMA from incremental exercise tests, a comparison of these methods to the objective determination using CWL tests will help to provide a basis for using these tests. It may be possible that during incremental tests, each workload has an

influence on the next and this may distort the data. Therefore a study of the gas exchange, HR, and lactate responses during CWL exercise at VT and Vd may contribute to the better understanding of the physiological consequences behind these concepts. Additionally, since exercise intensities relative to VT and Vd are often used by the runners to regulate continuous running training, the study of metabolic and respiratory responses to CWL exercise relative to VT and Vd may help in the evaluation of the physiological stress imposed by these workloads.

CHAPTER 2: REVIEW OF LITERATURE

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2.01: INTRODUCTION

The increasing interest (Farrell et al., 1979; Tanaka et al., 1984; Rodes and Mckenzie, 1984; Cunningham, 1991; Iwaoka, 1988; Kiousis et al., 1991; Conconi et al., 1982) in assessing onset of metabolic acidosis by using lactate, gas exchange, and heart rate indices stems largely from a series of papers over the last 18 years. These researchers reported that endurance exercise performance is well correlated to the highest workload or oxygen uptake that can be achieved during incremental exercise, either before an abrupt increase above resting value in blood lactate concentration, or before the breaking from linearity in the volume of expired air, together with changes of other subsidiary ventilatory variables, and/or before heart rate deflection point.

In recent years authors have challenged the "anaerobic threshold", concept and the methods of its detection. For the sake of clarity in the present discussion, thresholds will be identified by the parameters used to measure them (Bhambhani and Singh, 1985; Hughes et al., 1982). Therefore they will be referred to as lactate threshold (LT), ventilatory threshold (VT), and heart rate deflection point (Vd) when lactate, gas exchange, and heart rate indices have been used respectively. Furthermore, if either two ventilatory or lactate thresholds were determined, then the first will be referred to as a ventilatory or lactate threshold 1 (VT_1 or LT_1) and the second as ventilatory or lactate threshold 2 (VT_2 or LT_2).

The anaerobic threshold has been the target of considerable controversy. It is the word "anaerobic" which is been questioned as it does not describe adequately

the physiological status during intense exercise. It is well documented that hypoxia is not present (Brooks, 1985; Pirnay et al., 1972; Change and Quistorlf, 1978), at the so called "anaerobic threshold" (Wasserman and McIlroy, 1964) exercise intensity. Therefore it has been suggested that a more precise term is "onset of metabolic acidosis" (OMA), which better reflects the intracellular pH changes as a result of the increased exercise intensity (Walsh and Banister, 1988; Weltman et al., 1978; Hochacha and Momsen, 1983). Indeed the exercise intensity which is responsible for the intracellular pH decline due to H^+ (hydrogen proton) accumulation, produced from the glycolytically formed ATP hydrolysis (Hochacha and Momsen, 1983), corresponds with OMA and the beginning of fatigue. Low intracellular pH has been shown to inhibit glycolysis, by inhibiting the activity of phosphofructokinase (Suton et al., 1981; Trivedi and Danforth, 1966; Linderman and Fahey, 1991). Since maximal exercise is dependent upon glycolysis to sustain power output, a decrease in glycolytic activity may contribute to the sensation of fatigue and the cessation of exercise. Finally, Chase and Kushmerick, (1988) reported that the recovery of tension for a muscle was dependent upon pH, the authors suggesting that low intracellular pH was directly related with fatigue.

The originators of LT, VT, (Wasserman and McIlroy, 1964) and Vd (Conconi et al., 1982) claim that these measures can define indirectly either the work load or $\dot{V}O_2$ just before OMA during an incremental exercise test.

Since the purpose of this study was to compare the $\dot{V}O_2$, $\%\dot{V}O_2max$, heart rate (HR), %HRmax, and the treadmill velocity at which VT and Vd occur, the validity and reliability of VT and Vd by test-retest comparison, and the the lactate, HR, and gas exchange kinetics during 30 minute constant work load (CWL) exercise at VT and Vd, this chapter reviews the research on each of these topics. Studies dealing with the relationship between the VT, Vd, and LT are also examined.

2.02: ONSET OF METABOLIC ACIDOSIS

Christiansen, Douglas and Haldane, (1914) were the first to show increase in blood lactate with increasing exercise intensity in man. They observed that $\dot{V}E$ and $\dot{V}CO_2$ also increased as work rate increased. Hill, Long, and Lupton, (1924) postulated that the increase in blood lactate concentration with increased work load was due to an inadequacy of oxygen available to aerobically produce energy for the working muscles. The first description of the lactate threshold was by Owles, (1930). This investigator observed that there existed a critical metabolic level below which blood lactate does not increase and above which it does.

Margaria, Edwards and Dill (1933) developed the theory that exercise used aerobic processes up to peak $\dot{V}O_2$, above which energy demands were met by anaerobic glycolysis. They also developed the concept of the alactacid and lactacid components of the oxygen debt and theorized that during steady state exercise, lactate production occurred only at the onset of exercise, during an oxygen deficit.

Wasserman and McIlroy, (1964) were the first to introduce the term "anaerobic threshold". They defined the "threshold of anaerobic metabolism" as the point at which the concentration of bicarbonate in arterial blood decreases and the concentration in lactic acid rises. They used breath by breath ventilatory measurements and arterial blood samples during incremental cycle ergometer tests.

Hollmann, (1961), working in Germany, independently described the same concept of anaerobic threshold and its non-invasive detection. Much of the interest in this area of Exercise Physiology in Europe and Scandinavia today undoubtedly stems from Hollmann's early work and its linkage of the onset of metabolic acidosis with endurance performance.

Wasserman et al., (1973) redefined the anaerobic threshold as the work load or

$\dot{V}O_2$ just below the onset of metabolic acidosis. These investigators explained the mechanism by an inadequate delivery of oxygen to the working muscles at higher work loads, leading to an increase in the rate of anaerobic glycolysis. This results in an increase in the muscle lactate production and accumulation of lactate in the blood. Blood is buffered by the bicarbonate system which increases the blood carbon dioxide and this additional carbon dioxide is expelled by the lungs. This can be seen as an increase, during incremental exercise, in $\dot{V}CO_2$ and $\dot{V}E$ plotted against time. Various gas exchange measurements were obtained from 85 normal subjects aged 17-91 years using breath-by-breath gas exchange techniques during incremental bicycle ergometer tests. The authors concluded that the most sensitive indicators of OMA by gas exchange were a non-linear increase in $\dot{V}E$ or $\dot{V}CO_2$, or a rise in FEO_2. Repeated studies using these gas exchange measurements showed that OMA was nearly identical in several measures taken over a nine month period of time.

2.03: LACTATE AND VENTILATORY THRESHOLD

Costill, (1970) suggested that blood lactate concentration is due to production, rate of cellular utilization, excretion, and rate of diffusion of lactate into adjacent tissues. Thirty one highly trained distance runners were used in this study, venous blood samples were taken five minutes after races varying from 1.61 to 42 km in length. Another eleven distance runners completed maximal and submaximal treadmill tests with expired gas being collected and analyzed during the tests, and blood samples after each test. Higher lactate values were found after the shorter

races while the longer races produced lower lactate values. When $\dot{V}O_2$ for running was less than 70% of peak $\dot{V}O_2$, little or no increase in blood lactate concentration was observed. Highly trained runners were able to exercise up to 90% of peak $\dot{V}O_2$ with only moderate lactate accumulation. Post-exercise blood lactate values were found to be related to the fractional utilization of peak $\dot{V}O_2$ above 70%$\dot{V}O_2$max. It was concluded that training for competitive distance running seemed to allow a greater fraction of the aerobic capacity to be utilized without lactate accumulation.

Jordeldft, Juhlin-Dannfeldt, and Karlsson, (1978) examined the release of lactate in relation to tissue lactate. They had four students complete an incremental bicycle ergometer test to peak $\dot{V}O_2$ and submaximal tests at 30, 50, 70, and 90% of peak $\dot{V}O_2$. Blood samples were taken from the femoral artery and vein while the subjects were supine after four and twelve minutes on the bike during the constant workload tests. Separate tests took muscle biopsies at minutes 4 and 12 during constant work load exercise. Douglas bags were used for gas collection. Muscle lactate did not increase significantly until the subjects were working at 70% of peak $\dot{V}O_2$. At 70% of peak $\dot{V}O_2$, muscle lactate leveled off, and after four minutes a significant difference between arterial and venous blood lactate was seen, indicating release. The investigators concluded that lactate release levels off at high muscle lactate concentration due to translocation hinderences for lactate within exercising muscle.

McLellan et al., (1981) designed a study to investigate the validity of the above designated lactate levels of 2 and 4 mmol/l as indicative values for the transition thresholds. The effect of work load duration on invasive (LT) and noninvasive (VT) determinations of the transition thresholds was examined. Six subjects underwent cycle ergometer tests while the work load was increased 15 watts each 1 or 2 minutes and 30 watts each 3 or 4 minutes. The first and second breakaway points in the plot of $\dot{V}E$ versus $\dot{V}O_2$ were used as a non-invasive

method for the determination of the so called "aerobic and anaerobic" thresholds while the work load at an initial rise in lactate and the onset of the rapid rise in lactate was used as an invasive method. The findings showed that relative "aerobic and anaerobic threshold" values were significantly higher for the 4 minute test than the 1 minute test (60.13% vs. 52.03%$\dot{V}O_2$max, and 85.09% vs.77.52%$\dot{V}O_2$max, for "aerobic and anaerobic" threshold, respectively). Lactic acid at the first lactate breaking point was significantly higher for the 3 minute test when compared to the 1 minute test but there was no difference in lactate levels at the second abrupt increase in lactate work load defined in this study as "anaerobic threshold". In addition, $\dot{V}O_2$ associated with these lactate values was found to be different from those at first and second breaking points when $\dot{V}E$ is plotted against $\dot{V}O_2$. Based on these findings the investigators concluded that although lactate levels of 2 and 4 mmol/l are usually found at the transition thresholds they are arbitrary and do not always apply. Relative transition thresholds values may vary significantly according to the test protocol.

Ivy et al., (1980) determined the lactate threshold as the point just before the onset of blood lactate accumulation. This study compared fibre type and muscle respiratory capacity to the lactate threshold (LT). Incremental cycle ergometer tests were completed by 13 males with increases in workload of 25 watts every minute until volitional exhaustion. Blood was sampled every minute from the antecubital vein. Gas exchange variables were measured every minute. Muscle biopsies were taken from the vastus lateralis and were used to measure respiratory capacity (QO_2) defined as the rate of pyruvate oxidation and the percentage of slow twitch (ST) muscle fibres. Relationships were found between the LT and the percentage of ST fibres (r=0.74) and between LT and QO_2 (r=0.93). The association between the LT and percentage of ST fibres may be due to a high mitochondrial density in ST fibres. It was postulated by these investigators that the concentration of blood lactate was

due to the balance between production and removal of lactate from the blood. The ventilatory threshold was measured by the non-linear increase in $\dot{V}E$ and no significant differences were found between VT and LT.

A study was conducted by Green et al., (1983) on five men and five women using two identical progressive exercise tests on a bicycle ergometer. The VT was determined from the initial breakpoint in the relationship between $\dot{V}E$ and O_2 uptake by a computer alogorithm and the LT was determined by an abrupt increase in arterial blood lactate. Three more tests were conducted and stopped below, at, and above the VT. Muscle biopsies were taken four to five seconds after each test was stopped. They found that the LT occurred before the VT. At 79% of VT, muscle lactate concentration had increased four times the resting value. Muscle lactate concentration was significantly higher than blood lactate values for all the tests. It was concluded that muscle glycolysis increased before the LT or VT and that LT and VT are not coincidental.

Donovan and Brooks, (1983) and Brooks, (1985), studied the LT concept from a different prospect. They hypothesized that the systematic increase in blood lactate observed for work rates greater than 50-60 $\%\dot{V}O_2max$ could not be due to an increased lactate production but instead to a reduced hepatic clearance. They postulated that an increased vasoconstriction, mediated by the sympathetic nervous system, occurs with progressively intense exercise and reduces blood flow to the liver. This would then diminish that organ's ability to remove lactate from the blood and would allow the production of lactate to outstrip removal. The above hypothesis is an extension of a study which used isotopic tracers in the rat before and after endurance training. They concluded that the reason blood lactate is reduced at a particular work rate is not due to a reduced lactate production by the muscle but to an increased lactate removal. However, several studies (Henricksson et al., 1977; Karlsson, 1971; and Saltin et al., 1976), using muscle biopsy, have

clearly demonstrated reduced lactate production following endurance training. This discrepancy raises questions about the validity of the isotopic tracer approach in the study of lactate kinetics. Finally Brooks, (1985) questions the oxygen limiting hypothesis of the LT. He is also categorical about the use of VT and its concordance with LT basing his arguments on several papers which will be discussed in a later section (2.05).

2.04: HEART RATE DEFLECTION POINT

The asymptotic nature at near maximum effort of the heart rate frequency (F_H) during an incremental exercise test, when plotted against either $\dot{V}O_2$ or exercise workload, was first shown by Wyndham et al., (1959). These researchers conducted a study to examine the accuracy of predicting peak $\dot{V}O_2$ by using Astrand-Ryhming's prediction nomogram which assumes a linear relationship between F_H and $\dot{V}O_2$. Wyndham and coworkers postulated that the O_2 intake curve reaches its asymptote more slowly than does the heart rate; hence if heart rate is plotted against O_2 intake and a straight line fitted and extrapolated to the maximal value of HR, the O_2 intake at this value is an underestimate of the actual measured maximum O_2 intake. They have also suggested two possible explanations for the observation that the curve of O_2 intake/work rate approaches its asymptote more slowly than does the curve of heart rate/work rate:

a) most individuals cannot force themselves to work hard enough to reach a

maximum level of O_2 intake

b) a further possible explanation may lie in a consideration of the circulatory parameters concerned in the transport of O_2 from the lung by heart and blood circulatory systems to working muscles. The uptake of O_2 is governed quantitatively by the equation:

$\dot{V}O_2$ (ml/min)=cardiac frequency(beats/min) * stroke volume (ml/beat) * (A-V O_2 difference vol%)

As has been shown (Astrand P-O and Astrand I, 1958), no further rise in stroke volume takes place beyond a F_H~120 beats/min, then at higher rates of work a further increase in $\dot{V}O_2$ will depend solely on the cardiac frequency and arteriovenous oxygen difference. There is no direct evidence in man to indicate which of these two variables is the first to reach limiting values at maximum effort. The results of this study, however, suggested that it is cardiac frequency, and that a small amount of extra oxygen is extracted by the muscles from the circulatory blood by widening the A-V O_2 difference. The ability of the body to increase the A-$\dot{V}O_2$ difference in severe exercise could be explained by the fact that there is a virtual shut-down of blood flow to kidney and other abdominal viscera as well as non-working muscles where normally the rate of extraction of O_2 per litre of blood is not high. The proportion of the total blood flow which normally goes to these areas could thus be diverted to working muscle where the rate of extraction of O_2 is high. The O_2 unsaturation of mixed venous blood would thus increase and, for a given level of cardiac output, the O_2 intake could increase and serve the energy demands of an increasing work rate without increasing F_H.

Another study conducted by Davies, (1968) to examine the accuracy of the Astrand-Ryhming, Margaria, and Wyndham peak $\dot{V}O_2$ prediction nomograms confirmed the asymptote of the F_H at near maximum effort and it is suggested that the same physiological mechanisms explain this phenomenon.

Conconi et al., (1982) designed a non-invasive field test to determine OMA. They examined 210 runners who ran continuously on the track from an initial velocity of 12-14 km/h up to submaximal velocities varying according to the runners' capability, while heart rates were recorded and plotted against running speeds. The velocity at which the linearity of running speed-heart rate relationship was lost was called the deflection velocity (Vd). It was shown that the Vd correlated very highly (r=0.99) with the OMA determined by blood lactate measurements, while the reproducibility of the Vd determination was also found to be highly significant (r=0.99). Furthermore, they reported very high correlations between Vd and competitive running speeds, r=0.93 in the 5000m, r=0.95 in the marathon, and r=0.99 in 1-hour races. The feasibility of the measure was also emphasised. Finally, the use of the running speed-heart rate relationship, an approach which can be carried out in the field, is said to be useful for the training and racing pace monitoring.

The relationship between velocity (V) and heart rate (HR) was determined in four canoeist, 42 cross-country skiers, 73 cyclists, 9 ice-skaters, 10 roller-skaters, 32 rowers, and 20 walkers by Droghetti et al., (1985). The athletes were asked to increase work intensity progressively, from low to submaximal velocities; HRs were determined by ECG in roller-skating, ice-skating, and walking, or read on a cardiofrequency meter in canoeing, cross-country skiing, cycling, and rowing. In all athletes examined the linearity of the V-HR relationship was maintained up to a submaximal speed (Vd) beyond which the increase in work intensity exceeded the increase in HR. Vd and OMA determined through lactate measurements, in only 19 athletes, were coincident (6 cross-country skiers, 3 cyclists, 2 roller-skaters, 3 rowers, and 5 walkers). Vd was correlated with the average speeds maintained in walking (20 km, n=13, r=0.88), cross-country skiing (15 km, n=20, r=0.80; 30 km, n=8, r=0.82; 12 km, n=7, r=0.86; 11 km, n=7, r=0.86) and cycling (1000 m, n=68,

r=0.83). They suggested that Vd was a limiting factor in those aerobic events.

Bunc et al., (1988) designed a study to examine if the point where the HR-exercise intensity relationship departs from linearity in an incremental exercise test may be employed as a predictor of VT. To examine this, 28 highly trained male long distance runners were tested on a treadmill and 17 untrained young male subjects were tested on a cycle ergometer using a continuous incremental protocol. The VT was determined from the dependence of $\dot{V}E$ on $\dot{V}O_2$ and/or $\dot{V}CO_2$. The $\dot{V}O_2$, HR and exercise intensity at VT were compared with the same parameters determined from the dependence of HR on exercise intensity. No significant differences were found between VT and Vd. They concluded that Vd coincides with VT.

Ballarin et al., (1988) investigated the validity by test-retest comparisons of Vd in children and adolescents. All tests were performed either outdoors on a 400 m track (n=159, 110 males and 49 females) or indoors on a 200 m track (n=115, 59 males and 56 females). The subjects increased their work intensity progressively from low to submaximal speeds. HR was determined by a heart rate monitor (Sport Tester TM PE 3000). In every subject examined, the linearity of the work load-HR relationship was lost at a speed called Vd, above which the increase in velocity exceeded the increase in HR. HR at Vd was defined as HRVd. The respective test-retest correlation coefficients for Vd, HRVd, and slope of the linear part of the graph were 0.99, 0.82, and 0.95 when determined outdoors and 0.99, 0.84, and 0.93 when determined indoors. The outdoor-indoor correlation coefficients were 0.93, 0.85, and 0.28, respectively for Vd, HRVd, and slope. The authors suggested that application of this test to children and adolescents in running may prove useful in cross-sectional and longitudinal studies of the development of aerobic power during growth.

2.05: LACTATE THRESHOLD, VENTILATORY THRESHOLD AND HEART RATE DEFLECTION POINT - CONCORDANCE AND RELIABILITY

(I) Lactate and ventilatory threshold comparison. Ventilatory threshold reliability.

The idea for VT assessment stems from the early belief that this measure can replace the invasive and more laborious LT determination. The non-invasive nature of VT and the encouraging results of the first studies that VT coincides with LT encompassed both measures under the same word "Anaerobic Threshold". However, several experimental manipulations dissociate the measures and some Sports Scientists claim that these are two different phenomena. The following section will review studies which support each of these arguments.

Davis et al., (1976) compared the lactate and ventilatory thresholds in nine male subjects. Gas exchange measurements and venous blood samples were taken every 30 seconds during incremental work on a cycle ergometer. The non-linear increase in $\dot{V}E$ and $\dot{V}CO_2$ and the rise in FEO_2 were found to occur within 30 seconds of one another. The average of these three measures expressed as a percentage of peak $\dot{V}O_2$ was used as the VT. An abrupt increase in venous lactate was used to detect LT. No significant differences were found between VT and LT,

which occurred at 59.8%$\dot{V}O_2$max and at 59.7%$\dot{V}O_2$max respectively. A correlation of r=0.95 was found between LT and VT.

In the same study, the VT was determined for three different modes of exercise including treadmill running, leg cycling, and arm cranking. VT was found at 46.5%, 63.8%, and 58.6% of peak $\dot{V}O_2$ respectively. Significant differences in peak $\dot{V}O_2$ were found between the three modes, yet no significant differences were found between the VT expressed as %$\dot{V}O_2$max for treadmill running and leg cycling. A significant difference in VT for arm cranking compared to the other two modes was found, yet this was postulated to have occurred due to the small amount of muscle mass involved and unfamiliarity with the exercise. The authors noted a limitation in using a subjective determination of the deflection points of $\dot{V}E$ and $\dot{V}CO_2$. They reported that these points were not always clear and investigators differed in the selection of some of this points. The VT measurements showed a test-retest correlation of r=0.77 for treadmill running and r=0.74 for leg cycling.

Davis et al., (1979) conducted another study to investigate VT alterations caused by endurance training in middle-aged men, and the validity of VT expressed as %$\dot{V}O_2$max. Nine sedentary middle-aged men underwent cycle endurance training 45 minutes per day for 9 weeks. Before and after the training period the subjects performed three cycle ergometer tests. VT was determined as the work rate or $\dot{V}O_2$ before systematic increase in $\dot{V}E/\dot{V}O_2$ without an increase in $\dot{V}E/\dot{V}CO_2$. Work rate was increased by 15 W/min to exhaustion in the first two tests; the third test consisted of constant load cycling at an O_2 uptake just below pre-training VT. After training, the VT increased significantly by 44%, expressed as absolute $\dot{V}O_2$, and by 15%, expressed relative to $\dot{V}O_2$max. The test-retest correlation coefficients for the VT expressed as %$\dot{V}O_2$max or absolute $\dot{V}O_2$ were 0.91 and 0.94 pre- and post-training respectively. It has been suggested that VT is profoundly influenced by endurance training and that it can be better expressed as absolute $\dot{V}O_2$.

Reinhard et al., (1979) proposed that an increase in the ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}O_2$) was a more sensitive measure of VT. A second criteria set along with the increase in $\dot{V}E/\dot{V}O_2$ was that there would be an increase in the ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$) at this point. This assured that the VT would occur during a period of isocapnic buffering, and would not be due to hyperventilation only. The VT was determined using this criteria, whereas LT was assessed by using venous blood lactate measurements. In eleven men and four women LT and VT were not different, and a correlation of r=0.94 was found between these measures. The authors concluded that VT coincides with LT and there is no necessity of invasive measurements.

Yoshida et al., (1981) compared VT measured by the open circuit Douglas bags method and LT measured by arterial blood lactate in 10 college males. An incremental cycle ergometer test was used with gas exchange measured each minute. Blood samples were taken from the radial or brachial artery each minute during exercise. The LT was determined as the point of departure of lactate concentration from resting values. The VT was determined from an abrupt increase in $\dot{V}E/\dot{V}O_2$ without a rise in $\dot{V}E/\dot{V}CO_2$. No significant differences were found between LT and VT. A significant correlation of r=0.87 was found between these measures. The authors concluded that the VT may be a good predictor of the LT and may be useful in evaluating submaximal exercise endurance.

In 1982, Caiozzo et al., tested 14 male and two female subjects on cycle ergometers to determine VT by various gas exchange measures and the LT. Gas exchange and venous blood lactate measurements were determined every 30 seconds throughout the incremental tests. Gas exchange indices measured to determine the VT included a non-linear increase in $\dot{V}E$, an abrupt increase in the respiratory exchange ratio (R), and an abrupt increase in $\dot{V}E/\dot{V}O_2$ without an increase in $\dot{V}E/\dot{V}CO_2$. The LT was determined as an increase in venous blood

lactate concentration above resting values. The mean VT using $\dot{V}E/\dot{V}O_2$ was 1.84 l/min and the mean LT was 1.85 l/min. The VT using $\dot{V}E/\dot{V}O_2$ was found to correlate highest with the LT (r=0.93). The test-retest correlation for the VT using $\dot{V}E/\dot{V}O_2$ was also the highest of the gas exchange indices (r=0.93). It was concluded that $\dot{V}E/\dot{V}O_2$ was the most sensitive and reliable measure of VT and was highly correlated with LT.

Aunola and Rusko, (1984) examined the reproducibility of first $\dot{V}E$ and $\dot{V}CO_2$ breaking points (VT_1) and the second $\dot{V}E$ and $\dot{V}CO_2$ breaking points (VT_2) in 33 men aged 20-50 years. They completed two maximal exercise tests on a bicycle ergometer 7 days apart. LT_1 was also determined as the workload or $\dot{V}O_2$ where lactate concentration increased distinctly from its resting level around 2 mmol/l, and LT_2 was determined as a starting point of accelerated lactate accumulation around 4 mmol/l. Two investigators first performed VT_1, VT_2, LT_1, and LT_2 determinations independently. After that they discussed the differences which appeared between VT_1-LT_1 and VT_2-LT_2 and agreed upon a compromised value for the final VT, LT_1 and VT, LT_2. Respiratory variables were measured with a computerized breath-by-breath method; samples of venous blood were drawn every 2 minutes and analyzed enzymatically for lactate.

The reproducibility of the VT, LT_1 (r=0.94) and of the VT, LT_2 (r=0.96) were equally good. It was suggested that either gas exchange or lactate indices can be used to define VT, LT_1 or VT, LT_2 during a 2 minute incremental exercise test. They both showed equal reproducibility: r=0.93 for the LT_1 and LT_2 and r=0.95 for the VT_1 and VT_2. The work rate and the measured physiological variables at the VT, LT_1 and VT, LT_2, except for the blood lactate concentration, were very reproducible. Age did not affect the thresholds' reproducibility. It was concluded that the poor blood lactate concentration reproducibility does not credit the use of blood lactate levels of 2 and 4 mmol/l as an indicator of any threshold phenomenon.

Sucec et al., (1989) investigated the validity of gas exchange indices as a measure of OMA on the treadmill. VT was estimated by using $\dot{V}E$, $\dot{V}CO_2$, $\dot{V}E/\dot{V}O_2$, and FEO_2 gas exchange variables. An abrupt and/or sustained increase in venous blood lactate (HLa) was considered as the criterion measure for LT estimation. Eighteen experienced runners performed a continuous incremental work test on a treadmill which commenced at a speed of either 180 m/min or 200 m/min, depending on the training history of each subject and was increased 10 m/min each 3 minute period. Two millilitres blood samples were drawn from an indwelling catheter in the midforearm, 15 seconds prior to each increase in work rate. The mean AT for gas exchange values and blood Lactate determinations were 2.79 l/min and 2.75 l/min $\dot{V}O_2$ respectively, with a validity coefficient (r) of 0.97. It was concluded that gas exchange indices are a valid measure of OMA on the treadmill.

Contrary to the preceding studies, many other studies, utilising a variety of methods, indicate that it is possible to dissociate LT from VT.

Yeh et al., (1983) studied six males and two females during two incremental cycle ergometer tests. Breath-by-breath gas analysis was used to determine the increase in $\dot{V}E/\dot{V}O_2$ and the increase in FEO_2 in both tests. Both arterial and venous blood lactate were measured every 30 seconds in the second test only. The arterial lactate curves seemed to rise smoothly with no abrupt increase, making the determination of a threshold difficult. Venous lactate began to rise two minutes after arterial lactate rose and lagged behind arterial lactate throughout the test. Reviewer variation was 0.51 l/min for $\dot{V}E/\dot{V}O_2$. A threshold phenomenon was not

detectable for the invasive measures and the reviewer variability was great in the non-invasive tests.

Five subjects each underwent an incremental bicycle ergometer test and four constant workload tests in a study by Simon et al., (1983). The incremental tests were used to determine the VT by an increase in $\dot{V}E/\dot{V}O_2$ and the LT by an increase in venous lactate concentration. In four of the subjects, VT was greater than LT. The constant workload tests were each 30 minutes in duration at intensities above and below VT and the respiratory compensation threshold (RCT). Above and below VT lactate concentrations and $\dot{V}CO_2$ maintained a steady state. Below and above RCT, lactate concentration and $\dot{V}CO_2$ increased throughout the test. The $\dot{V}E/\dot{V}O_2$ and FEO_2 peaked just before lactate peaked, just above the VT. Findings suggested that ventilatory measures do not necessarily indicate changes in blood lactate concentration during exercise.

Powers et al., (1984) found that LT and VT did not always occur simultaneously in 13 male subjects. Testing was done on a cycle ergometer with increments of 30 watts increased every three minutes until exhaustion. Measurements including $\dot{V}E$, $\dot{V}O_2$, $\dot{V}CO_2$, and venous blood lactate were determined for each workload. The LT was determined by a systematic increase in blood lactate concentration. The VT using $\dot{V}E$ (VT-$\dot{V}E$) was determined by a nonlinear increase in $\dot{V}E$. The VT using $\dot{V}E/\dot{V}O_2$ (VT-$\dot{V}E/\dot{V}O_2$) was determined by an abrupt increase in $\dot{V}E/\dot{V}O_2$ without an increase in $\dot{V}E/\dot{V}CO_2$. A paired t-test showed no significant differences between the LT (mean=1.68 l/min), VT-$\dot{V}E$ (mean=1.77 l/min), and VT-$\dot{V}E/\dot{V}O_2$ (mean=1.73 l/min). The correlation coefficients between LT and VT-$\dot{V}E$ and LT and VT-$\dot{V}E/\dot{V}O_2$ were r=0.67 and r=0.63 respectively. In five of the 13 subjects, LT did not occur at identical workloads as determined by VT-$\dot{V}E$ or VT-$\dot{V}E/\dot{V}O_2$. It was suggested that there were limitations using gas exchange measurements to determine the LT.

Neary et al., (1985) studied the relationship between the LT and VT in 10 healthy males during one-legged cycle exercise. Two pretests were done with one leg to determine LT and VT. Then, one leg was exercised at 75 to 85% of maximal heart rate for 60-75 minutes to reduce muscle glycogen. This was followed by a low carbohydrate diet and 30 hours later a one legged cycle ergometer test was conducted again. Both the pre-tests and post-tests consisted of 16 watt increments every three minutes until exhaustion. Muscle biopsies were taken to verify glycogen depletion. Blood samples were taken from the antecubital vein every work load and gas was analyzed every 30 seconds. The VT was determined by a non-linear increase in $\dot{V}E$ versus $\dot{V}O_2$. Lactate values were significantly lower for the prior exercise conditions at the LT and VT. The LT and VT occurred at the same $\dot{V}O_2$. The LT occurred at 62% $\dot{V}O_2$max for both pre- and post- tests. The VT occurred at 64% and 66% of peak $\dot{V}O_2$ for the pre- and post-tests, respectively. It was concluded that accumulation of lactate had no effect on VT supporting the contention that VT and LT are only coincidential, not cause and effect.

The present status of knowledge concerning lactate threshold and ventilatory threshold concordance does not favour the notion that blood lactate concentration is responsible for the abrupt increase in ventilation. Several factors such as training, substrate availability and exercise modality seem to dissociate LT and VT. The concordance of the measures reported from other studies may not be causal but rather coincidental. The VT phenomenon may have a physiological link with the lactate appearance in the blood, this alone can explain the concordance of the measures reported in the literature. However, before the physiological mechanisms behind these phenomena are fully understood LT cannot be used as the criterion measure for validating VT. VT can stand as a physiological measure and may be

included in the laboratory testing battery as it presents exceptionally high correlations with middle and long distance running performance. Unfortunately the problems of subjective determination which have not been solved as yet may limit the value of the measure.

(II) Lactate threshold, ventilatory threshold and heart rate deflection point comparative studies. Heart rate deflection point reliability.

This section will review studies dealing with the ongoing debate over the appearance or not of the Vd. Studies which support the phenomenon will be presented first.

Conconi's estimate of OMA was validated in 15 trained cyclists by Goodman et al., (1986). HR and metabolic data were analyzed while subjects increased speed 1 mph each minute on each subject's own cycle, mounted on a wind simulator. VT defined as the break from linearity in $\dot{V}E$ versus $\dot{V}O_2$, and Vd (the break from linearity in HR vs velocity) were observed in all subjects. VT (l/min), VT $\%\dot{V}O_2max$, and VTHR were significantly lower than Vd (l/min), Vd $\%\dot{V}O_2max$, and VdHR (2.55±0.53 vs. 2.84±0.66, 67.4±6.37 vs. 75.3±11.9, and 158±12.2 vs. 167±9.9, respectively $p<0.001$). VdHR was correlated with VTHR $r=0.71$ indicating a moderate degree of relationship despite a dissociation of all metabolic and HR data. It was concluded that Vd may represent a physiological or mathematical event

occurring systematically later than VT.

Gaisl and Wiesspeiner, (1988) examined the use of Vd as a non-invasive method to determine OMA in children. Seventy one 11-year-old male and female children were tested on a treadmill using a protocol consisting of 3 minutes work intervals and 2 km/h increments with an interruption of 10 seconds to take blood samples from the ear lobe. LT was fixed at a value of 4 mmol/l. After a break of 45 minutes an alternative method for determining OMA on a bicycle ergometer was used. Work increments of 10 watts/min were chosen. Vd was determined as the point where the HR-workload relationship lost linearity. Vd was highly related with LT (r=0.98). The authors postulated that it is possible to use the deflection from linearity of the HR in bicycle ergometer tests for training optimization in 11-year-old children if the proper degree of load is chosen. This simple method can substitute a more laborious invasive way of determining OMA.

Kispert et al., (1988) with 13 highly trained runners compared VT, LT, and Vd during treadmill running. The VT and LT were defined as the running speeds where the non-linear increase in ventilation and blood lactate occurred. The Vd was defined as the running speed where a decrease in the slope of the HR-running speed curve occurred. Mean HR values for the VT, LT, and Vd were 166±8, 165±7, and 166±7 bpm respectively. The VT and Vd appeared graphically as well-defined points, and had a high correlation (r=0.91). A specific LT was more difficult to determine, and the values did not correlate as well with either VT (r=0.68), or Vd (r=0.68). It was suggested that VT and Vd may be more precise measurements of submaximal exercise performance.

The use of Vd as an acceptable method for the noninvasive determination of LT was examined by Aro et al., (1988). The relationship between running velocity (RV) and HR was determined in 9 highly trained runners. Each subject performed a modified Conconi treadmill running protocol consisting of 400m stages. Initial RV

was 3.2 m/second with each successive stage duration decreasing by 5 seconds until exhaustion. In all subjects, a deflection from linearity (Vd) occurred at a submaximal RV beyond which the increase in RV exceeded the corresponding increase in HR. Oxygen uptake ($\dot{V}O_2$), RV, and HR at Vd were determined for each subject. To determine whether Vd occurs at the same exercise intensity as LT, the lactate steady state (SSLa) was determined for all subjects. SSLa was the highest exercise intensity that could be sustained for 30 minutes while maintaining a constant blood lactate concentration and was considered the criterion measure for LT determination. $\dot{V}O_2$, RV, and HR at SSLa were determined for each subject. Mean, $\dot{V}O_2$ (50.4±5.3 ml/kg/min) at Vd was not significantly different from SSLa (48.6±2.6), RV at Vd (4.2±0.2 m/s) was not significantly different from SSLa (4.1±0.3), and HR at Vd (170±12 bpm) was not significantly different from SSLa (169±8). It was concluded that Vd provides an acceptable noninvasive method for the determination of LT in highly trained runners.

A study to compare two noninvasive methods in the determination of OMA, using a single test in children, was designed by Baraldi et al., (1989). 70 children 7-14 years old performed a maximal exercise test on the treadmill for the determination of OMA by two methods: (1) VT was identified by the increase in the $\dot{V}E/\dot{V}O_2$ without a concomitant increase in the $\dot{V}E/\dot{V}CO_2$ and (2) Vd was identified as the point beyond which the increase in work intensity exceeded the increase in HR and the linearity of the work rate/HR relationship was lost. The protocol consisted of 2% per minute stepwise increases in treadmill inclination throughout the test until exhaustion. VT and Vd were correlated (r=0.80). However, despite the fact that the authors were not presenting data to support VT and Vd concordance, based on this correlation they confirm the validity of the simple Conconi method for the determination of OMA.

Two different noninvasive procedures (i.e. VT and Vd) have been used to

determine OMA in 15 long distance runners in a study conducted by Brettoni et al., (1989). VT was obtained on a treadmill using 0.5 km/h increments per minute and was determined as the work load before $\dot{V}E$ breaking point. Vd was assessed on a 400m track where the original Conconi protocol was employed. HR (166±9 vs 165±11 bpm) and running speed (15.16±1 vs 15.21±0.8 km/h) at VT and Vd were not different for the laboratory and field tests respectively. The authors suggested that when the laboratory test mimics as close as possible the field exercise usually performed by the athletes, it is a good predictor of the OMA in the field.

From the studies which have already been presented it seems that Vd is a reproducible measure and correlates with either the LT or VT but does not coincide. However, a number of studies which performed in the laboratory with a modified protocol failed to identified Vd in all of their subjects. Detailed description of the subjects and the experimental design which they were using will follow.

Ribeiro et al., (1985) conducted a study to investigate Vd and LT relationship and the Vd validity. They hypothesized that Vd was a better predictor of LT. To test this hypothesis, 11 subjects with different levels of conditioning were tested on a cycle ergometer with 30 watts/min increment to compare Vd and LT. Heart rate from ECG tracings and blood samples for lactate were taken every minute. Blood lactate values were plotted against power output and three straight lines were fitted so that two breaking points were identified. The point beyond which blood lactate began to increase systematically above resting value was defined as LT_1. The point immediately before the rapid rise in blood lactate was defined as LT_2. Vd was identified as the point at which heart rate started to deviate from linearity. When the results were expressed in watts the following significant correlation coefficients

were obtained: LT_1 and LT_2=0.92, LT_1 and Vd= 0.89, LT_2 and Vd=0.92. The LT_1 was significantly lower than Vd (166.4±52.6W and 234±69.5W). There were no significant differences between the LT_2 and Vd (240±67W and 234±69W). Based on these findings the authors concluded that Vd may coincide with the LT_2 and not the LT_1.

In the same study another group consisting of 16 physically active subjects performed two tests 1-2 weeks apart to evaluate the reproducibility of the Vd. After 3 minutes of warm up at 25W, increments of 25W were added every minute to volitional exhaustion. VT was defined as the work load before consistent decrease in the fraction of expired CO_2 ($FECO_2$). VT was noted in all tests, eight subjects failed to demonstrate Vd in at least one of the evaluations. The fact that the Vd could not be demonstrated with a documented VT in several subjects led the researchers to conclude that no causal relationship exists between the two, and that Vd may not be a generalizable physiological variable.

Nine trained athletes and four sedentary individuals were tested by Kuipers et al., (1988) to compare Vd and the 4 mmol/l LT when cycling on a cycle ergometer. The work load was increased by 10 watts every 45 seconds. The work load at which plasma lactate concentration equalled 4 mmol/l was assessed under steady state conditions on separate occasions. In addition, in three subjects the Vd and the 4 mmol/l lactate level under steady state conditions were assessed on a treadmill using 0.5 km/h increments and 30 seconds duration per stage. LT on the treadmill was determined employing 5 minutes stages and 1 km/stage increment. Blood sampling was performed during the last 30 seconds of each workload. On the cycle ergometer only 6 subjects demonstrated Vd. The workload at which HR departed from linearity in the progressive protocol did not coincide with the steady state 4 mmol/l workload, (2.86±32 W and 2.50±51 W) respectively. On the treadmill no Vd was observed. It was concluded that in cyclists Vd does not always occur, and

when it does, it does not coincide with LT determined under steady state conditions.

Francis et al., (1989) with nine untrained subjects aged 22-36 years investigated if there is a relationship between VT and Vd for this category of subjects, when cycling under well controlled laboratory conditions. The 100 watts initial power output was increased by 5 rpm every 30 seconds (11.1 watts) until the point of exhaustion. HR and expiration gases were collected at 30 seconds intervals. The results indicated that the heart rate and ventilatory response to increasing velocity as previously reported under field conditions does not exist under laboratory conditions. While there was a definite and statistically significant inflection in the ventilatory response to increasing velocity, heart rate remained linear. Researchers suggested that caution should be used when determined OMA from the single measure of HR response.

The study by Tokmakidis and Leger, (1987) with 22 elite male runners, is the only study which has been conducted in the field to investigate Vd validity as compared to LT under the same conditions. The runners performed two maximal multistage running field tests on a 183.9-m indoor track with inclined turns. The initial speed of 9 km/h was increased by 0.5 km/h every lap for the Vd test and by 1 km/h every 4 minutes (interspersed with a 45 seconds pause for finger tip blood sampling) for the LT test. Vd and LT criteria were the workload at which HR departed from linearity and the sudden sustained LA increase respectively. Results indicated higher Vd speed than LT speed (18.5±1.1 and 16.2±0.8 km/h) and yielded a low correlation (r=0.50). Vd speed was similar to the maximal speed achieved during the LT test (18.7±0.9 km/h), which usually corresponds to peak $\dot{V}O_2$ in such a protocol. This study failed to support the validity of Conconi's heart rate threshold. The authors felt that the modification in the protocol was responsible for this failure.

From the preceding section it is apparent that there is disagreement between the studies about the heart rate deflection point near maximal effort. The use of different cohorts, different exercise modes and protocols, and the subjective determination of the measure make the comparisons difficult and may explain partly the discrepancies observed between the studies. However, there is a tendency in the studies with highly trained runners to identify Vd in most cases, though this did not occur at the same exercise intensity as either VT or LT. Finally, until further research establishes the physiological rational behind this phenomenon, it is premature to suggest that this measure may be representative of a highly trained state.

2.06: RELATIONSHIPS OF THE LACTATE THRESHOLD, VENTILATORY THRESHOLD AND HEART RATE DEFLECTION POINT TO PERFORMANCE

Rhodes and McKenzie, (1984) examined the relationship between performance time predicted from the VT and actual performance time in a marathon. Eighteen male subjects ran on the treadmill to volitional fatigue. The VT was determined by a nonlinear increase in $\dot{V}CO_2$. A correlation coefficient of r=0.96 was found between the predicted and the actual marathon times. The investigators suggested that the VT may be more visible in trained than in untrained individuals, and the velocity at VT may be critical in determining efficient running speed during marathons.

Tanaka and Matsura, (1984) tested 12 trained male runners to determine the LT and the onset of blood lactate accumulation (OBLA) defined as the workload at an absolute lactate concentration of 4.4 mmol/l. The LT was determined as a systematic increase in venous blood lactate. Incremental, non-continuous treadmill tests were employed to make these measurements. The LT and OBLA were compared with the runners' velocities during a marathon race. Differences were found to exist between the velocity at OBLA (5.3 m/second) and the marathon velocity (4.57 m/second), but not between the velocity at the LT and the marathon velocity. The LT correlated with the velocity during a marathon (r=0.78). The investigators concluded that LT is more closely associated with marathon running performance and that the degree of the association is raised when peak $\dot{V}O_2$ is combined as additional information.

Maffulli et al., (1987) conducted a study to investigate the relation between 4

mmol LT (HLa_4) and Vd under well-controlled laboratory conditions using different exercise testing protocols (1, 2, and 4 minutes of exercise respectively). The data were also compared with the speed maintained during a marathon race. Eleven well trained subjects were tested on three different days, at least one day apart, on a treadmill. Treadmill speed was increased in steps of between 0.083-0.16 m/second. The speed was kept constant for a period of 1, 2, or 4 minutes according to the experimental procedure chosen. In every subject the 1 minute test preceded the 2 minute, and this one preceded the 4 minute. Oxygen uptake was determined by the Douglas bag method. On the basis of these tests six different velocities were chosen-two below, one on Vd, and three above it. Finger tip blood was collected during the last 30 seconds of each 5 minutes constant speed trials. LT was defined as the point at which a non-linear increase of blood lactate occurred. Furthermore, HLa_4 was determined. Vd was detectable for all tests and corresponded with 79.5±3.3 $\%\dot{V}O_2max$. HR and treadmill velocity at Vd were not different for the 1, 2, and 4 minutes work increment duration tests. Test-retest correlation coefficient using twice the 2 minutes experimental procedure was r=0.97. Vd correlated well with LT (r=0.95), while Vd and HLa_4 showed a correlation of 0.67. Marathon velocity was closer to Vd velocity than to HLa_4 velocity. The authors concluded that Vd is useful for the rapid non-invasive determination of the LT and can be used as a predictive tool for the marathon race pace on the assumption that the test conditions are well controlled.

Seling, (1988) investigated the relationship between treadmill speed measured at RCT (identified in this study as the deviation from linearity of $\dot{V}E$ or $\dot{V}CO_2$ when plotted against $\dot{V}O_2$) and average running speed achieved in a competitive marathon 14-15 days later. Forty-seven athletes underwent an incremental treadmill test with 0.25 km/h per 30 seconds speed increments in the critical range of speeds below and above RCT. For the full group (44 finishers) marathon speed was

correlated with velocity at RCT (r=0.87). For the 30 experienced athletes (at least 3 previous marathons) marathon speed was better correlated with RCT velocity (r=0.93). It was concluded that marathon performance can be predicted from a non-invasive gas exchange treadmill test, particularly if factors such as competitive experience and weather conditions are also considered.

Kiousis et al., (1991) conducted a study to investigate the relationship between VT treadmill velocity and the actual pace in the classical marathon course. 20 highly trained marathoners underwent an incremental treadmill test 15-7 days before the marathon race. Treadmill velocity was increased 0.5 km/h every minute until volitional exhaustion. VT was determined as the workload before $\dot{V}E$ breaking point and/or before $\dot{V}E/\dot{V}O_2$ systematic increase. The work load corresponded with the highest recorded $\dot{V}O_2$ was defined as $v\dot{V}O_2max$. VT and $v\dot{V}O_2max$ were highly correlated with the marathon pace for the 16 finishers, r=0.96 and r=0.92 respectively. It was concluded that physiological assessment on the treadmill can predict with fair accuracy optimal marathon pace in the classical marathon course.

From the studies presented so far it is apparent that VT, LT and Vd are highly related with the performance in the marathon race. Since these measures explain greater variance than either peak $\dot{V}O_2$ or running economy over this distance they may used to determine optimal marathon pace or for the prediction of the marathon time. Studies examining the correlation of these measures to shorter distances (i.e. 800m-20000m) will follow.

Farrell et al., (1979) found very high correlations in 18 highly trained distance runners between distance running performance and the onset of plasma lactate accumulation (OPLA). Each subject underwent a series of steady state treadmill tests each lasting 10 minutes. The tests were conducted at velocities corresponding

to the race paces from the marathon to 9.7 km. Blood lactate was determined from venous samples collected at the end of each steady state run and plotted on a graph. The increase in blood lactate was determined by visual inspection and linear regression for the portion of the graph above and below the visual detected point. The velocity at the OPLA correlated best with marathon performance at r=0.98. Correlations between OPLA and the other distances were r=0.97 for 19.3 km, r=0.97 for 15 km, and r=0.96 for 9.7 km.

Kumagai et al., (1982) studied the relationship between the LT, VT and the relationship between these measures and various running distances. The subjects were 17 male high school distance runners, and each underwent an incremental treadmill test to exhaustion. The VT was determined by an increase in $\dot{V}E/\dot{V}O_2$ and the LT by the first nonlinear increase in venous blood lactate concentration. The correlation coefficient found between VT and LT was r=0.91. The LT was also found to correlate highly with 5 km performance (r=0.95), 10 km performance (r=0.84), and 10 mile performance (r=0.84).

Powers et al., (1983) studied the relationship between the VT and 10 km running performance in nine male distance runners. VT was determined by a non-linear increase in $\dot{V}E$ and $\dot{V}CO_2$. The correlation coefficient between the VT and 10 km performance was r=0.94. Correlations between performance versus running economy and peak $\dot{V}O_2$ were also found at r=0.51 and r=0.38, respectively.

Tanaka et al., (1984) measured the LT by venous measurements in 21 trained male runners before and after nine months of training. The $\dot{V}O_2$ at LT increased significantly from 48.5 ml/kg/min to 50.2 ml/kg/min. The LT was also shown to correlate highly with 5 km performance (r=-0.76) before training and (r=-0.84) after training. The LT correlated with 10 km performance (r=-0.81) before and (r=-0.88) after the nine months of training. It was concluded that the LT was a critical determinant of distance running success.

Iwaoka et al., (1988) designed a study to examine the possible gender differences in LT, respiratory compensation threshold (RCT), and their relations to distance running performance. Ten male and eight female well trained distance runners performed an incremental running test on the treadmill. Treadmill speed was increased every three minutes by 20 m/min until exhaustion. LT was determined from an inflection point in blood lactate and RCT was determined from a marked increase in $\dot{V}E/\dot{V}CO_2$. LT was significantly higher in males than in females (49.2 vs 45.5 ml/kg LBM/min), while RCT was not significantly different between groups (51.5 vs 52.3 ml/kg LBM/min). No significant differences were observed either in LT or in RCT expressed as $\%\dot{V}O_2max$. Running velocity at RCT was strongly related to the run times of 800 and 1500m in females and 5000 and 10000m in males (r=-0.76~0.95). The authors concluded that there are no remarkable gender differences in LT and RCT when compared in relative terms and that RCT is a sensitive parameter for evaluating an endurance performance despite its controversial status.

The contribution of VT, peak $\dot{V}O_2$, and running economy (RE), to actual running time (ART) in a 5 km race was examined by Cunningham, (1991). 24 trained female runners underwent an incremental treadmill test where the treadmill slope increased by 2.5% every minute until runners indicated voluntary exhaustion. VT was determined by using the breakpoint from linearity of $\dot{V}E/\dot{V}O_2$ while $\dot{V}E/\dot{V}CO_2$ remained unchanged. The velocity at VT (vVT) and velocity at peak $\dot{V}O_2$ ($v\dot{V}O_2max$) were correlated with ART, r=0.78 and 0.77 respectively. It was concluded that the derived variables vVT and $v\dot{V}O_2max$ appear to explain significant variation in distance running performance among adolescent female cross country runners.

The preceding section presented a universal agreement regarding the relationship of VT, LT and Vd with running performance. High correlations have been reported here and elsewhere for distances ranging from 800m to ultra marathon running. These measures explain a significant variation in middle and long distance running performance. It is to the runner's advantage to meet either VT, LT or Vd at the highest possible speed and even strive to improve these through training. The application of these measures in regulating the training and race pace may prove very beneficial for the coach and the regular runner.

2.07: PERCEIVED EXERTION RATINGS RELATIVE TO THE ONSET OF METABOLIC ACIDOSIS

Simon et al., (1983) examined perceived exertion relative to LT in six highly trained and six sedentary untrained men. Borg scale rating of perceived exertion, lactate, and gas exchange measurements were taken throughout an incremental cycle ergometer test. Peak $\dot{V}O_2$ and LT for the trained group averaged 63.8 ml/kg/min and 60.6%$\dot{V}O_2$max, while those of the untrained group averaged 35.5 ml/kg/min and 45.1% $\dot{V}O_2$max, respectively. The trained subjects' perceived exertion at the LT (13.5) was significantly greater than that of the untrained subjects' (10.5). However, the mean perceived exertion corresponding to a lactate concentration of 4 mmol/l in the trained (16.2) and untrained (15.4) was similar which occurred at 85% and 80.1%$\dot{V}O_2$max, respectively. It was concluded that trained subjects rate the effort of work at the LT greater than untrained subjects but both groups rate it similarly at a lactate concentration of 4 mmol/l.

DeMello et al., (1987) conducted a study to investigate rating of perceived exertion at the LT (RPE_{LT}) in 10 male and 10 female trained distance runners, and 10 male and 10 female untrained subjects. A continuous, progressive, grade increment (2.5% every 2 minutes) treadmill protocol was used to measure peak $\dot{V}O_2$. LT (abrupt increase in blood lactate above base line level) and perceptual responses to various intensities were measured within 2 weeks using a discontinuous, progressive, treadmill protocol with 5%$\dot{V}O_2$max increases every 3 minutes separated by 3 minutes rest. The means for RPE_{LT} for the four groups (13.6±2.1, 13.5±1.6, 13.5±1.5, and 12.9±1.3, respectively) were not significantly different even though the ratings were given at markedly different levels of $\dot{V}E$, $\dot{V}O_2$ l/min, HR, and %$\dot{V}O_2$max. It was concluded that LT is an important physiological anchor point for perception of effort but is not affected by the state of training or gender. Trained and untrained men and women perceive the exercise intensity at LT as "somewhat hard" (RPE=13-14).

The effects of training on the rating of perceived exertion at the VT with 17 college students were studied by Hill et al., (1987). Subjects completed 18 interval sessions (5 x 5 minutes cycling at 90-100% $\dot{V}O_2$max) and 8 continuous training sessions (40 minutes running or cycling) in 6 weeks. Pre- and post-training, cardiorespiratory, metabolic, and perceptual variables were measured at the VT during graded cycle ergometer test. VT was determined as the point before $\dot{V}E/\dot{V}O_2$ systematic increase. Both before and after training, exercise at the VT was perceived as "somewhat hard" to "hard" (RPE=13-15). The relationship between peak $\dot{V}O_2$ and RPE was altered by training, with trained subjects having a lower RPE at any given %$\dot{V}O_2$max. It was concluded that RPE at the VT is not affected by training, despite the fact that after training the VT occurs at a higher work rate and is associated with higher absolute and relative metabolic and cardiorespiratory demands.

2.08: HEART RATE INDICES AND THE ONSET OF METABOLIC ACIDOSIS

Paton et al., (1979) examined five runners and 6 non-runners and found that the runners' LT occurred at higher absolute and relative levels of the peak $\dot{V}O_2$. However, the heart rate at LT for both groups was not significantly different (runners=181 bpm, non-runners=185 bpm). It was concluded that regardless of an individual's fitness level absolute heart rate measures provide a good indication of work level required for the LT.

Dressendorfer et al., (1981) examining 110 non-athletic men, 30 to 60 years of age, observed that the heart rates at LT were significantly higher than 85% of actual maximum rate (152 bpm) or 85% of the age predicted (220 minus age) maximum HR (151 bpm). However, heart rates at LT were not significantly different from 80% of maximum HR reserves (154 bpm) as calculated by the Karvonen equation. In 78 of the 110 subjects (71%), heart rate at LT was higher than 85% HRmax. These observations suggested that LT in healthy men frequently occurs above the upper limit of target heart rates commonly recommended for exercise to develop and maintain aerobic fitness.

Parkhouse et al., (1982) examined thirty-three men, 17-28 years of age who were divided into 3 groups; untrained, trained, and highly trained. They observed that the HR at LT for the three groups were almost identical (163.9, 164.4, and 167 bpm), as well as the %HRmax at LT (82, 84, and 86). In addition, 67% of all subjects had a heart rate at LT above 80% of maximum HR. The authors concluded that percentage of maximum HR may be used for exercise prescription, however, training at 80% of HRmax may be too low a stimulus for optimal improvement.

Dwyer and Bybee, (1983) investigated the HR response and percent HRmax

of VT in 20 young females. The subjects' VT, peak $\dot{V}O_2$, and HRmax were determined during an incremental cycle ergometer exercise test to fatigue, while the HR at VT was determined by regressing HR on $\dot{V}O_2$ using individual regression equations. The mean value for VT was 70.1%$\dot{V}O_2$max with corresponding HR of 158.4 bpm (86.3% HRmax). They observed that all subjects were below VT at 70% HRmax. However, a zone of non-uniform work stress with respect to VT was observed between 75-95%HRmax (58-75%$\dot{V}O_2$max). Within this zone a highly variable number of subjects exercised above their VT at any specific percent at HRmax. Furthermore, the low correlation of r=0.60 that was found between %HRmax of VT and %$\dot{V}O_2$max of VT did not allow, according to the authors, the translation of VT to a %HRmax or absolute HR figure for training prescription. The investigators concluded that standard values for %HRmax at VT, grouped by age or sex, should not be applied to individuals due to the wide range among homogeneous subjects in relative HR at the VT.

2.09: LACTATE RESPONSES TO CONSTANT WORKLOAD EXERCISE

Nagle et al., (1970) conducted four submaximal treadmill tests on five males. Peak $\dot{V}O_2$ was determined from treadmill tests lasting six minutes at speeds varying from 268 to 322 m/min. The submaximal tests were performed at approximately 70, 80, and 90%$\dot{V}O_2$max. Work at 90% lasted 30 minutes, at 80% for 40 minutes, and at 70% for 60 minutes. Expired gas was sampled and analyzed at minutes 2, 4, and 6 during the peak $\dot{V}O_2$ tests. A three minute post exercise blood sample was taken from the antecubital vein and analyzed for lactate. During the submaximal tests gas was sampled and analyzed for minutes 1, 2, 3, 5, 10, and 15 and in 5-10 minute intervals thereafter. Blood samples were taken during gas collection periods. The highest workload (86% $\dot{V}O_2$max) showed a continuous increase in lactate throughout the tests. The intermediate workload (77%$\dot{V}O_2$max) resulted in an increase in lactate which plateaued after the 10^{th} minute. At the least intense speed (71%$\dot{V}O_2$max), there was a small increase in lactate which levelled off. During the test at the highest workload, $\dot{V}O_2$ increased 5% at the end of the run. The results of the study indicate that lactate is elevated over the duration of 30 to 60 minutes runs in proportion to aerobic demands in excess of 65 to 75% of maximum. These observations led the investigators to conclude that lactate is continuously produced in work requiring 65 to 90%$\dot{V}O_2$max even when a reasonably steady state of O_2 consumption is attained.

Kinderman et al., (1979) designed a study to investigate lactate and HR kinetics when: 1) the HR corresponding to the 4 mmol LT, determined by an incremental treadmill test, was kept for a 30 minute running period 2) the treadmill speed corresponding to the 4 mmol LT was kept constant throughout the 30 minute period. Seven cross country skiers worked until exhaustion on the treadmill.

A constant grade of 5% was maintained throughout the exercise, and every 3 minutes the speed was increased until exhaustion. At the end of each period, the exercise was interrupted for 20 seconds for ear lobe blood sampling. The beginning of the step increase of lactate levels was defined as the 4 mmol LT according to Mader et al., (1976). On different days the test persons were subjected to two additional exercises, each with a duration of 30 minutes. During exercise with a constant HR, treadmill speed was reduced continuously to keep the HR level. In the beginning the treadmill speed was higher than that determined for 4 mmol lactate, but after 30 minutes it was lower than in the beginning. $\dot{V}O_2$ behaved correspondingly. Blood lactate concentration displayed an approximately twofold rise when compared to 4 mmol lactate up to 10 minutes and after was continuously reduced as a result of the reduction of the treadmill speed. During the exercise performed with constant speed, lactate concentration initially rose to the values of nearly 4 mmol/l and leveled off during the rest of the exercise. HR displayed a slight but permanent increase and was on the average above 170 bpm. The authors suggested a new classification of concepts for the OMA that will make possible the determination of optimal workload intensities during endurance training.

1) Aerobic threshold: approximately 2 mmol/l lactate-first significant elevation of blood lactate level, non-linear increase of $\dot{V}E$, $\dot{V}CO_2$.
2) Aerobic-anaerobic transition: approximately 2 to 4 mmol/l lactate.
3) Anaerobic threshold: approximately 4 mmol/l lactate-steep part of exponential increase in lactate concentration.

Scheen et al., (1981) evaluated VT and LT at constant workloads from 36-80% of peak $\dot{V}O_2$ and lasting 20 minutes in duration. Male subjects walked on the treadmill with venous blood lactate, $\dot{V}O_2$, $\dot{V}E$, and HR determined every 30 seconds during the tests. A total of one hundred tests were completed using 66 subjects.

The $\dot{V}O_2$ reached a delayed steady state at workloads of 63% $\dot{V}O_2$max and above. Below 48% $\dot{V}O_2$max, $\dot{V}E$ reached a steady state while above this, $\dot{V}E$ increased throughout the test. The VT occurred at 57%$\dot{V}O_2$max. Blood lactate concentrations showed increases at the lowest workload (43%$\dot{V}O_2$max) yet after increasing initially, blood lactate plateaued and then dropped. At 63%$\dot{V}O_2$max lactate concentration increased continuously throughout the test. At moderate intensities (48-57%), lactate increased progressively to a steady state level between minute 10 and 20. The hyperventilation threshold was found at 57%$\dot{V}O_2$max. The authors suggested that the LT, the steady state LT, and the hyperventilation threshold did not coincide.

Stegmann and Kinderman, (1982) compared prolonged exercise tests relative to IAT and OBLA. IAT was defined as the workload corresponding to the steady state between lactate diffusion into the blood and elimination, according to the model derived by Stegmann et al., (1981). Incremental cycle ergometer tests were performed by nine male and 10 female rowers, with 50 watts work increments every two minutes until volitional exhaustion. $\dot{V}O_2$ was measured continuously and arterialized blood from a hyperemic ear lobe was taken at the end of each workload. OBLA was determined as the workload at 4.0 mmol/l lactate. Prolonged exercise tests, 50 minutes in duration, were conducted at workloads corresponding to the IAT and OBLA. Blood was sampled from the ear every five minutes for the determination of lactate. On the basis of the results three groups were formed. Group I, 15 rowers, showed a lactate concentration lower at IAT than OBLA. None of the rowers could work for 50 minutes at OBLA. Lactate at IAT maintained a steady state level and continued to rise when working at OBLA. Group II, three rowers, had identical IAT and OBLA values, and at both of these workloads lactate maintained a steady state value for 50 minutes. Group III consisted of one rower whose IAT lactate value was higher than the OBLA. Both work loads at IAT and

OBLA maintained a steady state in lactate. The authors concluded that the IAT corresponded to the maximal lactate steady state.

A study to compare the $\dot{V}O_2$ and running velocity at which LT, VT, and the maximal lactate steady state (MSSLA), and the maximal $\dot{V}O_2$ steady state ($MSS\dot{V}O_2$) occurred in 11 trained male runners was conducted by Haverty et al., (1988). Each underwent an incremental treadmill test to exhaustion. The LT was defined by a systematic continuous increase in arterialized venous blood lactate; the VT was determined by an abrupt rise in $\dot{V}E/\dot{V}O_2$ without an increase in $\dot{V}E/\dot{V}CO_2$. Each subject also completed a series of steady state treadmill runs of 20 minutes duration. The MSSLA was determined as the highest velocity and $\dot{V}O_2$ at which lactate concentration increased by less than 0.2 mmol/l from minute 10 to minute 20. The $MSS\dot{V}O_2$ was determined as the highest velocity or $\dot{V}O_2$ at which a steady state in $\dot{V}O_2$ was not delayed for more than 3 minutes (with a steady state defined as $\dot{V}O_2$ within 0.2 l/min of the average $\dot{V}O_2$ over the last 10 minutes of each test). Each subject also completed a 5 km time trial to assess performance. No significant differences were found among the four variables expressed either as $\dot{V}O_2$ or velocity. Significant correlations were found between MSSLA and $MSS\dot{V}O_2$ (r=0.74) expressed as $\dot{V}O_2$, and between MSSLA and $MSS\dot{V}O_2$ (r=0.90), $MSS\dot{V}O_2$ and VT (r=0.70) and MSSLA and VT (r=0.67) expressed as treadmill velocity. It was concluded that (a) MSSLA and $MSS\dot{V}O_2$ are closely related, and (b) MSSLA is a good predictor of performance and may be an important objective measure of cardiorespiratory endurance capacity.

2.10: VENTILATORY RESPONSES TO CONSTANT WORK LOAD EXERCISE

Hagberg, Mullin, and Nagle, (1978) studied 18 male subjects during constant workload cycle ergometry. Peak $\dot{V}O_2$ was determined by an incremental bicycle ergometer test. Subjects cycled at 65% and 85% $\dot{V}O_2$max for 20 minutes each. Blood was sampled from the antecubital vein after minute 1, 3, 5, 8, 11, 14, 17, and 20 of exercise. $\dot{V}O_2$ was monitored continuously, breath-by-breath. A significant rise in $\dot{V}O_2$ from minutes 5-20 occurred in 76% of the tests at 65%$\dot{V}O_2$max, and 85% of the tests at 80%$\dot{V}O_2$max. $\dot{V}E$ increased by 15% and 20% from 5-20 minutes at 65% and 80%$\dot{V}O_2$max. Breathing frequency increased 14% and 30% from 5-20 minutes at 65% and 80% $\dot{V}O_2$max workloads. The average change in R from the 5^{th} to the 20^{th} minute of exercise was 0 and -0.02 at the 65% and 80%$\dot{V}O_2$max workloads. At 65%$\dot{V}O_2$max lactate concentrations stayed constant after minute 8, while at 85%$\dot{V}O_2$max, a continuous increase in lactate occurred throughout the test. The authors concluded that no steady state in $\dot{V}O_2$ was observed at the workloads tested, and a slow rise in $\dot{V}O_2$ may be due to a temperature effect.

Ribeiro et al., (1986) conducted a study to investigate the metabolic and ventilatory responses to steady state submaximal exercise on the cycle ergometer. Eight healthy subjects performed an incremental cycle ergometer test with increments of 30 watts every minute to determine peak $\dot{V}O_2$, LT_1, and LT_2 (according to the criteria used by Ribeiro et al., 1985). Blood was sampled during the last 15 seconds of each workload. A second test was carried out 3-7 days apart to identify the power output corresponding to the steady state $\dot{V}O_2$ at the following conditions: 1) the LT_1; 2) the LT_2; 3) the average of $\dot{V}O_2$ at LT_1 and LT_2 (referred as $LT_{1\text{-}2}$); 4) the average of $\dot{V}O_2$ at LT_2 and peak $\dot{V}O_2$ (referred as LT_2max).

Another 4 exercise tests lasting 40 minutes were performed on different days in a random order and were designed so that, after a 10 minutes adaptation period, a steady state $\dot{V}O_2$ corresponding to that at LT_1, LT_{1-2}, LT_2, and LT_2max was maintained. $\dot{V}E/\dot{V}O_2$ values were significantly higher for the LT_2 when compared to the LT_1. Blood lactate concentrations, $\dot{V}E/\dot{V}O_2$, and $\dot{V}E/\dot{V}CO_2$ demonstrated steady state values during the last 20 minutes of exercise at the LT_1, LT_{1-2}, and LT_2 intensities, but increased progressively until fatigue in the LT_2max trial (mean time=16 minutes). Serum glycerol levels were significantly higher at 40 minutes of exercise on the LT_{1-2} and LT_2 when compared to LT_1, while the respiratory exchange ratios (R) were not significantly different from each other. The R increased significantly from 5-10 minutes and thereafter tended to decrease from the former exercise intensities. HR increased and reached steady levels at 20 minutes of exercise at LT_1, LT_{1-2}, and LT_2. For the LT_2max HR increased progressively until exhaustion. HR values were significantly higher at the LT_{1-2} and LT_2 when compared to the LT_1. The authors suggested that metabolic and ventilatory steady state can be maintained during prolonged exercise at intensities up to and including the LT_2, and fat continues to be a major fuel source when exercise intensities are increased from the LT_1 to the LT_2 in steady state conditions. The blood lactate response to exercise suggests that, for the organism as a whole, anaerobic glycolysis plays a minor role in the energy release system at exercise intensities up to and including the LT_2 during steady state conditions.

2.11: SUMMARY

The recent studies on exercise concerning the onset of metabolic acidosis have improved our understanding of the physiological mechanisms underlying exercise. The term "onset of metabolic acidosis" is used in preference to "anaerobic threshold" which no longer adequately describes the physiological phenomena occurring at a certain exercise intensity.

Studies on exercise and lactate production found that lactate in the blood increases with increased exercise intensity. This was at first postulated to be due to an oxygen deficit in the muscles and an increased dependence on anaerobic glycolysis for ATP resynthesis. The lactate threshold was later defined as a systematic increase in blood lactate during incremental exercise. In 1973, Wasserman et al. created the theory behind the non-invasive detection of LT by gas exchange measurement. Conconi et al., (1982) based on previous observations of Wyndham et al., (1959) and Davis, (1968) of a heart rate deflection near maximal effort during incremental exercise, postulated that this corresponded with OMA. The proponents of Vd have demonstrated that this is a simple, reproducible and valid index of exercise performance but may not be detectable in everyone.

Since then, work has been done on each threshold phenomenon (i.e. VT, LT, Vd) to determine their relationships and characteristics. These concepts and their use as valid measures of the exercise intensity corresponding with OMA are being actively debated. LT, VT, and Vd have been found to coincide by several investigators while others claim these measures are separate phenomena. Several problems have been associated with the detection of these points including inconsistent methods for blood sampling, inconsistent gas exchange measures, subjective determinations, use of different testing protocols, and the use of different population cohorts.

Despite, however, the controversy surrounding these measures several attractive features have also been found. The LT was found to be highly related to muscle respiratory capacity. There are studies which have found very high correlations between performance and LT, VT, and Vd.

Several factors have been shown to influence these points. Endurance training, substrate availability, pedalling speed, inspired oxygen concentration, glycogen depletion, age, and different exercise testing protocols are among them. These studies indicate the care required in obtaining reproducible values for any of these thresholds, and also lead to questions concerning the accuracy with which a VT or Vd reflect the LT.

The perception of exertion at the VT and LT exercise intensities, during incremental treadmill testing, has been rated as "somewhat hard" (13-15 on the Borg scale). There are no studies concerning Vd.

There is not universal agreement regarding the $\%\dot{V}O_2max$ and %HRmax at VT and Vd either for trained or untrained individuals. This emphasizes the need for individual assessment of these measures if they are to be used to set the optimal race and training pace of the runner.

During constant workload exercise, lactate appears to rise continuously at high intensities of work yet plateau after the initial minutes at lower intensities.

Gas exchange kinetics have been studied during constant workload exercise. $\dot{V}O_2$ has been shown to have a delayed steady state at high workloads while at lower prolonged workloads after a steady state has been obtained a slow rise throughout the exercise period has been presented. $\dot{V}E/\dot{V}O_2$, $\dot{V}E$, and VF increase throughout the exercise period with more pronounced increases for the exercise performed above the 4 mmol/l LT. R after an initial increase, even for the low prolonged exercise intensities ($<50\%\dot{V}O_2max$), is followed by a continuous decrease throughout the exercise period.

2.12: STATEMENT OF THE PROBLEM

The preceding sections include the limited research associated with the determination and comparison of Vd and VT under well controlled laboratory conditions with highly trained runners. The lactate gas exchange and heart rate responses to 30 minutes constant workload exercise relative to VT and Vd have not been reported elsewhere. By evaluating the kinetics of these variables during constant work load exercise, either at Vd or VT, it may help to better understand the physiological consequences of these exercise intensities and this may have direct application in training the distance runner, since continuous runs relative to VT and Vd of thirty minutes duration during training are widespread and highly recommended (McDougal and Sale, 1981; Wenger and Bell, 1986; Sjodin et al., 1982). Therefore, the purpose of this study is to compare lactate, HR, and ventilatory responses to CWL exercise at VT and Vd. A second goal was to re-examine VT and Vd validity and reliability when they are expressed as $\dot{V}O_2$, $\%\dot{V}O_2max$, HR, %HRmax and TV by test-retest comparisons.

2.13: RESEARCH HYPOTHESIS

It was hypothesized that there would be no significant differences between $\dot{V}O_2$, $\%\dot{V}O_2max$, HR, %HRmax, and TV at which Vd and VT occur within and between tests for each individual. It was also hypothesized that lactate, gas exchange, and HR responses to CWL exercise either at Vd or VT were not significantly different.

In order to test these hypotheses two separate approaches were adopted. Firstly, two incremental treadmill tests were employed one day apart to test the reproducibility of the measures. Secondly two prolonged runs of 30 minutes duration either at VT or Vd in a random order were used on separate days to record lactate, HR, and ventilatory responses at these work rates. A detailed description of the methodology adopted to test the research hypothesis is contained in the next chapter.

CHAPTER 3: MATERIAL AND METHODS

3.01: SUBJECTS

3.02: EXPERIMENTAL DESIGN

3.03: INCREMENTAL TREADMILL TESTING PROCEDURES

3.04: CONSTANT TREADMILL VELOCITY PROCEDURES

CHAPTER 3: MATERIAL AND METHODS

3.01: SUBJECTS

Twenty well trained male runners volunteered to act as subjects for this study. Three subjects, due to illness (two) and injury (one), did not run the constant work load trials and it was not possible to find any heart rate deflection point for another subject during both maximal and submaximal tests despite measuring him on three different occasions. These four subjects were only excluded from the constant work load ventilatory threshold velocity (CWLVT), and constant work load heart rate deflection velocity (CWLVd) measures.

The criteria for selection of the subjects was that they had trained and competed regularly during the last three years on distances ranging from 800m up to the marathon and cross country. Fourteen subjects were good club level athletes and seven had made the national team for either cross country or middle and long distance track events. Most of the subjects were close to peak performance level as the laboratory data collection took place immediately after their racing season, during the transitional resting period (August-September). Table 1 presents selected characteristics of subjects including their racing distances.

Subjects were well informed about the experimental purpose and design of the study and each subject signed a consent form. All subjects were familiar with treadmill running as they had been physiologically assessed several times during their running career.

Table 1. Selected characteristics of subjects including their racing distances.

Subject	Age	Weight Kg	Height cm	Peak $\dot{V}O_2$ l/min	Peak $\dot{V}O_2$ ml/kg/min	Running event
A	22	75	178	4.45	59.4	M
B	19	65	175	3.81	58.5	M-L
C	21	64	182	4.02	63.3	L
D	21	68	176	4.27	62.8	M
E	21	63	176	3.85	61.1	M
F	21	68	178	4.00	58.9	M
G	25	64	175	3.50	54.7	M
H	23	64	173	4.47	69.8	L^+
I	21	63	172	*4.17	66.2	M^+
J	23	71	180	4.13	58.1	M
K	31	64	170	3.60	55.5	L
L	32	71	182	4.41	62.1	L
M	17	59	180	*4.00	67.6	$M\text{-}L^+$
N	27	78	188	4.70	60.2	L
O	23	66	181	4.62	70.1	$M\text{-}L^+$
P	26	71	176	4.30	60.5	M-L
Q	28	64	170	*4.22	65.9	L^+
R	18	68	180	3.95	58.1	M
S	21	56	168	3.88	69.2	L^+
T	26	66	175	*4.20	63.7	L^+
X	23.3	66.4	177	4.13	62.4	
SD	4.0	5.1	5.0	0.32	4.8	

Peak $\dot{V}O_2$ during first day 's testing (day 1, figure 2) for 16 subjects, and (*) day 3 for 4 subjects, (L) long distance runner, (M) middle distance runner, (M-L) middle and long distance runner, (+) National team level athletes.

3.02: EXPERIMENTAL DESIGN

Data collection took place on four sessions within five days (figure 1). During the first day each subject performed a peak $\dot{V}O_2$ test. After a day of rest a submaximum test was performed, i. e. 90-95% $\dot{V}O_2$max, to test reliability and reproducibility of the physiological variables which had been assessed from the first test. The submaximal test on day 3 to examine reproducibility was chosen because VT and Vd were expected to occur below 90%$\dot{V}O_2$max, as reported in the literature. Indeed, values ranging from 75%$\dot{V}O_2$max (Goodman et al., 1986), with trained cyclists, to 78 and 82%$\dot{V}O_2$max (Maffulli et al., 1987; Brettoni et al., 1989) with highly trained runners have been reported for Vd. For VT values as low as 50%$\dot{V}O_2$max and as high as 80%$\dot{V}O_2$max for recreational and elite runners respectively have been reported (Francois et al., 1987; Rhodes and McKenzie, 1984; Davis, 1985). Furthermore, the submaximal test was employed to prevent exhaustion and to minimize glycogen depletion as immediately the next day constant workload trials were scheduled. On day 4 subjects were randomly assigned to running for 30 minutes on their individual either at VT or Vd, with the alternate 30 minutes run the next day.

By placing the submaximal test and the two steady state runs on three successive days, mainly due to practical reasons, (Day 3, 4, and 5, figure 1) subjects were facing a possible muscle glycogen depletion (Costill et al., 1971) resulting from a low carbohydrate intake in conjunction with exercise. This might well influence the results as it is well established that alterations in blood lactate concentration and ventilatory threshold during exercise can be induced either by dietary

DAY	TEST
-2	
-1	
1	First physiological assessment $\dot{V}O_2max$ test
2	
3	Submaximal 90-95% $\dot{V}O_2max$
4	First CWL trial 30' running either on VT or Vd
5	Second CWL trial 30' running either on Vd or VT

Figure 1. A schematic representation of the experimental design.

Resting days.

(CWL) Constant work load.
(VT) Ventilatory threshold.
(Vd) Heart rate deflection velocity.

manipulations (Segal et al., 1979; Ivy et al., 1981; Jones et al., 1973) or exercise induced glycogen depletion (Mavrogiannis, 1985; Huges et al., 1982; Green et al., 1979).

On day 3 exercise was terminated when subjects were utilising 90-95% of their peak $\dot{V}O_2$, with only the last four minutes of the run above 85%$\dot{V}O_2$max, and a total exercise time of less than 18 minutes. The combination of this exercise intensity and time will result in only a 5-10% muscle glycogen stores reduction (Gollnick, 1973). On day 4 and 5 exercise intensities ranging either from 68-76%$\dot{V}O_2$max or 79-85%$\dot{V}O_2$max, for CWLVT and CWLVd, respectively were employed for 30 minutes. In this case subjects have experienced a 30-40% depletion in the total working muscles glycogen content (Gollnick, 1973).

Costill and Miller, (1980) and Sherman and Costill, (1984) have recommended increasing the daily intake of carbohydrates to 50-60% of total calories (more than 250 grams for 2500 calories) to prevent the gradual depletion of the body's glycogen stores with successive days of training. Therefore, subjects were instructed to consume a mixed diet with a carbohydrate content of more than 60% and a minimum of 250 grams. carbohydrates daily. It was easy for the athletes to achieve these targets as they were already consuming daily close to the aforementioned percentage and quantity of food. This was monitored by asking the subjects to fill dietary forms where they reported all drinks and food which they had consumed during the day preceding each test. Subsequent evaluation of dietary forms revealed that subjects met this criteria, indeed in 90% of cases carbohydrate intake exceeded 400 grams daily. This procedure has shown to result in similar pretest muscle glycogen concentrations within 24 hours, if the muscle glycogen reserves have been only moderately depleted (McArdle, Katch and Katch, 1986; Sherman et al., 1981; Miller et al., 1983).

Subjects were asked also to refrain from physical activity 48 hours preceding

the first test (day 1) and additional physical exertion during the testing days. The majority of the subjects acted as they were instructed but four of the national level athletes reported they had an easy run after testing. The fact that they were used to high weekly mileages (more than 80 miles) and this was lowered by half during the testing week further support the view that glycogen depletion or other aspects of fatigue did not influence their laboratory data. Finally, each test was performed after 12 hours fasting, mostly during morning hours.

3.03: INCREMENTAL TREADMILL TESTING PROCEDURES

(I): Treadmill protocol

Incremental exercise tests were performed on a motor-driven treadmill (Quinton). Treadmill speed was calibrated while the subject ran at different running speeds by counting the time it takes for the completion of 30 treadmill revolutions. The incremental exercise peak $\dot{V}O_2$ test consisted of 5 minutes warm up at a speed which had been previously determined during familiarisation with treadmill running and usually corresponded to a perceived exertion of less than 9 on the 15 grade Borg scale (Borg, 1985). For the less fit subjects (club level) this speed corresponded with 2.22 m.sec or 8 km/h treadmill velocity whereas for the better athletes (national team level) it corresponded with a treadmill velocity of 2.77 m.sec or 10 km/h. On the completion of warm up subjects were left free for five minutes to perform their usual pre-racing stretching routine.

The protocol consisted of 1 minute running stages with 0.5 km/h per stage increments while the treadmill elevation was kept horizontal. This protocol has been validated from other studies with runners for the determination of peak $\dot{V}O_2$, VT and Vd simultaneously (Aro et al., 1988; Kispert et al., 1988; Maffuli et al., 1987; Bunc et al., 1984; Peronet et al., 1987; Davis et al., 1984; Brettoni et al., 1989). The rationale for the use of this protocol is that it simulates as closely as is practically possible the training and competitive environments of the athletes during the test (Davis et al., 1984). However, this protocol produces lower peak $\dot{V}O_2$ values as compared with gradient increments at some stage or throughout the test (Weltman et al., 1990; Katch et al., 1976; Astrand and Rodahl, 1986).

(II): Gas analysis

$\dot{V}O_2$ was measured by the open circuit spirometry method. Expired gases were continuously sampled from a 5 litre mixing chamber by a Beckman Metabolic Measurement Cart (MMC). Fractional expired O_2 (FEO_2), fractional expired CO_2 ($FECO_2$), gas temperature, barometric pressure, volume of expired air ($\dot{V}E$), and breathing frequency were measured continuously and displayed at minute intervals using a system of instruments involving a volume turbine, OM-11 oxygen analyser and an LB-2 carbon dioxide analyser. Both analysers have a rapid response time (<1s) a resolution of 0.01% and an accuracy of ± 1.0% of full scale. This instrument has been validated by Willmore et al., (1976) and Farrell et al., (1979) and used extensively in the laboratory assessment of oxygen consumption.

Before and after each test the analysers were calibrated with mixed gases of known concentration. During the calibration procedure it was observed that the

OM-11 oxygen analyser demonstrated a small systematic downscale drift in the oxygen reading from the pre to post test values. This analyser is sensitive to the pressure of water vapour and it is suspected that the small decrease in the oxygen percentage from the pre- to post test calibration was the result of water condensation in the sample lines due to incomplete removal in the drying tube (Wilmore and Davis, 1976). This inconsistency in the calibration resulted in a minor $\dot{V}O_2$ over-estimation at some stage of the test. However the average decrease was less than 0.07% oxygen and this made little difference to the $\dot{V}O_2$ assessment.

Expired gas volume was measured with a biased flow turbine which was calibrated before and after each day's testing using a calibrated one litre syringe at the mouth piece. The MMC pressure transducer was checked daily and compared with an accurate wall mercury barometer. The monolithic chip temperature transducer was checked before each test by applying a portable thermometer in the volume transducer chamber.

The timing and sequence of measurements were under the control of an electronic interface and a programmable Monroe calculator. The electronic interface also digitises the signals from the analysers and transfers the digital values to the calculator every 400 ms.

(III): Data collection

The subjects breathed through a low resistance non-rebreathing 2 way Rudolf 2700 B valve. The expired gases passed through a 100 cm length of 340 mm diameter non-kinkable tubing into the 5 litre mixing chamber. Respiratory gas exchange data for each work load (i.e. $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}$Estpd and R) were determined

on a locally developed computer programme based on the computations described by McArdle, Katch and Katch, (1986) when $\dot{V}E$ atps, FEO_2, $FECO_2$ are known.

Strong verbal encouragement was given during the final stages of the peak $\dot{V}O_2$ test. The highest $\dot{V}O_2$ value obtained during the first incremental exercise test was recorded as the subject's peak $\dot{V}O_2$. Criteria for peak $\dot{V}O_2$ were:

1) A levelling off or decline of $\dot{V}O_2$ with increase in work load (McConnel, 1988; Taylor, 1955; Davis et al., 1984).
2) Heart rate maximal value (HRmax) to be within ±10bpm of the age-predicted HRmax (Gibson et al., 1979; Shephard et al., 1968).
3) Respiratory exchange ratio (R) greater than 1.05 (Davis et al., 1984; McMiken and Davis, 1976).
4) A score on the completion of the test equal or greater than 19 in the 15 grade Borg scale (Borg, 1985; Hammond and Froelicher, 1984).

Four subjects did not meet all of these peak $\dot{V}O_2$ criteria during the first test (table 1), therefore they were asked to perform a maximal test to volitional exhaustion two days later instead of a submaximal trial. They all met the aforementioned criteria during the second trial.

(IV): Ventilatory threshold assessment

VT was detected from individual plots of ventilatory and gas exchange variables (figure 2 and 3) at each work load during both incremental exercise tests for each subject according to the criteria of Wasserman et al., (1973); Davis et a., (1976); Caiozzo et al., (1982); Sucec et al., (1989); Davis et al., (1983); Iwaoka et al.,

(1988); Reinhard et al., (1979). These criteria can be summarised as follows:

1) a non linear increase in expired minute volume ($\dot{V}E$ atps)
2) a non linear increase in carbon dioxide production ($\dot{V}CO_2$ stpd) when oxygen uptake ($\dot{V}O_2$ stpd) continued to increase linearly
3) a systematic increase in the FEO_2 without a concomitant increase in the $FECO_2$.

$\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ were not used in this study to detect VT because increases in the $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ are analogous to increases in FEO_2 and $FECO_2$ respectively (Caiozzo et al., 1982; Reinhard et al., 1979; Davis et al., 1979). This was confirmed in this study.

The transformation of VT values from work load to $\dot{V}O_2$ or HR values was performed by computing the linear regression equation for $\dot{V}O_2$ and/or HR with work load. VT was assessed by visual inspection by two independent scientists, the ventilatory transients were plotted against work load. In the cases where the workload corresponding with the $\dot{V}E$ inflection point was not the same with the workload which corresponded with the point before FEO_2 systematic increases (usually observed differences of less than 0.5 km/h treadmill velocity) the work load before FEO_2 systematic increases was considered the VT (Caiozzo et al., 1982).

(V): Heart rate deflection velocity assessment

HR was measured by means of a Sport Tester HR monitor (PE3000) throughout peak $\dot{V}O_2$, submaximal and constant work load tests. Changes in HR

frequency were calculated and stored in the receiver's memory every 5 seconds for subsequent recall. The last three HR values of each minute, i.e. the ones which were recorded during 50", 55" and 60", were averaged and the mean HR value was used for drawing graphs (Table 2).

The treadmill running velocity-HR relationship obtained with the tests (maximal-submaximal) showed broken stick linearity (figure 2 and 3) for all the subjects but two, one, and one during the first, second, and both tests respectively. These two lines were drawn by subjective judgment using criteria described by Conconi et al., (1982); Gaisl et al., (1988); Maffulli et al., (1987); Borseto et al., (1989). The running speed which corresponded with HRVd was determined by interpolation (figure 2 and 3).

3.04: CONSTANT TREADMILL VELOCITY PROCEDURES

(I): VT and Vd constant treadmill velocities

VT velocity for the constant workload trials was the value which was obtained for each subject during maximal and submaximal tests. For the subjects where the first test VT was different from the one discerned during the second test, an average value was used for the constant work load VT test (i.e. 13.75 km/h, figure 2). Where this occurred it was due to minor drifts towards higher or lower speeds of less than 0.5 km/h from test to test.

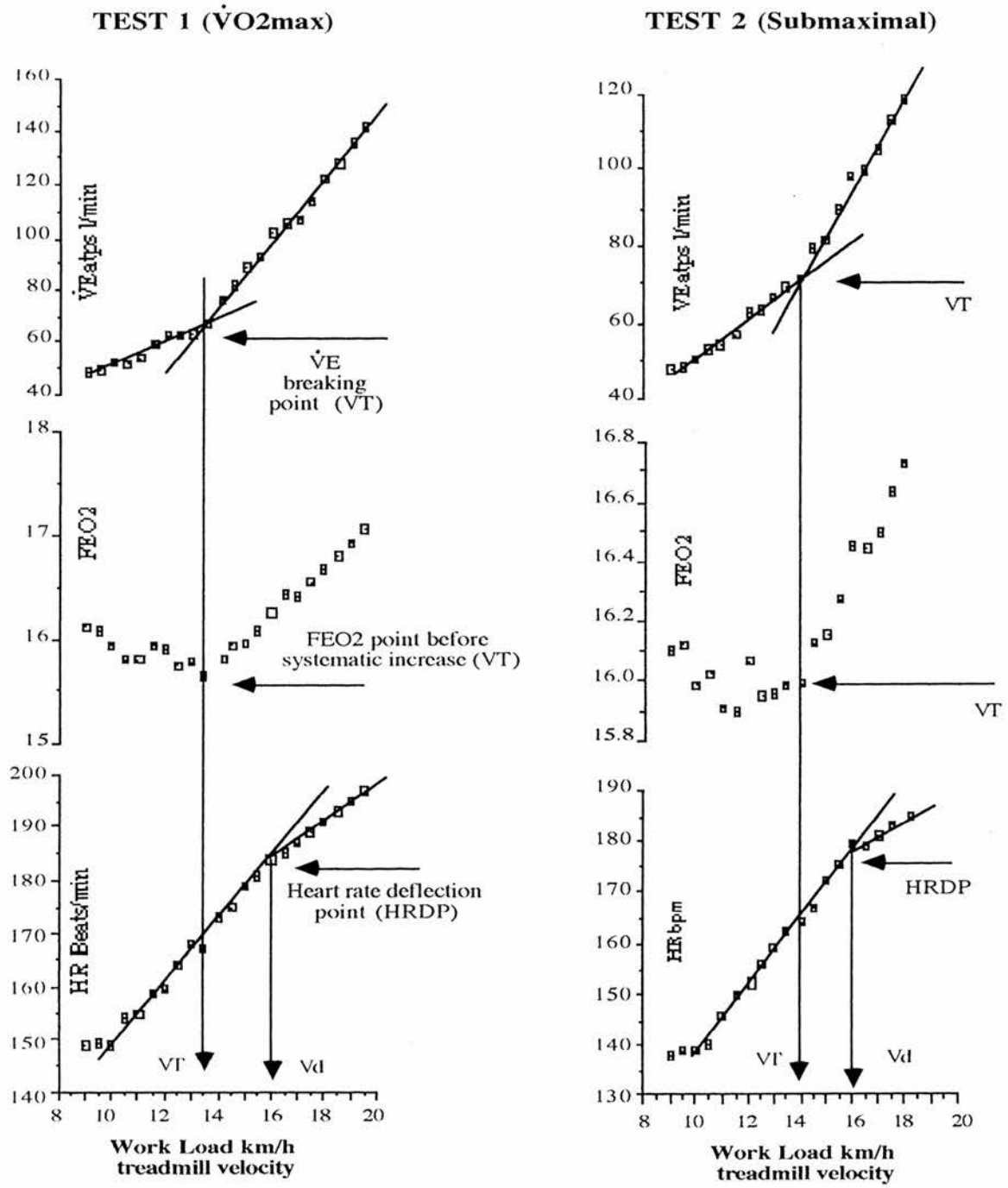

Figure 2. Graphical representation of changes in HR, FEO_2, and $\dot{V}E$(atps) during both physiological assessments (test 1 and 2) for subject A. Note that VT test-retest value was different (i. e. 13.5 and 14km/h) whereas Vd was met at the same work load (i. e. 16km/h).

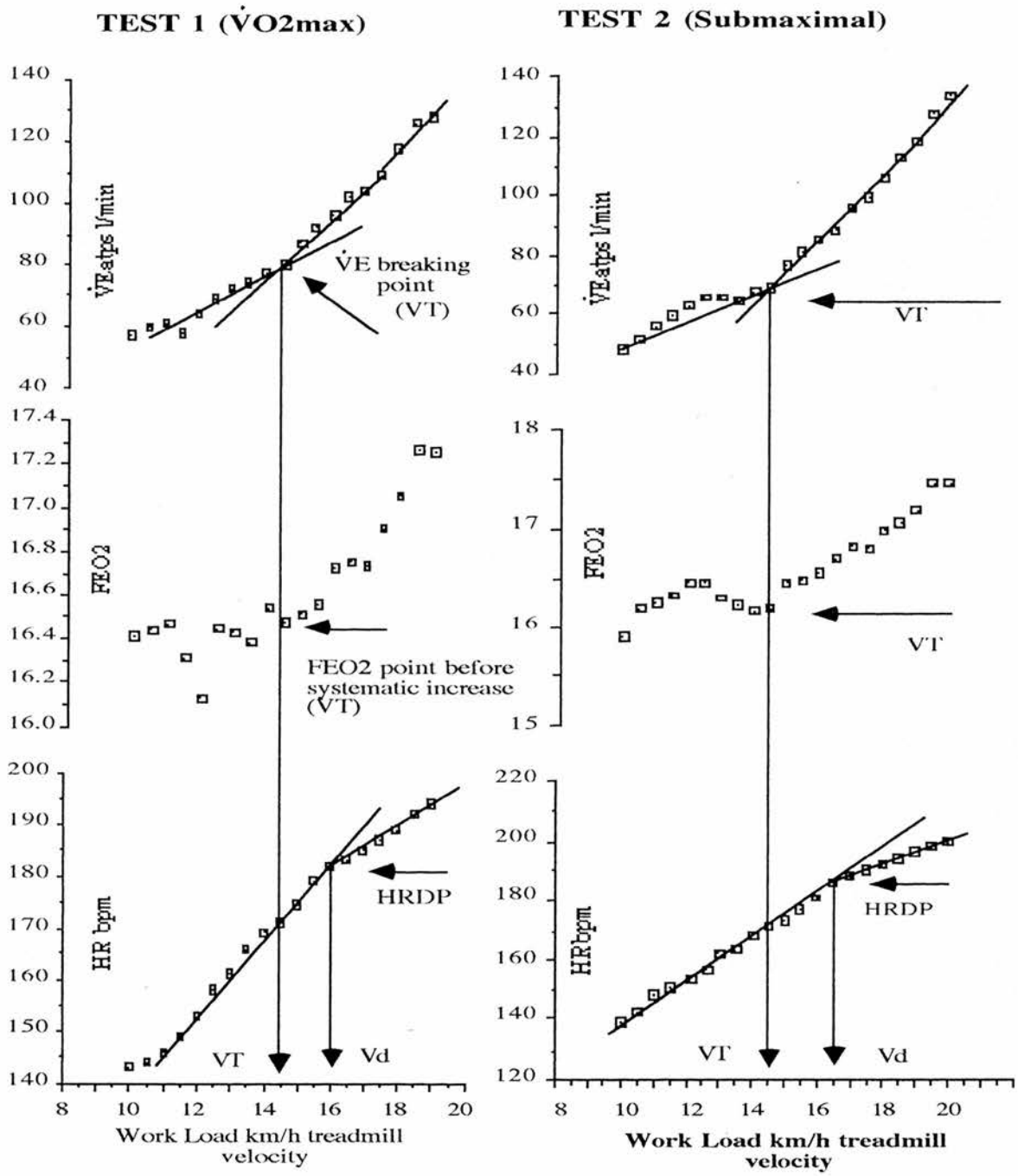

Figure 3. Graphical representation of changes in HR, FEO_2, and $\dot{V}E$(atps) during both physiological assessments (test 1 and 2) for subject E. Test-retest VT value (km/h) was the same (i.e. 14.5 km/h treadmill velocity) whereas Vd had a 0.5km/h drift toward a higher work load in the second physiological assessment.

Where test-retest HRVd was different, the mean value was also employed for the constant work load Vd trial (i.e. 16.25km/h, figure 3) for all subjects but the three which showed Vd in only one test. For these three subjects, this single value had to be used for the prolonged constant load trial relative to Vd.

(II): Gas exchange and heart rate measurements

During constant workload tests either at VT or Vd the gas exchange variables were measured between 8 and 10, 18 and 20, and 28 and 30 minutes of exercise. Subjects were asked to breath through the valve two minutes before each gas collection started. This resulted in at least 15 minutes running without having to wear the mouthpiece, which increases the airways' dead space and enhances the non-exercise induced ventilation. $\dot{V}$Estpd, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E/\dot{V}O_2$, $\dot{V}E/\dot{V}CO_2$, VF (ventilatory frequency), and R, were used for subsequent analysis. HR frequency was monitored and stored in a Sport Tester's memory every 5 seconds throughout the exercise period.

(III): Blood plasma collection

Arterialised capillary blood (Forster et al., 1972) was sampled by finger-prick. Fifty μl (microlitre) of blood were collected while the subject rested for 30 minutes on a bed (resting value), and by interrupting constant load exercise for a minute after ten, twenty, and thirty minutes of running during both steady state trials (Figure 4). Blood was collected in heparinised capillary tubes and immediately placed into a standardised 4 μl diluting solution to prevent coagulation. Samples

were centrifuged within 15 minutes. Thirty µl of the supernatant blood plasma were stored in a freezer for subsequent analysis.

(IV): Blood plasma analysis

Samples were diluted with 270 µl buffering solution mixed well and analysed enzymatically for lactate using lactate/640 Analyzer. Extensive testing programmes have established that the Roche Model 640 Lactate Analyzer in serum and plasma samples give essentially the same results as are obtained with classical enzymatic spectophotometric methods, based on the oxidation of lactate to pyruvate by NAD in the presence of lactate dehydrogenase (Racine et al., 1975; Guillot et al., 1976; Tsopanakis et al., 1986).

The measurement of lactate is achieved by monitoring the concentration of an electrochemically active substance liberated by the essentially irreversible oxidation of lactate to pyruvate in the presence of the enzyme cytochrome b_2. (Racine and Mindt, 1971; Williams et al., 1970). The instrument was calibrated before and after use with two standard solutions of lactate i.e. 1.0 mmol/l and 5.0 mmol/l. Sample plasma lactate values were accepted if they were reproducible within ± 0.15 mmol/l (2 to 3 times).

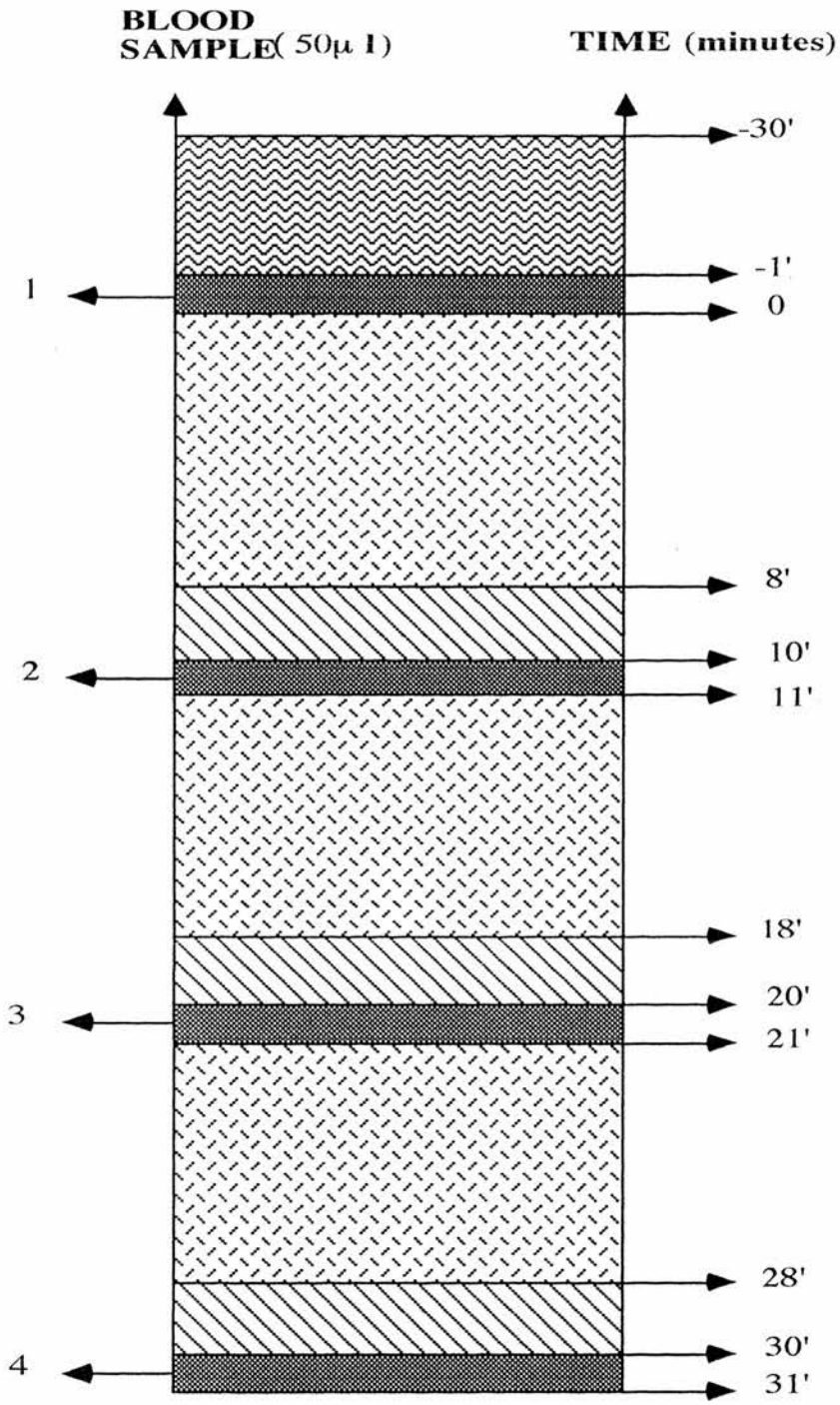

Figure 4. Schematic presentation of the experimental design for blood and gas collection during 30 minutes running on the treadmill either at VT or Vd.

Blood sampling period

Gas analysis during running phase

Bed resting period

Running time before blood collection

Table 2. A print out of the heart rate response as it was stored in the Sport Tester receiver's memory during incremental treadmill peak $\dot{V}O_2$ test for subject F. Note that an average value of the last three values which were stored each minute was used for subsequent analysis.

PULSE RATE LISTING

Copy right by Polar Electro

Source: RECEIVER MEMORY

Date : 18-09-90

Time (min) Starting Time: 0 : 0 : 0

													Work Load km/h	**Average HR bpm**
0	100	110	117	120	121	122	121	123	123	122	120	118	8	120
1	118	120	120	125	127	124	121	121	118	122	117	117	8.5	119
2	117	119	120	123	121	121	121	122	125	127	127	127	9	127
3	125	124	125	123	123	123	123	123	124	126	125	124	9.5	125
4	128	126	130	129	132	132	134	134	132	130	139	130	10	130
5	131	133	136	137	139	138	137	137	135	134	135	136	10.5	135
6	139	141	143	142	143	141	141	139	139	137	138	138	11	138
7	136	135	137	136	139	139	138	140	141	143	143	142	11.5	143
8	143	142	144	143	143	143	145	146	145	145	146	148	12	146
9	148	148	148	150	151	152	151	149	148	146	147	148	12.5	147
10	148	149	151	154	154	154	155	156	158	156	156	155	13	156
11	157	158	156	158	155	155	153	154	154	155	157	159	13.5	157
12	159	159	159	158	160	160	162	162	164	161	160	160	14	160
13	161	164	164	163	166	166	166	165	164	165	168	170	14.5	168
14	170	169	169	168	169	169	169	170	171	174	174	171	15	173
15	173	175	174	174	174	176	177	178	179	179	179	177	15.5	178
16	178	179	179	180	180	179	178	177	177	179	182	183	16	181
17	183	180	182	184	181	181	180	181	182	184	184	184	16.5	184
18	185	185	185	186	184	185	186	185	186	186	187	187	17	187
19	186	185	188	188	188	189	188	189	189	190	191	191	17.5	191
20	191	190	190	190	190	189	189	188	189	190	191	191	18	191
21	191	191	191	191	191	191	191	191	190	191	193	192	18.5	192
22	192	193	194	194	195	195	195	194	194	194	195	195	19	195
23	196	197	196	196	197	197	196	196	196	196	197	198	19.5	197
24	199													

CHAPTER 4 RESULTS

CHAPTER 4 RESULTS

4.01: STATISTICAL ANALYSIS

All statistical analyses were done by computer using the Statistical package for the Social Sciences. Analysis of variance (ANOVA) for repeated measures was used to evaluate differences within and among submaximal tests, where appropriate Scheffe's F post-hoc test was used to perform multiple comparisons.

The reproducibility of VT and Vd was evaluated by Pearson's Product Moment correlation coefficients and linear regression analysis. Similarity of the tests was implied by a regression intercept at zero and slope unity (Cohen and Holliday, 1982; Maxwell and Delaney, 1989). The significance of differences between the means was tested by a paired t test. In all statistical analysis the 0.05 level of significance was used.

4.02: RESULTS

In the preceding chapter two separate areas of research in this study have been identified, the first relating to VT and Vd, reliability and concordance and the second concerned with the lactate, heart rate, and ventilatory responses to CWL exercise relative to VT and Vd.

This chapter gives the results of both areas of concern. Data will be presented separately. This will allow three fundamental questions to be answered in relation to elite runners:

a) What is the validity of VT and Vd as systematic physiological phenomena when they are expressed as $\dot{V}O_2$ml/min, %$\dot{V}O_2$max, treadmill velocity (TV) km/h, HRbpm, and %HRmax?

b) Does the HR criteria introduced by Conconi et al., (1982) to estimate OMA coincide with the traditional VT?

c) How do physiological mechanisms such as lactate, HR, and ventilatory transients respond to a 30 minute constant load exercise either at VT or Vd?

4.03: VT REPRODUCIBILITY

Test-retest correlation coefficients for VT expressed as TV, $\dot{V}O_2$l/min, %$\dot{V}O_2$max, HRbpm, and %HRmax were: 0.95, 0.86, 0.75, 0.94, and 0.86, ($p<0.01$) respectively (Figure 5). Test-retest sample means for %$\dot{V}O_2$max at VT, HRbpm at VT, and %HRmax at VT were significantly different (Table 3). VT was 100% detectable (n=20) for

Table 3. The means, standard deviations, and ranges of variables at the VT, for both physiological assessments (test 1 and 2, n=20)

	Treadmill velocity km/h	$\dot{V}O_2$ l/min	%$\dot{V}O_2$max	HR bpm	%HRmax
Test 1 X	14.75	3.01	73.8	166	84.8
SD	1.59	0.31	5.2	10	3
Range	11.5-15.5	2.35-3.32	65-83	144-180	78-89
Test 2 X	14.67	2.96	72.1	161	83
SD	1.61	0.26	4.2	11	3.5
Range	11.5-17.5	2.35-3.32	64-82	139-180	76-89
P	ns	ns	<0.05	<0.05	<0.05

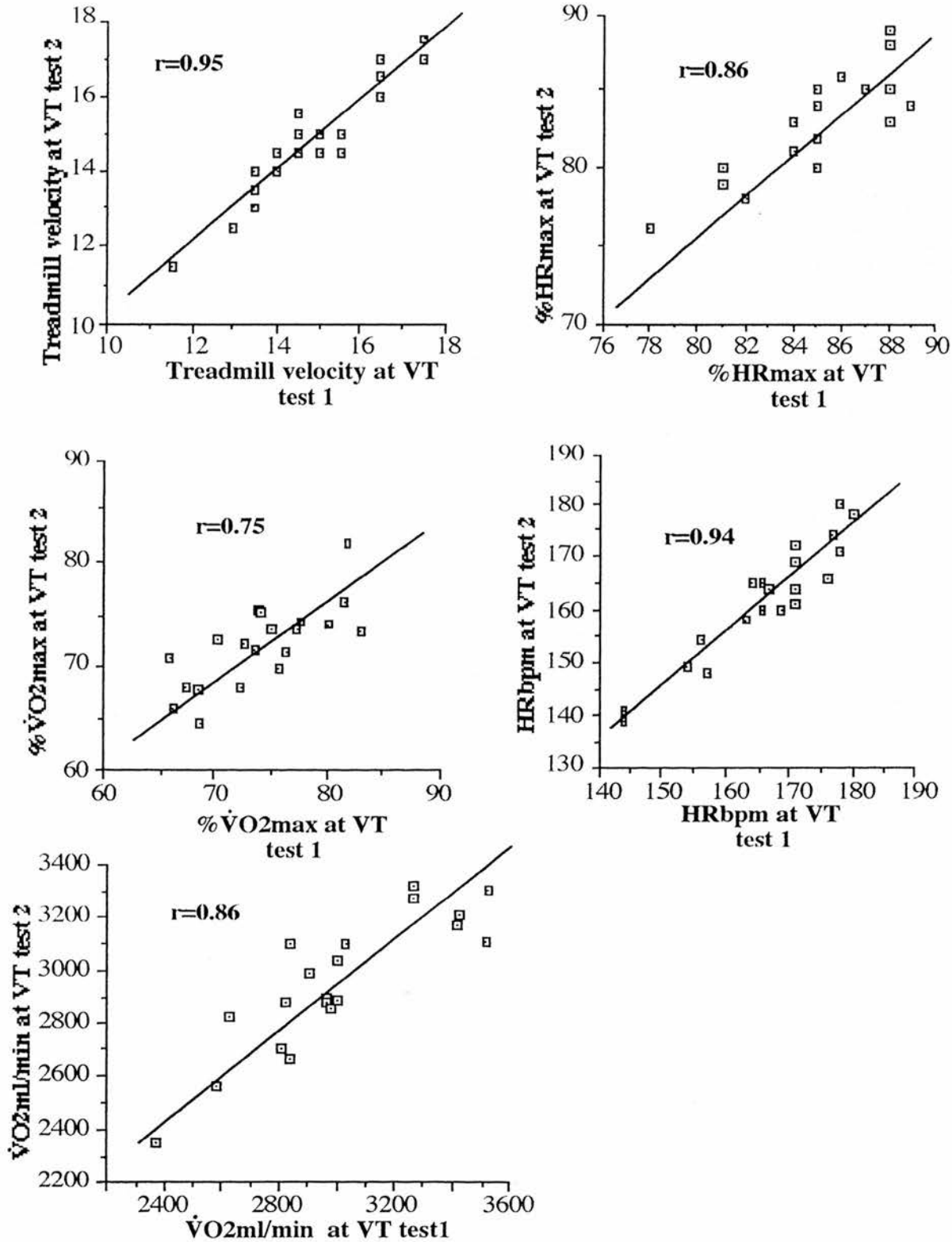

Figure 5. Reproducibility of treadmill velocity, %HRmax, %$\dot{V}O_2$max, HRbpm, and $\dot{V}O_2$ ml/min at VT for test 1 and 2.

both tests. $\dot{V}E$ breaking point and the point before systematic increase of FEO_2 were easily defined (Figure 2, 3, and 7).

4.04: Vd REPRODUCIBILITY

The reliability analysis by test-retest comparisons of TV, $\dot{V}O_2$, $\%\dot{V}O_2max$, HR, and %HRmax at Vd was: 0.69, 0.52, 0.07, 0.75, and 0.29, respectively (figure 6). There were no differences between mean values for test 1 and 2 for these variables (table 4).

Sixteen (80%) subjects demonstrated clear Vd in both exercise tests. Three subjects presented Vd in only one of the tests and one subject did not show Vd in either test. Figure 7, presents the three out of four most difficult graphs to assess Vd (subjects J, O, and K). Subject O did not show any clear deflection point during both tests, whereas for subjects K during test and J during re-test HR increased linearly throughout the incremental treadmill exercise tests. This raises questions about the validity of this measure as a consistent physiological phenomenon. For those subjects whose Vd was detectable at least in one test (n=17, and n=18 for test 1 and 2) their $\dot{V}O_2$l/min, HRbpm, and TV at VT correlated well with the corresponding Vd values (table 5). The high correlations found in this study does not mean that these variables are the same for each measure (i.e, VT, and Vd), just that they are related, (i.e. the higher or lower values of one variable are followed by similar values by the other variable). Indeed, by applying the paired t test statistic, it was shown that for each test Vd variables were significantly different from the corresponding variables at VT ($p<0.05$, table 5). All the preceding variables at Vd were in fact systematically greater compared with the respective variables at VT and above the onset of metabolic acidosis, suggesting that Vd does not coincide with VT.

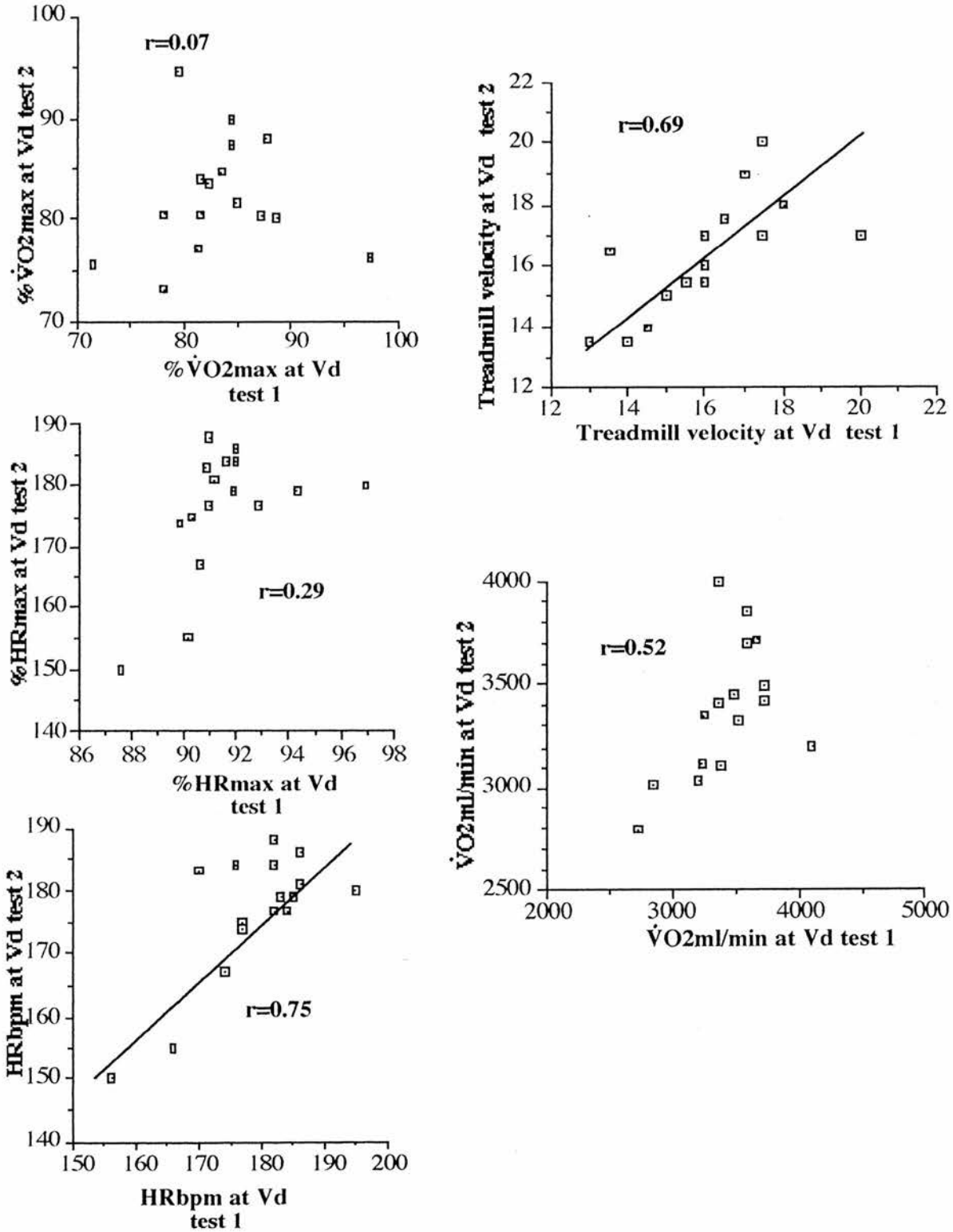

Figure 6. Reproducibility of %$\dot{V}O_2$max, treadmill velocity km/h, %HRmax, $\dot{V}O_2$ml/minute, and HRbpm at Vd during both incremental exercise tests.

Table 4. The means, standard deviations, and ranges of variables at the Vd for both physiological assessments (test 1 and 2, n=16).

	Treadmill km/h	$\dot{V}O_2$ l/min	%$\dot{V}O_2$max	HR bpm	%HRmax
Test 1 X	16	3.42	83.2	179	91.5
SD	1.8	0.34	5.7	9.2	2
Range	13.1-20	2.71-3.99	71.4-97.4	156-195	87.6-97
Test 2 X	16.3	3.37	82.2	176	90
SD	1.8	0.33	5.7	10.5	3.7
Range	13.5-20	2.8-3.39	73.7-94.6	150-188	84.2-98
P	ns	ns	ns	ns	ns

Table 5. Pearson's Product Moment correlations and Student's t values for VT and Vd as measured in test one and two.

FIRST TEST (n=17)			**SECOND TEST** (n=18)	
r	t	**VARIABLE**	r	t
0.82*	-8.3*	HRbpm	0.78*	-7.2*
0.77*	-7.6*	$\dot{V}O_2$l/min	0.60*	-6.2*
0.70*	-8.2*	%$\dot{V}O_2$max	0.41	-7.2*
0.43	-9.1*	%HRmax	0.54*	-7.7*
0.87*	-6.3*	Treadmill Velocity km/h	0.87*	-7.8*

*$p<0.05$

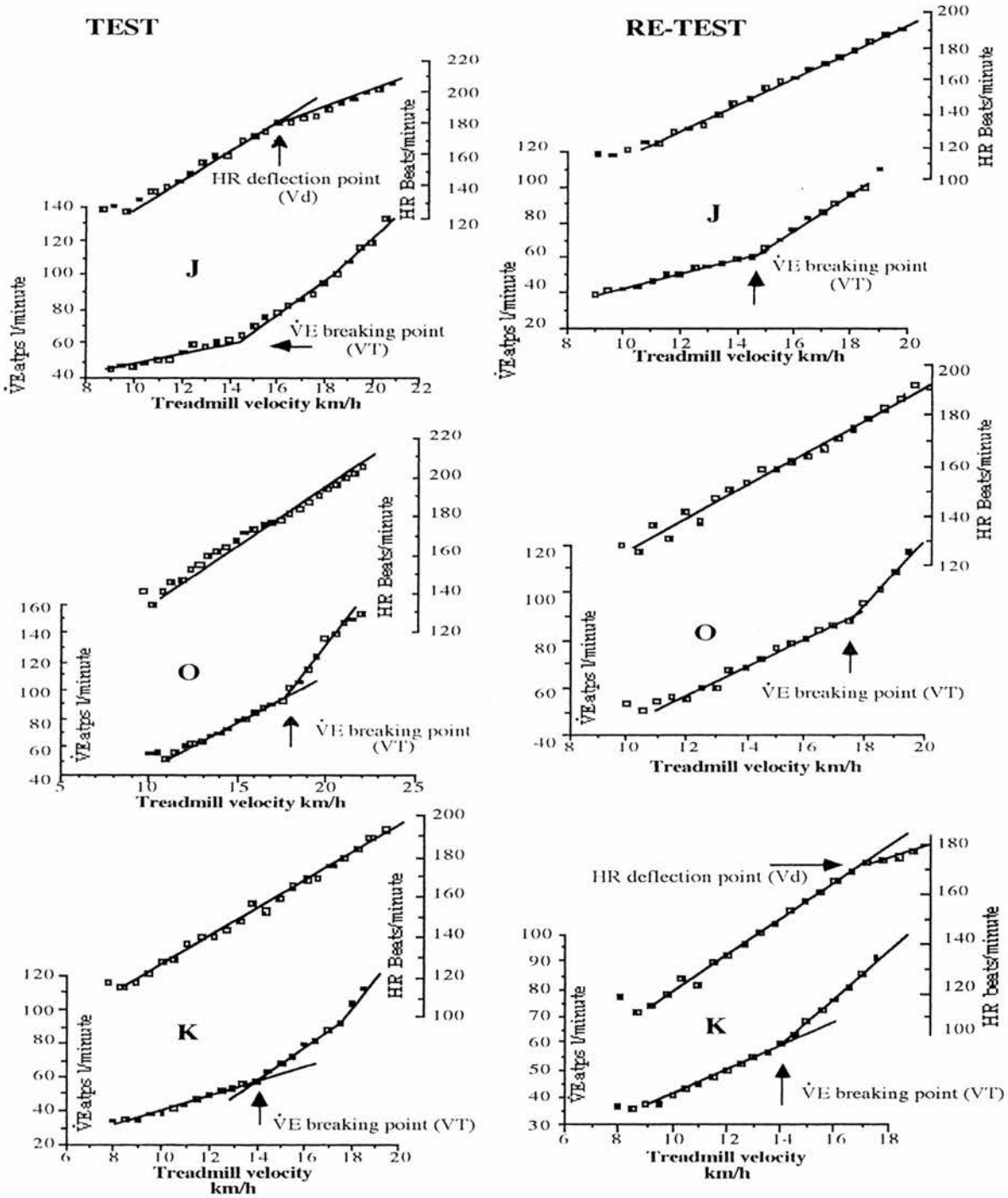

Figure 7. Representative heart rate (HR) and volume of expired air (V̇Eatps) curves for three subjects (J, O, and K). The left panel show the results in the first test and the right panel in the second test. Note that V̇E breaking point (VT) was evident for all trials whereas HR Vd either showed poor reproducibility or was not detectable.

VT and Vd when expressed as $\dot{V}O_2$ and HR appeared to correlate well within each test (r=0.60-0.82). However, when the VT and Vd data were transformed to $\%\dot{V}O_2max$ and %HRmax the correlations, for each test, between these same indices dropped to r=0.41-0.70. This suggests that the transformation of VT and Vd values to $\%\dot{V}O_2max$ and %HRmax increases (to varying degrees) the homogeneity of the data and thereby produces lower correlation coefficients.

The mean Vd (16.24±1.65, range 11.5-17.25), and VT (14.6±1.6, range 11.5-17.25) treadmill speeds (figure 8) which had been used for

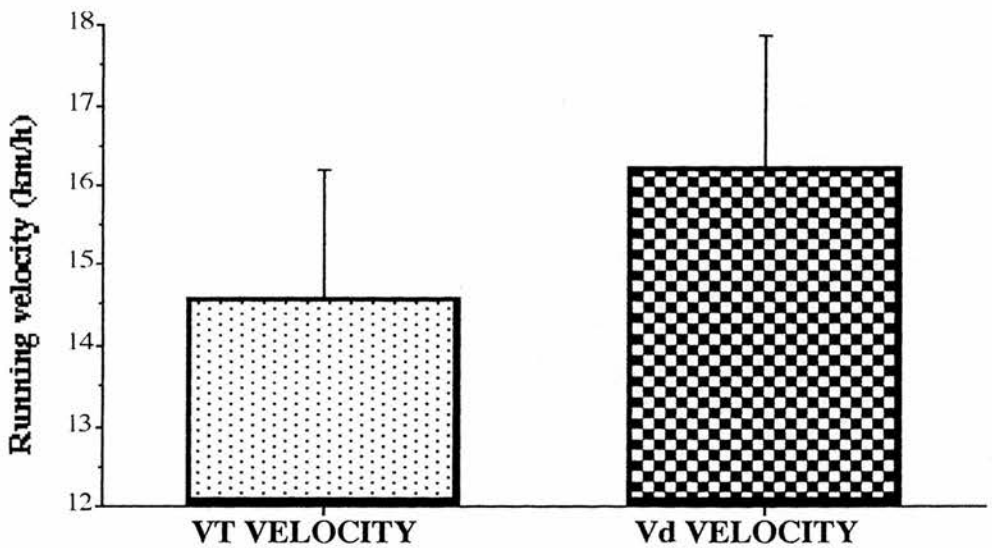

Figure 8. Comparison of the mean±SD, (n=16) VT (14.6±1.6km/h) and Vd (16.24±1.65km/h) running velocities which were finally used for the CWL exercise tests.

the CWL trials were highly correlated (r=0.94, $p<0.01$). This was expected as the test-retest TV at VT and Vd presented very small drifts of less than 1km/h and this did not affect the high correlation presented by this measure within tests (r=0.87)

for both tests. By using either VT or Vd treadmill speeds as the independent or dependent variable (figure 9) the following regression equations are produced (R=0.89 for both equations):

VT=0.915Vd-0.259 km/h

Vd=0.97VT+1.984 km/h

The first can be practically useful for the estimation of VT as it does not require a well equipped laboratory, whereas the second can be used for the estimation of exercise intensity corresponding with Vd, for racing and training pace application.

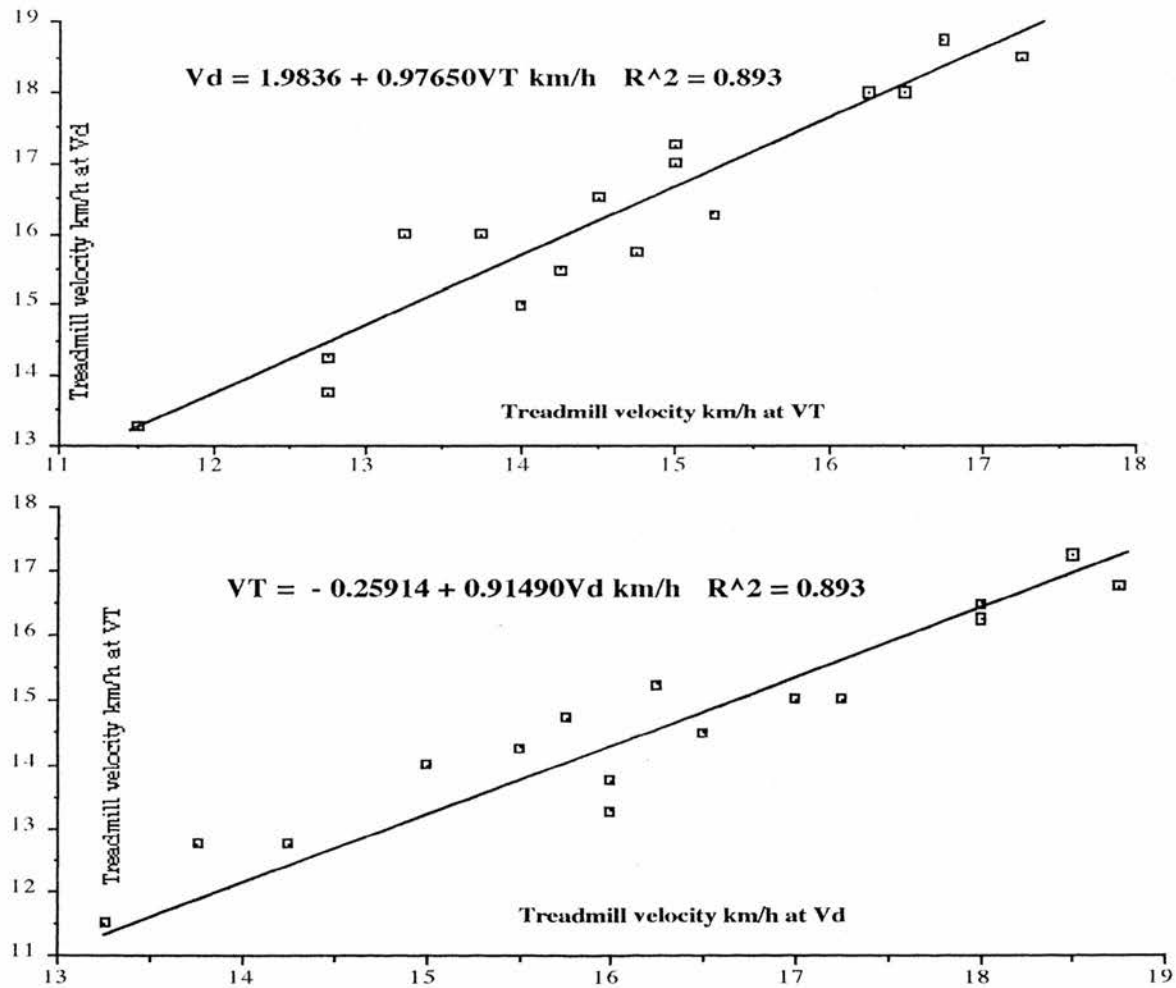

Figure 9. Regression equations with the work load for either VT or Vd as the dependent variable.

4.05: CONSTANT WORKLOAD RELATIVE TO VT AND Vd

This section describes the results of each of the 30 minutes constant work load exercise at VT and Vd. Lactate, gas exchange, and HR responses will be presented.

4.06: LACTATE RESPONSE

Figure 10 presents the blood lactate responses at each of the submaximal constant work load (CWL) tests which are given in table 6.

Table 6. Plasma lactate and heart rate responses during CWL exercise at VT and Vd. Values are expressed as means±SD.

	CWLVT		**CWLVd**	
Time	Lactate	HR	Lactate	HR
min	mmol/l	bpm	mmol/l	bpm
Rest	1.33±0.49	51.1±5.7	1.2±0.42	51±5.6
10	2.02±0.59	160±12.5	3.4±1.5	169±10.5
20	2.22±0.73	161±12	4.2±1.93	171.8±11.5
30	1.87±0.60	161±12	4.1±1.5	175±12

Note, that only resting values for all variables were statistically significantly different from the ones obtained at the 10^{th}, 20^{th}, and 30^{th} minute ($p<0.05$).

For both CWLVT and CWLVd, mean blood lactate concentrations measured at the 10^{th} minute of exercise were greater than the respective resting values. Mean lactate values measured at the time points 10, 20, and 30 minutes at CWLVT were not significantly different and mean lactate values measured at the same time points during CWLVd also were not different. The presence of more data points (i.e. more frequent sampling) could help in making a better evaluation of lactate kinetics throughout CWL exercise since figure 10 suggests that mean plasma lactate concentration may continue to increase between 10
and 20 minutes for both CWL tests.

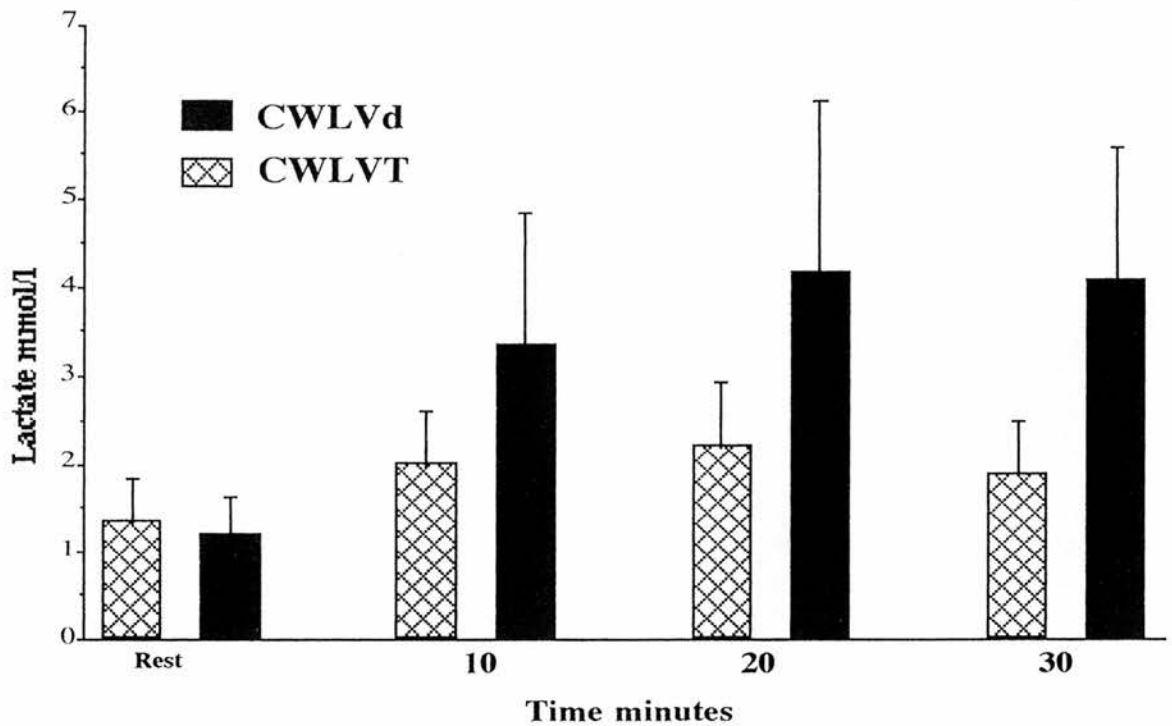

Figure 10. Plasma lactate concentration at CWLVT and CWLVd, values are means±SD.

Lactate concentrations were significantly higher for the time points 10, 20, and 30

minutes at CWLVd when compared to the corresponding CWLVT values.

Figure 11 presents the rate of lactate accumulation in the prolonged submaximal tests, calculated as the concentration at the end of the interval minus the concentration at the beginning of the interval divided by the time period in minutes. It is of note that during CWLVd

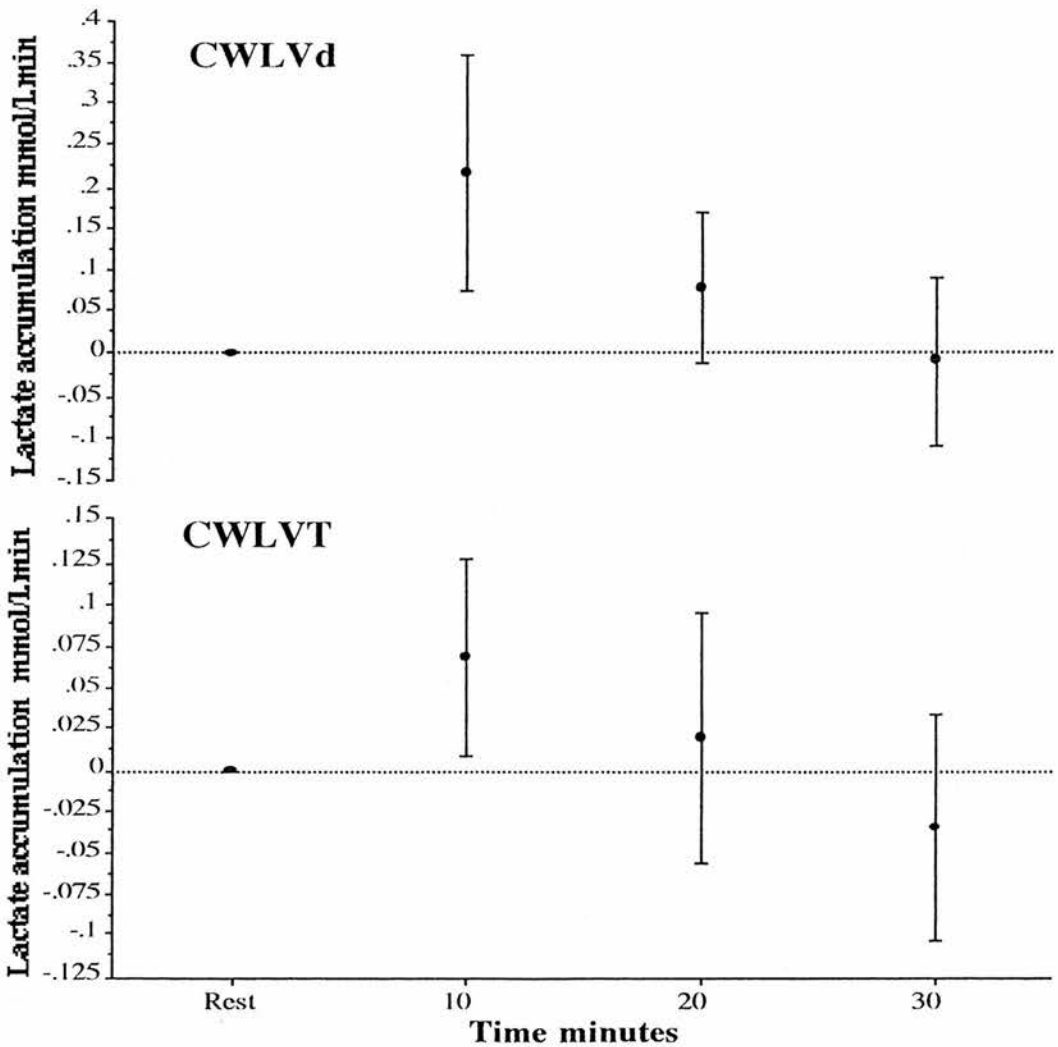

Figure 11. Mean (±SD) rate of plasma lactate accumulation during prolonged exercise tests relative to Vd (upper graph), and VT (lower graph).

and CWLVT, plasma lactate accumulation occurred only during the initial 10 minutes. Even though plasma lactate concentrations were higher at the CWLVd exercise after 10 minutes, accumulation was not significantly different. At the end of the 30 minute exercise period relative to VT, lactate uptake either by the working muscles or by the liver exceeds its rate of accumulation. This may be an indirect indication of the more efficient liver function (gluconeogenesis) during CWLVT as less blood had to be shifted from this organ to support the working muscles due to the lower exercise intensity.

The individual lactate concentrations (figure 12) during CWLVd for the time points 0 and 10 minute consistently increased, and resulted in a mean elevation of 2.8 times the resting value ($p<0.05$). Between time points 10 and 20 minute lactate values increased in the majority of the subjects ($n=11$) which resulted in a mean elevation of 21% ($p<0.05$)

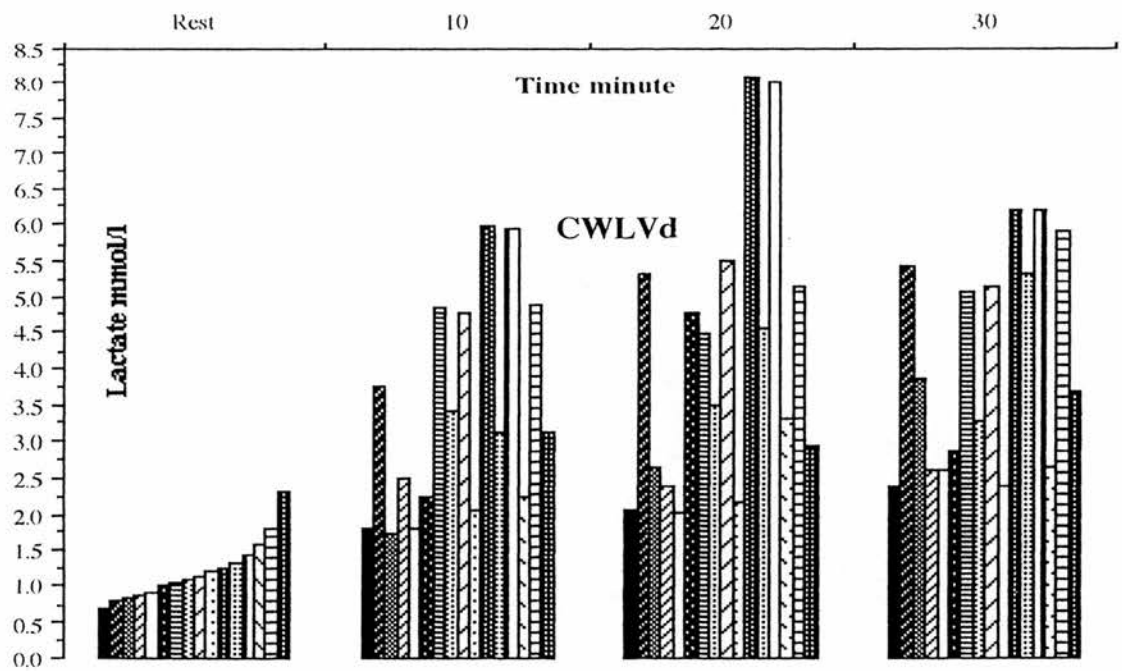

Figure 12. Individual lactate values during 30 minutes of running at Vd.

above the value at the 10th minute. However despite this mean trend for increased lactate values, individual lactate values for 5 subjects showed decreased lactate levels at the 20th minute. Mean plasma lactate concentration measured at the 30th minute was essentially stable compared with the mean value at the 20th, but there was no consistent trend in the individual lactate values. Plasma lactate concentrations decreased for 7 of the subjects, for another 7 they were still increasing, and for only 2 remained constant. Mean lactate concentration at the end of the 30 minute CWLVd trial was 4.09±1.5 (range 2.4-6.2) which, when compared to resting values (1.21±0.4, range 0.66-2.31) is significantly elevated ($p<0.05$).

The individual plasma lactate concentration values for the CWLVT for the time points 0 and 10 minutes does not show any substantial trend. Lactate values measured at the 10th minute were elevated in the majority of the subjects (n=13) giving a mean value of 2.02±0.59 mmol/l significantly above the resting value.

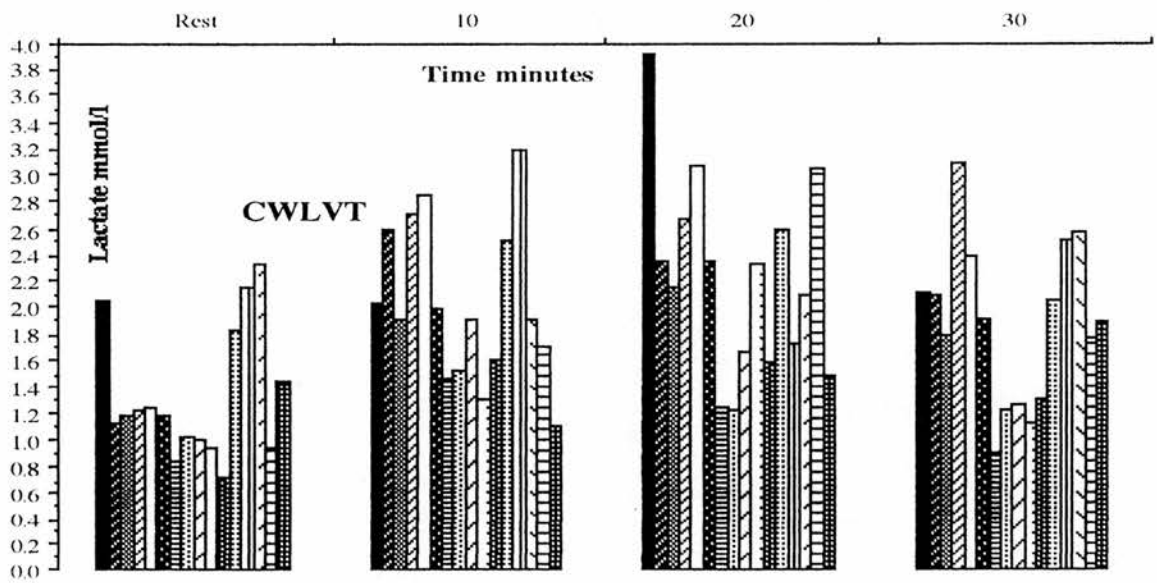

Figure13. Individual lactate curves during 30 minutes prolonged exercise test at VT.

Mean lactate concentration was not significantly different between 10, 20 and 30 minutes time points, although at the 20th minute mean increase in lactate resulted in an elevation of 9% above the value at 10th minute. Thereafter lactate concentration was followed by a non significant decrease of 15.5% by the end of the exercise period. Individual lactate values did not show consistency for the time points 10, 20, and 30 minutes. Individual plasma lactate concentrations at the end of the exercise period ranged between 0.9-3.1 mmol/l with a mean of 1.87±0.6mmol/l which was significantly greater than the resting value mean of 1.33±0.5 (range 0.73-2.34).

4.07: HEART RATE RESPONSE

The time course of the heart rate response at the onset of exercise is exponential. HR increases from its resting value rapidly, reaching after 10 minutes mean values of 159±11.2 bpm and 169±10.5 bpm for CWLVT and CWLVd respectively (figure 14). Beyond 10 minutes the increase in HR was less pronounced rising to a mean at the 30th minute of exercise of 161±12 bpm and 174.6±12 bpm for CWLVT and CWLVd. At each measurement time point beyond rest CWLVd HR was significantly higher when compared to the corresponding value of CWLVT.

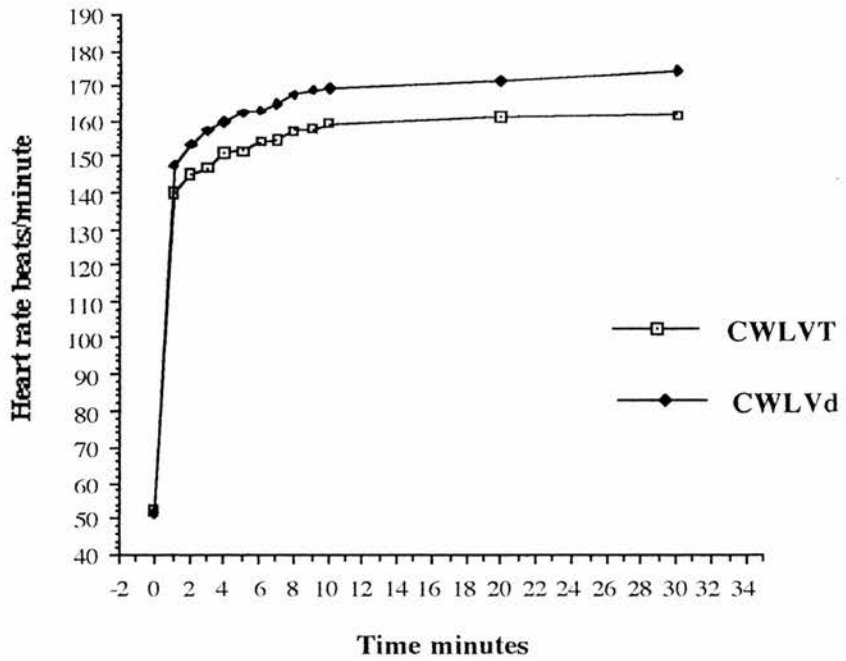

Figure 14. The effect of a 30 minutes run on HR (mean values, n=16) at a constant speed relative to VT and Vd.

4.08: VENTILATORY RESPONSE

Tables 7, presents the mean, and standard deviation of each of the measured variables at CWLVT and CWLVd. The ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$), ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$), VEatps, R, ventilatory frequency (VF), and $\dot{V}O_2$l/min values were significantly higher for the CWLVd when compared to the CWLVT ($p<0.05$) at all time points (i.e. 10, 20, and 30 minutes).

Mean $\dot{V}O_2$ values measured at the 10^{th} minute were significantly lower ($p<0.05$) from the values measured at the 20^{th} minute which was not different from the values measured at 30^{th} minute for both prolonged exercise tests (figure 15).

R values for the time points 10, 20 and 30 minutes followed a significant linear decrease throughout the constant load exercise test relative to VT. During CWLVd R values measured at 20th and 30th minute were not significantly different, although they were significantly lower than the one measured at the 10th minute (figure 16).

During prolonged exercise at VT, $\dot{V}E$ values measured at 10, 20, and 30 minute were not significantly different, although values at the 20th and 30th minute were slightly higher than the values at 10th minute (figure 17). In the CWLVd exercise test mean $\dot{V}E$ values at 10, 20, and 30 minute increased linearly, however only the 10th minute figure was significantly lower compared to the one measured at 30th minute. This phenomenon corresponds to the "ventilatory drift" observed during prolonged and intense exercise.

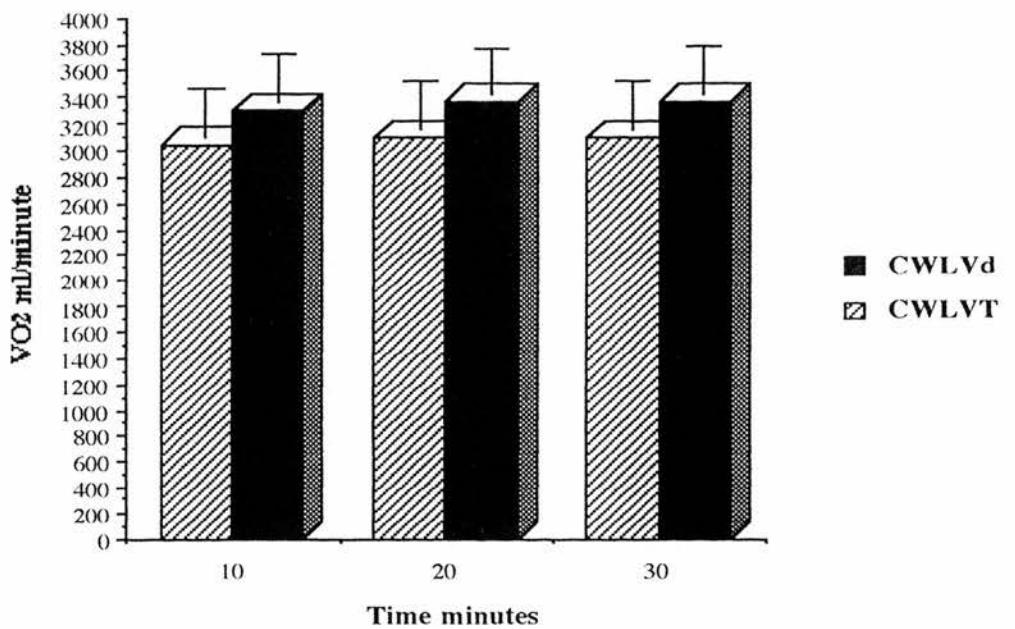

Figure 15. Mean(±SD), $\dot{V}O_2$ values for the time points 10, 20, and 30 minutes at VT and Vd constant work load exercise.

Table 7. Ventilatory changes during constant load exercise at VT, and Vd. Values are expressed as means±SD (n=16).

CWLVT							CWLVd					
Time	$\dot{V}E^+$	R^+	VF^+	$\dot{V}E/\dot{V}O_2{}^+$	$\dot{V}E/\dot{V}CO_2{}^+$	$\dot{V}O_2{}^+$	$\dot{V}E$	R	VF	$\dot{V}E/\dot{V}O_2$	$\dot{V}E/\dot{V}CO_2$	$\dot{V}O_2$
min.	l/min		breaths/min		l/min	l/min			breaths/min			l/min
10	72.3±9	0.91±0.05	38.6±6.5	20.4±1.8	22.4±2	3.04±0.35	83.7±12	0.93±0.05	41.3±7	21.7±2.5	23.2±2.9	3.30±0.35
20	73.7±10	0.89±0.05	40.2±5.8	20.4±1.7	22.8±1.7	3.1±0.387	85.2±13	0.91±0.04	43.3±6	21.6±2.1	23.6±2.1	3.36±0.38
30	73.1±9	0.87±0.06	40.8±6.7	20.2±1.8	22.9±1.7	3.1±0.366	88±14	0.92±0.05	46±6.7	22±2.2	24±2	3.37±0.36

$^+$ Values are different from the corresponding values of CWLVd ($p<0.05$)

] Significant differences within measures (columns) with ($p<0.05$,)

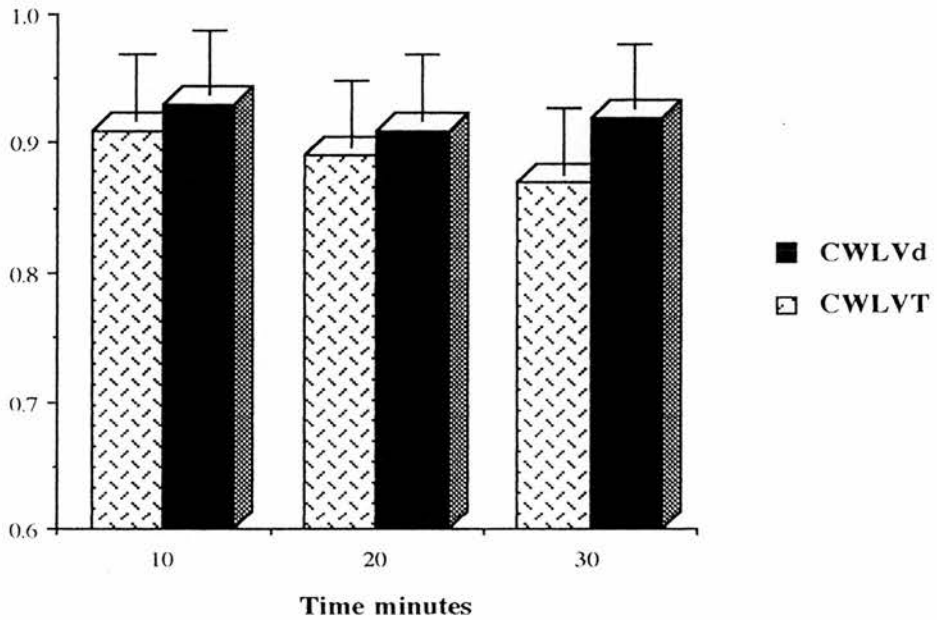

Figure 16. Mean (±SD) respiratory exchange ratio, values during prolonged exercise tests relative to VT, and Vd.

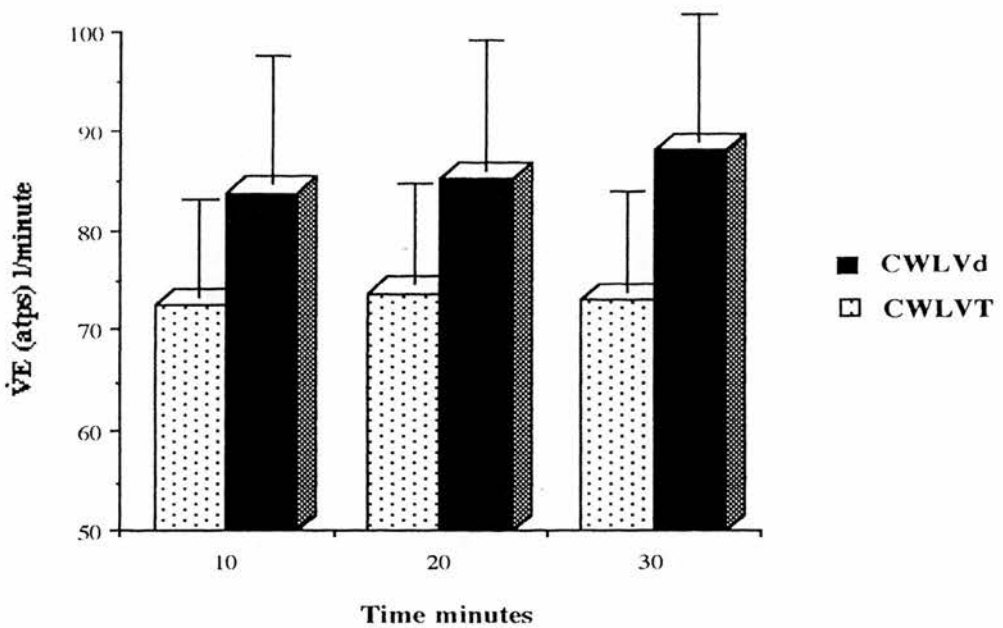

Figure 17. Volume of expired air for the time points 10, 20 and 30 minutes during constant load exercise relative to VT, and Vd (mean±SD).

VF increased significantly by 11% from 10th to 30th minute of exercise at CWLVd. VF values were significantly different from each other for the time points 10, 20 and 30 minutes at CWLVd, the value at the 20th minute being 5% greater and 6% lower than the one measured at 10 and 30 minutes respectively (figure 18).

$\dot{V}E/\dot{V}O_2$ values were not different within measured time points at the CWLVT and CWLVd exercise tests. However, CWLVd values were greater than the corresponding CWLVT values, suggesting that $\dot{V}E$ increases are not analogous to $\dot{V}O_2$ (figure 19).

$\dot{V}E/\dot{V}CO_2$ (figure 20), showed a similar response to the $\dot{V}E/\dot{V}O_2$. Mean $\dot{V}E/\dot{V}CO_2$ values at CWLVd were also significantly greater that those at CWLVT, suggesting that the $\dot{V}E$ increase is not analogous to $\dot{V}CO_2$, therefore, another factor may be responsible for regulating the respiratory centre under CWL exercise conditions.

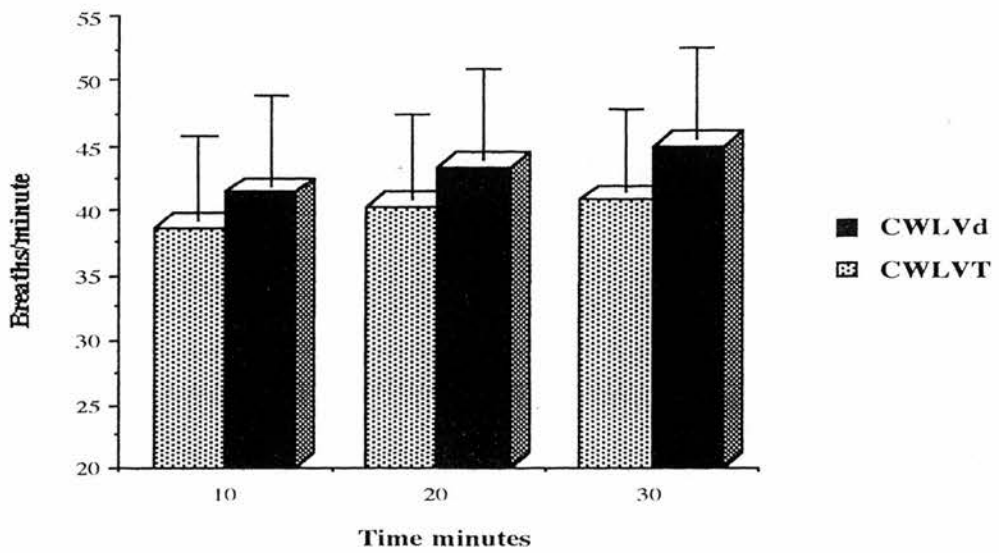

Figure 18. Mean(±SD) values for ventilatory frequency (VF) at CWLVT and CWLVd exercise.

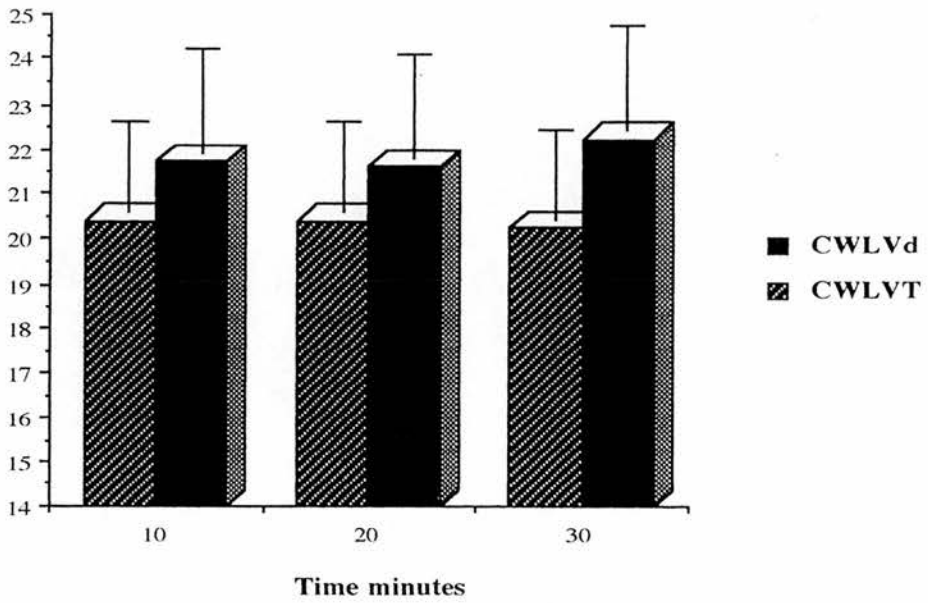

Figure 19. Ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$) during constant load exercise relative to VT and Vd values are means±SD.

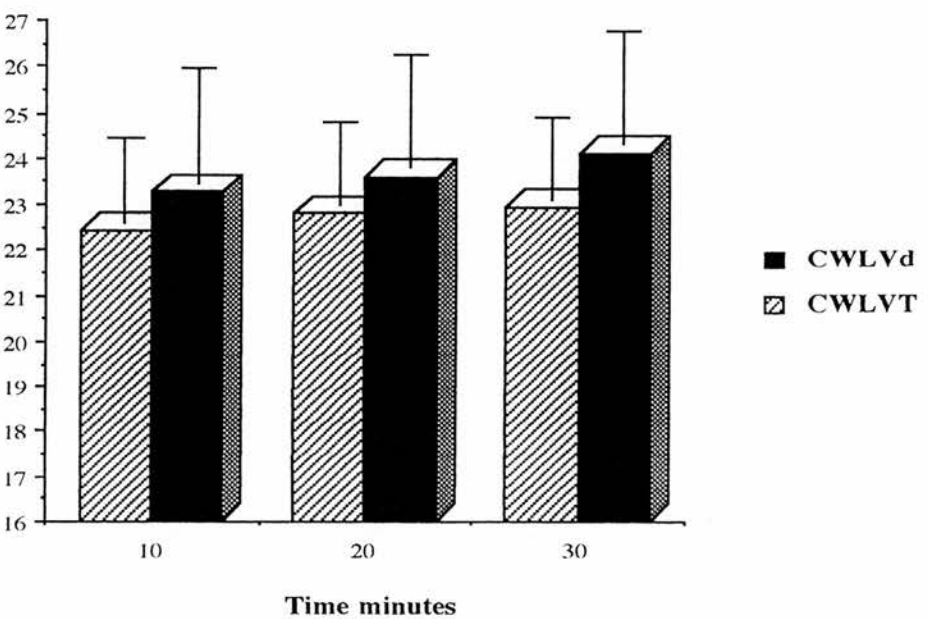

Figure 20. Ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$) response to CWL exercise relative to VT, and Vd for the time points 10, 20, and 30 minutes (mean±SD)

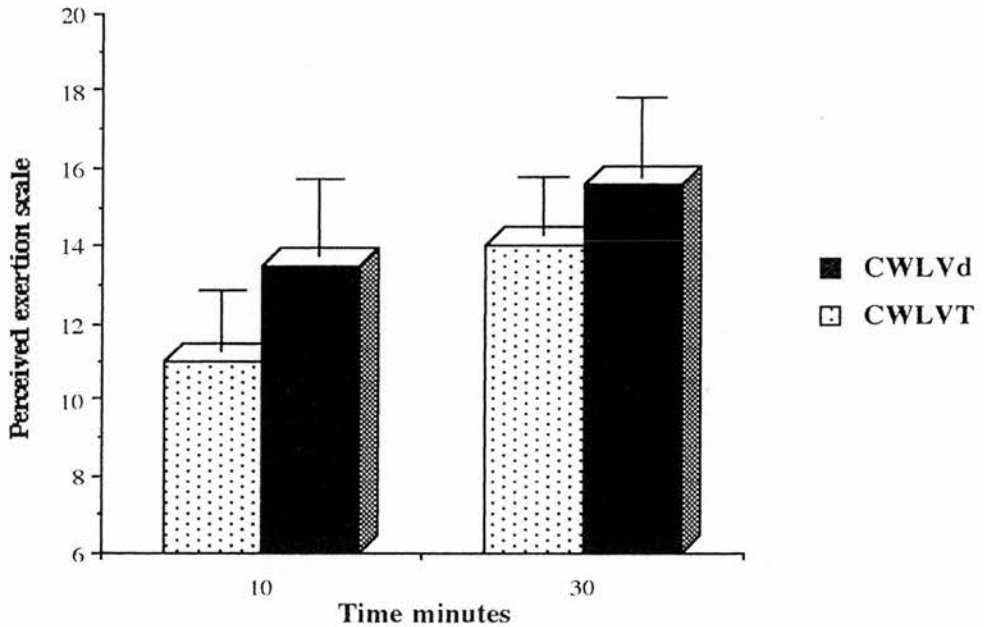

Figure 21. Perceived exertion rating for the time points 10 and 30 minutes for both constant load trials. Values are means±SD.

4.09: PERCEIVED EXERTION RATING

The mean Borg scale rating of perceived exertion increased from 11±1.6 and 14±1.2 at 10 minutes to 13.5±2.1 and 15.6±1.8 at 30 minutes of prolonged exercise relative to VT and Vd respectively (figure 21). All subjects stated that they would have been able to continue running if requested.

CHAPTER 5: DISCUSSION

5.01: TEST-RETEST RESULTS

5.02: CONSTANT WORKLOAD RESULTS

5.03: CONCLUSIONS

5.04: RECOMMENDATIONS FOR FURTHER STUDY

CHAPTER 5: DISCUSSION

5.01: TEST-RETEST RESULTS

VT reproducibility

The test-retest reliability of VT expressed as $\dot{V}O_2$, $\%\dot{V}O_2max$, HR, %HRmax and TV produced correlation coefficients of: 0.86, 0.75, 0.94, 0.86 and 0.95 respectively in this study. Other studies have examined the reliability of VT. A test-retest correlation coefficient of 0.72 for VT expressed as $\dot{V}O_2$l/min has been reported by Davies et al., (1976). The use of a different procedure to estimate VT (they were averaging $\dot{V}E$, $\dot{V}CO_2$, FEO_2 and RER points for the final VT value for the comparison), exercise modality and the training status of the subjects may largely explain the discrepancy between the studies. Findings reported in another study by Davis et al., (1979) showed a 0.95 and 0.91 correlation coefficient for the non-invasive OMA determination expressed as $\dot{V}O_2$ and $\%\dot{V}O_2max$ respectively. This study is compatible with the present study considering the criteria which have been used for the VT determination. However, the cycle ergometer and the sedentary middle aged subjects used in that study may contribute to the results' minor divergence. A test-retest reliability coefficient of 0.93 for VT, expressed as $\dot{V}O_2$, was reported by Caiozzo et al., (1982) by applying the same criteria as in the present study to detect VT. Finally, Aunola et al., (1984), with army personnel and judo practitioners using a cycle ergometer and the $\dot{V}E$ and $\dot{V}CO_2$ gas exchange variables to estimate VT expressed as $\dot{V}O_2$l/min, reported a 0.91 test-retest reliability.

Before drawing conclusions from comparing directly these studies on the

basis of absolute numerical values, several factors must be considered. The main factors reported to influence VT determination are: a) investigators subjectivity when examine plots of $\dot{V}E$, $\dot{V}CO_2$, $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$, b) exercise modality and c) the different training levels of the subjects. The cumulative effect of these factors may explain the differences of 5-10% between the independent studies reported here and it seems appropriate to conclude that the high test-retest correlations reported in the literature while VT is expressed as $\dot{V}O_2$l/min are confirmed in this study.

Surprisingly, reports in the literature regarding VT reproducibility with runners are scarce. Furthermore, the existing reports on this topic usually express VT as $\dot{V}O_2$ or $\%\dot{V}O_2$max. Most researchers have based their research on the results of work by Davis et al., (1983) and Davies, (1985), whose proposition was that VT was best measured in terms of the $\dot{V}O_2$ and not the workload or HR. It is not clear though why VT was expressed as $\dot{V}O_2$ and not as either the work rate which induces the particular $\dot{V}O_2$, or the HR needed by this exercise intensity to provide oxygen delivery. There have not been reports in the literature of VT values greater than $80\%\dot{V}O_2$max even with elite runners (Prietto et al., 1981). Since HR, $\dot{V}O_2$ and the work rate are linearly related for exercise intensities up to $85\%\dot{V}O_2$max (Davies, 1968; Wyndham, 1959) it seems safe to express VT as HR, %HRmax or work rate.

TV at VT showed the highest test-retest reproducibility in this study (r=0.95), followed by HR (r=0.94). VT expressed as $\dot{V}O_2$ showed the lowest test-retest correlation coefficient compared to the other absolute values. This may be explained by the day-to-day variations in running economy due to biological fluctuation (McConnell, 1988) and either the over- or under-estimation of the actual $\dot{V}O_2$ at VT caused by the drift between calibrations of the MMC analyzers (Wilmore et al., 1976). Indeed, the combined (technological and biological) intra-individual

day-to-day variation in a well-performed direct measurement of submaximal and maximal oxygen intake is between 4 and 6% (Boileau et al., 1977; Cunningham et al., 1977; Taylor et al., 1955). The technical problems account for at least as much as 10% of this variance, and problems with gas analysis can give rise to large and unsuspected systematic errors (Cotes and Woolmer, 1962), even for experienced personnel.

When the VT data were transformed to $\%\dot{V}O_2max$ and %HRmax, test-retest reliability between these same variables dropped to 0.75 and 0.86 respectively. It appears then that the transformation of an absolute VT value to a percentage increases (to varying degrees) the homogeneity of the data and thereby produces lower correlation coefficients. This is in agreement with the results obtained by Caiozzo et al., (1982) where the test-retest correlation coefficient of 0.94 (VT as $\dot{V}O_2$) was lowered to 0.91 when transformed to $\%\dot{V}O_2max$.

Mean HR value at VT during re-test was significantly lower compared with the mean value obtained during the first test. The explanation of this reduction may be the combined effect of the following factors: 1) the biological day-to-day variation in HR response to exercise, 2) the subjects were more accustomed to the testing during re-test, 3) the subjects were well aware of the experimental procedure and the anticipation of a less demanding physical exertion (submaximal test) may well have resulted in the release of lower quantities of catecholamines in the blood. These hormones are known to accelerate heart rate. Therefore, the extrinsic sympathetic influence on HR may have been less pronounced throughout the re-test (Grimby, 1966; McArdle, Katch and Katch, 1981). This phenomenon was also evident in the mean HR value at Vd during test and re-test. However, in that case the mean difference was only 3 bpm, resulting in a non-significant decrease, compared with the 6 bpm mean test-retest HR difference at VT.

Mean test-retest %HRmax at VT was 83.8 with a standard deviation of 3.1% and a 77-88.5% range. In fact, only one subject showed %HRmax at VT below 80%. He was the least trained at the time of the measurement, recovering from a long injury. This alone may explain his low value. During work at 80%HRmax three subjects were working at VT and 16 of them remained below VT. Between 80 and 85%HRmax 8 subjects were working at VT and for the remaining 8 subjects VT was measured between 85 and 88,5%HRmax. The national team members were all using more than 82%HRmax while working at VT, but it was not observed that they were grouping around 88%HRmax at VT (the highest observed in this study). Less successful but highly trained runners were also using higher percentages of their HRmax and showed the same variability regarding this measure, suggesting that the %HRmax alone at VT may be an indicator of a highly trained runner but can not distinguish the elite competitor from the good club runner.

The present study is in general agreement with the study of Dwyer and Bybee, (1983) as far as %HRmax at VT is concerned. However, the greater variability reported in that study regarding %HRmax at VT (70-90%HRmax) may be explained by the difference in training level (untrained to well trained), exercise modality and sex of the subjects.

In terms of $\%\dot{V}O_2max$ at VT in the present study, the mean±SD test-retest value was 73±4.3 with a range of 66-81.8$\%\dot{V}O_2max$. The high $\%\dot{V}O_2max$ values observed were expected as other studies using well trained or elite runners reported similar values (Prietto et al., 1981; Whithers et al., 1981; Peronnet et al., 1987). This is thought to be the result of specific training (Davis et al., 1979; Ready and Quinney, 1982). The zone of non-uniform work stress expressed as $\%\dot{V}O_2max$ appeared to cover a slightly greater range (15.7) compared with %HRmax (11.5). Seven subjects were using 66-71$\%\dot{V}O_2max$ at VT with the rest (13) encountering a non-uniform physiological response between 71 and 81$\%\dot{V}O_2max$. This observation suggests

that an exercise intensity selected from the literature and applied to these well trained runners may have limited usefulness in ensuring that a desired level of metabolic stress is attained in all runners. Training programmes using prescriptions based on $\%\dot{V}O_2max$ or %HRmax without consideration of individual values at VT create among the participants multiple training stimuli that, in turn, result in a wide range of improvement in cardiovascular and metabolic functions (American College of Sports Medicine, 1978; Pollock, 1973; Dwyer and Bybee, 1983). The low correlation between %HRmax and $\%\dot{V}O_2max$ at VT (r=0.67) precludes the translation of VT, expressed as $\%\dot{V}O_2max$ to a %HRmax or absolute HR figure for training prescription and/or activity regulation. This is of limited concern because when a peak $\dot{V}O_2$ test is carried out, VT assessment and the HR value at VT are usually included among the measures. Individual assessment of VT is essential for the runner to regulate the extent of the physiological stress encountered with the large amount of daily training.

Vd reproducibility

Vd did not show the same clarity and reproducibility as VT. The heart rate response to incremental exercise for one subject was linear throughout both tests. Three subjects did not present Vd in at least one of the tests. Reports pertinent with this topic appear to be divided in the literature. Conconi et al., (1982); the first to identify Vd, Drochetti et al., (1985); Maffuli et al., (1987); Bretoni et al., (1989); Goodman et al., (1986); Borsetto et al., (1989); Ballarin et al., (1989) and Kispert et al., (1987), have not reported cases of not presenting Vd in a large variety of sport activities. On the other hand, Tokmakides et al., (1987); Francis et al., (1989);

Kuipers et al., (1988); and Ribeiro et al., (1985), with elite runners, well trained runners, heterogeneous group and untrained cyclists respectively, were not able to show Vd in all of their subjects. The later studies failed to thoroughly validate Vd. In fact, in the study by Tokmakides et al., (1987) Vd was rated impossible to identify in 9% of the cases and doubtful in 36% of the cases. Francis et al., (1989) in the laboratory failed to show Vd in all of their subjects (n=9). Kuipers et al., (1988) and Ribeiro et al., (1985) reported 37 and 50% absence of Vd during testing. Difficulties in determining Vd were also pointed out by Lacour et al., (1987). In the present study, out of 40 tests only in five tests (12.5%) was it not possible to detect Vd. The remaining 35 tests presented a clear HR deflection point near maximal effort. This is in close agreement with the study by Tokmakides et al., (1987) who also used well trained runners. However, direct comparison of this study with those others which failed to show 100% identification and reproducibility of Vd can not be done due to different methodological approaches. Bunc et al., (1988) describe the conditions which must be fulfilled for Vd determination as follow: (I) the first intensity of exercise must be below approximately 50%$\dot{V}O_2$max; (II) the differences in intensity between steps must be approximately 5-10%$\dot{V}O_2$max. Indeed, by looking at the results of the incremental tests of those subjects which did not present Vd these conditions were fulfilled. The study by Kuipers et al., (1988) can probably be used for direct comparison with the present study as they used the same testing procedure in the laboratory for Vd determination. The small number of subjects which they used (n=9) may largely explain the difference (37 to 12.5%) in failure rates to identify Vd.

Several reasons may be responsible for the discrepancies between studies. The day-to-day variability in the heart rate response to exercise has been said to be one of them. Wyndham et al., (1959) the first physiologists to describe the asymptote in

the heart frequency near maximal effort, emphasized that it is important to be clear on a basic assumption made in fitting appropriate regression lines to the experimental data. The assumption is that, given a rate of work, repeated observations of heart rate will result in a series of values of this measurement which will scatter about some "true" mean value. The scatter is due to random errors of measurement, physiological variation and the like. In view of this limitation, their study was designed to measure heart rate on at least five separate occasions at each of a number of rates of work up to and above the maximum level of oxygen intake, to obtain the "true" mean HR response to exercise. Furthermore another report by Conconi, (1983) mentioned the need of repeated measures to obtain the true HR response to incremental exercise. If this is true, then the practicality of Vd is questioned. In fact, this limitation exists also when gas exchange indices are used to determine OMA. Davies et al., (1976) and Davies, (1985) stated that repeated tests are sometimes necessary in order to secure an accurate estimation of the parameter using the non-invasive gas exchange technique.

Another reason for the non-appearance of the Vd in some subjects may be the nature of the Conconi protocol. The studies which failed to identify Vd in every subject, including the present study, did not use the original Conconi protocol. This protocol increases the speed by 0.4-0.5 km/h every stage while keeping the distance constant (200m) and therefore the running time of each stage continuously decreases. Subsequent protocols which have been used in the laboratories usually keep the exercise time per stage constant (i.e. 1 minute) and increase the work rate by either 10-25 watts (cycle ergometer) or 0.5 km/h (treadmill). By using Conconi's protocol the curvilinear exponential function of speed in such a protocol enables higher speeds and permits more frequently the occurrence of a heart rate plateau as a function of the speed. Using the protocols modified arbitrarily for laboratory

testing, subjects have more time per stage to increase HR and lose the possible deflection point, since this may occur under different conditions if the increases of the treadmill speed were followed by the analogous concurrent decrease of the exercising time per stage.

Whether this phenomenon of HR described by Conconi et al., (1982) interrelates with the onset of rapid metabolic changes is unclear, nor has a physiological explanation been proposed from these researchers to justify it. Moreover, the method which Conconi et al., (1982) were using to validate Vd as an objective method for OMA determination, by comparing it with LT, has been heavily criticised. Since the LT was determined as the crossing point of two linear components corresponding to the three lower and three upper speeds, a mathematical bias ensures high correlations that could not be obtained using an "independent" and traditional protocol (Tokmakides et al., 1987).

Considering all the aforementioned limitations the low test-retest reproducibility of Vd expressed as: $\dot{V}O_2$ (0.52), $\%\dot{V}O_2max$ (0.07), HR (0.75), %HRmax (0.29) and TV (0.69), and even Vd non-appearance in some instances, may well be explained. However, even with the restrictions of this study Vd was identified in most cases and its reproducibility expressed either as HR or TV appears to be acceptable. Therefore, the determination of this variable, either in the field where it can be identified using the protocol proposed by Conconi et al. (1982) or in the laboratory using a modified protocol, may be included in the athletes' test results. Furthermore, Vd application to regulate training and racing pace in runners make the repeated tests worth trying.

VT and Vd comparison

VT values expressed as $\dot{V}O_2$, HR and TV were systematically lower than the corresponding Vd values. The results obtained in a study conducted by Bunc et al., (1988) with well trained middle and long distance runners showed that VT coincides with Vd. The conflicting results between these studies can only be explained by the different methods used to estimate the thresholds under examination. Bunc et al., (1988) were not accepting as VT the workload before systematic increase of $\dot{V}E/\dot{V}O_2$. Instead, they plotted $\dot{V}E$ versus $\dot{V}O_2$. VT was estimated as the intercept of two best fit lines and it was always between workloads, resulting every time in overestimation of VT. The estimation of Vd also followed different methodological approaches resulting in underestimation of the Vd according to the criteria used in the present study. These two factors contribute mainly to the differences between these studies. However, examining the results of this study and allowing for over- and under-estimations, VT expressed as HR, $\dot{V}O_2$ and TV was still slightly lower than the corresponding values at Vd. Another study by Baraldi et al., (1987) also compared VT and Vd in runners. They reported only a correlation of 0.80 between measures. Due to poor description of the methodological approaches used in their study interpretation of the results is difficult. Goodman et al., (1986) with trained cyclist also reported similar results. $\%\dot{V}O_2max$ and HR at VT were found to be significantly lower than the values at Vd. HR at VT correlated with HR at Vd (r=0.71) indicating a moderate degree of relationship. The corresponding correlation in the present study was 0.82 and 0.78 for the first and second test respectively, suggesting that the exercise modality may affect this measure. Ribeiro et al., (1985), despite failing to detect Vd in 50% of the subjects, found a coincidence of the rest of the subjects' Vd with the point where $FECO_2$ began to decrease. This is referred to as VT_2.

Finally, in the present study VT variables were lower than those at Vd yet they were correlated. Much higher relationships were found when VT and Vd were expressed as TV rather as $\dot{V}O_2$. This may be explained by the difference in running economy between subjects where running economy is defined as the $\dot{V}O_2$ cost of running at a fixed intensity (Cavanagh and Kram, 1982).

In summary, the present study following the traditional method for VT determination and a modified treadmill protocol for Vd detection failed to support concordance of these measures. If VT coincides with the OMA as is proposed by other studies, then Vd occurs above OMA and may induce higher physiological stress when employed for training purpose. Discussion of the results and the physiological consequences of the CWL trials at VT and Vd will follow.

5.02: CONSTANT WORKLOAD RESULTS

Lactate kinetics during CWLVT and CWLVd

Blood lactate increased for both CWL trials measured at the 10th minute compared with the resting value. The magnitude of the rise in lactate by the 10th minute of exercise was greater during CWLVd. If one accepts that increases in lactate during exercise reflects the rate of glycolytic ATP phosphorylation then the energy contribution from anaerobic sources during the initial adjustments to sudden energy demands of exercise were greater during CWLVd. The metabolic clearance of lactate is well known to increase during exercise (Hermansen and Stensvold,

1972). This was evident during both exercise intensities. After the initial increase in the rate of lactate accumulation, when the TCA cycle adapted to the increased energy demands of the exercise and started to support almost exclusively the ATP needed to keep the runner going, the rate of lactate accumulation was decreasing for the time points 20 and 30 minutes. More frequent data sampling may have revealed this decrease commencing sooner than 20 minutes. By the 30th minute at CWLVd energy contribution via anaerobic glycolysis was probably insignificant as the rate of blood lactate accumulation was back to the resting value. Findings by Brooks et al., (1984) using radiotracer techniques in rats demonstrated that even though blood lactate levels and lactate turnover rates were elevated, the anaerobic energy production contributed insignificantly to the total energy demands. Margaria et al., (1964) suggested that anaerobic glycolysis contributes significantly to the energy release systems only at exercise intensities above peak $\dot{V}O_2$. The results of the present study support indirectly this theory.

During CWLVT energy release by the oxidative pathway was adapted earlier to the energy demands of the workload clearing blood lactate at a faster rate. This is indicative of the greater physiological stress induced by the CWLVd exercise. Aro et al., (1988), with well trained runners, showed that Vd is the highest exercise intensity that could be sustained for 30 minutes while maintaining a constant blood lactate concentration (MSSLA-maximum steady state lactate). This may explain why mean lactate values measured at 20 and 30 minutes during CWLVd remained unchanged in the present study. If this is the case with Vd then its applicability as far as physical conditioning in runners is concerned may be enormous. It is well established that exercise which results in a continuous increasing blood lactate concentration can not be sustained for long period of time. Therefore, knowledge of the individual maximum lactate steady state pace becomes more important the

longer a certain workload has to be sustained. This is especially the case in middle and long distance events. Indeed, MSSLA has been shown to correlate highly with long distance running performance (Haverty et al., 1988). During both CWL trials individual steady state lactate concentrations ranged from 1.5-8 mmol/l (Table 8), a finding which is consistent with the observation of Stegmann and Kindermann, (1982).

Table 8. Individual plasma lactate concentrations for subject B and D during CWLVT and CWLVd.

TIME minutes	CWLVT lactate mmol/l		CWLVd lactate mmol/l	
Rest	1.11^{+}	1.23*	1.44^{+}	1.14*
10	3.95^{+}	2.70*	5.95^{+}	4.76*
20	5.55^{+}	2.67*	8.01^{+}	5.53*
30	4.36^{+}	2.85*	6.20^{+}	5.13*

$^{+}$Subject B, *subject D.

Ventilatory responses during CWLVT and CWLVd

A slow rise in $\dot{V}O_2$ (2%) during both CWL trials measured at the time points 10 and 20 minutes was evident in this study. The magnitude of $\dot{V}O_2$ rise is similar to those reported by Nagle et al., (1970) for treadmill running at 67-74 and 74-79%$\dot{V}O_2$max. Surprisingly, these investigators also did not report significant $\dot{V}O_2$ alterations after the 25th minute of exercise. This slow rise in $\dot{V}O_2$ must be caused by changes occurring after the 10th minute of exercise and can not be caused directly by the workload itself as the $\dot{V}O_2$ has been already adjusted to the exercise demands (Whipp and Wasserman, 1972). The cause of the rise in $\dot{V}O_2$ is unknown.

Henry, (1951) and Volkov et al., (1969) both theorized that it was due to an oxidative removal of lactate from blood. Data from Hagberg et al., (1978) indicate that the slow increase in $\dot{V}O_2$ is primarily a temperature effect, both directly as evident by the T_c (core temperature) effect, and indirectly, by causing a slight hyperventilation which increases the $\dot{V}O_2$ of the respiratory muscles. The present study supports this second theory as the magnitude of $\dot{V}O_2$ rise in both CWL trials was the same and the lactate concentration was far different. In view of the extra work demanded from the respiratory muscles to dissipate elevated body heat, the slow rise in heart rate may seem reasonable. Indeed, the elevation in HR was more pronounced during CWLVd, possibly due to the greater hyperventilation.

Considering the aforementioned speculations it seems reasonable that all subjects had greater $\dot{V}O_2$ and HR values by the 30th minute of exercise at CWL intensities compared with those obtained during incremental tests. This would suggest that the HR value which is representative of a threshold phenomenon, if it has been derived from an incremental test and is being used as a guide regulating prolonged running training exercise intensity, should be expected to be exceeded by the end of the exercise.

Wasserman, (1978) and James et al., (1989) showed that when exercise induces metabolic acidosis (i.e. above VT) the predominant cause of increased ventilation is an increase in ventilatory frequency. Data from the present study are consistent with this statement as during CWLVT, $\dot{V}E$ and VF remained fairly unchanged whereas the increases in $\dot{V}E$ during CWLVd were followed by increases in VF, suggesting that exercise was performed above OMA. Increases in $\dot{V}E$ throughout the exercising period at CWL intensities greater than 80%$\dot{V}O_2$max have been observed in other studies (Hagberg et al., 1978; Scheen et al., 1981; Nagle et al., 1970). The cause of this increased ventilation that occurs after the initial minutes of CWL exercise is unknown. The ventilation is increased beyond that required to

maintain arterial PCO_2 homeostasis (Hagberg et al., 1978) resulting in an arterial hypocapnia. One ventilatory control factor often implicated in mediating this hypocapnic ventilation is an increased temperature. Dempsey and Reddan, (1976) have demonstrated that abolishing the rise in core temperature during exercise eliminated the hypocapnic hyperventilation. In view of this, it is reasonable to suggest that the core temperature was greater during CWLVd as $\dot{V}E$ continuously increased compared to CWLVT where $\dot{V}E$ remained unchanged throughout the exercise. This is an indirect indication that these exercise intensities may differ in terms of the thermal strain imposed on the heat dissipatory mechanisms. Since an excessive rise in body temperature caused by the combined effect of a warm environment and the internal body metabolic heat production is a limiting factor to distance running performance (Lloyd, 1966), care may have to be taken by the runner to avoid dehydration and heat stroke when training or racing at a pace relative to Vd in a warm environment.

R measured at the lungs when hyperventilation occurs can not be indicative of respiratory quotient (RQ) at the muscles. However the change in substrate utilisation at CWLVT would have been evident from the change in R in this study as hyperventilation did not occur. R values measured at 20^{th} and 30^{th} minute of exercise were decreasing, showing a shift from carbohydrate to fat energy substrate. However, at CWLVd changes in substrate utilisation may have been evident if a progressive hyperventilation had not occurred. The disproportional increase of $\dot{V}CO_2$ (hypocapnia) versus $\dot{V}O_2$ due to hyperventilation may explain why R values measured at 30^{th} minute was greater than the 20^{th} minute values.

In an incremental exercise test the hyperventilation that occurs above VT is thought to be indicative (among other factors) of the excess $\dot{V}CO_2$ derived from the buffering of H^+ by the bicarbonate system and not of the $\dot{V}O_2$ (Newsholme and Leech, 1984; Wasserman et al., 1973). Considering the VT and Vd prolonged

exercise intensities in this study it seems reasonable that $\dot{V}E/\dot{V}O_2$ values should be greater at CWLVd. Ribeiro et al.,(1986) reported similar results for compatible exercise intensities.

$\dot{V}E/\dot{V}CO_2$ also presented greater values at CWLVd, contrary to the expectation that this measure may present equal values at CWLVT and CWLVd as it is thought that $\dot{V}E$ increases are analogous to $\dot{V}CO_2$. This may be because isocapnic buffering which occurs during an incremental test (Davies, 1985) is not evident during CWL exercise. At the 10, 20 and 30 minute time points excess CO_2 may have been already cleared. PCO_2 was not measured in this study and the blood lactate concentration may not be an accurate indicator of total H^+ accumulation in the muscles and blood (Walsh and Banister, 1988).

The Vd may represent the highest workload that can be reached where there is a balance between lactate elimination and production. Above this point, lactate may not be removed as fast as it is built up in the blood causing a metabolic lactic acidosis. The lactate acidosis produced and the inability to compensate for it by respiratory alkalosis may affect the ability to perform at a given intensity. This acidosis may result in the runner needing to slow down when this occurs and may be a limiting factor to endurance performance. Lactate and/or H^+ build up may be the result of diffusion hindrance, a block in lactate in the muscles, or by an increase in production (Brooks, 1985).

As it is confirmed from other studies that VT and Vd are closely related with performance it would be to a runners advantage to meet these measures at higher running velocities. An increase in either VT or Vd may be induced by increased muscle respiratory capacity through increased oxidative enzymes and the size and number of mitochondria (MacDougal and Sale, 1981). They might also increase with

increased blood flow to the muscles, increased lactate and/or H^+ clearance, or recruitment of more slow twitch muscle fibers. In order to stimulate these processes, training programs based on VT and Vd may be used. However, according to the theory that training prescriptions based on exercise intensities which induce different metabolic and respiratory responses will result in different training stimuli, varied improvements in cardiovascular and metabolic functions may occur (American College of Sports Medicine, 1978; Pollock, 1973). Therefore, running training intensities relative to Vd and VT are expected to produce different training adaptations. The practical question facing the coach and the athlete, however, is which training intensity is most effective in bringing about the adaptations desired? There appears to be no simple answer. The peculiarities of each event and the individual responses to training must always be considered among other factors.

Theoretically, the most effective form of training for stressing the runner's central oxygen transport system and for stimulating adaptive changes in the heart musculature would be continuous running involving as large a muscle mass as possible for long period of time, usually greater than an hour (MacDougal and Sale, 1981). It has been suggested that the exact intensity will depend upon the level of the runner's VT (MacDougal, 1977). There is also evidence (Conconi et al., 1983) to suggest that continuous running regimens of shorter duration, less than 45 minutes, improve performance to varied degrees. The results presented in this study indicate that running can be sustained for at least 30 minutes at an intensity (Vd) greater than VT. When continuous running intensity exceeds this threshold the accompanying acidosis will increase the subjective feeling of fatigue and eventually shorten the duration of the training session. There is no training study as yet to examine which of these exercise intensities contributes to better improvements in performance at varied distances.

5.03: CONCLUSIONS

Within the limitations of the present study the following conclusions were made from the results and observations.

I) Heart rate deflection point is usually but not always detectable when the experimental procedure used in this study is applied.

II) The HR, $\dot{V}O_2$ and TV at Vd were not equally reproducible as they were at VT.

III) The $\dot{V}O_2$, HR and TV at which VT occured were significantly lower than the corresponding values at Vd.

IV) The VT variables were correlated with those at Vd. TV showed the highest correlation among them.

V) Real differences exist in exercise-induced stress during CWL exercise relative to VT and Vd. In this regard greater values for Vd in blood lactate, $\dot{V}O_2$, HR, $\dot{V}E/\dot{V}O_2$, $\dot{V}E/\dot{V}CO_2$, $\dot{V}E$, VF, R and RPE have been observed.

VI) Training stimulus relative to VT and Vd may not induce the same physiological adaptations.

5.04: RECOMMENDATIONS FOR FURTHER STUDY

This research has indicated a number of issues which merit further consideration with regard to VT and Vd in well trained runners. Further experiments towards better understanding of the rationale behind HR response during incremental exercise tests and the appearance or not of the deflection point would be useful in determining the utility of this measure. Experiments designed to

produce more data points (eg. every minute) for lactate and gas exchange during CWL exercise relative to Vd and VT will help to draw more informative graphs and therefore better evaluate the kinetics of these variables. Effects of substrate availability, glycogen depletion, test duration and the mode of exercise would also provide useful information.

Study of these measures in other populations would also be a suitable area for future research. One other major area which should be researched is the physiological adaptations and the improvements in performance induced by employing training intensities relative to VT and Vd. This is a fascinating area of research as it has direct field applications.

Finally, more research involving the "onset of metabolic acidosis" concept using objective techniques is needed. This is a heavily researched area but the subjective determinations are open to criticism. As objective ways of determining lactate, gas exchange and heart rate responses are developed, together with the evolution of research techniques at the cellular level, evaluation of exercise tests by these measures can be made more reliable.

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