THE RELATIONSHIP BETWEEN EXCESS CO2 AND BLOOD LACTATE IN ELITE CYCLISTS

by

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ABSTRACT

This study examined the relationship between expired non-metabolic CO2 (EXCO2) and the accumulation of blood lactate, while emphasis was placed on the ventilatory (EXCO2 and VE/VO2) and lactate threshold relationship. Twenty-one elite cyclists (15 males, 6 females) performed a progressive intensity bicycle ergometer test (PIT) during which ventilatory parameters were monitored on-line at 15 second intervals, and blood lactate sampling occured on each minute. Threshold values were determined for each of the three indices: excess CO2 (EXTT), VE/VO2 (VVTT), and blood lactate (LATT). The three threshold values (EXTT, VVTT, LATT) all correlated significantly (P(0.001) when each was expressed as an absolute VO2 (l/min). A significant RM ANOVA (F=8.41, P(0.001) and post hoc correlated t-tests demonstrated significant diferences between the EXTT and LATT (P(0.001) and the EXTT and VVTT values (P(0.025).

The LATT occured at an average blood lactate concentration of 3.35 mmol/l, while the mean expired excess CO2 volume at the EXTT was 14.04 ml/kg/min. Over an 11 minute range across the threshold values (EXTT and LATT), which were used as relative points of reference, the expired EXCO2 volume (ml/kg/min) and blood lactate concentration (mmol/l) correlated significantly

(r=0.69, P(0.001). Higher individual correlations over the same period of time (r=0.82 - 0.96, P(0.001) stress the individual nature of this relationship. Expired EXCO2 volume appeared to track blood lactate levels over this 11 minute period when the significant threshold difference (1.35 min.) was taken into consideration. These results indicate a strong relationship between the three threshold values, although changes and expired EXCO2 precede changes in blood lactate concentration and the ventilatory equivelant (VE/VO2). Although changes in expired EXCO2 volume appear to track changes in blood lactate concentration, blood lactate concentration can not be accurately predicted from expired EXCO2 volume as the nature of this relationship varies between individuals and appears to be influenced by gender.

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CHAPTER ONE

INTRODUCTION AND STATEMENT OF THE PROBLEM

INTRODUCTION

As early as 1930 W.H. Owles recognized that there was a critical exercise intensity level above which the working muscles produced lactic acid. Owles also observed that accompanying an accumulation of blood lactate was an increase in CO2 excretion and ventilation. Harrison and Pilcher (1930) reasoned that the excess CO2 being produced was the result of bicarbonate buffering of acids being produced during anaerobic metabolism. Recent studies suggest that as much as 90% of the hydrogen ion produced during anaerobic metabolism is in fact immediately buffered by the bicarbonate system (Wasserman et.al, 1986).

In 1964 Wasserman and Whipp recognized that the onset of blood lactate accumulation correlated highly with break points in ventilation (VE), carbon dioxide excretion (VCO2), and the respiratory exchange ratio (R), parameters which could be monitored noninvasively. It was suggested that the initial onset of blood lactate accumulation reflected a shift from aerobic to anaerobic metabolism, a shift in the use of energy pathways, and that this point of transition could be accurately determined through the noninvasive measures investigated. It was at this time that they defined that point immediately preceding the non-linear increase in VE, VCO2, or the sudden increase in R as the "anaerobic threshold." This "threshold" point appears to

represent a critical intensity above which endurance performance is severely limited (Rhodes et al, 1981) and is postulated to represent a transition point in energy metabolism: a transition threshold.

Since its time of conception the "anaerobic threshold" (AT) has been put to many uses. In the past this 'transition threshold' has been used clinically in assessing exercise tolerance (Wasserman et. al, 1964), in exercise prescription (Davis et. al, 1981, Tanaka et. al, 1981), to characterize endurance athletes (Rusko et. al, 1980), and to predict endurance performance (Rhodes et al, 1981). The AT has been described as a key parameter which defines the ability to maintain high-intensity exercise (Whipp et. al, 1981), yet the concept of the AT is still extremely controversial. Although there does appear to be a "critical intensity" above which endurance performance is severely limited, and this intensity is concomitant with an increase in blood lactate accumulation, VCO2, EXCO2, VE, VE/VO2, and R, there is no conclusive evidence supporting the theory of the anaerobic threshold. It appears it is in the mechanisms of such increases, the nomenclature, and the original assumptions that the explanation for the AT is failing.

Many of the original ideas and assumptions of the AT are being challenged. It was originally surmised that the lactate threshold and ventilatory threshold were synonymous (Wasserman et al, 1964), and believed that only under hypoxic conditions do the tissues produce lactic acid (Margaria et al, 1933). The

appearance of lactate in the bloodstream was thought to reflect the production of lactic acid within the muscle cell, yet this idea is being challenged by many investigators who are finding lactate to be released under what they believe to be adequate tissue oxygenation (Connett et al, 1984, Jobsis et al, 1968), and finding that the lactate and ventilatory thresholds can be uncoupled (Segal et al, 1979). Other investigators believe that blood lactate levels do not accurately represent the cellular lactate production or concentration. Investigators are finding that a considerable portion of the lactate formed within the muscle may be oxidized within the active muscle tissue (Brooks, 1986), and that a translocation hinderance to the lactate molecule may be present (Stainsby, 1986). These findings suggest that the appearance of blood lactate may be delayed, not representing an increased reliance on anaerobic energy production or the cellular lactate concentration.

The relationship between blood lactate accumulation and EXCO2, or non-metabolic CO2 [VCO2 - (RQ x VO2)], has not yet been determined, although early research by Clode, Clark and Campbell (1961) and Issekutz and Rodahl (1961) reported high correlations between the lactate threshold and EXCO2. The earlier investigations suggest that EXCO2 permits changes in blood lactate concentration to be detected with reasonable accuracy. W.E. Hearst (1982, unpublished thesis) found that EXCO2 correlated highly with blood lactate at four specific running speeds (r=0.89). It has been postulated that expired EXCO2 volume may reflect intracellular production of lactic

acid. Recent studies suggest that as much as 90 - 94% of the lactic acid produced is immediately buffered by the bicarbonate buffering system (Wasserman et al, 1986). EXCO2 is then produced during periods when the rate of lactic acid is increasing, such as in a progressive intensity test. This study will examine the relationship between EXCO2 and blood lactate at all points during a progressive intensity test while examining the relationship between the EXCO2, blood lactate, and the VE/VO2 thresholds, their equality or difference, and the consistency of any discrepancies which may occur. This investigation will, in part, examine the concepts of both lactate and ventilatory threshold equality, and the reflection of cellular lactate production by blood lactate accumulation.

JUSTIFICATION

The use of the AT has become widespread in the field of exercise physiology as is thought to reflect one's aerobic work capacity. This transition threshold (TT) represents that critical intensity above which work capacity is severely limited. Examination of noninvasive measures at the TT allows one to examine the person's ability to transport oxygen to, extract and utilize oxygen in the working muscle mass. The work intensity at the TT is thought to represent a person's endurance capacity, and EXCO2 has been correlated highly with marathon performance times. The mechanisms underlying the TT are also mechanisms which will limit aerobic performance and induce

fatigue. Determining the TT accurately allows one to study limiting factors in a patient and athletic population, determine aerobic capacity, monitor training adaptations, and prescribe exercise that will stress the desired energy systems.

Most researchers opt to use noninvasive ventilatory and/or gas exchange variables in detecting the TT. Although some researchers have claimed to have validated the use of noninvasive measures and have found them to be reliable indices (Powers et.al. 1984, Caiozzo et.al. 1982, Davis et. al. 1976), many researchers still question the practice (Simon et.al. 1983, Hughes et.al. 1982, Hagberd et.al. 1982). Research on the topic has failed to investigate the relationship between blood lactate accumulation and the noninvasive ventilatory and gas exchange variables at points other than those of the threshold or "breakaway" points. The discussion of EXCO2 has been restricted to the relationship between EXCO2 and performance (Volkov et al, 1974, Rhodes et. al, 1984, Hearst, 1982 unpublished thesis).

This study is justified in that it examines the relationship between EXCO2 and blood lactate at all points during a progressive intensity test. This study will also examine the relationship between the lactate and ventilatory thresholds, their equality or difference, and the consistency of any discrepancies which may occur.

DEFINITIONS

Transition Threshold (TT) - that point where the aerobic

energy pathways can no longer maintain the tissues metabolic level and the increasing demands of the tissues are met through an increased reliance on anaerobic metabolism, with lactate accumulation exceding its removal.

Lactate Threshold (LATT) - that work rate just below the point at which there is an abrupt increase in venous blood lactate; that work rate below the breakaway point on the lactate/time curve.

Ventilatory Threshold (VT) - that work rate just below the point where there is a non linear increase in EXCO2 (EXTT), and an abrupt increase in the ratio VE/VO2 (VVTT).

Excess CO2 (EXCO2) - the nonmetabolic CO2 produced through the buffering of acids by the bicarbonate buffering system. EXCO2 can be calculated by comparing the CO2 produced and the O2 consumed. It is commonly calculated by the following equation:

$$EXCO2 = VCO2 - (\text{resting RQ} \times VO2)$$

HYPOTHESIS

1. there will be a significant correlation between EXTT and LATT.
2. there will be a significant difference between EXTT and LATT.
3. EXCO2 levels will parallel blood lactate levels through all stages of the progressive intensity test.

SECONDARY HYPOTHESIS

1. there will be a significant correlation between EXTT and VVTT.
2. there will be a significant difference between EXTT and VVTT.
3. there will be a significant correlation between VVTT and LATT.
4. there will be a significant difference between VVTT and LATT.

RATIONALE

Approximately 90 - 94% of the hydrogen ion produced in the working muscle mass from the dissociation of lactic acid will be buffered immediately by the bicarbonate system producing non-metabolic (excess) CO2 and water (Wasserman et.al. 1986). EXCO2 will be generated as long as the rate of lactic acid production is increasing as there will be additional hydrogen ion to buffer (Wasserman et.al. 1986). In a progressive intensity test (PIT) with increasing workloads lactic acid production will continue to rise, slowly at first and then rapidly in a curvilinear fashion throughout the duration of the test producing a relative increase in EXCO2.

The hydrogen ion and CO2 produced within the muscle readily move across the muscle membrane into the blood stream, enhanced by the presence of carbonic anhydrase in the capillary

endothelial cells. The lactate molecule is not, however, readily diffusable across the muscle membrane (Stainsby 1986) and may be removed by the oxidaitve fibers within the active muscle bed (Brooks 1986). Because of lactate's delayed release into the blood stream EXCO2 will precede the accumulation of lactate in the blood and may offer a more accurate prediction of cellular lactate production and accumulation (Issekutz and Rodahl 1961).

The ventilatory parameters most often used in the determination of the transition threshold, VE and VE/VO2, are directly linked to the production and release of CO2 and H+ from the active muscle mass. The prime driving forces for increases in ventilation are an increase in the PCO2 and decrease in the pH of the blood. Because the release of CO2 and H+ provide the driving force for ventilation, increases in expired CO2 will precede an increase in ventilation with the VE and VE/VO2 threshold points overestimating the onset of anaerobic metabolism and lactate accumulation within the working muscle mass.

DELIMITATIONS

This study is delimited by:

a) This subject sample size (N=21).

b) The sample type, consisting of elite cyclists.

c) The sampling rate of CO2 (15 s intervals) and blood lactate (1 min. intervals).

LIMITATIONS

This study's results are limited by:

a) Data collection capabilities of the Beckman Metabolic Measurement Cart and interfaced Hewlett Packard Data Acquisition system.

b) The individual's metabolic response to the protocol.

c) The blood lactate sampling and measurement technique.

d) The determination of the transition threshold through visual inspection.

CHAPTER TWO

SELECTIVE REVIEW OF THE LITERATURE

A CRITICAL REVIEW OF BLOOD LACTATE AND VENTILATORY METHODS OF DETECTING THE TRANSITION THRESHOLD

INTRODUCTION

As early as 1930 W.H. Owles recognized that there was a critical exercise intensity level above which the working muscles produced lactic acid. Accompanying an accumulation of blood lactate Owles observed an increase in CO2 excretion and ventilation. Harrison and Pilcher (1930) reasoned that the excess CO2 being produced was a result of bicarbonate buffering of acids being produced during anaerobic metabolism. This critical intensity, dubbed the 'anaerobic threshold' by Wasserman and Whipp in 1964, appears to represent a work intensity above which endurance performance is severely limited, and is postulated to represent a transition point in energy metabolism - a 'transition threshold'.

Physical exercise requires a balance between the production and consumption of energy within the working musculature. The coupling of these two elements depends on the individual's limited response of the cardiovascular and respiratory systems to exercise. There seems to be a 'critical intensity' above which the cardiovascular and/or respiratory response is of an insufficient magnitude, failing to supply the energy demanded through the aerobic pathways, and work capacity is severely limited (Knuttgen, 1962). Work performed below this critical intensity may be performed for indefinite durations as the energy required for muscular contraction is being supplied

predominately through unlimited aerobic energy sources while waste products are being adequately removed. Work intensities above this critical intensity result in rapid fatigue due to a reliance on limited anaerobic energy sources and an accumulation of inhibitory waste products.

The determination of this transition point has been the subject of many investigations and has found mixed reviews. The object of this review will be to present previous research findings pertaining to the aerobic-anaerobic transition, the accumulation of blood and muscle lactate, and the subsequent release of non-metabolic carbon dioxide. Emphasis will be placed on the determination of the critical intensity at which there is a sharp increase in intracellular lactic acid formation.

1.0 MODELS OF TRANSITION

The concept of a critical intensity above which there is an increase in blood lactate was introduced in the early 1900's by Christiansen et al (1914) and later by Owles (1930). It was also recognized at this time that changes in ventilation accompanied this phenomena (Douglas, 1927). It was long believed that these changes were associated with a relative shortage of oxygen at the muscular level (Hill et al, 1924) which lead to the concept of the "anaerobic threshold" forwarded by Wasserman and McIlroy (1964) (Davis, 1985). In more recent years the concept of a transition threshold has been under close scrutiny and the controversy around the concept has lead to the formulation of three separate models of aerobic-anaerobic transition.

1.1 SINGLE BREAKAWAY MODEL

The classical model of aerobic-anaerobic transition during progressive intensity testing is the single breakaway model stemming from early work which identify a "critical intensity" above which there was an accumulation of blood lactate. Margaria and colleagues (1933 and others) greatly influenced the initial model in that they established the idea that lactic acid was formed and blood lactate accumulated during times of local muscle hypoxia: at the onset of exercise and during times of oxygen deficit. (An idea questioned to date, first refuted by Hubbard, 1973)

In 1964 Wasserman and McIlroy introduced the term "anaerobic threshold" to denote that work rate at which the tissues oxygen supply first fell below demand and the "excess" energy needs were supported through anaerobic metabolism. They first defined this point as that point immediately preceding a disproportional increase in plasma lactate levels above resting levels. They also suggested that this point could be determined by decreases in blood bicarbonate or pH, or an abrupt increase in the respiratory exchange ratio (R). It was Wasserman and colleagues that later further investigated and refined the non-invasive ventilatory measures of the anaerobic threshold. They more recently supported the single threshold phenomena by employing a log-log transformation of lactate concentration and VO2 (Beaver et al, 1985).

1.2 DOUBLE BREAKAWAY MODEL

A three phase, double breakaway model was forwarded by

Kindermann, Simon and Keul (1979) in response to reported threshold lactate values of both 2 and 4 mmol/l. These authors found that exercise could be maintained at high load intensities, producing a steady-state lactate concentration of approximately 4 mmol/l, for prolonged durations. These authors suggested that the anaerobic threshold proposed by Wasserman et al (1964), which occured in the range of 2 mmol of lactate per liter of blood volume, reflected the upper limit of exclusive aerobic metabolism, and suggested that this 'threshold' should be refered to as the 'aerobic threshold'. In their framework the 'aerobic-anaerobic transition' period occured between blood lactate concentrations of 2 and 4 mmol/l, and that a blood lactate concentration of 4 mmol/l represented the true 'anaerobic threshold' - a value above which lactate production exceeds its removal and endurance exercise is severely limited.

The model and terminology proposed by Kindermann et al (1979) was further developed by Skinner and McLellan in 1981. These authors, drawing information from past research articles, attempted to explain each stage of the three phase double breakaway model and the physiological mechanisms underlying the events which occured.

Skinner and McLellan (1981) described the initial phase, the aerobic phase, as being predominantly aerobic with a heavy reliance on type I (ST or SO) muscle fibers and free fatty acids (FFA) as the metabolic substrate. The aerobic threshold, following, leads into the aerobic-anaerobic transition phase which involves the recruitment of type IIa (FOG) fibers and the

appearance of lactate in the blood. Lactic acid in turn decreases blood and intracellular pH and causes an increase in excess CO2, ventilation (VE), R and a disproportional increase in VE/VO2. There is an increase in the fractional concentration of expired O2 (FeO2) and a continued rise in the fractional concentration of expired CO2 (FeCO2). The aerobic-anaerobic transition phase ends at the anaerobic threshold where lactate production equals its removal capacity. The anaerobic phase, the third phase, follows. This phase involves the recruitment of type IIb (FG) fibers with a rapid rise in lactic acid production. Lactate production exceeds its removal with a rapid increase in blood lactate and VE, and a decrease in FeCO2.

1.3 EXPONENTIAL MODEL

The exponential model described by Yeh et al (1983) and Hagan and Smith (1984) has had breif appearances in the literature since it was first described by Jervell in 1929. Hughson et al (1987) found that blood lactate concentration increased as a continuous function during progressive exercise. In contrasting the continuous function model and the single breakaway model (the log-log model of threshold determination) described by Beaver et al (1985) Hughson et al (1987) found that the mean square error was approximately 3.5 times larger (P,0.001) when the single breakaway model was used as compared to the continuous function plus constant model they employed. The authors suggested that a lactate slope index would be a prefered indicator of 'fitness' replacing the previously applied threshold concept. Their model suggests that there is no

"breakaway" point at which there is an increased reliance on anaerobic metabolism, but that there is an exponential increase in the production of energy through the anaerobic energy pathways from the onset of incremental exercise. In this model all parameters - blood lactate, VE, VCO2, and R - are thought to increase in a curvilinear fashion from the onset of incremental exercise. This model challenges the very exsistence of critical intensities, lactate and ventilatory thresholds, upholding a belief that anaerobic energy sources supply an ever increasing proportion of the total energy expenditure from the onset of incremental exercise.

2.0 THRESHOLDS

During the transition from aerobic to anaerobic metabolism an abrupt increase in blood lactate concentration, expired CO2 (VCO2), and ventilation may be observed, and have been reported throughout the last century. The critical intensity at which this transition occurs has been widely researched appearing in the literature under a wide variety of nomenclature. This 'transition threshold' has been refered to as the 'anaerobic threshold' (Wasserman et al, 1964), the 'aerobic threshold' (Kindermann et al, 1979), and the 'aerobic-anaerobic threshold' by Mader et al (1976) (Jacobs, 1986). This transition threshold represents a combination of both a lactate and a ventilatory "threshold."

The use of the transition, or 'anaerobic' threshold has become widespread in the field of sport science and exercise

physiology. The transition threshold represents that critical intensity above which work capacity is severely limited. Examination of the transition threshold has enabled investigators to examine a person's ability to transport oxygen to, extract and utilize oxygen in the working muscle mass. The work intensity at the transition threshold is thought to be representative of a person's endurance capacity (Rusko et al, 1980), and has been correlated highly with marathon performance times (Rhodes et al, 1981). The proposed mechanisms underlying the transition threshold are mechanisms which limit aerobic performance and induce fatigue. Determining the transition threshold accurately would allow investigators to study limiting factors in both patient and athletic populations (Wasserman et al, 1964), determine aerobic capacity (Farrell et al, 1979), monitor training adaptations (Ready et al, 1982), and prescribe exercise which will stress the desired energy systems (Davis et al, 1981; Tanaka et al, 1981; Kinderman et al, 1979). The 'anaerobic threshold' has been described as a key parameter which defines the ability to maintain high-intensity exercise (Whipp et al, 1981), yet the concept of the 'anaerobic threshold', or any combination of the lactate and ventilatory thresholds, remains extremely controversial. It appears it is in the mechanisms of such increases, the nomenclature, and the original assumptions that the transition threshold is failing.

Many of the original ideas and assumptions of the 'anaerobic threshold' are being challenged. It was originally surmised that the lactate threshold and ventilatory threshold were synonymous

(Wasserman et al, 1964), and believed that only under hypoxic conditions did the tissues produce lactic acid (Margaria et al, 1933). The appearance of lactate in the blood stream was thought to reflect the production of lactic acid within the muscle cell. These ideas are being challenged by investigators who are finding lactate to be released under what they believe to be adequate tissue oxygenation (Connett et al, 1984; Jobis et al, 1968), and finding that the lactate and ventilatory thresholds can be uncoupled (Segal et al, 1979). Other investigators are finding that blood lactate levels do not accurately represent the cellular lactate production or concentration. Investigators are finding that a considerable portion of the lactate formed within the muscle may be oxidized within the active muscle tissues (Brooks, 1986), and that a translocation hinderance to the lactate molecule may be present (Stainsby, 1986). These findings suggest that the appearance of blood lactate may be delayed, not representing an increased reliance on the anaerobic energy production or the cellular lactate concentration.

2.1 LACTATE THRESHOLD

Almost all tissues of the body can produce lactic acid, but the best example is the exercising skeletal muscle. Lactate is produced in order to supplement aerobic energy supply. The presence of lactate in the blood stream is believed to represent an increased reliance on glycolytic pathways of energy production (Jones et al, 1981). Pyruvate is the key intermediary (Jobis et al, 1968; Wasserman et al, 1986). An imbalance between pyruvate formation and its oxidation in the Krebs cycle will

cause pyruvate accumulation and its subsequent conversion to lactate. The conversion of pyruvate to lactic acid allows the oxidation of NADH and the continuation of glycolysis (Stainsby, 1986; Jones, 1980). Lactic acid will immediately dissociate in the physiological pH range (pKa=3.9) forming two lactate and two hydrogen ions. The lactate which is formed may then be transported to, and taken up by other tissues with adequate oxygen supply and utilized as a source of fuel (Brooks, 1986; Stainsby, 1986).

The lactate threshold, representing the aerobic-anaerobic transition threshold, has been observed since the early 1900's and has been defined in many ways since its introduction. The lactate threshold has been set as an absolute blood lactate concentration such as 2mM (Hughson et al, 1982) or 4mM (Sjodin et al, 1981; Kinderman et al, 1979), at the initial increase in blood lactate above resting levels (Wasserman et al, 1973; Davis et al, 1976), at that point where there is an abrupt increase in lactate accumulation (Aunola et al, 1984), at that point where there is a systematic increase in blood lactate concentration (Ciaozzo et al, 1982), or at a set slope value such as 51 or 45 degrees (Jones et al, 1982). Brooks (1985) defined the lactate threshold as that workload at which there was an abrupt increase in, or disproportionally high, non-linear increase in blood lactate concentration. Davis (1986) defined the lactate threshold as that workload immediately proceding a progressive increase in blood lactate concentration. It appears that the later definitions best apply as account for inter-subject

variability, although may be criticized as having a wide margin of inter-observer error in threshold determination.

At the onset of a progressive intensity test there is a slight initial increase in blood lactate (Brooks, 1986) which should not be mistaken as the transition threshold. This level will be maintained, or increase very little, up to a critical workload which varies between individuals. At this critical point there will be a disproportional or abrupt increase in the blood lactate concentration. During progressive increases in intensity of exercise a stage is reached at which there is an ever increasing reliance on anaerobic metabolism and the release of lactate into the blood (Davis et al, 1983). A gradual increase in blood lactate may be noted prior to an abrupt increase in blood lactate which occurs at a point representing the lactate threshold (Davis et al, 1983; Brooks, 1985; Wasserman, 1986).

2.1.1 Tissue O2 Supply and Lactic Acid Production

It was initially believed that lactate was produced during periods of insufficient oxygen supply (Hill et al, 1924; Margaria et al, 1933). If there is an inappropriate response to the level of muscular activity by either the cardiovascular or respiratory system and the oxygen supply of the muscle is not met, the deficit in energy demand is met through anaerobic energy production (Alpert, 1965; Jones, 1980) with the breakdown of glucose and/or glycogen ending with the formation of lactic acid. Dill et al (1932) warned, however, that the accumulation of lactic acid alone was inconclusive evidence of an oxygen

deficit (Gollnick, 1986). Research has taken many directions in the quest for conclusive evidence supporting either adequate or inadequate oxygen supply.

Jobis and Stainsby (1968) looked toward NAD/NADH levels for an answer and concluded that the high NAD levels during lactic acid formation indicated adequate oxygen supply. Graham (1978), however, found no relationship between NAD concentration and blood lactate levels. Stainsby later concluded (1986) that lactate production without hypoxia was related to the more rapid activation of glycolysis over oxidative phosphorylation.

Holloszy (1976) looked at changes in blood lactate levels and oxygen utilization at submaximal workloads in order to determine the presence or absence of local tissue hypoxia. Holloszy found that after training there was a lower blood lactate content at the same submaximal workload while the oxygen utilization did not change. It was concluded that muscle hypoxia could not be present as changes in blood lactate could not be attributed to increases in VO2. Davis (1985) suggested that this was a correct assumption for submaximal workloads where adequate oxygen supply is undisputed but did not however reveal information regarding oxygen supply above the anaerobic threshold.

Much controversy still exsists around the hypothesis that hypoxic conditions are present when lactic acid is being produced in the working tissues. Recent articles by Brooks (1985), Davis (1985), Gollnick et al (1986), and Wasserman et al (1986) reitterate the concern. Production of lactate does

augment the cellular supply of ATP, but Brooks (1985) and Gollnick et al (1986) are firm believers that this occurs in the presence or absence of adequate oxygen supply while Wasserman and colleagues (1986) are firm believers that lactate accumulation is oxygen dependent and requires a change in the redox state of the cell.

2.1.2 Lactate Production vs. Removal

Cohen et al (1976) suggested that lactate accumulation was not only due to local tissue hypoxia but to an imbalance between the rate of lactate production and its removal. The fate of the lactate molecule has been a topic of investigation since the early 1900's. Recent research suggests that as much as 75% of the lactate produced is removed within the working muscle mass (Brooks, 1986; Hermansen et al, 1976). Earlier research found that the lactate released into the blood stream could be removed by well oxygenated skeletal muscle (Jorfeldt, 1970), by the liver (Cori and Cori, 1929) or kidney (Yudkin et al, 1975) via gluconeogenesis, by the heart (Carlsten, 1961), or the brain (Belcastro et al, 1975). Recognizing the importance of the liver in the removal of blood lactate Donovan and Brooks (1983) suggested that reduced hepatic blood flow, and hence hepatic clearance of lactate, may play an important role in the lactate threshold. Whichever mechanisms limit lactate removal, the consequence of an imbalance between lactate production and its removal is an increase in both muscle and blood lactate levels (Skinner et al, 1981; Brooks, 1985; Davis, 1985; Wasserman, 1986), both having major physiological implications.

2.1.3 Muscle Fiber Recruitment Patterns

Muscle fiber type and fiber type recruitment patterns may be important factors which contribute to the lactate threshold (Skinner et al, 1981). Jorfeldt (1970) suggested that type II fibers are net producers of lactate while type I fibers are net consumers, therefore lactate accumulation would depend on muscle fiber type and recruitment patterns. Type II fibers have an abundance of M-LDH which favors the reduction of pyruvate to lactate (Sjodin, 1976) and are more likely to become hypoxic as have a low capillary density, mitochondrial concentration and rate of oxidative phosphorylation (Tesch et al, 1981). Type I fibers favor the oxidation of lactate to pyruvate having an abundance of H-LDH (Sjodin, 1976). Graham (1978) found type I fibers to have three times the amount of lactate as compared to type II fibers.

2.1.4 Lactic Acid and Fatigue

Lactic acid produced during anaerobic glycolysis immediately dissociates producing two hydrogen ions and two lactate molecules. The hydrogen ion liberated from lactic acid formation is predominately buffered by the bicarbonate buffering system, while the lactate molecule is an important form of stored fuel for energy production, and supplies precursors for blood glucose (Cori et al, 1929; Brooks, 1986). If, however, the biproducts of lactic acid are allowed to accumulate within the working tissue there will be a rapid onset of fatigue. Increased lactate production will cause increased H+ release and a subsequent decrease in muscle and blood pH (Wenger et al, 1976). A decrease

in muscle and blood pH will limit the production of energy through anaerobic glycolysis through the inhibition of the rate limiting enzymes PFK (phospho-fructokinase) (Danforth, 1965), and phosphorylase (Hultman et al, 1980). A decreased cellular pH may also cause fatigue by altering membrane permeability (Wenger et al, 1976) or interfering with the Ca++ binding at the actomyosin binding sites (Wenger et al, 1976). Increased lactate production may play a role in impairing the aerobic energy supply by inhibiting FFA mobilization from the adipose tissue, limiting the supply of fuel for aerobic energy production (Issekutz et al, 1962).

2.2 VENTILATORY THRESHOLDS

Noninvasive measures of the onset of metabolic acidosis, the transition threshold, have allowed wide use of the concept. The refinement of noninvasive measures to determine the transition threshold has been an ongoing process since the 1930's when Owles (1932) recognized that expired volume (Ve) and volume of expired CO2 (VCO2) increased disproportionally above a critical intensity at which plasma lactate began to rise. Turrell and Robinson (1942) believed that the increase in CO2 production was the result of bicarbonate buffering of the metabolic acids being produced (A.), with the rapid dissociation of the newly formed carbonic acid (B.):

A. $HLa + NaHCO3 = NaLa + H2CO3$ (carbonic acid)

B. $H2CO3 = H2O + CO2$

The increased CO2 production, an associated fall in blood bicarbonate, and a rise in arterial pH are now believed to be

prime stimulators of ventilation, accounting for the rise in expired volume observed by Owles in 1932.

2.2.1 Ventilatory Control

Under normal conditions changes in metabolic demand are met with changes in minute ventilation of a magnitude which will maintain the arterial oxygen and carbon dioxide partial pressures at relatively constant values. Changes in the rate and depth of breathing are made in order to allow a matching of aveolar ventilation to blood perfusion, and allow for the correction of venous PO2 (Sutton et al, 1979). This ventilatory control is accomplished through the central nervous system. Input to the central nervous system through neurogenic and humoral stimuli allow sensitive control of the ventilatory response.

At the onset of exercise ventilation increases on the first full respiratory cycle due to a neurogenic component involving cerebral irradiation and reflexive stimuli from mechanoreceptors in the limbs (Powers et al, 1985). Humoral stimuli work above this initial increase and allow sensitive respiratory control ensuring appropriate aveolar ventilation. The humoral stimuli, PO2, PCO2, and pH, cause a gradual increase in respiration although their exact values do not increase significantly; the ventilatory response prevents wide fluctuations in the humoral variables from occuring.

It appears ventilation is more closely linked to CO2 output than any other variable (Sutton et al, 1979, Wasserman et al, 1975). Arterial PCO2 is regulated by ventilation, with

ventilation increasing in proportion to CO2 production (Swanson, 1977), a fact that has lead some to believe that PCO2 is not a primary regulating factor (Asmussen, 1983). The CO2 production will increase during exercise due to aerobic metabolism of fats and carbohydrates up to the "anaerobic threshold" after which an additional CO2 load will be introduced due to the production and buffering of lactic acid by bicarbonate. Up to the "anaerobic threshold" ventilation will increase in proportion to the increases in CO2 production, however, above this point increases in ventilation above those responsible for PaCO2 compensation do not allow for the complete compensation for the pursuing lactic acidosis (Wasserman et al, 1975). The hydrogen ion entering the blood stream will have an additional independent ventilatory stimulus stimulating the central chemoreceptors (Sutton et al, 1979), and ventilation increases in an attempt to lower the hydrogen ion content of the blood. Hyperpnea will drive PaCO2 lower than normal constraining the buffering capacity of the blood, and pH will drop (Whipp et al, 1980).

2.2.2 VCO2, RQ, and 'Excess' CO2

Increased CO2 production at high intensity exercise lead researchers to look toward the respiratory quotient (VCO2/VO2) to answer questions in regards to exercise capacity and accumulation of lactate in the bloodstream. Balke et al (1954) observed comparible increases in VCO2 and VO2 up to a specific point at which VCO2 exceded VO2. They took this point, where R was greater than 1.0, as indicating the upper limits of aerobic metabolism. In 1961 and 1962 Issekutz and Rodahl investigated

changes in RQ and related them back to aerobic work capacity and maxVO2. They concluded that changes in RQ best represented the degree to which anaerobic glycolysis participated in the total production of energy - its contribution to the total energy expenditure. They considered changes in RQ above 0.75 to be an index of inadequate oxygen supply to the working musculature, which was later supported by Naimark et al (1964). Wasserman and McIlroy (1964) concluded that the threshold of anaerobic metabolism could be observed without invasive measures or maximal effort in cardiac patients when R was measured continuously during a progressive intensity test, represented by an abrupt increase in R. The nomenclature, threshold of anaerobic metabolism, was soon known and refered to as the "anaerobic threshold."

Wasserman and colleagues (Naimark et al, 1964) found changes in R at different workloads to reflect the balance between oxygen supply and oxygen demand at the level of the working musculature. They recognized that R rose appreciably at the onset of blood lactate accumulation, and that the fall in blood bicarbonate was highly correlated with the production of non-metabolic CO2 (r = 0.98). Issekutz and Rodahl (1961) had suggested that the production and displacement of bicarbonate (nonmetabolic) CO2 reflects anaerobic metabolism more closely than blood lactate due to its rapid diffusion from the muscle cell. Using a resting RQ between 0.70 and 0.80 they calculated the nonmetabolic CO2,

$$\text{Excess } CO_2 = VCO_2 - (RQrest * VO_2)$$

which highly correlated (r = 0.92) with changes in blood lactate levels, and increased in proportion to increases in ventilation. The results indicated that ventilation was highly influenced by the accumulation of metabolic acids and the production of excess CO2 (Issekutz et al, 1961).

Using healthy trained and untrained males Bouhuys et al (1966) could not duplicate the excellent correlations found by Issekutz and Rodahl in 1961. Their investigation found that the increase in lactic acid was of a greater magnitude than the decrease in standard bicarbonate, with the differences being intensified with increasing workload. They concluded that increases in both R and excess CO2 were associated with increases in lactic acid accumulation (r = 0.622 and r = 0.796) but the reverse was not always true. They suggested that R and excess CO2 only provided a rough estimation of the degree of exercise induced acidosis while direct lactate determination was still preferable.

It had been suggested that ventlatory and gas exchange variables measured in volumes (ie. VCO2 and Ve) rather than those presented as ratios (ie. R and Ve/VO2) would be more proportional to blood lactate levels (Naimark et al, 1964, and Wasserman et al, 1964). Clode and Campbell (1969) provided supporting evidence for this hypothesis when investigating CO2 balance in the body and found that blood lactate could be estimated accurately by VCO2 when changes in tissue PCO2 are taken into consideration. They concluded that using a CO2 balancing technique changes in blood lactate concentration could

be estimated with reasonable accuracy.

2.2.3 Comparing Noninvasive Ventilatory Measures

In 1973 Wasserman et al defined the anaerobic threshold as "the level of work or O2 consumption just below that at which metabolic acidosis and the associated changes in gas exchange occur (pg. 236)." In comparing noninvassive gas exchange variables using breath-to-breath gas analysis during incremental exercise these investigators found that the transition threshold could be determined as that work rate or VO2 immediately preceding a nonlinear increase in Ve or VCO2, an increase in R, or that point where end-tidal O2 increases without a corresponding decrease in end-tidal CO2. Of these measures R was found to be the least sensitive as the metabolic RQ increases during incremental tests at increased workloads producing increasing amounts of metabolic CO2. The metabolic production of CO2 is much greater than the production of excess CO2 in normal subjects with changes in R being overshadowed.

J.A. Davis et al (1976) investigated the validity and feasability of using noninvasive laboratory measures in the detection of the 'anaerobic threshold' in three modes of exercise. As Wasserman et al (1973) these investigators found R to be the least sensitive measure. Using Ve alone to predict the lactate threshold point produced a correlation coefficient of 0.88, and when all gas parameters were used in correlating both lactate and gas analysis transition thresholds a correlation coefficient of 0.95 was found. They suggested that the major limitation in detecting the transition threshold from changes in

gas exchange variables is the subective determination of the threshold point from a time-based plot, although questioned the use of increased blood lactate levels above resting values as the criterion measure, being an indirect index of muscle lactic acid production.

The common ventilatory and gas exchange measures used in 'anaerobic threshold' detection were studied in 1982 by Caiozzo et al to determine which indices provided the most accurate and reliable determination of the lactate transition threshold. The investigators used four separate noninvasive measures (Ve, VCO2, R, and Ve/VO2) to determine the ventilatory threshold point and each was correlated to the criterion measure, the lactate threshold. It was found that Ve/VO2 provided the highest correlation ($r = 0.93$, $P < 0.001$) and provided the best test - retest correlation ($r = 0.93$, $P < 0.001$). R was the least sensitive measure ($r = 0.39$) while VCO2 and Ve appeared to provide excellent predictions of the lactate threshold ($r = 0.83$, $r = 0.88$). Multiple correlational analysis did not significantly improve the threshold detection over Ve/VO2 values, while the threshold points expressed as a %VO2max produced lower correlation coefficients than did expressing the threshold point as an absolute VO2 (l/min).

Powers et al (1984) were not able to reproduce the high correlations found earlier and concluded that the lactate threshold can not be accurately determined by gas exchange indices in all subjects. Using a slightly different protocol, with work increments of three minutes, and a blood lactate

sampling rate of once every three minutes (their criterion reference), these investigators found a poor correlation (r = 0.63) between the transition threshold when determined through Ve/VO2 and through blood lactate.

2.2.4 Non-Metabolic CO2 and Performance

Approximately 90 - 94 % of the hydogen ion produced within the working muscle mass from the dissociation of lactic acid will be immediately buffered by the bicarbonate buffering system producing non-metabolic (excess) CO2 and water (Wasserman et al, 1986). Excess CO2 (EXCO2) will be generated as long as the rate of lactic acid production is increasing as there will be additional hydrogen ion to buffer (Wasserman et al, 1986). In a progressive intensity test (PIT) with increasing workloads lactic acid production will continue to rise, slowly at first and then rapidly after the transition threshold in a curvilinear fashion producing a relative increase in excess CO2 [EXCO2 = VCO2 - (RQrest * VO2)].

The relationship between blood lactate accumulation and EXCO2 has not yet been determined, although early research by Clode, Clark and Campbell (1961) and Issekutz and Rodahl (1961) reported high correlations between the lactate threshold and EXCO2. These earlier investigations suggest that the calculation of EXCO2 permits changes in blood lactate to be detected with reasonable accuracy. The hydrogen ion and CO2 produced within the muscle cell readily diffuse across the muscle membrane into the blood stream, enhanced by the presence of carbonic anhydrase in the capillary endothelial cells. The lactate molecule,

however, may be removed by the oxidative fibers within the active muscle bed (Brooks, 1986) and has a translocation hinderance (Stainsby, 1986) to movement across the cell membrane. Because of these mechanisms increases in expired EXCO2 may be detected before a significant rise in blood lactate is detected; EXCO2 may offer a more accurate prediction of cellular lactate production and accumulation (Issekutz et al, 1961).

In 1975 Volkov et al suggested that the measurement of excess CO2 during a maximal oxygen consumption test allows accurate noninvasive determination of a subject's anaerobic power. The investigators thought that the excess CO2 index is directly related to the magnitude of lactate production through the glycolytic pathways and the organism's buffering capacity. They found that there is a consistent increase in excess CO2 when a critical intensity, or the threshold of anaerobic metabolism, is surpassed, and that this transition threshold point depends on the subject's state of training.

In 1981 Rhodes and McKenzie found that the running speed at which a breakaway point in expired EXCO2 occured could be used to predict marathon running performance. These authors found a highly significant correlation ($r=.94$, $P<0.01$) between predicted and actual marathon times suggesting that the rapid increase in expired EXCO2 may be an indication of the critical intensity at which the onset of anaerobisis occurs. These results were supported by Hearst (1982, unpublished thesis) who found that expired EXCO2 correlated highly ($r=.89$) with blood lactate accumulation at four specific running speeds.

3.0 PROTOCOLS FOR ELUCIDATING TRANSITION

Underlying investigations of the transition threshold are progressive intensity tests (PIT). In 1975 Wasserman and Whipp described the responses one could expect to obtain when using different forcing functions. In general, at work rates below the transition threshold mean arterial pH and PCO2 are maintained by increases in alveolar ventilation which are proportional to the increases in CO2 production. At work rates above the transition threshold the compensation for metabolic acidosis is accomplished through a disproportionally high increases in ventilation over those mediated by increased CO2 production causing arterial PCO2 to fall while pH remains unchanged. At even greater work rates yet (80% max +) increases in ventilation are unable to compensate for increases in blood pH due to the loss of blood bicarbonate and blood pH falls. (Wasserman and Whipp, 1975)

During a progressive intensity test with 1 - 4 minute increments the following trends may be observed:

1. VO2 increases linearly throughout the test until a maximal value is reached at which VO2 will plateau.

2. Ve and VCO2 will increase linearly with VO2 up to a critical intensity, the anaerobic threshold, at which lactic acid is produced causing VCO2 to increase faster than VO2.

3. Ve will increase in proportion to VCO2, therefore increases in Ve equal increases in VCO2 which are both greater than increases in VO2.

4. R increases since increases in VCO2 are greater than

increases in VO2.

The difference between the two different forcing functions, 1 or 4 minutes, is that while using 1 minute increments end-tidal PO2 will increase at the anaerobic threshold while end-tidal PCO2 remains constant, while using 4 minute increments end-tidal PO2 increases at the anaerobic threshold while end-tidal PCO2 decreases. (Wasserman and Whipp, 1975)

Wasserman and Whipp (1975) concluded that progressive intensity tests using one minute increments had several advantages over using four minute increments when noninvasive determination of the 'anaerobic threshold' was a major objective. This conclusion was based on five major points:

1. 1 minute increments allowed a more specific definition of the anaerobic threshold.

2. the test is much shorter.

3. lactate values do not reach such high values allowing quicker recovery from exhaustive exercise.

4. is easier to reach and determine the maxVO2 if so desired.

5. the 'anaerobic threshold' can be expressed as either a VO2 or work rate without large errors in estimation.

It appears that the transition threshold can be discerned readily from tests using 1 or 4 minute workload increments, or values inbetween, as long as one recognizes that different responses occur. Similar transition threshold values have been reported during tests using 1 and 4 minute workload increments (Wasserman et al, 1975; Yoshida, 1985). McLellan (1987)

attributes much of the conflicting data to methodological errors in threshold detection, using changes in ventilatory and gas exchange parameters observed in a 'fast' increment test to determine the anaerobic threshold during a 'slow' increment test and visa-versa.

4.0 CONCLUSIONS

Amidst the controversy surrounding the equality and cause-and-effect relationship of the ventilatory and lactate transition thresholds there are still many firm believers in the phenomena (McLellan, 1987; Wasserman et al, 1986; Davis, 1985). Many reviewers (Hughson et al, 1987; Brooks, 1985; Stainsby 1985), however, still question the relationship. It appears that there is inconclusive evidence supporting either position, in a large part due to an insufficient method of determining intracellular events, suggesting a need for further research in the area utilizing some of the new technologies.

CHAPTER THREE

METHOD AND PROCEDURES

METHODS AND PROCEDURES

Subjects

Twenty-one national team cyclists (15 males and 6 females) participated in the study. The subjects were tested over a three day period just prior to the national team time trials; all subjects were in a highly trained state. The subjects were asked to refrain from heavy exercise 24 hours prior to the test and perform on an empty stomach.

Testing Procedures

The baseline measures of height and weight were taken prior to testing.

Testing was performed at U.B.C. in the J.M. Buchanan Fitness and Research Center. Subjects were asked to warm up 15 minutes prior to being tested, and were allowed only a limited warmup with the testing apparatus in place.

The test protocol consisted of a continuous progressive intensity test on a Monarch stationary bicycle. Following a two minute warmup at a work rate of 50 or 100W (female/male) the cyclists pedalled against an initial resistance of 100 or 150W (female/male) with increases of 25W every second minute (figure 1.0). A cadence of 90 rpm was maintained throughout the test. The test was terminated upon volitional fatigue; that point where the athlete was unable to maintain or regain the 90 RPM frequency.

Table 1.0 The progressive loading scheme.

stage	time (min)	work rate (W) male	work rate (W) female	frequency (RPM)
wmup	0 - 2	100	50	
1	2 - 4	150	100	90
2	4 - 6	175	125	90
3	6 - 8	200	150	90
4	8 - 10	225	175	90
5	10 - 12	250	200	90
6	12 - 14	275	225	90
7	14 - 16	300	250	90
	etc.			

Testing measures included VO2, VE, VCO2, R. EXCO2, blood lactate and HR. Respiratory gas exchange variables were monitored online using a Beckman Metabolic Cart interefaced with a Hewlett Packard Data Acquisition System. Variables were determined or calculated over 15 second time intervals.

HR was monitored in the last 10 seconds of each minute through the use of a three lead V5 ECG recording.

Blood samples were taken via finger tip venous sampling at minute intervals throughout the duration of the progressive intensity test. The blood samples were taken, immediately heamolysed using cooled perchloric acid, and analyzed. Total blood lactate was determined using a Kontron Medical Lactate Analyzer 640.

The breakaway threshold points were determined by three independant observers through visual inspection of the EXTT, VVTT and LATT curves. Each set of curves were analyzed separately while the subjects identity was withheld. The proposed breakaway points, or transition thresholds, were then discussed by the group of three observers and an exact breakaway point was agreed upon.

Experimental Design and Data Analysis

There were three dependent variables (blood lactate, EXCO2 and VE/VO2) and one independant variable (progressive intensity test).

A regression analysis was performed comparing EXTT and LATT, EXTT and VVTT, LATT and VVTT. Correlation coefficients and an r* value were obtained for the data.

Significant differences were determined using a repeated measures ANOVA. A significant F value was further investigated using post hoc procedures.

The degree to which the curves paralleled each other was investigated by graphing average absolute EXCO2 and lactate values for five minutes pre and post threshold. A correlation coefficient was determined for all data points pre/post threshold.

CHAPTER FOUR

RESULTS AND DISCUSSION

RESULTS

Twenty-one Canadian National caliber cyclists participated in this study just prior to the Canadian National team time trials. The descriptive subject data is presented in Table 1.

TABLE 1. Descriptive subject data for all subjects grouped, males and females

	GROUP	MALE	FEMALE
	N = 21	n = 15	n = 6
Age (yr)	23.19 ± 3.34	23.26 ± 2.79	23.00 ± 1.95
Weight (kg)	70.10 ± 8.46	73.57 ± 7.30	61.43 ± 3.34
Height (cm)	175.5 ± 1.57	177.9 ± 6.90	169.6 ± 1.59
MaxVO2 (ml*kg*min)	63.41 ± 5.9	65.38 ± 5.0	58.50 ± 5.1

Aerobic - anaerobic transition threshold values were determined by using each of three single indices (ie. LATT - blood lactate, EXTT - excess CO2, and VVTT - the ventilatory equivalent for VO2, VE/VO2). The mean threshold VO2 values (±S.D.) for each method of AT detection, reported in l/min, were 3.65 ± 0.57, 3.46 ± 0.62, and 3.58 ± 0.51 for each of LATT, EXTT, and VVTT respectively. Table 2 presents a correlation matrix for the reported indices. Diagrams 1 through 3

TABLE 2. A correlation matrix between absolute VO2 AnT values as determined through different methods

	LATT	EXTT	VVTT
LATT	1.0		
EXTT	0.95 *	1.0	
VVTT	0.91 *	0.92 *	1.0

* significant at P<0.001

represent plots comparing the three indices and report the regression equations. The highest correlation between transition thresholds determined through different methods was found between LATT and EXTT (r = 0.95, P(0.001). The lowest correlation between two measures was found between LATT and VVTT (r = 0.91, P(0.001).

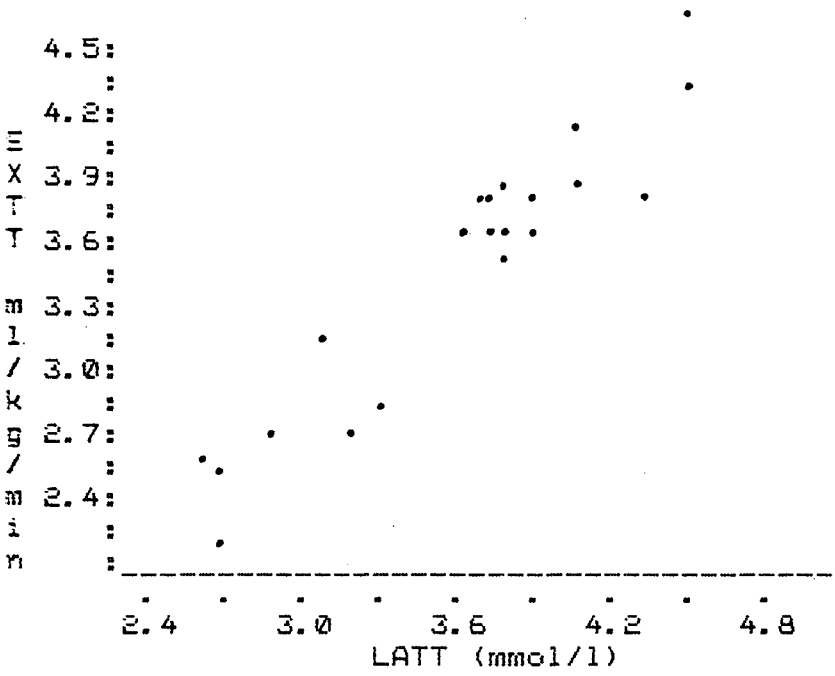

FIGURE 1. LATT (mmol/l) vs EXTT (ml/kg/min)
N = 21
R = 0.95
P(R) (0.001

X = 0.8613*Y + .6653

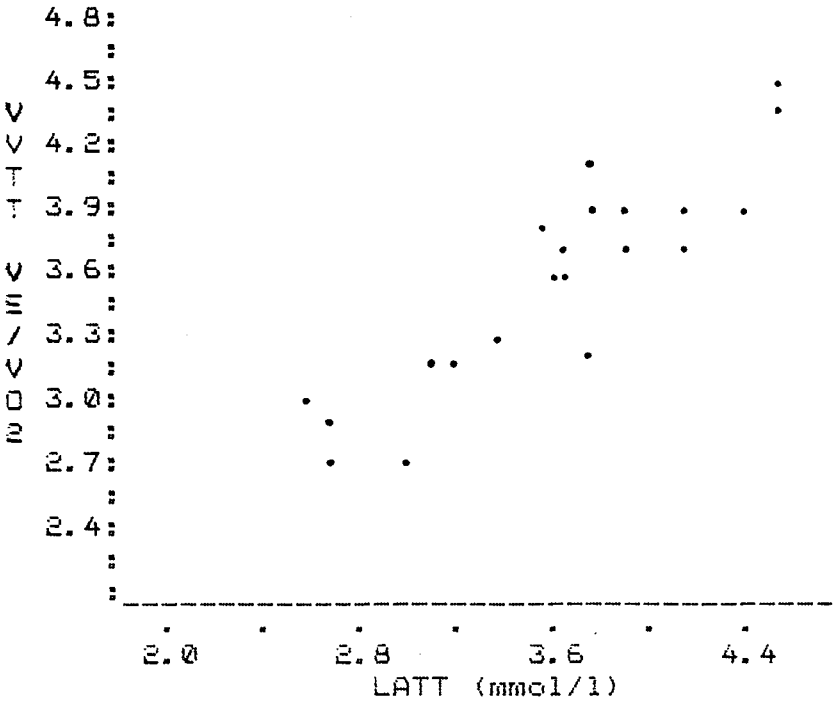

FIGURE 2. LATT (mmol/l) vs VVTT (VE/VO2)

N = 21
R = 0.91
P(R) (0.001

X = 1.0256*Y - .029

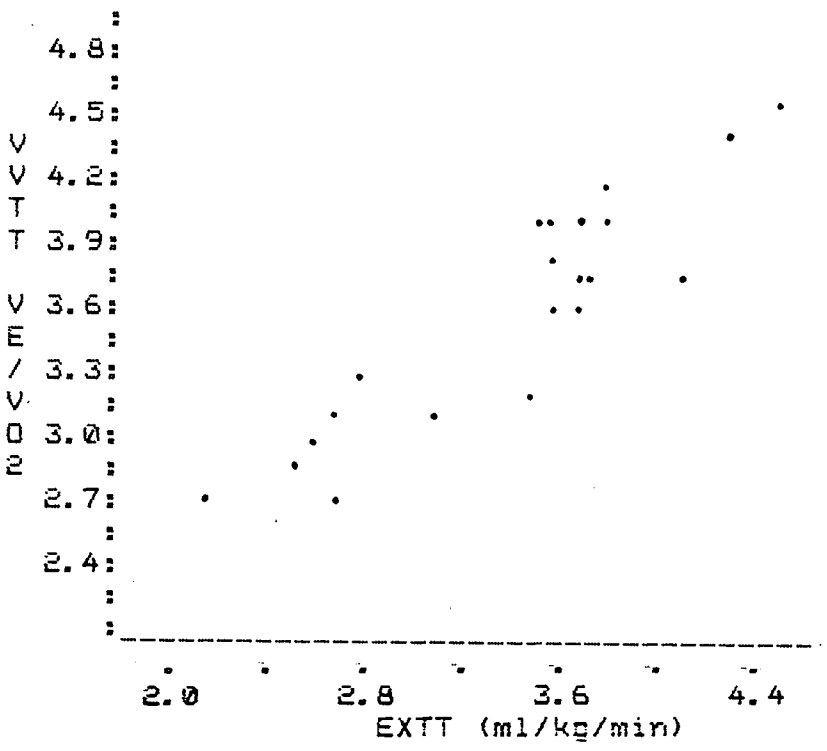

FIGURE 3. EXTT (ml/kg/min) vs VVTT (VE/VO2)

N = 21
R = 0.92
P(R) (0.001

Y = .7462*X + .9974

The equality of the three transition threshold VO2 values (l/min) was determined through the use of a repeated measures ANOVA which revealed a significant difference between cell means ($F = 8.41$, $P(0.001$). Post hoc comparisions revealed significant differences between LATT and EXTT means ($t = 4.287$, $P(0.001$) and between EXTT and VVTT means ($t = 2.212$, $P(0.025$), but a nonsignificant difference between the LATT and VVTT means (see TABLE 3). There was a general trend for the EXTT to precede the VVTT, which preceded the LATT. Although the trends for the VVTT were inconsistent, the trend for the EXTT to precede the LATT held true in 19 of 21 subjects.

As VO2 (l/min) and time are linearly related differences

between VO2 means represent differences between the means when represented as a time (minutes). The mean times (min) at which the transition thresholds occured were approximately 12 ± 3 minutes (LATT), 10.5 ± 3 minutes (EXTT), and 11.5 ± 2.5 minutes (VVTT). Using correlated t-Tests significant differences were found between both LATT and EXTT means ($t = 5.01$. $P<0.001$) and EXTT and VVTT means ($t = 2.00$, $P<0.050$). The blood lactate transition threshold (LATT) was significantly delayed, as compared to the ventilatory transition threshold detected through the increased release of EXCO2 in expired air, with an average time delay of 1.4 minutes.

TABLE 3. RM ANOVA and Post Hoc comparisions of the threshold VO2 values (l/min) as detected through different means

TT INDEX	POST HOC CORRELATED T-TESTS. LATT	EXTT	VVTT	N	MEAN VO2 (l/min)
LATT	--	t=4.29	t=1.38	21	3.65 ±0.57
EXTT	P<0.001	--	t=2.21	21	3.46 ±0.63
VVTT	P>0.05*	P<0.025	--	21	3.58 ±0.51
ANOVA	F=8.41	P<0.001	df = 2		

* nonsignificant difference

Individual subjects were compared over a relative period of time pre and post thresholds, using the EXCO2 and blood lactate threshold points as relative points of comparision. This procedure also allowed for the significant time delay in the appearance of blood lactate to be accounted for. The subject's expired EXCO2 volume and blood lactate concentrations were compared over a five minute pre threshold and five minute post threshold period, for a total of eleven minutes. Absolute values

of lactate (mmol/l) and excess CO2 (ml/kg/min) correlated significantly (r = 0.69, P(0.001) (see DIAGRAM 4) over this 11 minute range. With an r* of 0.48, approximately 50% of the variance in blood lactate levels can be accounted for by chances in expired excess CO2 when using the TT as a relative point for comparision. Individual correlations over the same time period produced highly significant correlations (ranging from .82 to .96, P(0.001), with approximately 80 - 90% of the variation in blood lactate being accounted for through changes in EXCO2. The influences of both individual differences and gender differences may account for the lower grouped correlation obtained.

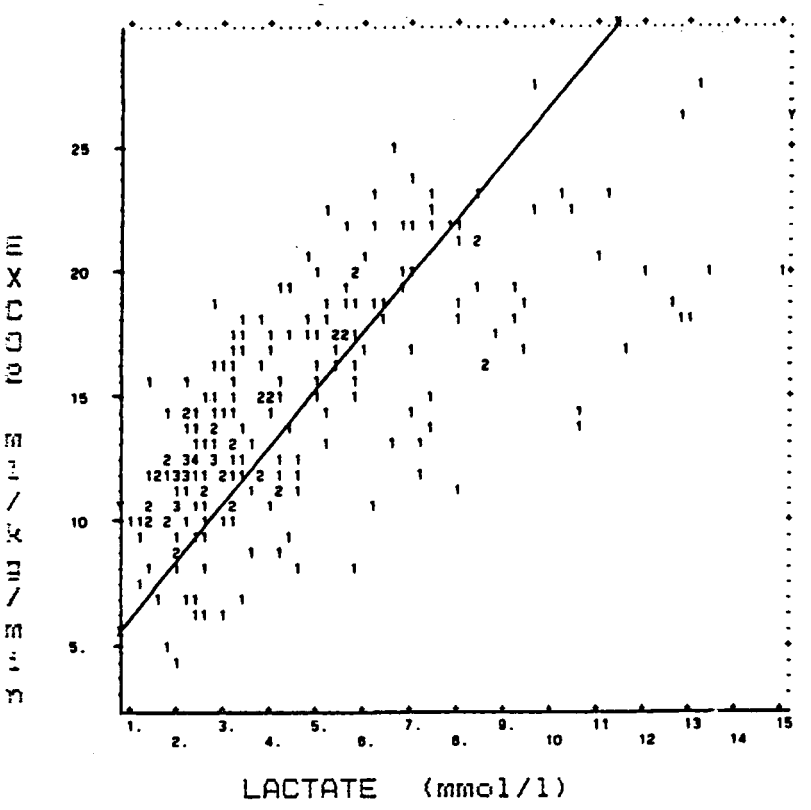

FIGURE 4. Lactate (mmol/l) vs expired excess CO2 (ml/kg) over an 11 minute range (5 min. pre & post AT) using the AT as a relative point of reference

n = 224
R = 0.69
P(0.001

X = .4391*Y - 1.7895

Changes in EXCO2 appear to track changes in blood lactate levels when the thresholds are used as relative points, although the nature of this relationship varies between individuals and gender. Both average male and female lactate values were similar pre threshold after which the female's blood lactate and EXCO2 increased at an increased rate over the male's. The significant individual correlations of blood lactate and EXCO2 over this period of time suggest the two are interrelated.

DISCUSSION

While previous studies (Caiozzo et al, 1982; Davis et al, 1976; Wasserman et al, 1973; Wasserman et al, 1964) have demonstrated a relationship between ventilatory and blood lactate thresholds, and earlier studies have suggested that EXCO2 may reflect changes in blood lactate (Issekutz et al, 1961; Clode et al, 1961), the exact relationship between excess, non-metabolic CO2 and blood lactate accumulation has yet to be elucidated. The purpose of this study was to determine the relationship between blood lactate and EXCO2 throughout a progressive intensity test in twenty-one elite level cyclists, while particular emphasis was placed on the relationship between the blood lactate and the ventilatory (EXCO2, and VE/VO2) thresholds. Although many studies have examined the aerobic-anaerobic transition threshold utilizing a bicycle ergometer progressive intensity test, an extensive review of the literature has not revealed any previous studies investigating these specific ventilatory measures or utilizing such an elite level subject pool.

The results of this study demonstrated significant correlations between each of the three indices used in determining the transition threshold (EXCO2, VE/VO2, and blood lactate). A correlation of r=0.91 was found between the lactate and VE/VO2 thresholds, consistent with earlier findings (Caiozzo et al, 1982; Davis et al, 1976; Wasserman et al, 1973). These

previous authors have suggested that VE/VO2 may offer the most sensitive measure of the lactate threshold.

This study found the EXCO2 threshold to correlate best with the lactate threshold (r=0.95), although the EXCO2 threshold did not represent the actual occurance of the lactate threshold. A significant (P<0.001) difference was found between the average EXCO2 and lactate thresholds, represented by a 1.38 minute time delay in the appearance of blood lactate. As the VE/VO2 threshold point did not significantly differ from the lactate threshold point (P>0.05), VE/VO2 may be utilized, as previous researchers have suggested, to reflect the point at which there is an abrupt increase in blood lactate (Caiozzo et al, 1982; Davis et al, 1976; Wasserman et al, 1973).

Studies which have used blood lactate as their criterion measure must be interpreted with caution. Recent studies have suggested that blood lactate may not reflect intracellular lactate production or concentration (Stainsby, 1986; Brooks, 1986). Although VE/VO2 and blood lactate have been found to breakaway at similar points, these similarities may in no way reflect the accumulation of intracellular lactate. In this study the non-significant VE/VO2 and lactate turnpoint difference may be due to the erratic VE/VO2 and lactate turnpoint relationship. In 13 of 21 cases the VE/VO2 turnpoint was equal to or preceded the lactate turnpoint, while in 8 of 21 cases the VE/VO2 turnpoint occured after the lactate turnpoint (a range of 3 min. pre to 2 min. post lactate turnpoint). Although the average VE/VO2 turnpoint was not significantly different from the

average lactate turnpoint, the wide range of VE/VO2 turnpoint values, spanning the lactate turnpoint, may be largely responsible for this finding.

EXCO2 turnpoints were found to be more consistently related to the lactate turnpoints, preceding the lactate turnpoint in 19 of 21 cases. Not only were the EXCO2 and lactate turnpoints significantly correlated (r=0.95) a significant difference was found between them (P(0.001). This data suggests that EXCO2 diffuses across the muscle membrane more readily than the lactate molecule is released from within the cell, supporting evidence of a translocation mechanism (Stainsby, 1986). This hypothesis is further supported by the results documented by Caiozzo et al (1982) who found the VCO2 turnpoint was equal to, or preceded the lactate turnpoint in 13 of 16 subjects, although the significance of such a difference was not investigated.

The mean absolute lactate value was found to be 3.35 mmol/l (±0.829), occuring over a 1.72 - 5.30 mmol/l range. These results would suggest that individual differences must be recognized (Stegmann et al, 1981), and that individual determination of the lactate threshold at a set value of 2 or 4 mmol/l (Kindermann et al, 1979; Sjodin et al, 1981) is not possible, being subject to large factors of error.

The relationship between blood lactate and EXCO2 was investigated over an eleven minute range (5 min. pre and post turnpoint) in the present study. In order to compare subjects the EXCO2 and lactate turnpoints were used as relative points for comparision. When the two curves were compared in this

manner it was found that changes in EXCO2 closely tracked changes in blood lactate, although the relationship was different for both males and females. Approximately 50% of the variance in blood lactate concentration could be accounted for by changes in expired excess CO2 when using the turnpoints as relative points of reference (r=0.69, P(0.001). Individual correlations over the same period of time ranged from 0.82 to 0.96 (P(0.001). This data is in agreement with the earlier findings of Bouhuys et al (1966) who found a correlation coefficient of r=0.80 between changes in blood lactate concentration and changes in excess CO2, although the grouped correlation is much lower than those results found by Issekutz and Rodahl (r=0.92, 1961). The lower correlation coefficient found for the group in this study was largely influenced by the combination of the two sexes (male r=0.72, female r=0.78), while Bouhuys et al (1966) found age to be an influencing factor. The reflection of intracellular lactate concentrations by pooled venous blood lactate levels must be acknowledged as a source of error (Brooks, 1986), while the CO2 storage capacity of the individual must also be acknowledged (Clode et al, 1967). Previous studies have suggested however, that the use of a CO2 balancing technique does allow changes in blood lactate to be determined with reasonable accuracy (Clode et al, 1969; Clode et al, 1967; Bouhuys et al, 1966; Issekutz et al, 1961). This study suggests that expired EXCO2 may be reflecting intracellular lactate production, as suggested by Volkov et al (1975). The significant differance between an increase in expired EXCO2 and

blood lactate accumulation may reflect the delayed release of lactate into the blood stream. While EXCO2 is freely diffusable across the cell membrane a translocation hinderance may prevent the rapid diffusion of lactate across the cell membrane as suggested by Stainsby (1986). Although the blood transit time to the collection site must be considered as contributing to the delay, during strenous exercise the transit time is decreased and would only account for a very small portion of the observed difference.

The average excess CO2 threhold value was 14.04 ml/kg/min (±2.72). The mean absolute lactate concentration at the threshold point was 3.35 mmol/l (±0.829), in close agreement with Hearst (1982 unpublished data). The wide range of threshold values, 1.72 - 5.30 mmol/l, suggests that this point is highly dependent upon the individual and the setting of predetermined threshold values would not allow for this inter-individual variation.

Previous studies investigating the relationship between EXCO2 production and performance (Hearst, unpublished 1982; Rhodes et al, 1981; Volkov et al, 1975) have found that a significant relationship does exsist between these two variables. The previous studies have suggested that the index of EXCO2 is directly related to the magnitude of lactic acid production through the glycolytic pathways and the organism's buffering capacity. The EXCO2 turnpoint may represent the onset of metabolic acidosis, as suggested by the relationship between the turnpoint and performance variables, while the relationship

between metabolic acidosis and fatigue has been well documented (Wenger et al, 1976).

VE/VO2 threshold points are harder to discern than those threshold points in the expired EXCO2 and blood lactate accumulation. The VE/VO2 threshold points do, however, allow the blood lactate threshold points to be predicted with a high degree of accuracy. EXCO2 may offer a valuable noninvasive method of determining the point where intracellular lactate production and consumption are unbalanced and the rapid onset of metabolic acidosis occurs. Further investigation into the relationship between intra-cellular lactic acid production and the release of EXCO2 may allow performance variables to be determined with greater care and accuracy. New research technologies will allow this relationship to be better documented in the future.

CHAPTER FIVE:

SUMMARY AND CONCLUSIONS

SUMMARY

Significant correlations were found between the threshold points as determined through three independent indices (blood lactate, excess CO2, and the ratio VE/VO2). The best correlation between any two of the threshold points was found between the threshold points as determined through the blood lactate index and the excess CO2 index (r=0.95). A correlation was found of r=0.91 between the threshold points as determined through the blood lactate index and the VE/VO2 index, while a correlation of r=0.92 was found between the VE/VO2 index and the excess CO2 index.

A significant F ratio was found when comparing the threshold points as determined through the three independent indices (F=8.41, P<0.001). Post hoc tests revealed a significant difference between the threshold points as determined through blood lactate and through excess CO2 (P<0.001), and between the excess CO2 index and the VE/VO2 index (P<0.025). There was no significant difference found between the blood lactate and VE/VO2 indices (P>0.05).

The mean absolute lactate concentration at the threshold point was 3.35 mmol/l (±0.829), while the range of threshold values varied from 1.72 - 5.30 mmol/l. The average excess CO2 threshold value was 14.04 ml/kg/min (±2.72).

Blood lactate concentration correlated significantly (r=0.69, P<0.001) with expired excess CO2 volume over an 11

minute range across the threshold points. Higher (0.82 - 0.96) correlation coefficients obtained for each individual express the independent nature of the relationship between EXCO2 and blood lactate. Excess CO2 volume appeared to track blood lactate levels throughout the progressive intensity test, although excess CO2 volume increased at a significantly lower level (P(0.001) than did blood lactate, with an average time delay in the appearance of blood lactate of 1.35 minutes (VO2 difference of 0.193 l/min).

The relationship between expired excess CO2 volume and blood lactate concentration appeared to be sex dependant. The females (n=6) had a lower excess CO2 volume for each unit of lactate concentration as compared to the males (n=15). When compared on a relative scale, with the zero point at the threshold point as determined through each of the two indices, the relationship between blood lactate concentration and excess CO2 volume can be visualized.

Table 1.
Hypotheses:

variables	sig. correlation		sig. difference	
	sig./non	P	sig./non	P
LATT vs EXAT	significant	P(0.001	significant	P(0.001
EXTT vs VVTT	significant	P(0.001	significant	P(0.025
VVTT vs LATT	significant	P(0.001	non-signif.	P)0.050

Changes in expired excess CO2 volume appear to track changes in blood lactate concentration (r=0.69) when the two variables are put on a relative scale (accounting for the significant time difference between the two threshold points), although there is a wide variation between individuals in the exact nature of this relationship ie. blood lactate can not be predicted from excess CO2 with reasonable accuracy.

CONCLUSIONS

1. There is a strong relationship (r=0.91 - 0.95) between the transition thresholds as determined through the three indices.

2. Changes in expired excess CO2 volume precede changes in blood lactate concentration and the ventilatory equivalent (VE/VO2).

3. Changes in expired excess CO2 volume appear to track changes in blood lactate concentration (r=0.69, P(0.001), although the exact nature of this relationship depends on the individual and appears to be influeced by gender.

4. Blood lactate concentration can not be accurately predicted from expired excess CO2 volume.

5. Further investigation is required to determine the relationship between intra-cellular lactate concentration and expired excess CO2 volume.

6. The blood lactate concentration at the transition threshold may vary widely between individuals (3.35 mmol/l ±0.829), and should not be set at a predetermined value.

7. Changes in blood lactate concentration may not be reflecting changes in intra-cellular lactate concentration, with blood lactate thresholds not reflecting the rapid onset of metabolic acidosis. Excess CO2 may reflect increased intra-cellular lactate production with greater accuracy, making studies using blood lactate threshold points as their criterion reference suspect to wide margins of error.

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APPENDIX A

LACTATE THRESHOLDS:

A SUMMARY OF OBSERVER ESTIMATES

EXPRESSED IN MINUTES

N=21	OBSERVER			ACCEPTED	RANGE
#	#1	#2	#3	VALUE (MIN)	WIDTH
1	13.0	13.0	13.0	13.0	0.0
2	18.5	18.0	18.0	18.0	0.5
3	10.5	10.0	10.0	10.0	0.5
4	15.5	15.5	15.0	15.5	0.5
5	14.0	14.5	--	14.5	0.5
6	10.5	10.5	10.5	10.5	0.0
7	13.0	13.0	--	13.0	0.0
8	15.5	13.5	14.5	15.0	2.0
9	16.5	16.5	17.0	16.5	0.5
10	7.0	7.5	--	7.5	0.5
11	14.5	14.5	14.5	14.5	0.0
12	12.5	12.5	12.5	12.5	0.0
13	11.5	12.0	11.5	11.5	0.0
14	10.0	10.5	11.0	10.5	0.5
15	11.0	10.5	--	10.5	0.5
16	9.0	9.0	8.5	9.0	0.5
17	10.0	10.0	9.5	10.0	0.5
18	10.0	10.0	--	10.0	0.0
19	12.0	12.0	12.5	12.0	0.5
20	9.0	9.0	10.0	9.0	1.0
21	10.0	10.0	10.0	10.0	0.0
				AVERAGE	<0.5

EXCO2 THRESHOLDS:

A SUMMARY OF OBSERVER ESTIMATES

EXPRESSED IN MINUTES

N=21	OBSERVER			ACCEPTED	RANGE
#	#1	#2	#3	VALUE (MIN)	WIDTH
1	11.0	11.5	9.5	11.0	2.0
2	18.5	18.0	18.0	18.0	0.5
3	9.0	9.0	8.5	9.0	0.5
4	12.0	12.0	12.0	12.0	0.0
5	11.5	11.5	11.5	11.5	0.0
6	10.5	10.5	10.5	10.5	0.0
7	12.5	12.5	12.5	12.5	0.0
8	14.0	14.5	12.5	14.5	2.0
9	15.0	15.0	15.0	15.0	0.0
10	5.0	5.0	4.5	5.0	0.5
11	13.0	13.0	12.5	13.0	0.5
12	11.5	12.0	11.0	11.5	1.0
13	10.0	10.0	--	10.0	0.0
14	10.0	10.5	10.0	10.0	0.5
15	9.0	9.0	9.0	9.0	0.0
16	5.0	5.0	5.0	5.0	0.0
17	9.0	9.0	9.0	9.0	0.0
18	9.0	9.0	9.0	9.0	0.0
19	10.0	9.0	9.0	9.5	1.0
20	8.0	8.0	8.0	8.0	0.0
21	9.5	10.0	9.5	10.0	0.5
				AVERAGE	(0.5

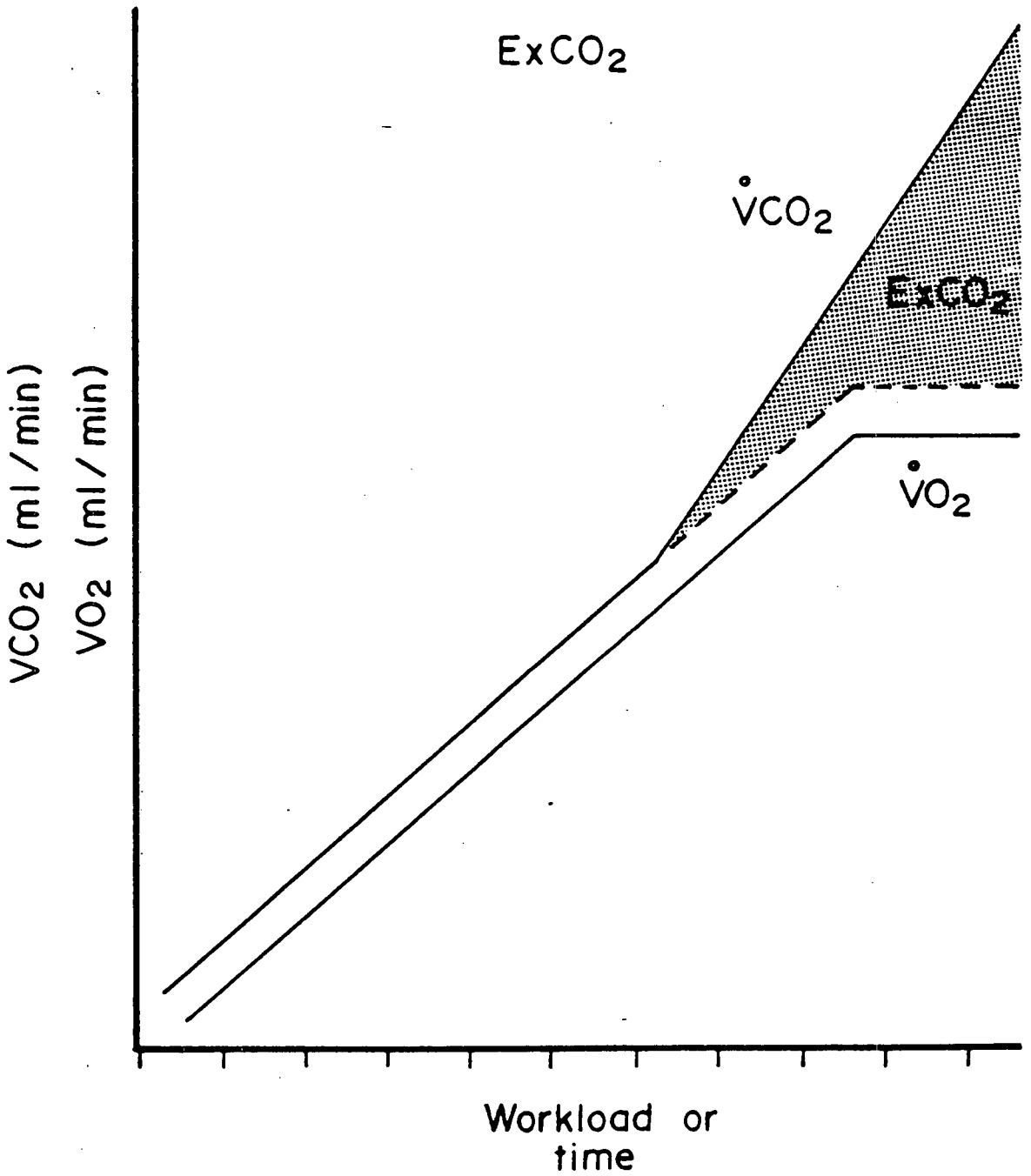
VE/VO2 THRESHOLDS:

A SUMMARY OF OBSERVER ESTIMATES

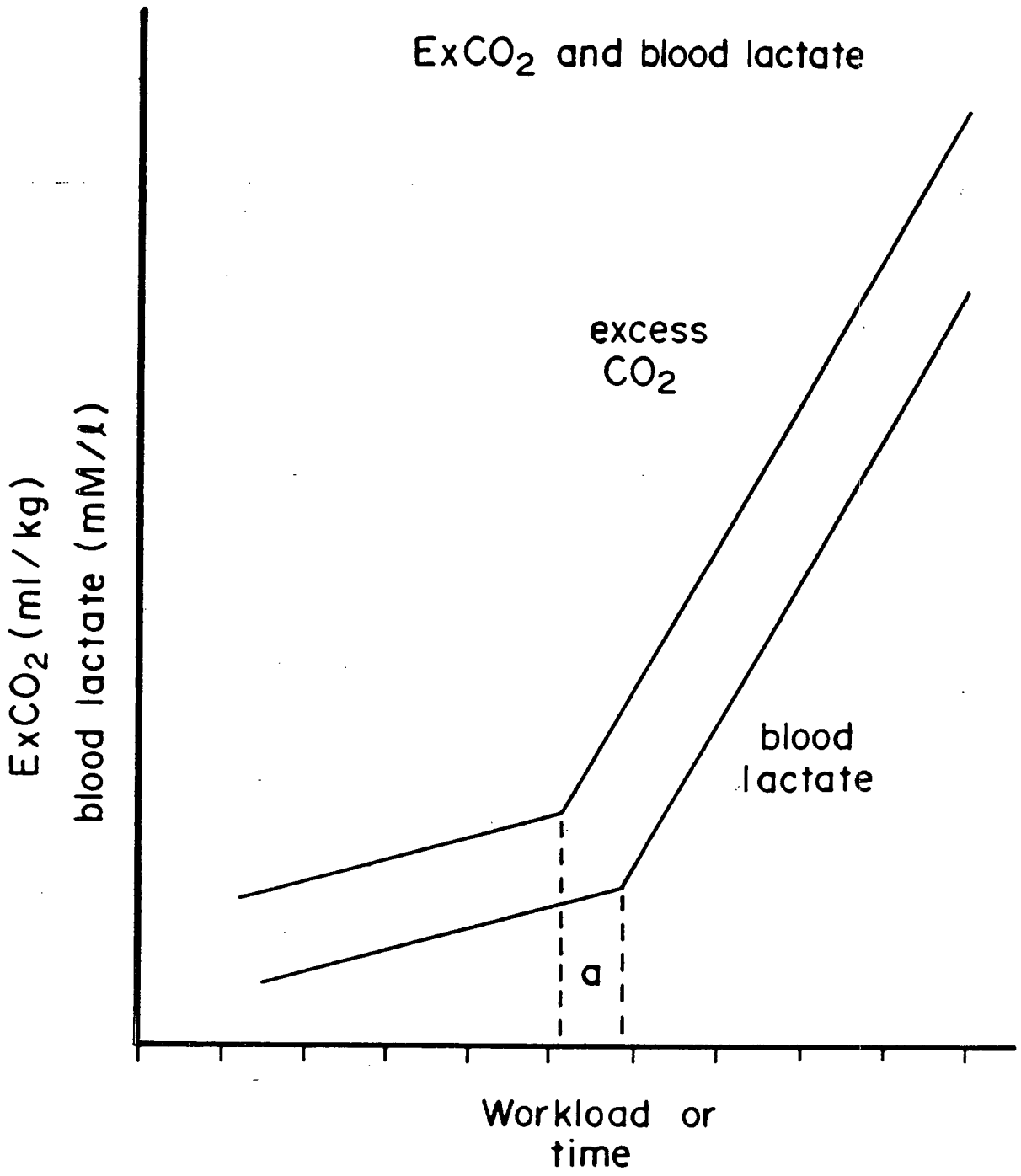
EXPRESSED IN MINUTES

N=21	OBSERVER			ACCEPTED	RANGE
#	#1	#2	#3	VALUE (MIN)	WIDTH
1	12.0	12.0	11.5	12.0	0.5
2	18.5	18.5	18.5	18.5	0.0
3	9.0	9.0	--	9.0	0.0
4	13.0	13.0	12.5	13.0	0.5
5	11.5	12.0	11.5	11.5	0.5
6	12.5	13.0	12.5	12.5	0.5
7	12.0	13.0	12.5	12.5	1.0
8	12.0	12.0	11.5	12.0	0.5
9	15.5	15.0	14.5	14.5	1.0
10	7.0	7.0	7.0	7.0	0.0
11	12.5	13.0	--	13.0	0.5
12	13.0	13.5	15.5	13.5	2.5
13	10.0	9.5	9.5	9.5	0.5
14	9.0	9.0	11.5	9.0	2.5
15	11.0	11.5	11.5	11.5	0.5
16	6.0	9.0	9.0	9.0	3.0
17	9.0	8.0	8.0	8.5	1.0
18	11.5	12.0	11.5	11.5	0.5
19	11.0	11.0	11.0	11.0	0.0
20	10.0	10.0	12.0	10.0	2.0
21	10.0	10.0	9.5	10.0	0.5
				AVERAGE	<1.0

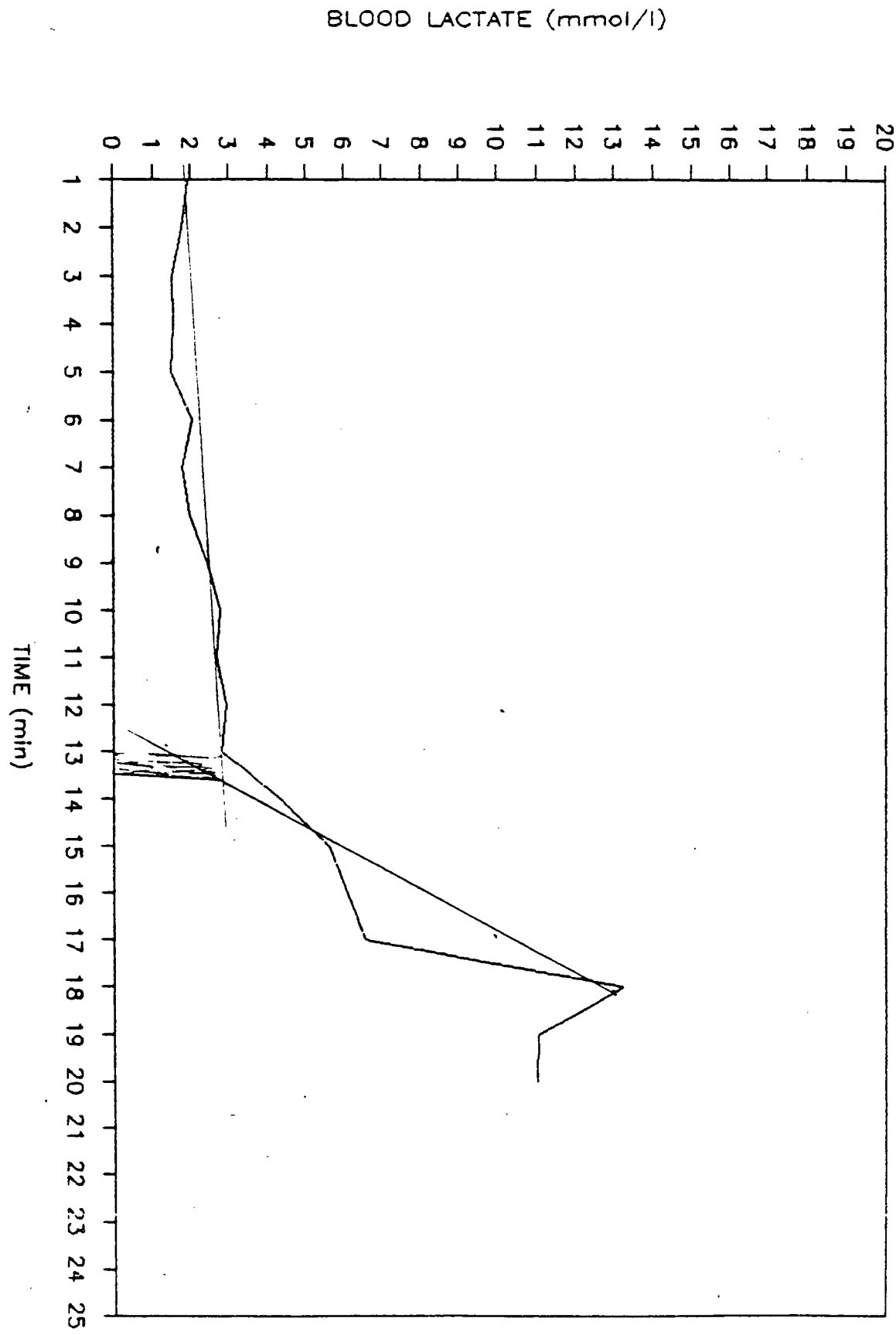
APPENDIX B

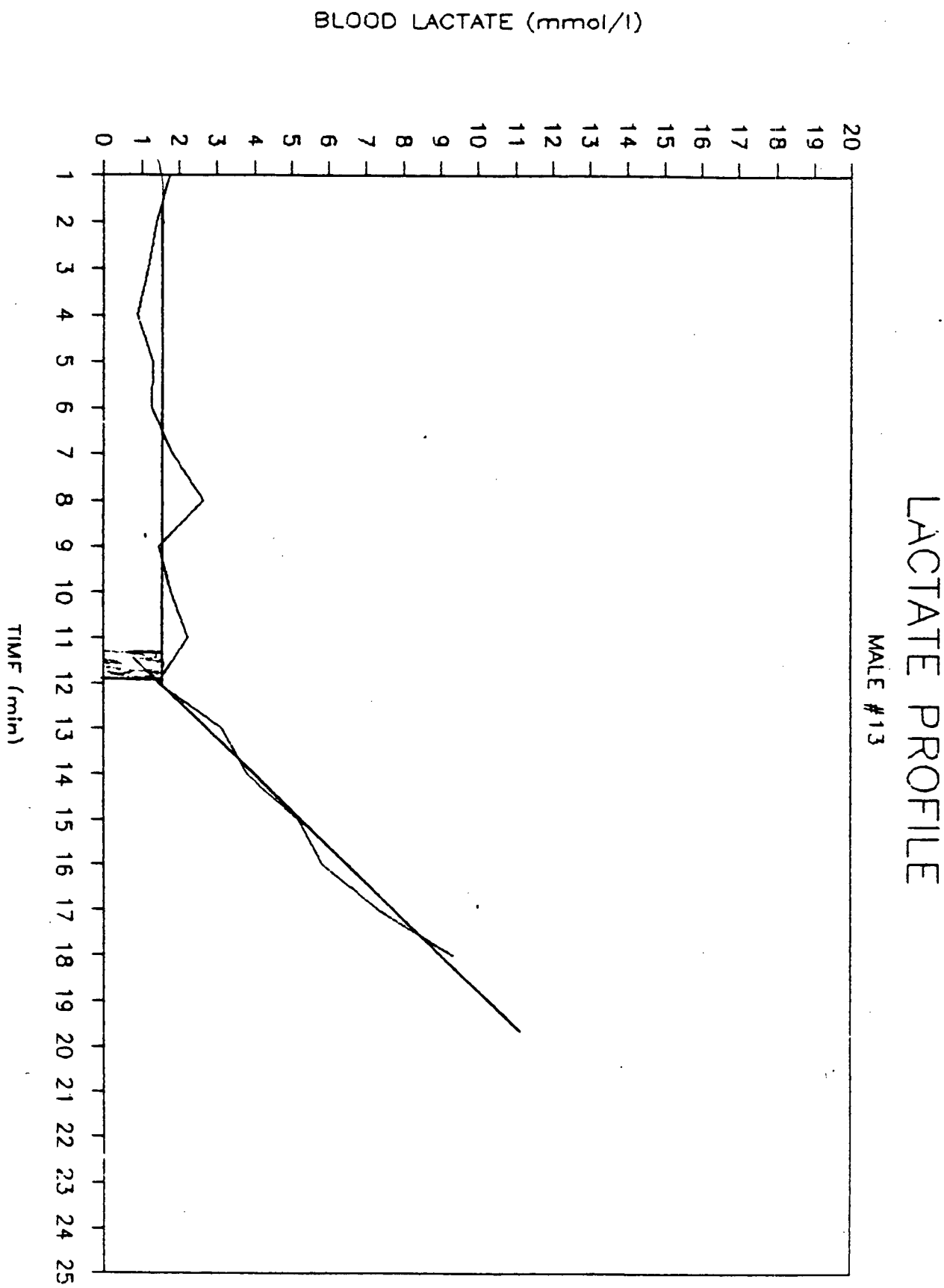

ExCO2
V̇CO2
ExCO2
V̇O2
VCO2 (ml/min)
VO2 (ml/min)
Workload or
time

APPENDIX C


ExCO2 and blood lactate
excess CO2
blood lactate
a
ExCO2 (ml / kg)
blood lactate (mM/l)
Workload or time

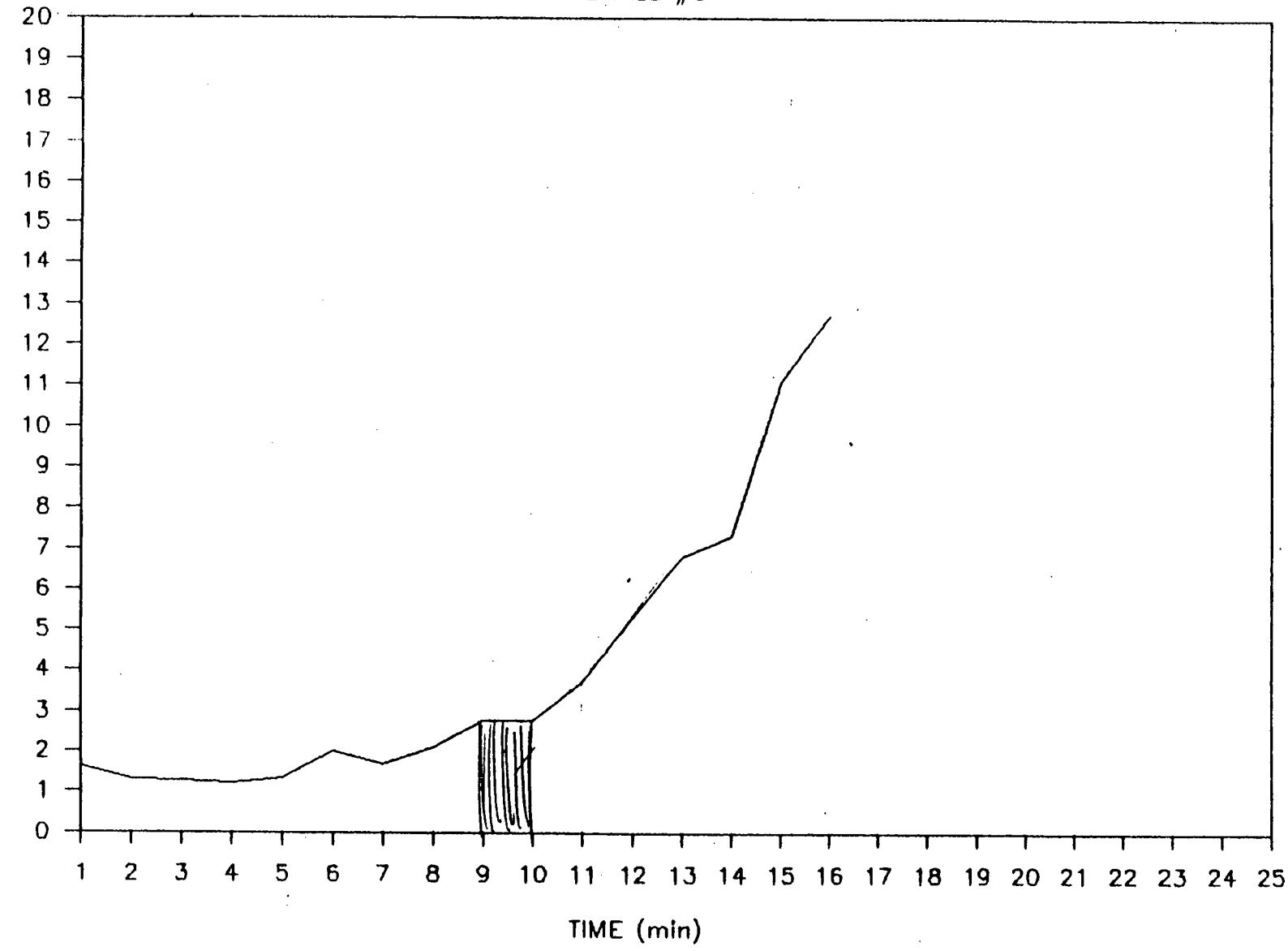
APPENDIX D


LACTATE PROFILE
MALE #1
BLOOD LACTATE (mmol/l)
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
TIME (min)
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25


LACTATE PROFILE
MALE #13
BLOOD LACTATE (mmol/l)
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
TIME (min)

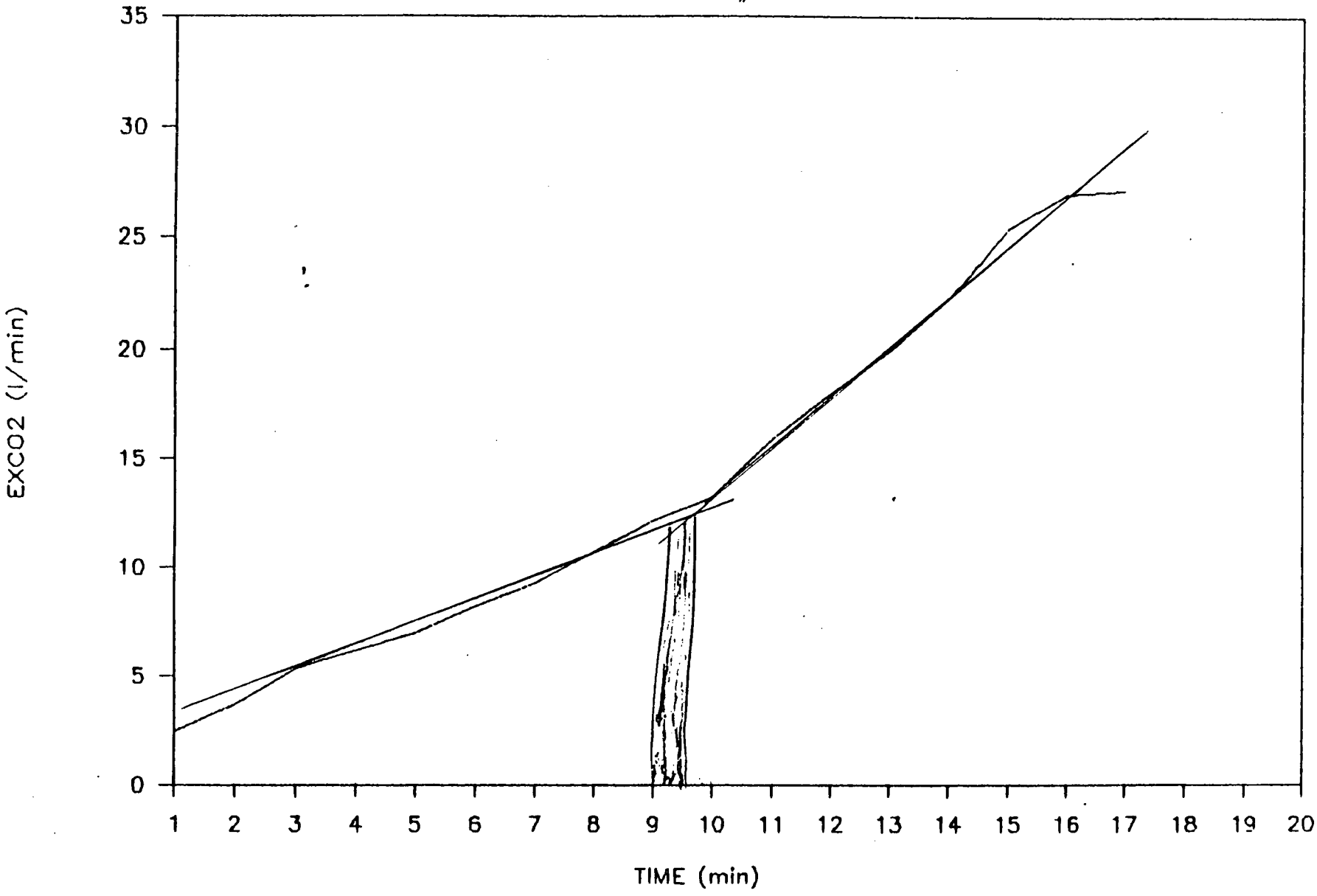
LACTATE PROFILE

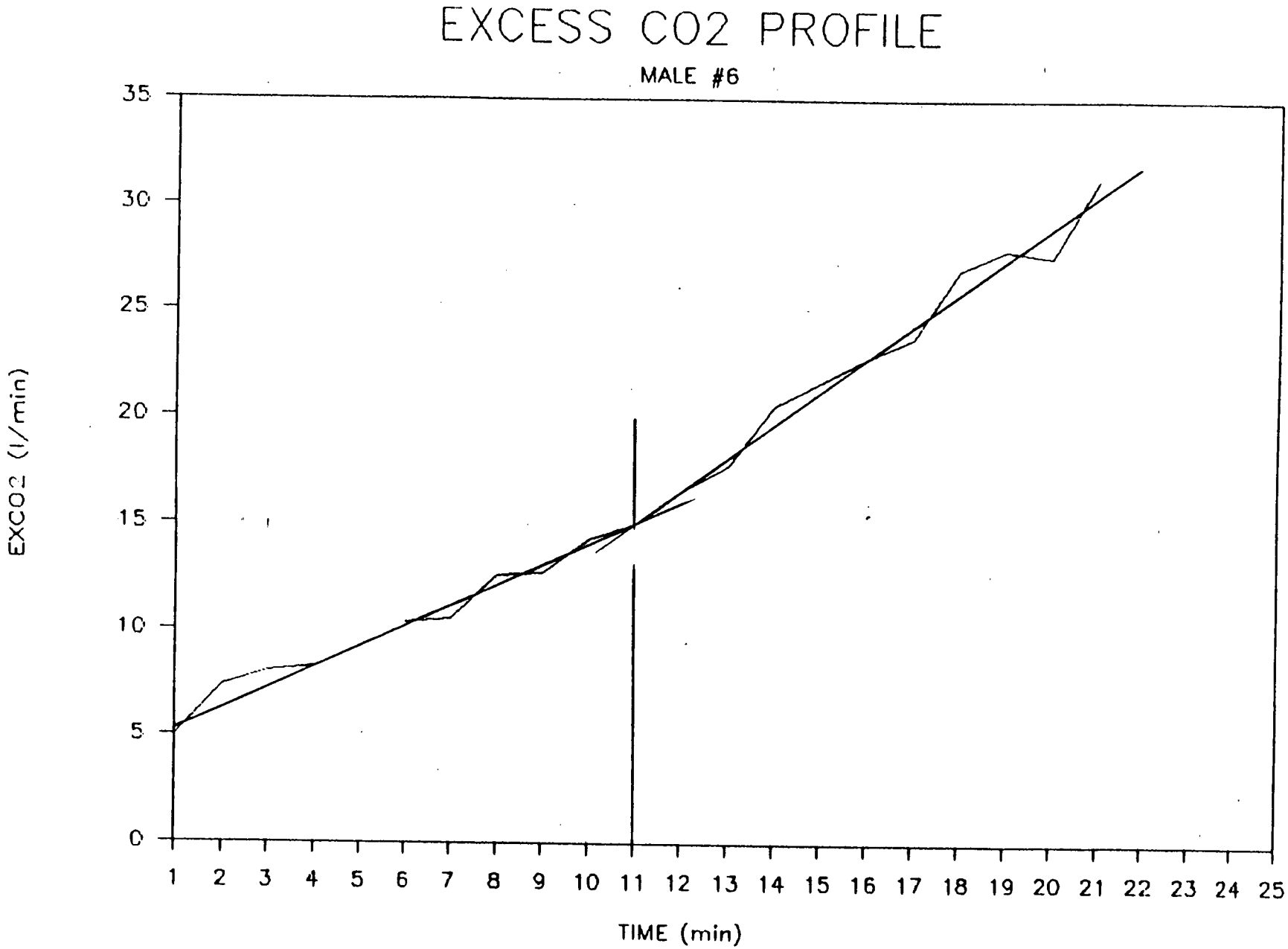
FEMALE #6

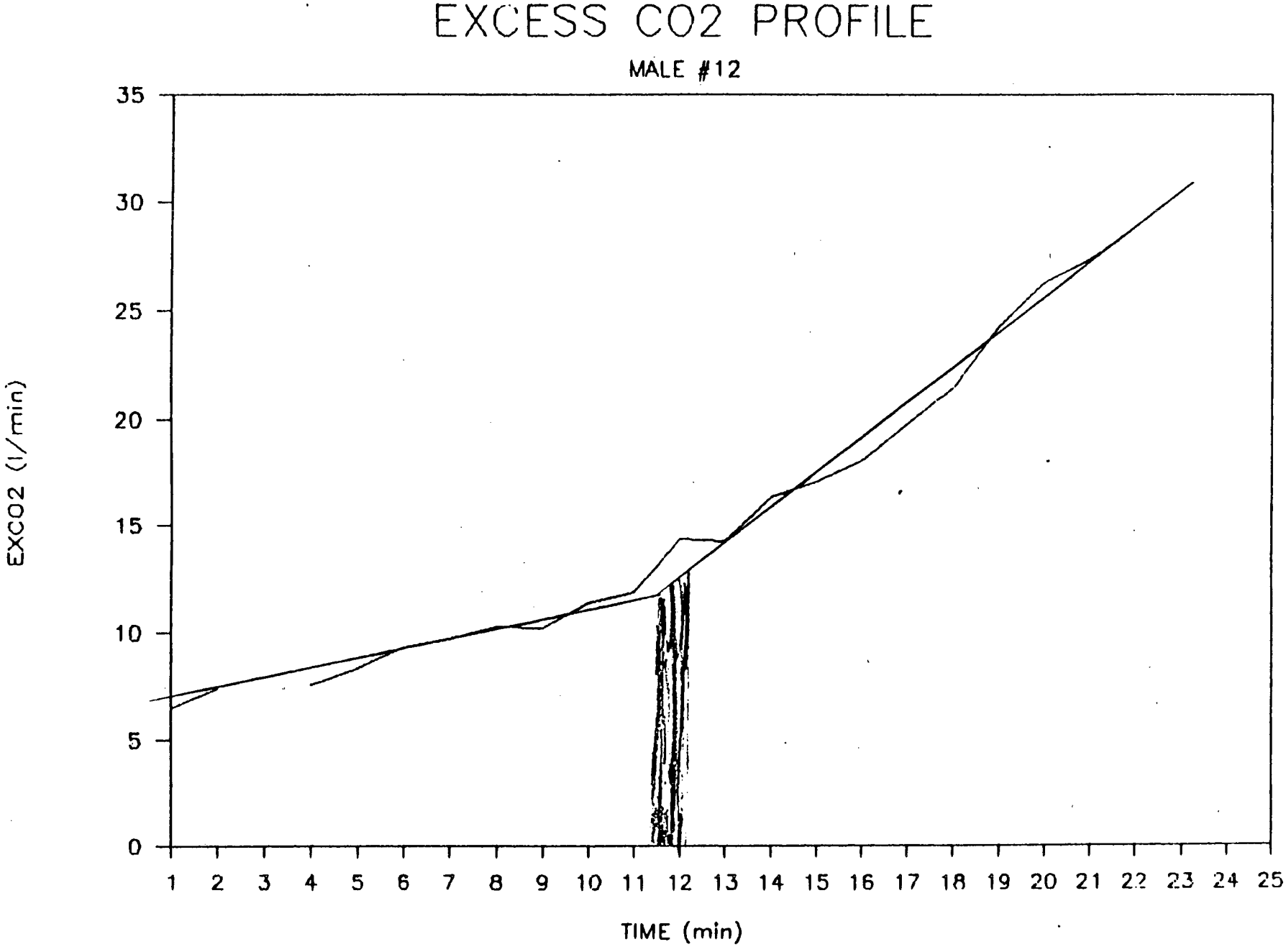


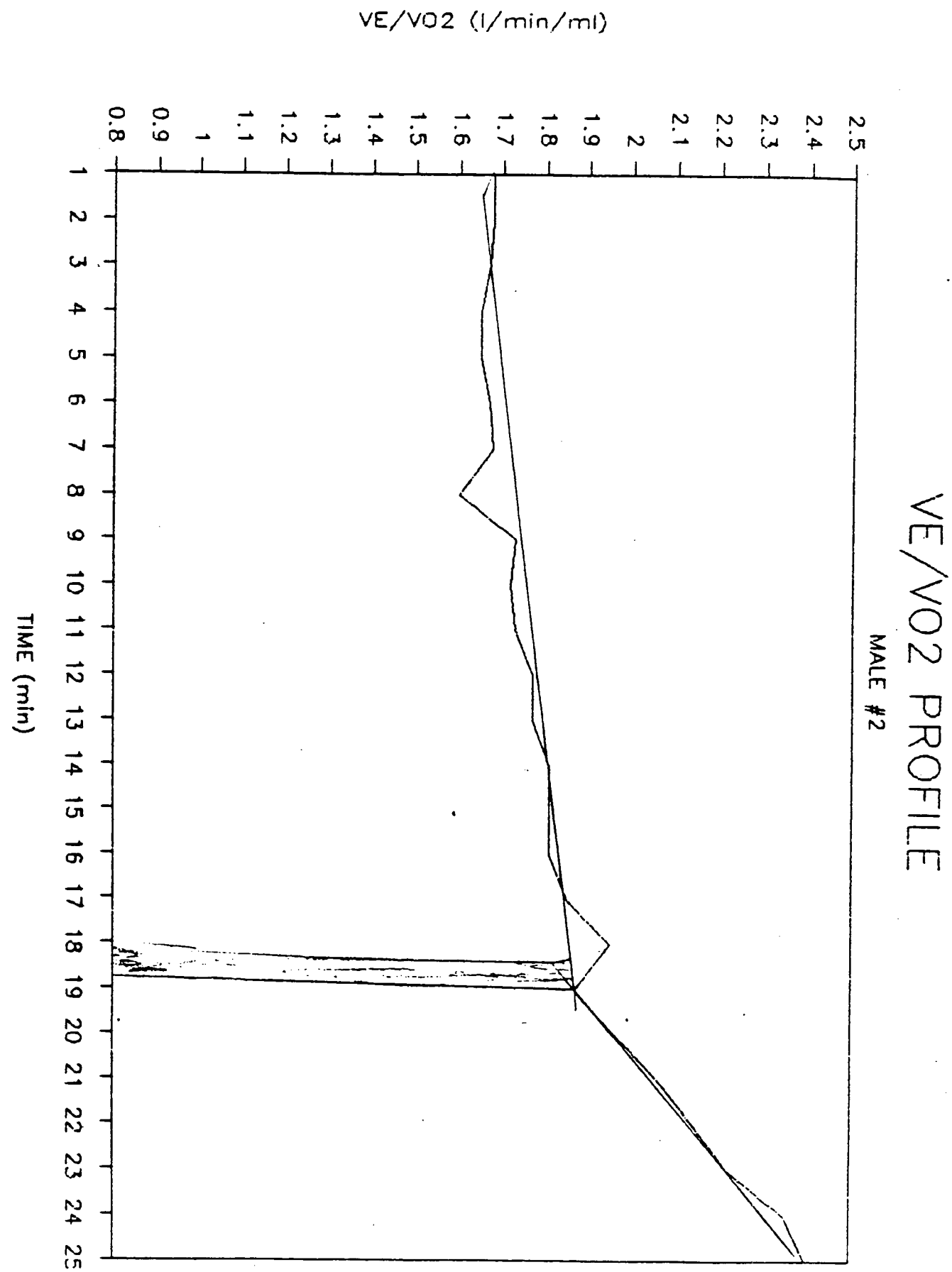
EXCESS CO2 PROFILE

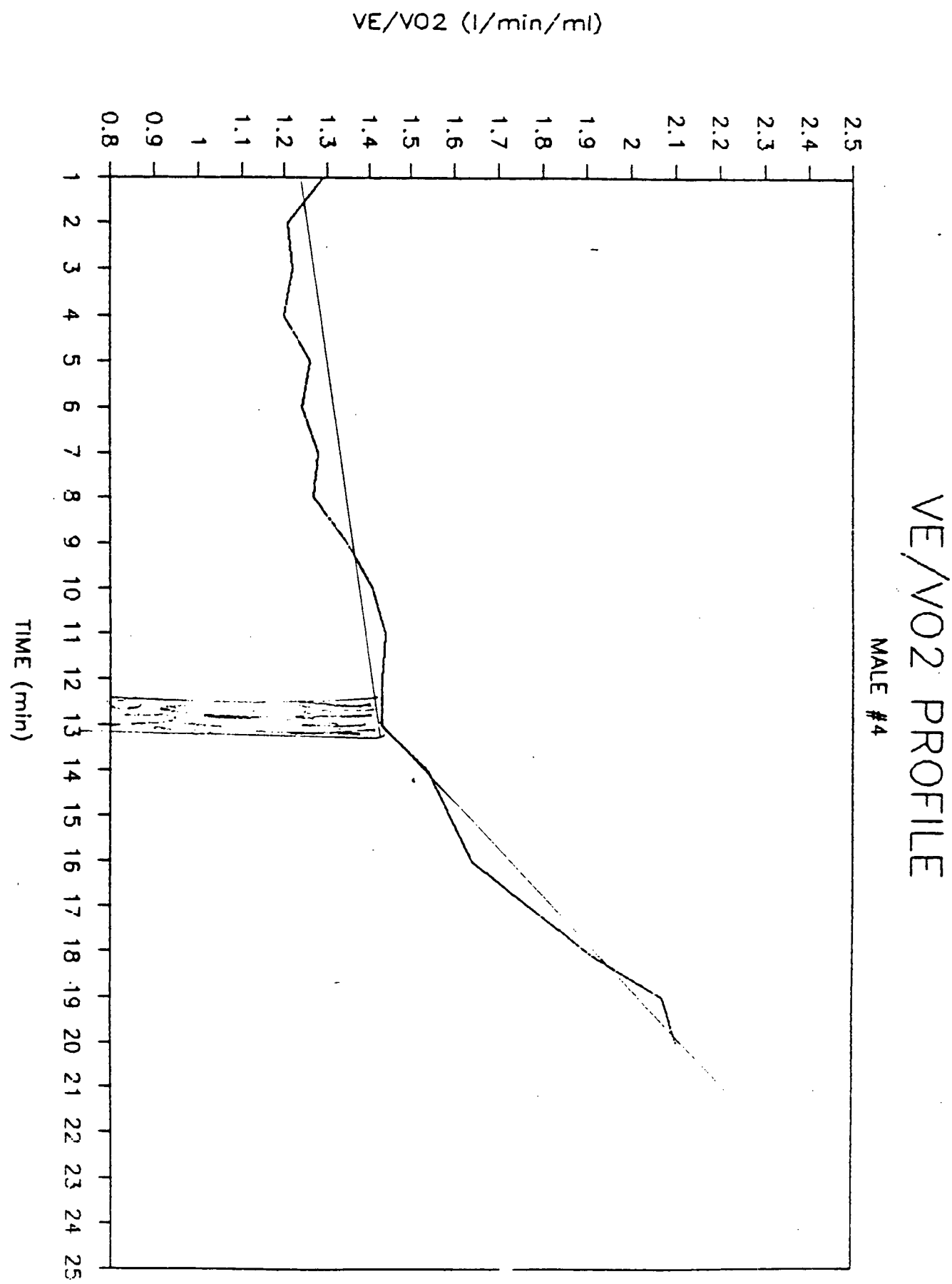
FEMALE #6

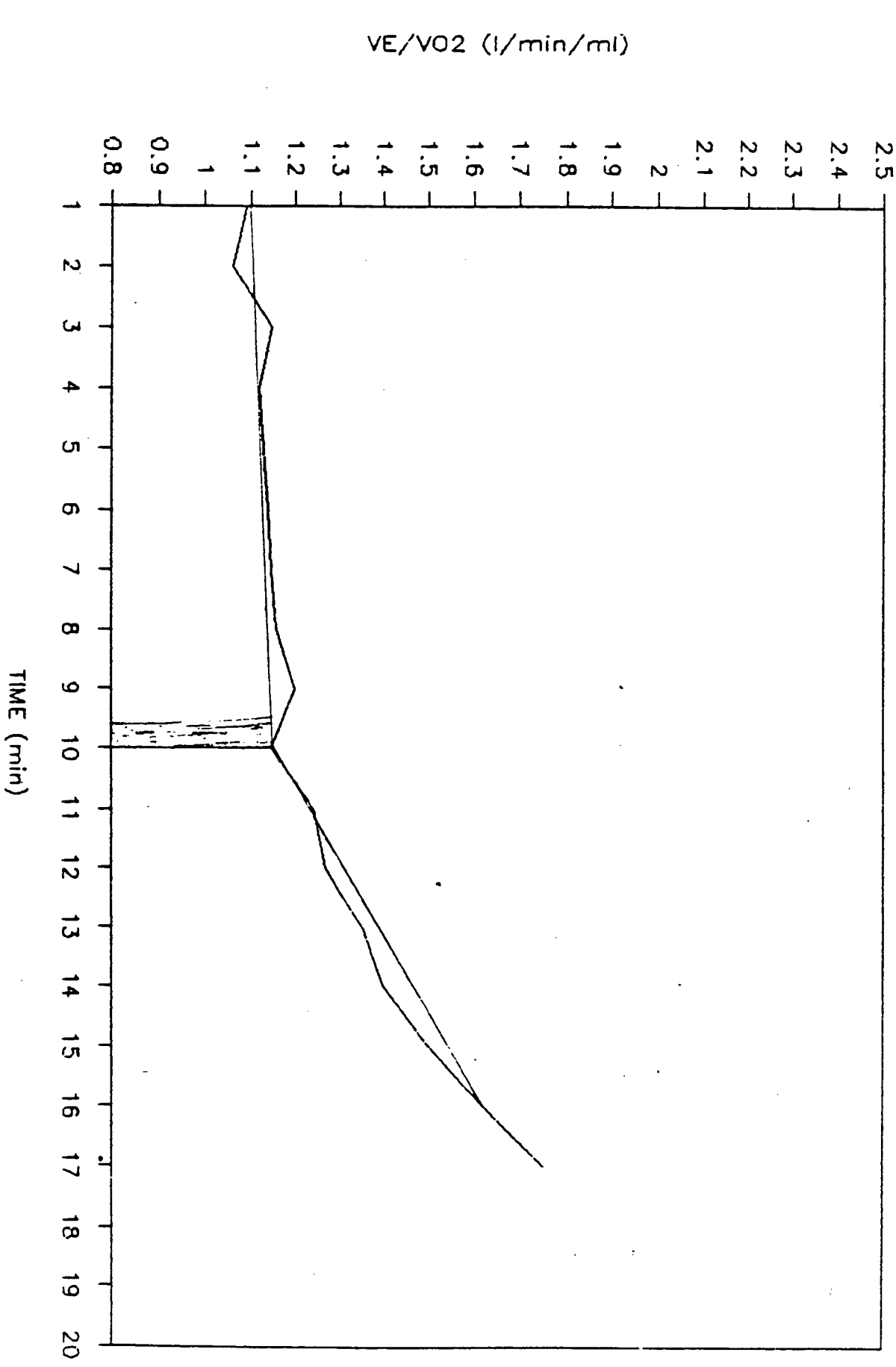



EXCESS CO2 PROFILE
MALE #6
EXCO2 (l/min)
0
5
10
15
20
25
30
35
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
TIME (min)

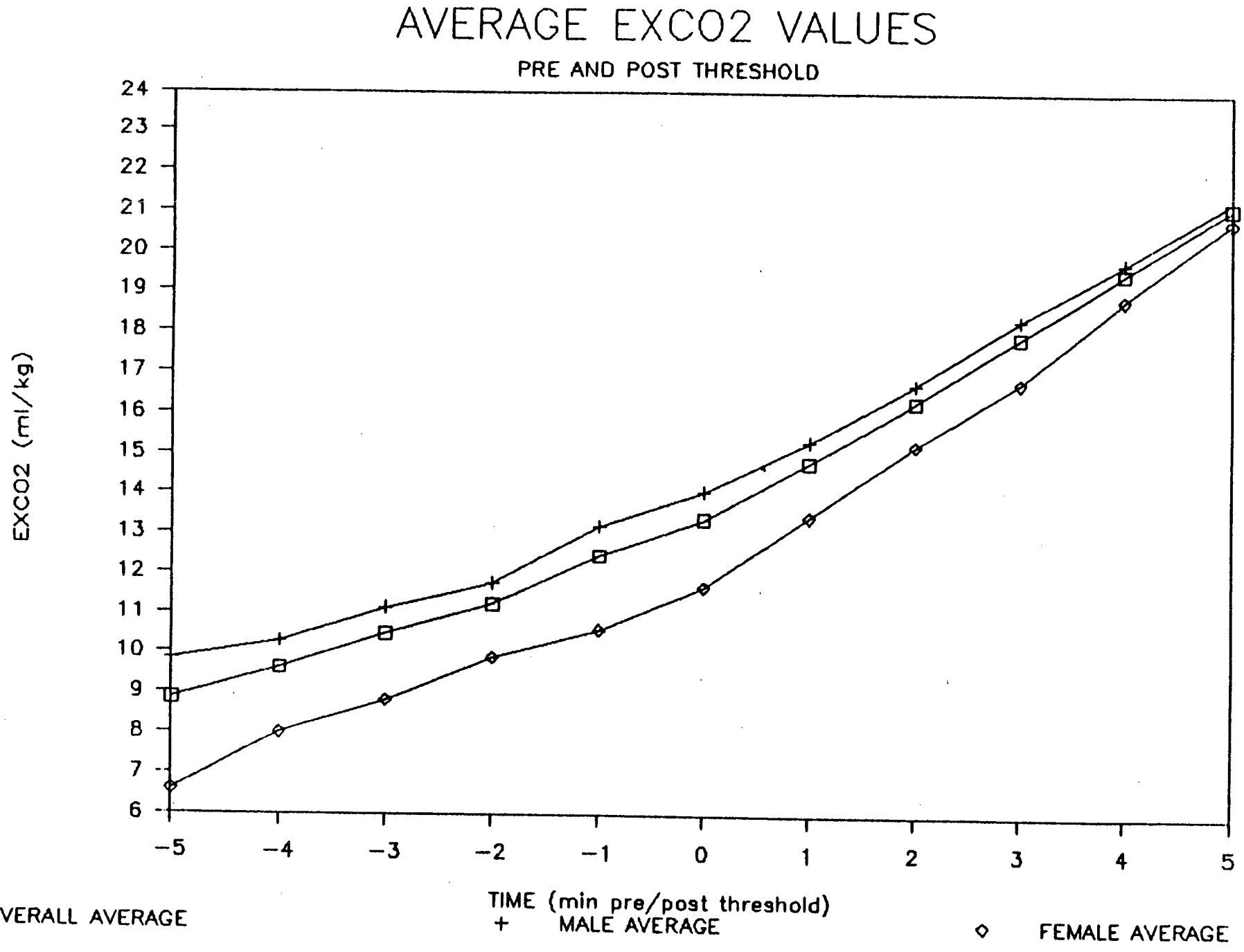

EXCESS CO2 PROFILE
MALE #12
EXCO2 (l/min)
0
5
10
15
20
25
30
35
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
TIME (min)


VE/VO2 PROFILE
MALE #2
VE/VO2 (l/min/ml)
2.5
2.4
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
TIME (min)


VE/VO2 PROFILE
MALE #4
VE/VO2 (l/min/ml)
2.5
2.4
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
TIME (min)


VE/VO2 PROFILE
FEMALE #6
VE/VO2 (l/min/ml)
2.5
2.4
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
TIME (min)
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20

APPENDIX E


AVERAGE EXCO2 VALUES
PRE AND POST THRESHOLD
EXCO2 (ml/kg)
24
23
22
21
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
−5
−4
−3
−2
−1
0
1
2
3
4
5
TIME (min pre/post threshold)
OVERALL AVERAGE
MALE AVERAGE
FEMALE AVERAGE

AVERAGE LACTATE VALUES

PRE AND POST THRESHOLD