

REPRODUCIBILITY AND VALIDITY OF TWO ANAEROBIC
THRESHOLD METHODOLOGIES WITH OVERWEIGHT WOMEN

by

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(ABSTRACT)

The twofold purposes of this investigation were: to determine the reliability and validity of two noninvasive methods for determining anaerobic threshold (AT) and; to examine the effect of an aerobic training program on the AT. Twelve sedentary, overweight women participated in an 8 wk walk/jog program 3 d•wk⁻¹ at 70% $\dot{V}O_{2max}$ with a concurrent hypocaloric diet. A computerized polynomial regression and four independent reviewers were used in an attempt to decrease the subjectivity of noninvasive AT estimation from data plots. Duplicate plots allowed determination of intra-reviewer variability for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ estimation. All reviewers fell within the 2-10% variability range accepted for research and clinical purposes,

respectively. Evaluation of variability among reviewers revealed that 100% and 83% of the Spearman Rho correlation coefficients were significant ($p < 0.05$) for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$, respectively. However, test-retest reliability (different days) of mean AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ estimates from the same reviewer demonstrated poor reproducibility; this implies that the observed variability is attributable more to physiological factors than to AT estimation technique of reviewers. Additionally, neither AT_{FEO_2} nor $AT_{\dot{V}E/\dot{V}O_2}$ demonstrated statistically significant correlations with a criterion variable of AT_{HL_a} . The $AT_{\dot{V}E}$ -computer regression method provided a reliable ($P = 0.85$, $p < 0.01$) and valid method ($P = 0.94$, $p < 0.01$) of AT_{HL_a} estimation in untrained individuals. Dependent t-tests revealed no significant differences for invasive and noninvasive AT values pre- to post-training. In summary, these results do not support the use of multiple reviewers to increase objectivity of noninvasive AT estimation.

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Chapter I

INTRODUCTION

The concept that an exercise-induced metabolic acidosis occurs at a particular oxygen uptake during incremental exercise was put forth over fifty years ago. Early work by Owles (1930) suggested that an exercise intensity exists above which the exercising muscles begin to produce large amounts of lactic acid. This increased rate of blood lactate accumulation is suggested to reflect a shift from predominantly aerobic to more anaerobic metabolism (Skinner & McLellan, 1980). The onset of this process was termed the "anaerobic threshold" in 1964 (Wasserman & McIlroy) and is currently defined as the oxygen uptake above which aerobic energy production is supplemented by anaerobic mechanisms resulting in a significant increase in lactate (Wasserman, 1984).

This increase in blood lactate is also correlated with ventilatory and respiratory compensation for metabolic acidosis. In 1973, Wasserman and colleagues suggested that the onset of metabolic acidosis (anaerobic threshold) could be discerned noninvasively from these ventilatory and gas exchange alterations. Today, use of noninvasive techniques to determine AT has become widespread.

However, anaerobic threshold (AT) is currently the subject of considerable controversy. Its underlying concept, terminology, and methods of detection are being actively debated. Disagreement exists concerning the validity of using noninvasive ventilatory and gas exchange measures to discern an intracellular event (Brooks, 1985). Yet, despite the controversy, the AT appears to be an invaluable concept with important implications to those in the fields of exercise science and rehabilitative medicine.

Current applications of the anaerobic threshold include identification of the optimal intensity for exercise prescriptions (Dwyer & Bybee, 1983), evaluation of the capacity for prolonged submaximal exercise (Farrell, Wilmore, Coyle, Billing, & Costill, 1979; Powers, Dodd, & Garner, 1984; Sjodin & Jacobs, 1981; Weltman, Katch, Sady, & Freedson, 1978), determination of endurance training effects (Davis, Frank, Whipp, & Wasserman, 1979), and the diagnosis and treatment of both cardiac and pulmonary patients (Wilson, Bell, & Norton, 1980).

Perhaps the most intriguing aspect of the anaerobic threshold is its relation to endurance performance. Current research indicates that the AT is a parameter that can predict the highest metabolic rate which can be maintained for extended periods. Thus, it is a critical factor in

determining an athlete's potential for prolonged exercise. Coaches and exercise rehabilitation specialists can optimize the metabolic and cardiopulmonary benefits of chronic exercise through the use of the AT to improve individualized prescriptions of exercise training intensity (Davis, 1985).

One aspect of AT related research currently receiving considerable attention involves refining the definition of this parameter and standardizing the protocols and techniques for its measurement. Another area focuses on determination of the optimal duration and training intensity necessary to increase the AT, and demonstration of its significance for exercise and work performance. The results of this investigation will add to the body of knowledge concerning the reliability and validity of two currently popular methods of anaerobic threshold determination.

Statement of the Problem

Recently, much attention has been given to the issues of reliability and validity of AT prediction, particularly through comparisons of invasive and noninvasive techniques (Ivy, Withers, Van Handel, Elger, & Costil, 1980; Reinhard, Muller, & Schmulling, 1979). Several investigators have supported the idea that AT can be effectively determined solely by ventilatory or gas exchange measures (Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976; Davis et al., 1979; Ivy et

al., 1980; Yoshida, Nagata, Muro, Takeuchi, & Suda, 1981; Caiozzo, Davis, Ellis, Azus, Vandagriff, Prietto, & McMaster, 1982; Reybrouck & Ghesquiere, 1984) while others have questioned this practice (Green, Hughson, Orr, & Ranney, 1983; Simon, Young, Gutin, Blood, & Case, 1983; Yeh, Gardner, Adams, Yanowitz, & Crapo, 1983; Powers et al., 1984; Brooks, 1985). A need for additional data on this issue is clearly indicated (Jones & Ehram, 1982).

The primary purpose of this investigation is to determine if two different methods accurately and reliably determine the anaerobic threshold as indicated by a blood lactate (HLa) of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$. The first method (AT-reviewer) involves the examination of data curves by four independent, outside reviewers. The second method (AT-computer) employs a two-component polynomial regression computer program to aid in estimation of the anaerobic threshold. Computer regression programs and the use of independent, outside reviewers represents two methods currently thought to decrease the subjectivity involved in visual discernment of the anaerobic threshold from data plots.

Secondly, the response of invasive and noninvasive anaerobic threshold indices to a short-term aerobic training and dietary intervention will be examined in a group of sedentary, overweight women. A review of the AT literature

failed to yield any previous research on untrained, overweight women undergoing such a treatment.

Research Hypotheses

HO₁ : Using the ventilatory threshold ($AT\dot{V}_E$ determined via a two-component polynomial regression computer method), there is no significant difference between anaerobic threshold values obtained on the same subject on two different days of testing, pre-training or post-training.

HO₂ : There is no significant difference between AT_{FEO_2} and $AT\dot{V}_E/\dot{V}O_2$ determined by informed reviewers using explicit criteria for AT determination based upon ventilatory and gas exchange indices, pre-training or post-training.

HO₃ : For those noninvasive AT estimation methods determined to be reliable, there is no difference between the indices for exercise time at AT correlated with a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ lactate concentration, pre-training or post-training.

HO₄ : Given reliable and valid methods of anaerobic threshold determination, there is no difference pre- to post-training in the exercise time associated with $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ lactate concentration following an 8 wk walk/jog program performed at 70% of subjects' peak oxygen consumption.

HO₅ : Given reliable and valid methods of anaerobic threshold determination, there is no difference pre- to

post-training in the HLa measured one minute before and after predicted AT during the submaximal treadmill test.

Significance of the Study

The results of this investigation will indicate the reliability (objectivity) for two currently popular methods of AT determination. Furthermore, the results will address the validity of these noninvasive methodologies for detecting the HLa level $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ (AT criterion). Establishment of reliable and accurate methods of AT detection would further encourage use of AT in future exercise testing, prescription, and evaluation.

It is generally known that the anaerobic threshold of an individual may be altered by physical training. However, the exact duration and intensity of training needed to produce this change is unknown (Skinner & McLellan, 1980). This study will assess the alterations in the invasive and noninvasive indices of the anaerobic threshold (HLa) that occur occur with aerobic training and dietary intervention in formerly sedentary, overweight women.

Delimitations

The following delimitations were imposed:

1. The sample was limited to twelve sedentary, overweight (body fat; \bar{x} = 32.2%) female volunteer (age; \bar{x} = 23.3 yrs) college students.
2. The training program was conducted only 3 d \cdot wk⁻¹ for 8 wk.
3. The training sessions consisted of aerobic exercise; walking/jogging at 70% of subjects' maximal oxygen capacity for 30 min.
4. Only three noninvasive indices for AT determination were utilized.
5. The invasive definition for AT was accepted as the exercise intensity associated with a 4 m \cdot mol \cdot L⁻¹ HLa.
6. During each submaximal exercise test, only two fingertip HLa measurements were taken at exercise times anticipated to yield 4 m \cdot mol \cdot L⁻¹ HLa from visual inspection of \dot{V}_E , $F_{E O_2}$, and $F_{E CO_2}$ plots. These sampling times were calculated from pre-training maximal exercise test data only. They were not recalculated post-training from maximal exercise test data taken at that time.
7. The lack of more than two blood lactate samples during exercise does not allow the determination of lactate "breakpoint" via regression analysis. Therefore, an

"idealized" lactate curve, derived from the literature, was utilized by the investigator to aid in determination of AT_{HLa} .

Limitations

The following limitations restrict the generalizability of the findings:

1. Due to a small sample size, results cannot be generalized to the population of overweight females as a whole.

2. Adherence to dietary and exercise requirements of this study was strictly voluntary, so variation may have occurred.

3. Due to the specificity of the exercise testing and training protocols, results from this study can be applied only to individuals involved in a walk/jog exercise training program performed at 70% $\dot{V}O_2$ max who were exercise tested utilizing a ramp-type treadmill protocol.

4. Findings are limited to three noninvasive indices currently used for AT detection.

5. The criterion measure of the anaerobic threshold in this study, $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa in fingertip blood samples, is only an indirect reflection of the rising anaerobic demands of active tissues during graded exercise.

6. Interpretation of changes in blood lactate concentration is limited because lactate concentration is the net result of synthesis, mobilization, and catabolism and does not reflect turnover rate (Brooks, 1985).

7. An "idealized" blood lactate curve, derived from the literature, was utilized in comparison of each subject's two fingertip blood lactate samples taken during the submaximal exercise tests to the AT criterion, $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration.

Definitions and Symbols

Terms and symbols requiring clarification for use in this study are as follows:

Aerobic/Anaerobic Metabolism. During progressive exercise, energy supply from aerobic metabolism (oxidative phosphorylation) may not be adequate and therefore, anaerobic metabolism (anaerobic glycolysis) must be utilized to supplement the energy supply (Yoshida et al., 1981).

Anaerobic Threshold (AT). During exercise, the oxygen consumption above which aerobic energy production is supplemented substantially by anaerobic mechanisms, resulting in a significant increase in active tissue and blood lactate (Wasserman, 1984).

AT-Computer. Determination of $AT\dot{V}_E$ values via a computerized two-component polynomial regression method.

AT_{FEO_2} . Anaerobic threshold determined as occurring at the point of systematic increase in F_{EO_2} without a corresponding decrease in F_{ECO_2} during increasing work (Withers, Sherman, Miller, & Costill, 1981).

AT_{HLa} . The criterion standard of the AT, a blood lactate concentration of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$.

AT-Reviewer. Determination of AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$ values by four independent, outside reviewers.

$AT_{\dot{V}_E}$. Ventilatory threshold for anaerobiosis is determined at the point just below a nonlinear rise in the $\dot{V}_E\text{-}\dot{V}O_2$ relationship (Wasserman et al., 1973, 1975; Davis et al., 1976, 1979).

$AT_{\dot{V}_E/\dot{V}O_2}$. Anaerobic threshold determined as occurring at the point of a systematic increase in $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ during increasing work (Caiozzo et al., 1982; Davis, Caiozzo, Lamarra, Ellis, Vandagriff, Prietto, & McMaster, 1983).

Exercise Prescription. The components of an exercise prescription include duration, frequency, type, and intensity based on physiological variables measured during a graded exercise test (ACSM, 1986).

Fraction of Expired Oxygen (F_{EO_2}). The percentage of oxygen in an individual's expired air (Wilson et al., 1980).

Fraction of Expired Carbon Dioxide ($F_{E}CO_2$). The percentage of carbon dioxide in an individual's expired air (Wilson et al., 1980).

Lactate or Lactic Acid (HLa). The end product of anaerobic glycolysis (Lamb, 1984) and glycogenolysis (Brooks, 1985). The net result of lactate production and lactate removal is a measurable lactate concentration in the blood (HLa), usually expressed in $m \cdot mol \cdot L^{-1}$ (Lamb, 1984).

Maximal Graded Exercise Test. An incremental ramp-type treadmill test designed to exhaust subjects in 8-12 min eliciting maximal physiological data (Whipp, Davis, Torres, & Wasserman, 1981). Also referred to as GXT_1 or GXT_3 .

Maximal Oxygen Uptake or Consumption ($\dot{V}O_{2max}$). The maximal rate at which oxygen can be consumed per minute by an individual during exhausting exercise; the power or capacity of the aerobic system, expressed in $liters \cdot min^{-1}$ or $ml \cdot kg^{-1} \cdot min^{-1}$ (Fox & Matthews, 1981).

Minute Ventilation (\dot{V}_E). The volume of air expired (\dot{V}_I) or inspired (\dot{V}_E) in one minute; usually it refers to the expired amount (Fox & Matthews, 1981), expressed in $liters \cdot min^{-1}$.

Nutritional Program. Is a weekly program designed to give subjects information regarding nutritional principles,

caloric content of foods, and behavior modification techniques (Toepfer, 1984).

Respiratory Exchange Ratio (R). The ratio of the volume of carbon dioxide expired per minute to the volume of oxygen consumed during the same time interval (i.e., $\dot{V}CO_2/\dot{V}O_2$) (Fox & Matthews, 1981).

Submaximal Graded Exercise Test. A treadmill exercise test that consists initially of approximately five increments of increasing speed and/or grade identical to the maximal graded exercise test and then continues at a constant speed and grade corresponding to 70% of subjects' $\dot{V}O_{2max}$ for as long as subject can continue or for 20 min (Toepfer, 1984). Also referred to as GXT₂ or GXT₄.

Ventilatory Equivalent for Oxygen ($\dot{V}_E/\dot{V}O_2$). The ratio of expired minute ventilation to the volume of oxygen uptake (Wilson et al., 1980).

Ventilatory Equivalent for Carbon Dioxide ($\dot{V}_E/\dot{V}CO_2$). The ratio of expired minute ventilation to the volume of carbon dioxide produced per minute (Wilson et al., 1980).

Oxygen Uptake ($\dot{V}O_2$). The amount of oxygen consumed per minute (Fox & Matthews, 1981).

Weight Reduction Program. A 1200-1400 kcal·d⁻¹ diet based on the diabetic exchange list diet (American Diabetes

Association, Inc., 1976). The diet is designed to result in weight loss of approximately $1 \text{ kg}\cdot\text{wk}^{-1}$ (Toepfer, 1984).

Basic Assumptions

1. Subjects exercised to a maximal physiologic level on the maximal treadmill tests.

2. Subjects adhered to their respective exercise prescriptions and did not engage in endurance activities other than the treatment program.

3. Subjects adhered to their hypocaloric diets.

4. A blood HLa of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$. is indicative of exercise intensity causing a substantial rise in active tissue anaerobiosis.

5. The "idealized" blood lactate curve formulated for use in this investigation represents the typical blood lactate response to incremental treadmill exercise.

Chapter II

REVIEW OF THE LITERATURE

The literature pertinent to this investigation is presented in three major sections. The initial section includes a background review on aerobic-anaerobic metabolism and possible mechanisms related to the concept of anaerobic threshold. The second section addresses the invasive and noninvasive indices of AT and employment of two different methods of "objectifying" AT determination. Research related to the "optimal" protocol to aid in AT determination is also included. The third and final section summarizes the effect of aerobic training on the anaerobic threshold.

Physiological Basis of the Anaerobic Threshold

The concept that an exercise-induced metabolic acidosis occurs at a particular oxygen uptake during incremental exercise was put forth over fifty years ago (Hill, Long, & Lupton, 1924); yet this concept was first termed the "anaerobic threshold" in 1964 (Wasserman & McIlroy).

Since 1964, the definition of "anaerobic threshold" has undergone numerous revisions. It is currently defined as the oxygen uptake above which aerobic energy production is supplemented by anaerobic mechanisms resulting in a significant increase in lactate (Wasserman, 1984).

When exercise intensity is progressively increased, energy supply from aerobic metabolism (oxidative phosphorylation) may not be adequate to sustain levels of ATP regeneration. Therefore, anaerobic metabolism (anaerobic glycolysis) must be utilized to supplement the energy supply. The acceleration in anaerobic glycolysis leads to an elevated muscle lactate concentration and a consequent metabolic acidosis (Sahlin, 1978). Therefore, the onset of an exercise-induced metabolic acidosis is thought to be "near the level where non-oxidative [anaerobic] metabolism begins to play a more predominant role in energy production" (Sady, 1980, p. 41). It is also thought that the increased blood lactate concentration levels reflect this shift, indicating an increased dependency on anaerobic glycolysis (Gollnick, 1973; MacDougall, 1977).

Energy Metabolism. Metabolism can be divided into aerobic and anaerobic phases, depending upon the availability of oxygen. The "supposedly clear-cut differences between the cellular conditions associated with either pyruvate or lactate being the end products of glycolysis have led to the terms aerobic and anaerobic glycolysis" (Karlsson & Jacobs, 1982, p. 190).

For the purpose of providing background information, a brief overview of aerobic and anaerobic metabolism is

provided at this point. Both aerobic and anaerobic metabolism occur within the muscle cell. Anaerobic metabolism uses only carbohydrates (glycogen or glucose) as its fuel whereas aerobic metabolism can also utilize fats (free fatty acids). The last step of aerobic metabolism is oxygen-dependent while anaerobic metabolism can occur despite oxygen lack. Aerobic metabolism is identical to anaerobic metabolism in terms of the steps which lead to the formation of pyruvate via glycolysis. When the oxygen supply available to the mitochondria of the muscle cells is sufficient, the pyruvate formed by glycolysis enters the Krebs cycle, and subsequently, the electron transport system. When the oxygen supply is insufficient, electron transport and oxidative phosphorylation are slowed (Stainsby, 1986). The pyruvic acid formed by glycolysis while oxidative phosphorylation is slowed, does not continue to be degraded and enter the Krebs cycle but is converted instead to lactate, and diffuses out of the muscle cell into the blood (Karlsson & Jacobs, 1971).

In the aerobic processes, oxygen serves as a hydrogen acceptor. If oxygen is not present in sufficient quantities to oxidize the NAD^2H , pyruvic acid acts as a temporary hydrogen acceptor. In the presence of the enzyme lactic dehydrogenase, the pyruvic acid is reduced to lactic acid, while NAD^2H is simultaneously reoxidizing allowing glycolysis

to proceed. However, "the fact that lack of adequate O_2 can cause such an imbalance of glycolysis relative to oxidative phosphorylation does not mean that lactic acid production always means there is a lack of O_2 " (Stainsby, 1986, p. 341).

Aerobic metabolism involves oxidation of glucose or glycogen via three major series of chemical reactions. During the first series of reactions, called glycolysis, pyruvic acid is formed and 2-3 ATP resynthesized. During the second series of reactions, known as the Krebs cycle, pyruvate enters the cell's mitochondria, attaches to coenzyme A, and enters the Krebs cycle where CO_2 is produced and electrons in the form of hydrogen atoms removed. The CO_2 diffuses into the blood and is carried to the lungs where it is exhaled. The final series of chemical reactions, involving the electron transport system, takes place in the inner membrane of the mitochondria. Hydrogen ions and electrons are "transported" to the oxygen by "electron carriers" (coenzymes NAD and FAD) in a series of enzymatic reactions; water is formed, and in the process ATP is resynthesized. Each pair of hydrogen atoms (electrons) carried down the chain produces three ATP for those attached to NAD and two ATPs for those attached to FAD. Overall, 12 pairs of electrons are removed from one mole of glycogen, resynthesizing 36 moles of ATP. The key to aerobic

metabolism is the ability of the electron transport system to accept the hydrogens (electrons) and pass them along the chain to combine eventually with oxygen. On the other hand, anaerobic metabolism (anaerobic glycolysis) takes place in the cytoplasm of the muscle cell (Gollnick & Hermansen, 1973) and involves the degradation of glycogen or glucose into pyruvic acid with a net gain of 2-3 ATPs per molecule. Thus, far more energy is produced as a result of aerobic metabolism (39 moles ATP) than that of anaerobic metabolism (2-3 moles ATP).

The breakdown of fatty acids (triglycerides) is strictly aerobic while carbohydrate breakdown may proceed either aerobically or anaerobically. When fatty acids are used as fuel for ATP resynthesis, they go through glycolysis, called beta oxidation, the Krebs Cycle, and finally, the electron transport system.

The Role and Importance of Lactate Production. The role of lactate production is to release some of the energy contained in glucose and transfer it to ADP for regeneration of ATP, which in turn, is used to fuel muscle contractions. Lactate production can serve as a supplement to aerobic energy production or as "an emergency method for ATP production" (Gollnick et al., 1986, p. 325) when insufficient oxygen is available. When oxygen flow to the mitochondria

is unable to meet all of the oxidative requirement for energy formation, anaerobic glycolysis supplements aerobic metabolism (mitochondrial oxidation), thereby conserving the oxygen that is ordinarily used in the mitochondria. This "sparing" of mitochondrial oxygen is achieved by the reoxidation of NAD²H by pyruvate rather than by the electron transport system. This allows maximal use of mitochondrial oxygen for ATP regeneration via the electron transport system. Thus, the production of lactate from pyruvate is an "oxygen-conserving" mechanism that allows the cell to obtain energy from glycogen or glucose even when oxygen supply is inadequate. The production of lactate serves as an essential anaerobic mechanism for the performance of prolonged and severe exercise. Yet, an individual can only exercise for a time that is dependent upon his ability to incur high lactate concentrations since muscle contractile and metabolic activity is limited by high lactate levels (Green, Houston, Thomson, Sutton, & Collnick, 1979; Issekutz, Shaw, & Issekutz, 1975).

Aerobic-Anaerobic Transition. The relative importance of the aerobic and anaerobic phases of metabolism to ATP resynthesis appears to be a junction of the intensity and duration of the exercise bout. Most investigators agree that anaerobic glycolysis and oxidative phosphorylation are the

primary metabolic processes involved in short-term, high-intensity exercise and prolonged, low-intensity exercise, respectively. There is some controversy, however, about the relative importance of each during the transition from aerobic to anaerobic metabolism during exercise of progressively increasing intensity. It is held by many proponents that the anaerobic threshold (AT) marks the aerobic-anaerobic transition point. In 1980, Skinner and McLellan proposed a hypothetical model to explain the aerobic-anaerobic metabolism transition. They identified three phases during the progressive transition from exercise of low to maximal intensity.

During Phase I, linear increases in $\dot{V}O_2$, \dot{V}_E , $\dot{V}CO_2$, and heart rate were observed. Little or no blood HLa accumulation occurred and R reached values of .7-.8. Thus, Skinner and McLellan concluded that this first phase (< 40% $\dot{V}O_{2max}$) primarily involved aerobic metabolism.

Phase II was said to occur when exercise intensity reached a point between 40-60% $\dot{V}O_{2max}$ and was characterized by a nonlinear increase in \dot{V}_E and $\dot{V}CO_2$, an increase in F_{EO_2} without a corresponding decrease in F_{ECO_2} , plus a rise in blood HLa from resting to a value lower than $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$.

The third and final phase occurred between 60-90% $\dot{V}O_{2max}$. At the onset of this phase, blood lactate is around

4 m•mol•L⁻¹ and then increases more rapidly until the subject attains his $\dot{V}O_2$ max. There is also a further increase in \dot{V}_E and a continuous rise in $\dot{V}CO_2$. F_{EO_2} decreases while F_{ECO_2} continues to rise.

In conclusion, Skinner and McLellan suggest that Phase III be designated the anaerobic threshold. The authors noted that both Phase II and III have been previously designated as the anaerobic threshold by various investigators (Green, 1979; MacDougall, 1978; Wasserman et al., 1973), depending upon their definitions of the anaerobic threshold.

Blood Lactate Accumulation. It has been known for years that lactate is produced during muscle contractions (Hill et al., 1923). Blood lactate concentration has been shown to increase virtually simultaneously with muscle lactate increases (Karlsson & Wigetz, 1971; Knuttgen & Saltin, 1972). It has been shown that lactate accumulation in the blood usually begins to occur at intensities representing 50-75% of subjects' $\dot{V}O_2$ max (Hermansen & Stensvold, 1972). The intensity of exercise that elicits an increase in lactate concentration in muscle and blood is highly variable and influenced by a number of factors such as the rate of lactate production, the rate of lactate diffusion from the cells to the blood, and the rate of lactate removal from the blood. Factors which influence the rate of lactate production and

removal include the amount of total muscle mass activated, the exercise intensity and duration, the pre-exercise concentration of glycogen in the working muscles, and the state of training of the involved musculature (Gollnick et al., 1986). Factors which are thought to determine the rate of lactate accumulation in the blood are the availability of oxygen, the number and type of muscle fibers recruited, the active muscle's potential for lactate formation, release, removal, and oxidation (Karlsson & Jacobs, 1981; Tesch, 1980) and the potential for other tissues to remove and metabolize blood lactate (Jorfeldt et al., 1978). Yet, despite much research in this area, the precise cause of the rise in blood lactate concentration above the anaerobic threshold remains unknown.

Possible Mechanisms of Lactate Increase During Exercise

Different viewpoints regarding the physiological mechanisms underlying the AT have been reported (Brooks, 1985; Karlsson & Jacobs, 1971; MacDougall, 1977; Skinner & McLellan, 1980; Stainsby, 1986; Wasserman and colleagues, 1964, 1967, 1973, 1984, 1986; and Yoshida et al., 1980, 1981). A brief overview of the mechanisms currently postulated include:

- 1) At the AT, lactate production results from oxygen-limited ATP production (local hypoxia).

- 2) At the AT, "the oxidative capacity of muscle is exceeded" (Davis, 1985, p. 14) despite adequate oxygen delivery.
- 3) The AT is a function of muscle fiber composition and pattern of recruitment.
- 4) The AT is the result of an exercise intensity at which there is a reduced hepatic clearance of blood lactate, not increased lactate production.
- 5) The AT is somehow a function of the shift in metabolic substrates used for energy production.

For detailed descriptions of possible mechanisms for the increase in blood lactate during exercise denoting the AT, refer to Brooks, 1985, 1986; Davis, 1985; Gollnick & Hermansen, 1973; Gollnick, Bayly, & Hodgson, 1986; Jones & Ehram, 1982; Karlsson & Jacobs, 1982; Stainsby, 1986; and Wasserman and colleagues, 1984, 1986. For purposes of this review, the five aforementioned mechanisms are summarized in the following pages.

Mechanism One: Lactate Production Results from Oxygen-Limited ATP Production. Until recently, exercise physiologists have attributed lactate production during exercise to "anaerobic glycolysis" secondary to local muscle hypoxia (Hollooszy, 1973; Karlsson et al., 1971; Saltin et al., 1971; Wasserman et al., 1973). This was based on the

close relationship observed between lactate accumulation in the blood and oxygen deficit during exercise.

In the early part of this century, several researchers observed that subjects could perform low to moderate exercise without an increase in blood lactate concentration above the resting value. However, it was observed that at higher exercise intensities, subjects reached a critical metabolic point (or "threshold") beyond which a progressive increase in blood lactate concentration occurred (Bang, 1936; Owles, 1930; Hill et al., 1924).

Hill et al. (1924) were among the first to postulate that blood lactate increased during exercise because of an inadequate supply of oxygen available to meet the energy requirements of the working muscles. In 1930, Owles reviewed the evidence which indicated an association between the blood lactate increase and the decrease in plasma bicarbonate resulting in what he referred to as the "metabolic acidosis of exercise." Margaria and colleagues (1933, 1964) developed the theory that one could exercise utilizing energy produced through aerobic metabolism up to a maximal level ($\dot{V}O_2\text{max}$). Above this point, additional energy demands were met via anaerobic glycolysis. In 1964, Wasserman and McIlroy first introduced the term "anaerobic threshold" and later defined it (Wasserman et al., 1973) as "the level of work or oxygen

consumption just below that at which metabolic acidosis and the associated changes in gas exchange occur" (p. 235). This metabolic acidosis was generally thought to be the direct result of local muscle hypoxia. Out of these classical studies grew the concept of a point or "threshold" occurring during incremental exercise beyond which further increases in exercise intensity were largely accomplished anaerobically with blood lactate increasing due to lack of adequate oxygen supply to the working muscle cells.

Recent controversy, however, centers on whether the exercise-induced lactate increase "relates at all to the adequacy of oxygen availability for energy production in the exercising muscle" (Wasserman et al., 1986, p. 344). A number of investigators have challenged the classical concept of the anaerobic threshold in light of current research (Brooks, 1985, 1986; Donovan & Brooks, 1983; Green, Hughson, Orr, & Ranney, 1983; Holloszy, 1973; Jones & Ehrsam, 1982; Karlsson & Jacobs, 1982; Stainsby, 1986) which demonstrates that there is always adequate oxygen in the muscle mitochondria during exercise and that the increase in lactate is independent of oxygen availability.

Holloszy (1973) and others (Brooks, 1985, 1986; Skinner & McLellan, 1980) have argued against the oxygen availability concept because they do not believe that lactate implies

hypoxia or that increased oxygen delivery can be responsible for training-induced decreases in blood lactate at a given submaximal work rate. Their reasoning goes like this: If untrained muscles are hypoxic during submaximal exercise because oxygen delivery is inadequate (hypoxia), and if improved oxygen delivery is responsible for lower muscle lactate production (via reduction of hypoxic condition), then the trained subject should have a higher oxygen consumption ($\dot{V}O_2$) than the untrained subject at a given submaximal workrate.

Since it is well documented that oxygen consumption ($\dot{V}O_2$) at a given submaximal workload is not increased by training, local muscle hypoxia cannot be the reason for changes in lactate concentration (Astrand & Rodahl, 1977; Divine-Patch & Brooks, 1980; Holloszy, 1976; Skinner & McLellan, 1980).

In an attempt to decrease any hypoxia occurring during exercise, Welch & Pedersen (1981) had subjects inhale oxygen-enriched gas mixtures while undergoing submaximal exercise. They found that the $\dot{V}O_2$ of their subjects did not increase during submaximal exercise possibly indicating that the subjects were not hypoxic during exercise.

A second argument against the oxygen availability (hypoxia) concept is proposed by those who cite evidence that

lactate is produced by well-oxygenated muscles; and therefore, increases in lactate are independent of oxygen availability (Brooks, 1985, 1986; Connett, Gayeski, & Honig, 1984; Jobsis & Stainsby, 1968; Stainsby, 1986).

One indicator of the availability of oxygen is the mitochondrial $[NAD]/[NADH]$ ratio (Stainsby, 1986). Hypoxia during muscle contractions causes the ratio to fall. Jobsis and Stainsby (1968) fluorometrically measured intracellular $[NAD]/[NADH]$ ratio despite lactate production. It was concluded that the muscles were well-oxygenated and that lactate production was not caused by "hypoxic stimulation of anaerobic glycolysis" (Jobsis & Stainsby, 1968, p. 292). The results of an investigation conducted by Carlson & Pernow (1961) indicate that lactate is produced regardless of a relatively high oxygen content and oxygen tension in the femoral venous blood during maximal exercise. Doll and co-workers (1968) reported that when a group of young men underwent strenuous exercise, the contracting muscles produced large amounts of lactate despite a more than adequate oxygen tension of approximately 21 mmHg in the femoral vein. This value is well above the critical oxygen tension (~ 21 mmHg) in the femoral vein, demonstrating that the increase in lactate production was apparently not due to local muscle hypoxia. Recent isotope tracer experiments on

isolated dog gracilis muscle (Connett et al., 1984) and with human subjects (Hetenyi, Perez, & Vranic, 1983) have shown that lactate production occurs under fully oxygenated conditions. This strongly suggests that lactate is produced in muscle for reasons other than lack of oxygen.

Wasserman (1984, et al., 1986), on the other hand, has hypothesized that alteration of the exercise oxygen requirement-availability balance does indeed influence lactate increase. To support the concept of muscle hypoxia during submaximal exercise, Wasserman (1984) cited research showing that lactate increase during exercise is influenced by acute changes in hemoglobin concentration, inspired oxygen concentration, and blood volume. He pointed out that experimental interventions that acutely lower oxygen delivery such as acute isovolemic anemia (Woodson et al., 1978), anemia (Whipp et al., 1981), and an increase in carboxyhemoglobin (COHb) (Vogel & Gleser, 1972) lower the anaerobic threshold and raise the level of blood lactate concentrations at submaximal workrates, providing support for the oxygen delivery mechanism of lactate increase (AT). Manipulation of blood flow to isolated contracting muscle also alters blood lactate levels (Idstrom et al., 1985). Conversely, inspiration of oxygen-enriched gas mixtures

(Hughes et al., 1968), decreases lactate concentration at submaximal workrates and increases the anaerobic threshold.

Wasserman (1984) pointed out that neither blood nor muscle lactate was measured in the study done by Jobsis and Stainsby (1968). Also, that the fluorometry method used was not sensitive enough to detect changes in the NADH/NAD ratio. Therefore, Wasserman concluded that this study does not "directly contradict the concept that lactate and the lactate/pyruvate ratio increase [during exercise] in the presence of relative oxygen lack" (p. 537).

Wasserman also refuted the conclusions put forth by Holloszy (1973); arguing that Holloszy "fails to recognize two [crucial] points in the anaerobic threshold-lactate increase concept" (p. 537). First, that lactate contributes toward the metabolic mechanisms only during the period of rising lactate concentration. And secondly, that during the period when lactate is increasing, the untrained compared to the trained have slower oxygen uptake kinetics for the same workrate (Whipp & Wasserman, 1972).

But how do Wasserman (1984) and others (Davis, 1985; Wasserman et al., 1986) account for the studies which demonstrate that oxygen flow into and out of the working muscle (Carlson & Pernow, 1961; Doll et al., 1968) is not low enough to suggest whole muscle hypoxia? They suggest that

"global adequacy [of oxygen] does not preclude local hypoxia" (Davis, 1985, p. 14) and that "the adequacy of oxygen for all muscle fibers in a contracting muscle cannot be assessed by methods depending on mean or predominant muscle PO_2 " (Wasserman et al., 1986, p. 344).

Mechanism Two: Overstimulation of Glycolysis. Some researchers use the term "anaerobic" to imply that anaerobic metabolism occurs because oxygen supply to the muscle is not sufficient to meet demand. Others use the term "anaerobic" to describe a metabolic pathway which does not require oxygen. These latter researchers feel that insufficient oxygen supply no longer seems to be the probable cause of lactate production during exercise and suggest that lactate in the blood results from an imbalance between the glycolytic and the oxidative activity of the cell (Gollnick, 1973; Holloszy, 1973; Rusko et al., 1980). This controversy over the meaning ascribed to the term "anaerobic" has prompted several investigators to define the "onset of metabolic acidosis" as the "anaerobic threshold", which is meant to serve as a functional definition without implying causality (Davis et al., 1979; Reybrouck & Ghesquire, 1984).

It is known that lactate production can occur in the presence as well as the absence of oxygen (Connett, et al., 1984; Jobsis & Stainsby, 1968). Lactate production has been

shown to occur in individuals at rest (Hetenyi et al., 1983) and during the initial workloads of exercise (Davis & Gass, 1981; Yeh, 1983). Therefore, the presence of blood lactate is not always indicative of muscle anaerobiosis (Brooks, 1985; Gollnick, 1986; Jones & Ehram, 1982).

Today it seems accepted that lactate production or accumulation can occur in the presence of oxygen due to an "overstimulation" of the glycolytic pathway with subsequent pyruvate formation and some reduction to lactate (Holloszy, 1976). According to Karlsson and Jacobs (1982), this "overstimulation" of glycolysis with subsequent lactate formation occurs at the onset of exercise and other transient conditions as well as under steady-state conditions when oxygen delivery is "strained".

Some contend that "overstimulation" of the glycolytic pathway possibly causes lactate to increase because of the limitation of the mitochondria to handle the products of glycolysis. This is sometimes referred to as the "mitochondrial enzyme availability concept" (Wasserman, 1984, p. 538). However, there is no direct evidence that a shortage of mitochondrial enzymes occurs thereby preventing adequate utilization of pyruvate. Possibly changes occur in the balance between the various regulatory enzymes, leading to the formation of pyruvate at a faster rate than that of

its utilization (oxidation) in the Krebs cycle (Jobsis & Stainsby, 1968).

Wasserman et al. (1986) hypothesizes that muscle lactate can increase because of two mechanisms. First, a rise in muscle lactate may occur because glycolysis increases so rapidly that the mitochondria cannot utilize pyruvate rapidly enough to prevent its elevation in the cytosol. The second proposed mechanism of lactate increase suggests that the mitochondrial membrane shuttle (electron transport system), which transfers protons and electrons to mitochondrial co-enzymes for subsequent combination with oxygen, becomes rate limited.

In summary, supporters of this explanation emphasize that lactate production is not caused by "hypoxic stimulation of anaerobic glycolysis" but results instead from an imbalance between glycolysis and the rate of pyruvate utilization in the Krebs cycle due to an inability of the oxidative enzymes and mitochondria to process the delivered oxygen at high exercise intensities. It is known that endurance training results in a rise in the number and size of mitochondria (Kiessling, 1971) and increases in the capacity of the oxidative enzymes (Holloszy, 1973). Supporters of this explanation suggest that these increases

may correspond with the increases in anaerobic threshold that are observed during post-training.

Mechanism Three: Muscle Fiber Type and Pattern of Recruitment. There is a growing tendency to attribute the pattern of blood lactate increase during incremental exercise to the sequential recruitment of different muscle fiber types. Skeletal muscle is composed of fibers possessing different contractile and metabolic properties as well as different recruitment patterns (Karlsson, 1979; Karlsson et al., 1981). Two predominant fiber types have been identified in human muscle: Type I or slow-twitch (ST) and Type II or fast-twitch (FT). The two fiber types are distributed in a mosaic pattern throughout the muscle (Jennekens et al., 1971). Research has shown that the relative proportions of the two fiber types varies from individual to individual (Gollnick et al., 1972).

They are identified as Type I and Type II on the basis of myofibrillar ATPase activity (Brooke & Engel, 1969). Type I and Type II fibers have low and high myosin ATPase activity, respectively. It has been demonstrated in animals that fibers with low myosin ATPase activity display slow contractile characteristics and those with high myosin ATPase activity, fast contractile characteristics (Barnard et al., 1971). The ST fibers have a higher overall lactate oxidative

capacity than FT fibers (Sjodin, 1976) due to high mitochondrial density (Hoppeler et al., 1973) and mitochondrial enzyme activity (Gollnick, 1973) which favor oxidation of lactate to pyruvate for use in the Krebs cycle. The metabolic profile of FT fibers, on the other hand, clearly demonstrates a higher glycolytic capacity for energy production than that of the ST fibers (Essen, Pernows, Gollnick, & Saltin, 1975).

The production and removal of blood lactate are influenced by the content of lactate dehydrogenase (LDH) in the sarcoplasm of the muscle fibers. This LDH can be present as heart-specific (H) or muscle-specific (M) isozymes. M-LDH facilitates the reduction of pyruvate to lactate whereas H-LDH favors the oxidation of lactate to pyruvate (Sjodin, 1976). The ST muscle fibers have a greater H-LDH activity (Sjodin, 1976) than FT. Consequently, the FT muscle fibers have a greater potential for lactate formation than the ST fiber, as indicated by a higher activity of LDH and a more muscle-specific LDH isozyme pattern (Sjodin, 1976).

Jorfeldt (1970) suggested that FT fibers would tend to produce lactate while ST fibers would tend to extract and oxidize lactate from the blood and from FT fibers. In 1978, Tesch and co-workers found that lactate was higher in FT fibers following maximal exercise. Others reported a

significant correlation ($r=.54$) between percent ST fibers and the rate of blood lactate removal after heavy exercise (Bonen, Campbell, Kirby, & Belcastro, 1978). Biochemically, the ST fiber appears suited for prolonged endurance exercise where their oxidative potential can be used to the fullest while FT fibers appear best suited to rapid, high intensity work of short duration.

In addition to muscle fiber type and ratio of oxidative to glycolytic enzyme activities, differences in other characteristics of the contracting muscle, such as capillary-muscle fiber ratio and respiratory capacity are associated with a delayed onset of blood lactate accumulation (Jacobs, 1981; Sjodin et al., 1982). The ability to keep the blood lactate concentration at a low level might be related to the capacity of the muscles to produce lactate (Rusko et al., 1980). Furthermore, blood lactate levels are also influenced by the type of muscle fiber being recruited at any given time. During the various stages of incremental exercise, there appears to be preferential recruitment of specific fiber types. At low-to-moderate exercise intensity (below 60-70% $\dot{V}O_2\text{max}$), the ST fibers are predominantly recruited (Gollnick et al., 1973). Progressively more FT fibers are recruited as the exercise intensity increases

(Gollnick et al., 1973) with a resulting increase in lactate production and accumulation (Karlsson & Jacobs, 1971).

Skinner and McLellan (1980) identified three phases during the progressive transition from exercise of low to maximal intensity. They hypothesized that Type I (ST) and possibly some Type IIa (fast, oxidative/glycolytic) muscle fibers are recruited during the first phase of exercise which primarily involves aerobic metabolism (below 40% $\dot{V}O_2\text{max}$). During this phase, blood lactate concentration rises from resting value to around 2 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$. With increasing intensity of exercise (40-60% $\dot{V}O_2\text{max}$), a greater recruitment of Type IIa muscle fibers and possibly some Type IIb (fast, glycolytic) fibers is seen. Consequently, a rise in blood lactate above 2 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ is seen. Skinner and McLellan (1980) refer to this intensity of incremental exercise as Phase II.

As the exercise intensity continues to increase (Phase III - 65-90% $\dot{V}O_2\text{max}$), a larger number of Type IIb fibers will be recruited (Essen, 1977). The M-LDH isozyme pattern of these fibers will lead to a greater production of lactate. The onset of Phase III is characterized by a sharp rise in blood HLa concentration from a level of about 4 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$. Confirming the velocity of these concepts, glycogen depletion studies have revealed a greater loss of glycogen in (ST)

fibers at low exercise intensities (Essen, 1977). As the duration and exercise intensity increased, more FT fibers evidenced depletion of glycogen. The exercise intensity at which the initial increase in blood lactate concentration occurs has been positively correlated to the percentage of ST fibers in the exercising muscle in both cycling (Ivy et al., 1980; Tesch et al., 1981) and running (Sjodin & Jacobs, 1981) studies. Therefore, it appears that the production, release, and oxidation of blood lactate by muscle is influenced by muscle fiber composition and the type of muscle fiber being recruited at any given time.

Mechanism Four: Reduced Hepatic Lactate Clearance. The fourth postulated mechanism suggests that the systematic increase in blood lactate could not be due to an increased lactate production but instead is related to a reduced hepatic clearance. Blood lactate concentration is the net result of lactate production and removal. Thus, a rise in blood lactate concentration may not necessarily indicate the onset of increased lactate production. It is known that lactate can be removed from the blood by the liver (Wahren, Hagenfeld, & Felig, 1975), active (Jorfeldt, 1970), as well as by nonactive skeletal muscle (Essen et al., 1975), kidney (Krebs, Hens, Weideman, & Speake, 1966), and heart (Keul, Doll, & Keppler, 1972). Proponents of this explanation

(Brooks, 1985, 1986; Brooks & Fahey, 1984; Donovan & Brooks, 1983) have hypothesized that the increased vasoconstriction that occurs with incremental exercise acts to reduce the blood flow to the liver. This reduced blood flow to the liver then reduces the ability of the liver to remove lactate from the blood, thus allowing the production of lactate to occur at a faster rate than its removal.

Donovan and Brooks (1983) used isotope tracers to study lactate in the rat before and after endurance training. The results indicated that blood lactate concentration is reduced at a particular submaximal workload post-training due to an increased lactate removal from the blood by the liver, not due to decreased muscle lactate production. In contrast, Karlsson et al. (1972) and Saltin et al. (1976) measured lactate production directly through muscle biopsy before and after endurance training and found that lactate production was indeed diminished post-training. These contradictory results have led some investigators to question the validity of the isotope tracer techniques in the study of lactate metabolism (Davis, 1985; Freminet, Poyart, & Leclerc, 1980).

Mechanism Five: Anaerobic Threshold, A Function of Metabolic Substrates. Researchers have successfully altered substrate availability by diet (Costill, Coyle, Dalsky, Evans, Fink, & Hoopers, 1977; Ivy, Costill, Van Handel,

Essig, & Lower, 1981) or exercise (Hughes et al., 1982) or the combination of diet and exercise (Heigenhauser, Sutton, & Jones, 1983). These experimental strategies have been prompted by the evidence which suggests that an increasing rate of free fatty acid (FFA) oxidation during prolonged submaximal exercise serves to inhibit muscle glycogen utilization (Rennie, Winder, & Holloszy, 1976). This greater reliance on FFA oxidation rather than glycolysis consequently results in decreased blood lactate concentration and R values (Costill et al., 1977). The net effect of either pre-exercise treatment (i.e., dietary intervention or exhaustive exercise) was to reduce muscle glycogen, utilization, thus reducing the potential for anaerobic glycolysis and increasing the anaerobic threshold. For example, Costill et al. (1977) assessed the effect of increased pre-exercise FFA levels in the blood on the rate of muscle glycogen utilization during 30 min of submaximal exercise. Pre-exercise FFA levels in the blood were increased by consumption of a fatty meal 4.5-5 h before exercise testing and injection of heparin to promote breakdown of triglycerides. Significantly less carbohydrate was used when blood FFA levels were elevated.

Heigenhauser et al. (1983) studied the responses of five male subjects performing two graded exercise tests, one

during control conditions and the other after reduction of muscle glycogen content by repeated maximal exercise and a high fat-protein diet. At rest, blood lactate concentrations were $0.73 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ lower during conditions of reduced muscle glycogen content than during control conditions. Blood lactate increased curvilinearly with increased exercise intensity under both conditions but was significantly lower during conditions of reduced muscle glycogen content compared with control conditions.

Anaerobic Threshold Determination from Blood Lactate Concentration

It is generally known that "lactate concentration increases in the blood during exercise secondary to increased muscle lactate production" (Wasserman et al., 1986, p. 347). It is also widely accepted that blood lactate concentration reflects muscle lactate concentration (Karlsson, 1971; Karlsson et al., 1972; Jacobs, 1971; Noble, Borg, Jacobs, Ceci, & Kaiser, 1983) and thereby provides an extramuscular index of anaerobic metabolism. Research has demonstrated that blood lactate concentration is accepted, at present, as the most objective index for determining muscular metabolism except for the direct measurement of cellular lactate via muscle biopsy (Powers et al., 1984; Yoshida et al., 1981). In fact, blood lactate (HLA) concentration has served as the

criterion measure of AT in numerous investigations (Caiozzo et al., 1982; Davis et al., 1976; Davis et al., 1979; Ivy et al., 1980; Powers et al., 1984; Reinhard et al., 1979).

However, it is important to remember that changes in blood lactate concentration, unlike muscle lactate, are the result of the balance between lactate production, release, distribution, and elimination (Kindermann & Keul, 1977).

Therefore, anaerobic threshold values determined from blood lactate concentration merely reflect the point where lactate production exceeds removal, not simply the point where lactate production begins to increase (Brooks, 1985).

Despite widespread use of the AT concept, controversy exists concerning the level of blood lactate concentration determined to reflect the point of metabolic acidosis. Researchers have traditionally couched the definition of the anaerobic threshold within the context of blood lactate elevations above resting levels. For example, the anaerobic threshold has been variously defined as the highest oxygen consumption that can be achieved during progressive exercise before:

- (1) a slight increase (Kindermann, Simon, & Keul, 1979);

- (2) a systematic increase (Caiozzo et al., 1982;

Farrell, Wilmore, Coyle, Billing, & Costill, 1979; Powers et al., 1984; Tanaka & Matsuura, 1984; Wasserman, 1981);

(3) a marked or nonlinear increase (Pendergast, Cerretelli, & Rennie, 1979; Simon et al., 1983);

(4) a sustained increase (Reinhard et al., 1979; Whipp & Mahler, 1980);

(5) an abrupt increase (Davis et al., 1976); or

(6) an exponential increase (McLellan, Feroah, & Skinner, 1981) in blood lactate above the resting value occurs.

Recently, however, some investigators have departed from defining the anaerobic threshold in such terms in favor of using a fixed, absolute blood lactate concentration. Selection of AT based on an absolute blood lactate concentration involves less subjectivity than identification of AT as the oxygen consumption just before a "systematic" increase in blood lactate occurs. Secondly, several investigators have suggested that the AT corresponds to a blood lactate of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ concentration based on empirical evidence (Costill, 1970; Kindermann et al., 1979; Mader, Heck, & Hollman, 1978; Rusko et al., 1980; Skinner & McLellan, 1980). Primarily, absolute concentrations of either $2 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ (Hughson & Green, 1982; LaFontaine, Londeree, & Spath, 1981; Wasserman et al., 1973; Yeh et al., 1983) or $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ (Costill, Thomason, & Robert, 1973; Karlsson et al., 1982; Kindermann et al., 1979; Mader et al.,

1978; Rusko et al., 1980; Sjodin & Jacobs, 1981; Skinner & McLellan, 1980; Tanaka & Matsuura, 1984) have been used as criteria for blood lactate AT.

Several investigators advocate the use of the 2 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration because it represents, on the average, the first significant increase of blood lactate above resting levels (Hughson & Green, 1982; Kindermann et al., 1979; Yeh et al., 1983). A concentration of 2 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa is approximately twice the average resting venous lactate concentration (Yeh et al., 1983). However, one drawback to utilization of the 2 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration to represent AT is that it denotes the upper range of normal resting values for venous lactate for some individuals (Kreisberg, 1980).

Other investigators suggest that a 4 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration should be used to represent AT based on a number of studies which have shown that endurance athletes are able to exercise for prolonged periods at a blood lactate concentration of 4 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ (Costill, 1970; Costill et al., 1973; Farrell et al., 1979; Kindermann et al., 1979; Rusko et al., 1980; Sjodin & Jacobs, 1981). In addition, Rusko and associates (1980) have suggested that the ventilatory and gas exchange alterations which are used for noninvasive detection of AT are not observed until a blood lactate concentration

of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ is reached. Use of the $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration is limited by the fact that some subjects fail to reach this concentration, even during exhaustive exercise (Caiozzo et al., 1982).

Opponents of the absolute blood lactate AT indices argue that "a physiological basis of why the anaerobic threshold should occur at some specific blood lactate concentration has not yet been elucidated" (Davis et al., 1983; p. 92). Davis et al. (1983) recently compared the gas exchange AT to three lactate criteria (lactate "breakpoint", $2 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa, $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa) for AT estimation and found that during graded exercise, $AT_{\dot{V}_E/\dot{V}O_2}$ corresponds to the lactate "breakpoint" and not to a fixed, absolute blood lactate of either 2 or $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$. Furthermore, Skinner and McLellan (1980) acknowledge that although some of the literature (also Phase III of their hypothetical model) suggests that a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentration is appropriate for AT, that this level is a rather "arbitrary" value which probably does not reflect the same degree of anaerobiosis in all individuals.

In conclusion, it is generally accepted that blood lactate is the most objective available index of the anaerobic threshold. However, a great deal of debate currently centers on what level of blood lactate concentration to use for AT determination. In order to

reduce observer error involved in AT determination, some researchers have relied upon an absolute blood lactate concentration (i.e., 2 or 4 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$) rather than identification of a point of curvilinear increase in blood lactate above resting values.

Anaerobic Threshold Estimation from Ventilatory and Gas Exchange Indices

A number of investigators have established that the intensity of exercise associated with a metabolic acidosis can be validly and reliably estimated from the ventilatory and gas exchange alterations consequent to buffering of blood lactate (Caiozzo et al., 1982; Davis et al., 1976, 1979, 1983; Reinhard et al., 1979; Wasserman et al., 1973). The increased rate of carbon dioxide production and appearance of unbuffered hydrogen ions (H^+) in the blood which result from lactate accumulation produce highly specific changes in ventilation and gas exchange. For example, the buffering of blood lactate (i.e., the reaction of H^+ with HCO_3^-) results in the formation of "excess" CO_2 beyond that generated from aerobic metabolism and this results in an abrupt increase in $\dot{V}\text{CO}_2$ and R. Below the anaerobic threshold, ventilation (\dot{V}_E) increases linearly with $\dot{V}\text{O}_2$ during graded exercise in healthy individuals. However, above the AT, the nonmetabolic production of CO_2 , at least in theory, acts as a strong

ventilatory stimulus, causing the $\dot{V}_E\text{-}\dot{V}O_2$ relationship to deviate from linearity (Wasserman et al., 1967). In addition, two other gas exchange variables (i.e., $\dot{V}_E/\dot{V}O_2$ and $P_{ET}O_2$) are also altered in response to buffering of blood lactate. A decrease in both the $\dot{V}_E/\dot{V}O_2$ and $P_{ET}O_2$ will be observed during the initial stages of an incremental exercise test "due to the decrease in the physiological dead space to tidal volume ratio (V_D/V_T)" (Davis, 1985, p. 11). As the exercise intensity increases, the decrease in V_D/V_T is slowed. At some point during the progressive exercise test, $\dot{V}_E/\dot{V}O_2$ and $P_{ET}O_2$ begin to systematically increase (Davis, 1985).

Numerous investigations have shown that the AT occurs at the same oxygen uptake whether measured via gas exchange or blood lactate (Caiozzo et al., 1982; Davis et al., 1976; Ivy et al., 1980; Reinhard et al., 1979; Wasserman et al., 1973; Yoshida et al., 1981). Consequently, the close agreement between the AT determined noninvasively vs that by blood lactate assessment has been used to establish the noninvasive approach as an acceptable alternative. For example, Davis and colleagues (1976) found a correlation coefficient of 0.95 between noninvasive AT measurements and that determined by evaluation of venous blood lactate response curves. Also, a high correlation ($r = 0.87$) was

demonstrated by Yoshida and co-workers (1981) between blood lactate AT and noninvasive AT in 10 male college students. In later work, Reybrouck, Ghesquiere, Cattaert, Fagard, and Amery (1983) found excellent agreement ($r = 0.98$) between blood lactate AT and AT determined from noninvasive indices. Unfortunately, the majority of investigators have restricted their research to male subjects. Further research is clearly needed to determine if such high correlation coefficients exist in the female population as well.

The noninvasive AT indices in use today have evolved over the course of the past 25 years. Initially, the AT was suggested to occur at the point of abrupt increase in the respiratory exchange ratio (Naimark et al., 1964; Wasserman & McIlroy, 1964). During incremental exercise, R was accepted as an accurate reflection of the buffering of lactate in the extracellular body fluids. In 1973, Wasserman and co-workers defined the AT as the point (intensity) at which minute ventilation (\dot{V}_E) and carbon dioxide ($\dot{V}CO_2$) departed from a linear relation with $\dot{V}O_2$ and where an increase in end-tidal PO_2 ($P_{ET}O_2$) and an abrupt increase in R occurred during graded exercise. Davis et al. (1976) expanded this definition to include the point of increase in fractional utilization of oxygen (F_{EO_2}). Investigators today continue to use the point of nonlinearity in $\dot{V}_E/\dot{V}O_2$ curve and

abrupt increase in $F_{E}O_2$ to detect blood lactate AT. The respiratory exchange ratio, however, is not currently in use as it has more recently been demonstrated to be an unreliable indicator of the onset of metabolic acidosis, or AT (Davis et al., 1976; Wasserman et al., 1973).

While \dot{V}_E and $\dot{V}CO_2$ "breakpoints" (i.e., departure from linearity with $\dot{V}O_2$) are valid AT indices, they are no longer considered to be technically optimal "because it is often difficult to judge the $\dot{V}O_2$ value at which \dot{V}_E ... [or $\dot{V}CO_2$ curves] ... begin to increase more steeply" (Davis, 1985, p. 11). Therefore, many investigators (Caiozzo et al., 1982; Davis et al., 1979; Poole & Gaesser, 1985; Reinhard et al., 1979; Yoshida et al., 1981) have recently suggested that AT detection is improved through the use of "dual" gas exchange indices which allow determination of AT "as the 'breakpoint' from a variable that is decreasing or is relatively unchanging over a number of work rates before it begins to increase" (Davis, 1985; p. 11). The two variables which seem most effectively to demonstrate this pattern of response during graded exercise are $\dot{V}_E/\dot{V}O_2$ and $P_{ET}O_2$ (Davis, 1985). For example, during incremental exercise, $\dot{V}_E/\dot{V}O_2$ typically decreases initially, flattens, and then rises steadily at the AT (Caiozzo et al., 1982) while $\dot{V}_E/\dot{V}CO_2$ remains stable at the AT point. This "triphasic" pattern of $\dot{V}_E/\dot{V}O_2$ facilitates AT

detection in contrast to the \dot{V}_E and $\dot{V}CO_2$ response curves "which rise continuously throughout ... [progressive exercise], leaving less confidence about where the nonlinear 'breakpoint' occurs" (Caiozzo et al., 1982; p. 1187). A systematic increase in $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ was first used to detect AT by Davis and co-workers in 1979. Others have subsequently reported high correlations between $AT\dot{V}_E/\dot{V}O_2$ and blood lactate AT (Caiozzo et al., 1982; Davis et al., 1983; Reinhard et al., 1979). For example, Reinhard et al. (1979) found a correlation coefficient of 0.94 when comparing $AT\dot{V}_E/\dot{V}O_2$ with blood lactate AT. Caiozzo et al. (1982) also found $AT\dot{V}_E/\dot{V}O_2$ to provide the most accurate and reliable detection of blood lactate AT when compared with three other noninvasive AT indices currently in use (i.e., nonlinearity in $\dot{V}_E-\dot{V}O_2$ relationship, abrupt increase in R, and nonlinear increase in $\dot{V}CO_2$). Caiozzo et al. (1982) favors the use of $AT\dot{V}_E/\dot{V}O_2$ for noninvasive AT detection over nonlinear increases in \dot{V}_E or $\dot{V}CO_2$ or abrupt increases in R for several reasons: (1) $AT\dot{V}_E/\dot{V}O_2$ yields the highest correlation with blood lactate AT ($r = 0.93$), (2) $AT\dot{V}_E/\dot{V}O_2$ yields the highest test-retest correlation coefficient ($r = 0.93$), and (3) $AT\dot{V}_E/\dot{V}O_2$ provides a "dual" criterion of AT which is easily derived from standard ventilatory and gas exchange measures. Davis et al.

(1979) and Poole and Gaesser (1985) have also reported test-retest correlation coefficients of 0.94 and 0.96, respectively, for $AT\dot{V}_E/\dot{V}O_2$.

In addition, it has been demonstrated that the exercise intensity at which $P_{ET}O_2$ increases without a corresponding decrease in $P_{ET}CO_2$ may also serve as a sensitive index of blood lactate AT (Wasserman et al., 1973). However, it requires the ability to measure gas exchange breath-by-breath during an exercise test. Lack of instrumentation for breath-by-breath gas analysis has prompted many investigators to detect AT via changes in $F_{E}O_2$ or $\dot{V}_E/\dot{V}O_2$. An abrupt increase in $F_{E}O_2$ previously has been shown to coincide with an abrupt increase in $P_{ET}O_2$ (Davis et al., 1976) and $\dot{V}_E/\dot{V}O_2$ (Reinhard et al., 1979).

In contrast, several investigators have questioned the validity of noninvasive determination of the AT (Gladden, Yates, Stremel, & Stamford, 1983; Green et al., 1983; Hughes, Turner, & Brooks, 1982; Powers et al., 1984; Simon et al., 1983). In general, they disagree with the hypothesis that an intracellular metabolic event (onset of "global" anaerobiosis in active skeletal muscle) can be detected from noninvasive measures (ventilatory changes). Each critic supports their point of view by citing studies which have demonstrated that invasive and noninvasive estimates of AT

do not always coincide during graded exercise under a variety of conditions (Green et al., 1983; Gladden et al., 1983; Hughes et al., 1982; Simon et al., 1983). For example, both Hughes, Turner, and Brooks (1983) and Heigenhauser, Sutton, and Jones (1983) have observed that ventilatory AT and blood lactate AT could be manipulated independently of each other. In the study by Hughes et al. (1983), nine male subjects performed incremental exercise on a cycle ergometer in both a normal glycogen state and in a glycogen-depleted state to determine if alterations in muscle glycogen content would affect their invasive and noninvasive AT values. The glycogen-depletion exercise test elicited a significant divergence between the two thresholds shifting the ventilatory AT to a lesser and the blood lactate AT to a greater exercise intensity relative to the normal glycogen state. Heigenhauser et al. (1983) had five male subjects perform two graded exercise tests, one during control conditions and the other after reduction of muscle glycogen content by repeated maximum exercise and a high fat-protein diet. Reduction in pre-exercise muscle glycogen was associated with a significantly higher \dot{V}_E and significantly lower blood lactate concentration at any given exercise intensity. Thus, conditions associated with glycogen depletion result in opposite changes in lactate AT

(increased) and ventilatory AT (decreased). In addition, Hughson and Green (1982) have shown a break in the relationship between blood lactate AT and ventilatory AT in response to two markedly different ramp protocols. Using a fast ramp protocol on a cycle ergometer, they were able to clearly dissociate the $AT\dot{V}_E$ from the blood lactate AT in 6 subjects. A comparison of the $\dot{V}O_2$ at $2 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate to the $\dot{V}O_2$ at the gas exchange AT indicated no difference for the slow ramp tests, but observed a higher $\dot{V}O_2$ in the fast ramp test for the point at which blood lactate reached $2 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$. Therefore, the close relationship between blood lactate AT and ventilatory AT may be dependent on the rate of increase in the exercise test protocol. Furthermore, the "uncoupling" of ventilatory AT and blood lactate AT has also been demonstrated in McArdle's syndrome patients (Hagberg, Coyle, Carroll, Miller, Martin, & Brooke, 1982). McArdle's syndrome patients lack the enzyme phosphorylase which plays an important role in regulation of the rate of glycolysis. During incremental exercise, Hagberg et al. observed that the McArdle's syndrome patients failed to experience a rise in blood lactate yet demonstrated a definite $AT\dot{V}_E$.

Based on the results of the aforementioned investigations, the critics suggest that the ventilatory

response to progressive exercise most likely results from a combination of neural and humoral inputs, not solely increases in muscle and blood lactate. Brooks (1985) agrees noting that in "the absence of a humoral signal (as in McArdle's patients), in response to a modified humoral signal (as in glycogen depletion), or in response to different input from higher centers or from peripheral proprioceptors (such as when the speed of movement is varied), the respiratory center in the brain will still produce an appropriate ventilatory response" (p. 26). Brooks (1985) argues therefore that the failure of noninvasive AT indices to accurately and reliably track changes in blood lactate concentration "renders the AT concept unusable" (p. 24).

In addition, recently it has been suggested that a "threshold" for blood lactate accumulation does not occur during graded exercise (Yeh et al., 1983). Yeh et al. (1983) simultaneously examined the arterial and venous blood lactate responses to incremental exercise in eight healthy subjects. For each subject, the arterial blood lactate curves appeared to rise smoothly throughout the exercise test without exhibiting any clear onset or "threshold" of lactate accumulation. The rise in venous lactate lagged behind the rise of arterial lactate by about 1.5 minutes. They

concluded, therefore, that AT is not even detectable using invasive methods.

The foregoing point of view runs contrary to the results of numerous studies which have demonstrated that noninvasive determination of the AT ($AT_{\dot{V}E}$, AT_{FEO_2} , $AT_{\dot{V}E/\dot{V}O_2}$) provides a simple, reproducible, and valid index of blood lactate AT (Caiozzo et al., 1982; Davis et al., 1976, 1979; Ivy et al., 1980; Reinhard et al., 1979; Yoshida et al., 1981). Gardner and Yeh (1984) have dismissed the existing supportive validation studies, arguing that even demonstration of an excellent correlation between lactate and noninvasive AT "does not prove the existence of and does not establish the accuracy of an anaerobic threshold" (p. 611). Additionally, Green and Hughson (1985) strongly suggest that "these studies are not validity studies as purported, but rather represent correlational studies between two fundamentally different variables" (p. 621). Brooks (1985) agrees, describing the anaerobic threshold as "an inappropriate and overly simplistic explanation of indirectly related phenomenon" (p. 22). Despite this controversy, it is important to emphasize that "the AT concept has been and continues to be instrumental in stimulating research that will undoubtedly enhance our understanding of the control of ventilation and

the interrelationship between a number of [physiological] systems during exercise" (Green & Hughson, 1985, p. 622).

In summary, various methods of noninvasive AT detection are currently in use. It appears that a systematic increase in $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ provides the most sensitive index of an exercise-induced metabolic acidosis, or AT. However, the subjectivity involved in AT estimation via the assessment of noninvasive responses remains a major limitation. Moreover, there are strikingly opposite and equally defensible viewpoints expressed by well-known scientists regarding the validity of the anaerobic threshold hypothesis.

Two Methodologies Currently Employed to Decrease Subjectivity of Anaerobic Threshold Determination

Despite use of what many regard to be physiologically acceptable indices, estimation of anaerobic threshold from blood lactate and gas exchange data is limited by the lack of objective techniques. Traditionally, the anaerobic threshold has been estimated via visual inspection of time-based plots of blood lactate and exchange variables in progressive exercise tests (Caiozzo et al., 1982; Davis et al., 1976; Simon et al., 1983; Wasserman et al., 1973; Yeh et al., 1983). This approach, based on the researcher's judgement, allows a great deal of subjectivity to enter into

the AT determination process. In an attempt to decrease subjective bias, investigators have recently employed two separate strategies for AT assessment.

The first method involves the use of independent reviewers to visually inspect plots of lactate and gas exchange alterations resulting from incremental exercise (Davis, Caiozzo, Lamara, Ellis, Vandagriff, Prietto, & McMaster, 1983; Gladden et al., 1983; Orr, Green, Hughson, & Bennett, 1982; Powers, Dodd, & Garner, 1984; Simon, Young, Gutin, Blood, & Case, 1983; Yeh et al., 1983). A review of the literature reveals that researchers have used anywhere from one (Caiozzo et al., 1982; Davis et al., 1983; Murray et al., 1983) or two (Miller et al., 1983; Poole & Gaesser, 1985; Powers et al., 1984; Simon et al., 1983) independent reviewers to as many as four (Orr et al., 1982; Yeh et al., 1983) and, in one study, nine (Gladden et al., 1983) to visually assess noninvasive or invasive ATs. As yet, however, no proficiency standard for AT determination by reviewers exists (Gardner, Yeh, Crapo, Yanowitz, & Adams, 1984). Qualifications of the reviewers used in the literature range from one or more of the primary investigators involved in the study (Davis, et al., 1983; Poole & Gaesser, 1985; Simon et al., 1983) to utilization of four independent doctoral degree level exercise

physiologists, not associated with the investigation, who had a minimum of 10 years experience with evaluation of exercise response measurements (Yeh et al., 1983).

Agreement Among Reviewers for Estimation of Noninvasive AT. In general, the agreement among independent reviewers for noninvasive AT estimations has been poor. For example, both Poole and Gaesser (1985) independently determined $AT_{\dot{V}_E/\dot{V}O_2}$ from coded photocopies of the data plots. The mean (\pm SE) difference in AT values between investigators was $54.8 \pm 5.8 \text{ ml} \cdot \text{min}^{-1} \dot{V}O_2$. Gladden et al. (1983), utilizing nine independent investigators, found the agreement (correlation coefficients) among the reviewers to range from 0.37 to 0.96 (median $r=0.66$) for noninvasive AT based on gas exchange variables. In a study by Powers et al. (1984), two of the investigators independently estimated AT following single blind review of the data plots. The two investigators differed in their choice of the blood lactate AT and $AT_{\dot{V}_E}$ on approximately 20% of the tests. Yeh and associates (1983) asked four doctoral level exercise physiologists to determine the GXT time at which $AT_{\dot{V}_E/\dot{V}O_2}$ and AT_{FETO_2} occurred. The results revealed that the AT estimations varied considerably among the reviewers for a given subject resulting in an average range of 16% reviewer variability. The time ranges among the four reviewers for a given subject varied from 0.9

minutes ($0.16 \text{ L}\cdot\text{min}^{-1} \dot{V}\text{O}_2$) to 4.2 minutes ($0.89 \text{ L}\cdot\text{min}^{-1} \dot{V}\text{O}_2$). The SD for the four reviewers for a given subject varied from 0.41 to 2.23 minutes, corresponding to oxygen consumptions of from 0.07 to $0.48 \text{ L}\cdot\text{min}^{-1}$. Each minute corresponded to a 20 W increment in workload. Therefore, Yeh et al. (1983; p. 1178) concluded that the current noninvasive assessment of AT has such a large range of between-reviewer variability that it is "unsuitable for clinical use". However, high test-retest correlation coefficients have been demonstrated by others. Poole and Gaesser (1985) found a test-rest correlation coefficient of 0.96 for their individual assessments of $\text{AT}\dot{V}_{\text{E}}/\dot{V}\text{O}_2$. This is in good agreement with the test-retest correlation coefficient of 0.93 demonstrated by Murray et al. (1983) for $\text{AT}\dot{V}_{\text{E}}/\dot{V}\text{O}_2$ using a single reviewer. Caiozzo et al. (1982) also found a test-retest correlation coefficient of 0.93 for $\text{AT}\dot{V}_{\text{E}}/\dot{V}\text{O}_2$ estimates selected by an independent investigator.

It has been suggested that the large range of between-reviewer variability for noninvasive AT estimation by several investigators may be due to provision of vague, unclear, or inadequately defined data assessment criteria (Homer, 1984; Yeh et al., 1983) or observer failure to correctly apply specified AT criteria (Davis, 1984). Furthermore, the physiological "noise" in the

breath-by-breath gas analysis measurements may make it difficult for reviewers to discern similar AT values (Davis et al., 1979; Yeh et al., 1983). It has also been suggested that periodic fluctuations in the ventilatory or gas exchange data may cause "different reviewers . . . [to] select thresholds after varying degrees of smoothing over different lengths of the curve" (Homer, 1984, p. 613).

Agreement Among Reviewers for Estimation of Invasive AT. Yeh and co-workers (1983) asked four highly-trained independent reviewers to determine blood lactate AT based on the exercise intensity where arterial lactate began accumulating; plots of lactate concentration vs exercise time were used. The between-reviewer exercise time range of AT as selected by the four reviewers varied from 0.2 to 5.0 minutes, corresponding to an oxygen consumption range of 0.50 to 0.93 L \cdot min⁻¹. The average range of variation was 2.2 minutes or 0.5 L \cdot min⁻¹ of oxygen consumption. The between-reviewer SD varied from 0.1 minutes to 2.06 minutes. Each minute corresponded to a 20 W increment in workload. Yeh et al. (1983) attributed this large range of reviewer variability to the absence of a threshold phenomenon for blood lactate during incremental exercise.

Comparison of Invasive and Noninvasive Thresholds Estimated by Independent Reviewers. A few recent studies

(Caiozzo et al., 1982; Ivy et al., 1980; Powers et al., 1984) have shown that the invasive and noninvasive anaerobic thresholds selected by independent reviewers show good agreement. For example, Caiozzo et al. (1982), using a single reviewer, found a high correlation between reviewer-selected gas exchange anaerobic threshold ($AT\dot{V}_E/\dot{V}O_2$) and reviewer-selected blood lactate anaerobic threshold ($r=0.93$). However, when noninvasive AT values ($AT\dot{V}_E$) chosen by two reviewers were compared with reviewer-selected blood AT values in a study by Powers and associates (1984), only a modest correlation coefficient ($r=0.67$) resulted.

Utilization of Computerized Methods to Determine the Anaerobic Threshold. The relatively large variability reported by some investigators for the "independent reviewer" method of AT detection has prompted several teams of investigators (Murray et al., 1983; Orr et al., 1982; Yeh et al., 1983) to utilize computer programs in an attempt to "simplify the determination of AT and minimize variations due to subjective human detection" (Yeh et al., 1983; p. 1179). In 1982, Orr and colleagues developed a computerized algorithm. This algorithm models the ventilatory response to exercise using multiple linear regressions. The first intersection point of the linear segment of the model was

considered to be the $AT\dot{V}_E$. The computer-determined $AT\dot{V}_E$ values were subsequently compared with $AT\dot{V}_E$ values selected by four independent reviewers after visual inspection of the same data. The four reviewer's estimates were averaged to yield a single value for each subject, resulting in a mean value of $2.26 \pm 0.69 \text{ L} \cdot \text{min}^{-1} \dot{V}O_2$ for $AT\dot{V}_E$. The $AT\dot{V}_E$ values derived from the computer algorithm averaged $2.21 \pm 0.65 \dot{V}O_2 \text{ L} \cdot \text{min}^{-1}$. A correlation coefficient of 0.94 was demonstrated between $AT\dot{V}_E$ "reviewer" vs "computer" methods.

Unfortunately, Orr et al. (1982) did not validate the algorithm against any independent criterion measure of AT, e.g., blood lactate changes. The high correlation ($r=0.94$) demonstrated between $AT\dot{V}_E$ -computer and $AT\dot{V}_E$ -reviewer methods does not validate the detection of the actual AT, rather it merely "validates the computer pattern recognition against human pattern recognition" (Orr et al., 1982, p. 1352). Using the same algorithm, Green et al. (1983) compared the $AT\dot{V}_E$ -computer method with blood lactate AT values also selected via the computer method. Their results indicate that noninvasive AT occurred at a significantly higher oxygen consumption than the blood lactate AT. Davis (1985) discussed possible reasons why use of this algorithm resulted in significant overestimation of AT with respiratory parameters. He acknowledged that this computer algorithm

(Orr et al., 1982) is a "useful first step in an attempt to improve anaerobic threshold detection" (p. 623). He recommends, however, that further refinement is necessary in order for the algorithm to provide a more accurate estimation of $AT\dot{V}_E$ in comparison with blood lactate AT.

Comparison of the Reviewer and Computer Methods of AT Selection. Murray et al. (1983) compared the reviewer and computer methods of noninvasive AT assessment. The reviewer method relied upon an observer to identify the point of systematic increase in $\dot{V}_E / \dot{V}O_2$ when graphed as a function of progressive exercise time. The computer method utilized a multi-segment linear regression program developed at the University of Waterloo to regress \dot{V}_E on $\dot{V}O_2$, identifying the first departure from linearity as $AT\dot{V}_E$. The results indicated that the two methods of $AT\dot{V}_E$ estimation provided reproducible but significantly different results. More comparisons of this nature are needed to establish the accuracy of either of these two methods of AT estimation.

Advantages and disadvantages have been identified for both the reviewer and computer AT assessment methods. One advantage of the reviewer method is that humans "can often recognize differences in [gas exchange] patterns that prove distressingly difficult to reduce to simple rules" (Homer, 1984, p. 613). A disadvantage is that periodic fluctuations

in the ventilatory or gas exchange data may cause "different reviewers . . . [to] select anaerobic thresholds after varying degrees of smoothing over different lengths of the curve (Homer, 1984, p. 613). The primary advantage of the computer method is that AT selection is less prone to observer error than by the reviewer method. However, a computer program will always select a threshold value, even when one may not be present (Green & Hughson, 1985). In addition, the \dot{V}_E - $\dot{V}O_2$ relationship is in reality curvilinear; thus, it is hard to detect the point of nonlinearity via a computer method based on linear regression (Green & Hughson, 1985). Furthermore, the final selection of AT via the computer method involves a somewhat subjective decision by the investigator. The search continues for AT detection methods that are objective, valid, and reproducible.

Optimal Protocol for Anaerobic Threshold Detection

Investigators have found the anaerobic threshold to be a reproducible parameter, independent of total test duration, using staged protocols (Buchfuhrer, Hansen, Robinson, Jue, Wasserman, & Whipp, 1983; Whipp, Koyal, & Wasserman, 1974) and ramp protocols with different slopes (Davis et al., 1980, 1982; Whipp et al., 1981). However, a standard protocol for the measurement of the anaerobic threshold has not been adopted. Work increments and exercise stages during

progressive exercise tests on either the cycle ergometer or the treadmill have recently ranged between 15 W (Whipp et al., 1981) and 30 W (Powers et al., 1984; Ready & Quinney, 1982) and from 15 s (Fairshter et al., 1983) to 2-3 min (Powers et al., 1984; Ready & Quinney, 1982; Simon et al., 1983; Weltman et al., 1978). Elevation of the exercise intensity by 0.5 METS at 30 s intervals in the present investigation is within the limits established in the literature.

Protocol Design. Initially, long duration steady-state exercise protocols designed to assess $\dot{V}O_{2\max}$ were employed to detect the AT (Davis, Whipp, & Wasserman, 1980; Powers et al., 1984; Taylor, Buskirk, & Henschel, 1955; Wasserman & Whipp, 1975). These steady-state protocols, consisting of at least 3-6 min of exercise at each exercise intensity (stage), were employed for AT detection because it had been suggested that the delay in diffusion of lactate from muscle to the blood would result in decreased accuracy in AT_{HLA} determinations for shorter stage durations (Stamford, Weltman, & Fulco, 1978).

Investigators eventually began experimenting with reduced-duration incremental exercise tests in an attempt to discover how brief a stage duration could be used without sacrificing the accuracy of AT detection. As a result, both

the total test time and the time increment per stage have been reduced as studies have revealed that stage duration did not influence the $\dot{V}O_2$ at the AT (Davis et al., 1976; Fairshter et al., 1983; Hughson & Green, 1982; Wasserman et al., 1973).

For example, Wasserman et al. (1973) found that the $\dot{V}O_2$ at AT occurred at the same exercise intensity whether 1 min or 4 min stage durations were employed during cycle ergometry. As a result, they elected to use a 1-min incremental exercise test as a standard protocol in their laboratory. Davis et al. (1976) demonstrated that tests employing increments in exercise intensity every minute while cycling or treadmill walking have a test-retest reliability similar to that of longer exercise tests. Fairshter and associates (1983), using both male and female subjects, compared 15 s and 1-min incremental test protocols on the treadmill and found no significant difference between the protocols for AT or $\dot{V}O_{2\max}$ values. In addition, the comparisons between protocols were not systematically different in men versus women nor in large versus small individuals. These findings support the use of stage durations of less than 1 min duration in the present protocol.

The gradual reduction in stage duration eventually resulted in the development of what is referred to today as the "ramp" protocol. A ramp protocol is one in which the exercise intensity is continuously increased throughout the test (Hughson & Green, 1982; Whipp, Torres, & Wasserman, 1981; Yeh et al., 1983). Thus, the protocols currently in use today consist of either a series of equal increases in exercise intensity at set intervals (i.e., once per minute) or a steady ramp increase. A lack of agreement exists concerning which duration of stage or amount of increase in exercise intensity per stage should be employed to optimize detection of the anaerobic threshold. Differences in the physiological implications of each test protocol have not as yet been adequately established.

Buchfuhrer et al. (1983) studied the effect of varying incremental workrate patterns on the estimate of the AT and found the $\dot{V}O_2$ at AT to be independent of the load increment on the cycle ergometer or the treadmill. In addition, Davis et al. (1980) also found that AT occurred at the same $\dot{V}O_2$ during ramp testing in which the mean slopes ranged from 20 to 50 $W \cdot \text{min}^{-1}$.

Wasserman et al. (1979) suggested that the shorter the stage duration or the steeper the ramp increase, the sharper the definition of the point at which blood lactate

concentration increases. Later, Whipp and colleagues (Green et al., 1983; Hughson & Green, 1982) advocated the use of the ramp protocol as a valid means of assessing the AT. Some have hypothesized that one of the advantages of utilizing a ramp protocol is that it avoids the abrupt step changes in exercise intensity observed in staged incremental tests. They have suggested that these step changes may suddenly alter recruitment patterns of motor units subsequently inducing "abrupt increases in ATP demand and a continual need for readjustment of processes supporting O₂ delivery" (Green et al., 1983; p. 1032). In summary, it seems that short-duration incremental protocols appear to provide a reliable and practical means for assessing the anaerobic threshold.

Data Collection Intervals. Data collection intervals are closely related to the stage duration employed during an exercise test. In the past, data collection intervals during exercise tests for AT assessment have ranged from every 2-3 min (Powers et al., 1984; Simon et al., 1983; Weltman et al., 1978) to every 15-30 s (Davis et al., 1976; Ready & Quinney, 1982). Recent advances in technology have enabled investigators to measure expired ventilation and oxygen and carbon dioxide tensions breath-by-breath through the use of rapidly responding oxygen analyzers and on-line computer

processing. This allows researchers to compute and visualize the AT as it occurs during the performance of an exercise test (Sue et al., 1980; Wasserman et al., 1973).

In 1982, Caiozzo and co-workers had subjects perform two maximal cycle ergometer tests using a $20 \text{ W}\cdot\text{min}^{-1}$ incremental staged protocol. Ventilatory and gas exchange measurements were made every 30 s throughout each test. They concluded that the one sample interval (i.e., 30 s) error contributed to the deviations in $AT\dot{V}_E/\dot{V}O_2$ vs blood lactate AT and that this might have been reduced in their investigation by using shorter data collection intervals (i.e., 15 s). That same year, Hughson and Green (1982) continuously collected respiratory data over 15 s intervals during fast ramp tests ($65.4 \text{ W}\cdot\text{min}^{-1}$) and over 30 s intervals in slow ($8.3 \text{ W}\cdot\text{min}^{-1}$) ramp tests. No significant difference was observed between the AT as assessed by gas exchange for the fast and slow ramp protocols.

Wasserman (1984) has suggested that a relatively short progressive exercise test in which gas exchange is measured breath-by-breath would allow rapid determination of the $\dot{V}O_2$ at AT because it allows on-line measurement of the characteristic gas exchange phenomena associated with developing metabolic acidosis. However, it has been previously demonstrated that gas exchange AT can be

accurately detected using less sophisticated methods than the breath-by-breath technique described by Wasserman and colleagues (Beaver, Wasserman, & Whipp, 1973; Wasserman et al., 1973). Davis et al. (1976) hypothesized that if the anaerobic threshold could be shown to be detectable using less complex methods of gas exchange measurement, the noninvasive detection of AT could become a routine laboratory measure. The Davis et al. (1976) approach involved determining AT values from 15 s data using the nonlinear increase in \dot{V}_E and $\dot{V}CO_2$ and abrupt increase in F_{EO_2} as indices of AT. A significant correlation ($r = 0.95$) was reported between the noninvasive AT and the AT as determined from venous blood lactate concentration. Others (Caiozzo et al., 1982; Ivy et al., 1980), utilizing open circuit spirometry rather than breath-by-breath analysis, have also reported high correlations between noninvasive and invasive AT values. Fairshter et al. (1983) compared ventilatory measurements made on a breath-by-breath basis, averaged, and reported for each 15 s interval with ventilatory measurements obtained simultaneously by collecting expired gas in a Tissot spirometer. Close agreement was demonstrated between the two methods. Thus, these findings support the use of open circuit spirometry to collect ventilatory and gas exchange

data every 30 and 15 s, respectively, in the present investigation.

Specificity of the AT. Several studies have shown that subjects achieve higher anaerobic threshold and $\dot{V}O_{2\max}$ values when exercise tested on the modality which was specific to their exercise training (Davis et al., 1976; Hagberg, Giese, & Schneider, 1978; Withers et al., 1981). For example, Withers et al. (1981) found that runners had significantly higher anaerobic thresholds than the cyclists when tested on the treadmill. These findings support the specificity of training concept, indicating that perhaps the adaptive responses to exercise are in part a function of the specific local muscular adaptations and recruitment patterns used in exercise training. It is known that only those muscles that are stressed during endurance training exhibit increased capillary density (Anderson & Henriksson, 1977), increased number and size of mitochondria (Kiessling et al., 1971) increase in mitochondrial and Krebs cycle enzymes (Gollnick et al., 1973), and increased glycogen storage (Hermansen, Hultman, & Saltin, 1967). It appears crucial, therefore, that the exercise test used to assess AT replicate the movement patterns of exercise training.

Mode of Exercise. The anaerobic threshold seems to vary as a function of mode of exercise (i.e., arm cranking, cycle

ergometry, treadmill) and quantity of active muscle mass. Research has shown that the blood lactate concentration is higher during exercise with small muscle groups (i.e., arm exercise) than during work with large muscle groups (i.e., leg exercise) at the same oxygen uptake (Asmussen & Nielson, 1946). Lactate production has also been shown to be higher at the same oxygen uptake during cycle ergometry than during treadmill exercise (Hermansen & Saltin, 1969). Furthermore, Davis et al. (1976) found that gas exchange AT values observed during arm cranking were significantly lower than those observed during treadmill walk-running. In addition, both the anaerobic threshold and $\dot{V}O_{2max}$ during incremental treadmill testing have been shown to be approximately 10% greater than those achieved during incremental cycle ergometry in sedentary subjects (Buchfuhrer et al., 1983; Wasserman, 1984; Withers et al., 1981).

It is speculated that the greater degree of metabolic acidosis demonstrated during cycle ergometry is due to the smaller muscle mass actively involved at a given exercise intensity in contrast to treadmill exercise. It appears that the smaller muscle mass used in cycling to generate the same power output as the equivalent treadmill exercise causes anaerobiosis to become manifest at a lower oxygen uptake. This would account for the greater metabolic acidosis

observed during cycle ergometry than during treadmill activity at equivalent exercise intensities (i.e., the same level of $\dot{V}O_2$) (Koyal et al., 1976). Other possible explanations involve unfamiliarity with the task and differences in recruitment patterns of motor units during leg cycling versus incremental treadmill walk-running. Since the subjects in the present investigation were trained via walk-jog exercise, they were exercise tested using a treadmill rather than a cycle ergometer.

The Effects of Aerobic Training on the Anaerobic Threshold

It has been well documented that endurance training reduces the accumulation of muscle and blood lactate during exercise at submaximal exercise intensities (Hurley, Hagberg, Allen, Seals, Young, Cuddihee, & Holloszy, 1984; Ivy et al., 1980; Karlsson et al., 1972; MacDougall, 1977; Saltin & Karlsson, 1971). For example, Hurley et al. (1984) found that endurance training $3 \text{ d} \cdot \text{wk}^{-1}$ via cycling and running induced lower blood lactate concentrations during exercise requiring the same percentage of $\dot{V}O_{2\text{max}}$ in the trained, as compared to the untrained state. The results of a study by Karlsson et al. (1972) suggest that the decreased concentrations of blood lactate during submaximal exercise in the trained state is secondary to a lower lactate concentration in the exercising muscles. Others support this

finding with results demonstrating that lactate production was diminished in the muscles of the trained relative to the untrained leg during exercise with both legs (Anderson & Henriksson, 1977; Saltin et al., 1976). It has also been demonstrated that endurance athletes are characterized by lower blood lactate concentrations than nonathletes at the same relative exercise intensity (Hermansen, 1971). A decreased lactate accumulation during submaximal exercise following training has been interpreted to mean that the AT has increased (Davis et al., 1979). However, the extent to which the AT is altered by endurance training has not been well established, especially in women. Most training studies have been conducted using men as subjects. The lack of information on women prompted this investigator to examine the effects of short-term aerobic training on the AT in twelve previously sedentary, overweight females.

How does aerobic training result in lower blood lactate concentrations at the same submaximal exercise intensity in the trained state? The physiological mechanisms responsible for a decreased accumulation of lactate and consequently an increased AT after endurance training remain unclear. Many possible mechanisms have been suggested; these fall into two basic categories, lower lactate production by the trained muscles or increased rate of lactate removal from the blood.

Chronic aerobic exercise causes several adaptations to occur within the exercised muscle which would enhance oxidation of pyruvate and reduce the rate of lactate production (Holloszy & Coyle, 1984). These changes ultimately result in an increase in the capacity for aerobic metabolism, thereby increasing the absolute intensities at which the AT is manifested. For example, they include:

(1) Increased number and size of muscle mitochondria which serves to increase the oxidative capacity of the active muscles (Kiessling et al., 1971; Morgan, Cobb, Short, Ross, & Gunn, 1971),

(2) Increased capillary density of trained skeletal muscle which serves to decrease the diffusion distance between the blood and muscle fibers (Anderson & Henriksson, 1977),

(3) Increased muscle respiratory capacity via training-induced increases in oxidative cellular enzymes (Gollnick et al., 1973), and

(4) Increased resting concentrations of ATP in the trained muscle (Karlsson et al., 1972).

Other major training-induced adaptations which act to delay onset of blood lactate accumulation include diminished catecholamine release (Winder, Hickson, Hagberg, Ehsani, & McLane, 1979) and alteration of substrate utilization

patterns (Hermansen, Hultman, & Saltin, 1967). A diminished catecholamine release consequently results in decreased stimulation of glycogenolysis during exercise (Richter, Ruderman, Gavras, Bulur, & Galbo, 1982) thereby, decreasing lactate release from muscle. Lactate production may also be reduced in physically trained individuals as a result of a shift from carbohydrate toward greater use of fat as a source of energy (Robinson & Sucec, 1980). This shift is evidenced by decreased R values (Saltin & Karlsson, 1971) and lower rates of glycogen depletion (Hermansen, Hultman, & Saltin, 1967; Karlsson et al., 1972) at the same absolute submaximal exercise intensity after training. This benefit of endurance training serves to increase AT by exerting a "glycogen-sparing" effect (Gollnick et al., 1986).

Additionally, it has been postulated that a possible effect of endurance training is to increase lactate removal, relative to the blood lactate concentration (i.e., increased metabolic clearance rate) allowing for a lower blood lactate concentration at any given lactate production in the trained compared to the untrained state (Donovan & Brooks, 1983). However, the literature is in disagreement regarding the fate of the lactate removed from the blood. Some suggest that most of the lactate is oxidized (Brooks & Fahey, 1984; Donovan & Brooks, 1983), while others (Hermansen & Vaage,

1977; McLane, 1979) hold that some of the lactate removed from the blood by active muscle may be resynthesized to glycogen.

Despite the foregoing debate, it is generally agreed that neither increased availability of oxygen nor increased blood flow to the active muscles is responsible for the diminished lactate response during submaximal exercise in the trained state. Furthermore, it is well known that oxygen consumption at submaximal exercise intensities is not increased by training. Despite increased capillarization, blood flow to the active muscles also does not increase as a result of endurance training (Varnauskas, Bjorntrop, Fahlen, Prerovsky, & Stenberg, 1970). In fact, blood flow to the active muscle is lower in the trained than in the untrained state (Hollooszy, 1973) at the same absolute submaximal exercise intensity. The active muscles compensate for the lower blood flow in the trained state by extracting more oxygen (Hollooszy, 1973).

Training Intensity and the Anaerobic Threshold.

Research has demonstrated that the anaerobic threshold can be increased through exercise training (Davis et al., 1979; Hurley et al., 1984; Ready & Quinney, 1982; Robinson & Sucec, 1980; Yoshida et al., 1982). For example, Davis et al. (1979) found the AT to be elevated (i.e., by 44% when

expressed as absolute $\dot{V}O_2$ and by 15% expressed relative to $\dot{V}O_{2max}$) in previously sedentary, middle-aged men after cycle ergometer training 45 min per day, 5 days per week, for nine weeks. The subjects were trained at exercise intensities of 50% $\dot{V}O_{2max}$ for the first 4 weeks and then 70% $\dot{V}O_{2max}$ during the last 5 weeks of the study. Ready and Quinney (1982) trained 21 males (\bar{x} = 25 yr) on a cycle ergometer at 80% $\dot{V}O_{2max}$ for 30 min four times per week for 9 weeks. Elevations of 70.4% (from 2.13 to 3.63 $L \cdot min^{-1}$) and 19.4% occurred following training in AT expressed as absolute $\dot{V}O_2$ and percent of $\dot{V}O_{2max}$ respectively. The 70% increase in AT expressed as $L \cdot min^{-1}$ following 9 weeks of training is approximately 25% greater than that found by Davis et al. (1979). Ready and Quinney attribute this discrepancy to the fact that middle-aged men (\bar{x} = 43.0 yr) served as subjects in the Davis et al. (1979) study while they used young men (\bar{x} = 25 yr) as subjects. Hurley et al. (1984) examined the alterations in AT (i.e., characterized by changes in blood lactate concentrations) in eight untrained males and eight distance runners following a 12 week intensive exercise training program. The program consisted of high-intensity interval training on a cycle ergometer 3 days per week (i.e., six 5-min exercise bouts at $\sim 90-100\% \dot{V}O_{2max}$) and running for 40 min three days per week at $\sim 75\% \dot{V}O_{2max}$. Lactate

concentrations at the same relative exercise intensities (in the 55-75% of $\dot{V}O_{2max}$ range) were significantly lower after training for both groups. Robinson and Sucec (1980) examined the effect of a 12 week training program on the AT in 21 normally active men. The subjects were divided into three groups: (1) distance group which trained by running for 30 minutes 3 times per week at $\sim 85\% \dot{V}O_{2max}$; (2) interval group which trained by running intervals of 100m, 200m, and 300m three times per week at $\sim 125\% \dot{V}O_{2max}$; and (3) control group which continued their usual activity pattern. The AT increased 15.6% for $\dot{V}O_2$ at AT for the distance-trained subjects, 11.3% for the interval-trained subjects, and 6.5% for controls. The results indicate that both moderate ($\sim 85\% \dot{V}O_{2max}$) and intensive ($\sim 125\% \dot{V}O_{2max}$) training increases the AT.

Few investigators have examined the effects of various training intensity levels on the AT in women (Gibbons, Jessup, Wells, & Werthman, 1983; Kilbom, 1971; Rivera, Metz, & Robertson, 1980). Gibbons et al. (1983) examined the effect of a continuous running program on the AT of 29 untrained females using various training intensity levels which were established based on individual anaerobic threshold measurements. The subjects were randomly assigned to one of three possible training intensities for 20 min four

days per week throughout the 8 week study. The AT+ group trained at 40% above their AT, the AT group trained at their AT, while the AT- group trained at 40% below their AT. The post-training AT was increased only in the group which trained at their AT level. It was suggested that if the objective is to increase the AT, it is more effective to train at an exercise intensity corresponding to one's AT than to train above or below that level. Unfortunately, AT's identified via ventilatory and gas exchange criteria were not correlated with blood lactate, the criterion measure of AT. Kilbom (1971), studying the effect of a low-intensity (70% $\dot{V}O_{2max}$), short-duration (7 wk) training program in untrained women, reported that blood lactate levels at comparable submaximal $\dot{V}O_2$ levels decreased post-training. Rivera, Metz, and Robertson (1980) examined the effect of 6 weeks of interval swimming training on the AT and performance times of 24 female age-group swimmers. The subjects were randomly assigned to two training groups. One group trained at ~ 84% of best performance time while the second group trained at an intensity that elicited an accumulation of 4 $m \cdot mol \cdot L^{-1}$ blood lactate (AT). From a tethered swimming test, maximal aerobic capacity, maximal alactacid capacity and maximal lactacid capacity were determined. The results indicated that both training groups improved performance times in 100

meter and 400 meter timed swims with the AT group improving at a faster rate. These data support the theory that AT training enhances the efficiency of aerobic and anaerobic metabolic systems and that the AT is influenced by swimming training.

Following a detailed review of the results of training studies, Wells (1985) concluded that the "metabolic adaptations to training appear to be related to exercise intensity while variations in exercise frequency and duration appear to be of lesser importance" (p. 243). Despite numerous investigations, the optimal level at which exercise training should be performed to result in an increased AT has yet to be clearly defined.

Summary

Over the past fifty years, both the concept and the definition of the anaerobic threshold have undergone numerous revisions. Currently it is defined as the oxygen uptake above which aerobic energy production is supplemented by anaerobic mechanisms resulting in a significant increase in lactate (Wasserman, 1984). Production of lactate serves as an "oxygen-conserving" mechanism that allows the cell to regenerate ATP even when oxygen supply is insufficient. Thus, the production of lactate is an essential mechanism for the performance of prolonged exercise. However, an

individual can only exercise for a period that is dependent upon his ability to incur high lactate concentrations since muscle contractile and metabolic activity is limited by high lactate levels (Green et al., 1979). Five possible mechanisms for the increase in blood lactate during exercise have been postulated.

Blood lactate concentration is currently accepted as the most objective index for determining the anaerobic threshold except for the direct measurement of cellular lactate via muscle biopsy. Controversy exists, however, concerning the level of blood lactate concentration determined to reflect the point of metabolic acidosis, or AT.

Numerous investigations have shown that the AT occurs at the same oxygen uptake whether measured invasively or noninvasively (Caiozzo et al., 1982; Davis et al., 1976; Wasserman et al., 1973). Based on these results, it has been suggested that the AT can be accurately and reliably estimated from either blood lactate levels of ventilatory and gas exchange alterations consequent to buffering of blood lactate. However, disagreement exists concerning the validity of AT estimation from noninvasive measures (Brooks, 1985; Green et al., 1983). Several research teams have demonstrated that invasive and noninvasive estimates of AT

do not always coincide during incremental exercise (Green et al., 1983; Hughes et al., 1982).

Furthermore, a major limitation of AT assessment is lack of objective techniques. Traditionally, the AT has been estimated via visual inspection of data plots by the primary investigator(s). Recently, researchers have begun to employ two methods, computerized regression programs and independent reviewers, in an attempt to decrease the subjectivity involved in AT estimation.

The AT has been found to be a reproducible parameter, independent of total test duration, using both staged protocols (Buchfuhrer et al., 1983) and ramp-type protocols (Davis et al., 1980). A standard protocol for the enhancement of AT estimation has not yet been identified.

Endurance training reduces the accumulation of muscle and blood lactate during exercise at submaximal intensities, therefore serving to increase the AT. The extent to which the AT is altered by endurance training is not well established, especially in women.

Chapter III

Journal Manuscript

Reliability and Validity of Two Anaerobic
Threshold Methodologies with Overweight Women

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(abbreviated title for running head)
Reliability and Validity of Two Anaerobic
Threshold Methodologies

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ABSTRACT

The primary purpose of this investigation was to determine the reliability and validity of two noninvasive methods of determining anaerobic threshold (AT). Twelve sedentary, overweight women participated in an 8 wk walk/jog program 3 d \cdot wk $^{-1}$ at 70% $\dot{V}O_{2\max}$ with a concurrent hypocaloric diet. A computerized polynomial regression and four independent reviewers were used in an attempt to decrease the subjectivity of noninvasive AT estimation from data plots. Assessment of duplicate plots by reviewers revealed a 2-7% intra-reviewer variability range (within acceptable limits for research and clinical purposes). Evaluation of variability among reviewers revealed that 100% and 83% of the Spearman Rho correlation coefficients were statistically significant ($p < 0.05$) for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$, respectively. However, test-retest reliability (different days) of mean AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ estimates from the same reviewers demonstrated poor reproducibility; this implies that the observed variability is attributable more to physiological factors than to AT estimation technique of reviewers. Additionally, neither AT_{FEO_2} nor $AT_{\dot{V}E/\dot{V}O_2}$ demonstrated statistically significant correlations with criterion variable 4 mmol \cdot l $^{-1}$ blood lactate (AT_{HLa}). However, the

AT_{VE} -computer method provided a reliable ($p=0.85$, $p<0.01$) and valid method ($p=0.94$, $p<0.01$) of AT_{HLa} estimation in untrained individuals. Thus, these results do not support the use of multiple reviewers to increase objectivity of noninvasive AT estimation.

ANAEROBIC THRESHOLD, COMPUTERIZED POLYNOMIAL REGRESSION,
HYPOCALORIC DIET, BLOOD LACTATE, RELIABILITY, VALIDITY,
TRAINING EFFECTS

Introduction

It has become common practice in the literature to estimate the AT based on ventilatory and/or gas exchange alterations consequent to a metabolic acidosis during incremental or ramp-type exercise. However, disagreement exists as to the validity of this practice. Several investigators support the idea that AT can be effectively determined via ventilatory and/or gas exchange indices (3, 4, 2), while others have questioned this practice (15, 19, 22). A need for additional data on this issue is clearly indicated (13). More research is also needed to establish to what extent AT is altered by short-term aerobic training. Thus, the purposes of this investigation were twofold: to determine the reliability and validity of certain noninvasive methods of AT estimation and; to evaluate the effects on the AT of an 8 wk aerobic training program administered to sedentary, overweight women who were consuming a hypocaloric diet.

Methods and Procedures

Subjects

Twelve sedentary, overweight (body fat; \bar{x} = 32.2%) females (age; \bar{x} = 23.3 yr) were selected from a pool of volunteers after completion of initial screening. Body fat (%) was estimated using the skinfold technique described by

Durnin and Womersley (7). Important physical characteristics are presented in Table 1.

Insert Table 1 Here.

Experimental Protocol

Subject Screening. Prior to participation, each subject was informed of the nature of the experiment and its inherent risks. Each completed a written informed consent document in accordance with the policy statement of the American College of Sports Medicine and the University Human Subject Committee Regulations. Completion of a detailed medical history questionnaire was required before participation. Subjects were then familiarized with the data collection equipment and procedures.

Maximal Exercise Test. A maximal graded exercise test (GXT₁) was first administered to collect noninvasive data needed for determination of each subject's exercise training prescription and for determination of exercise blood sampling times. A ramp-type treadmill protocol was used in which a warm-up was provided at 3.4 mph/0% grade. Thereafter, speed and/or grade increased every 30 s to achieve an overall increase of 0.5 METS/stage (1).

Submaximal Exercise Test. Within 3 d, a submaximal graded exercise test (GXT₂), identical to the earlier stages of GXT₁, was administered primarily for the purpose of establishing test-retest reliability of noninvasive data and collection of resting and submaximal blood HLa data. A blood sample (5 ml) was drawn prior to the submaximal test via venipuncture of an antecubital vein to ensure that pre-exercise lactate concentrations were not elevated. Duplicate capillary blood samples (200 ul) collected via finger puncture were taken 1 min before and after the test stage presumed to represent each subject's AT (based on ventilatory "breakaway" determined from each subject's GXT₁ data). All blood samples were immediately analyzed for lactate concentration using the YSI Scientific lactate analyzer (Model 23L).

Procedures and Instrumentation

Throughout each exercise test, heart rate, blood pressure, rating of perceived exertion (RPE), minute ventilation (\dot{V} , $l \cdot \text{min}^{-1}$, BTPS) and fractions of expired oxygen ($F_{E}O_2$) and carbon dioxide ($F_{E}CO_2$) were measured. Minute ventilation was recorded every 30 s using a Parkinson-Cowan CD-4 dry gas meter or a Hewlett-Packard pneumotach (Model 47303A). Expired $F_{E}O_2$ and $F_{E}CO_2$ were recorded every 15 s using Beckman model OM-11 and LB-2

analyzers, respectively. A 15 s phase delay was accepted between V measurement and stabilization of $F_{E}O_2/F_{E}CO_2$.

Anaerobic Threshold Estimation. The indices used to determine the AT values for each subject were \dot{V}_E , $\dot{V}_E/\dot{V}O_2/\dot{V}_E/\dot{V}CO_2$, $F_{E}O_2/F_{E}CO_2$ and HLa, the latter serving as the invasive criterion measure. The following criteria were employed in estimation of the AT:

1. The GXT time which occurred at the point just below a nonlinear rise in the \dot{V}_E - $\dot{V}O_2$ relationship;
 2. The GXT time which occurred at the point wherein $F_{E}O_2$ increased without a corresponding decrease in $F_{E}CO_2$;
 3. The GXT time which occurred at the point wherein a systematic increase in $\dot{V}_E/\dot{V}O_2$ was observed without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$;
 4. The GXT time corresponding to an HLa value of $4 \text{ mmol}\cdot\text{l}^{-1}$.
- A microcomputer facilitated the graphic presentation of the ventilatory and gas exchange data versus incremental exercise time.

To satisfy purposes of this investigation, a two-component polynomial regression method was then applied to the data for estimation of AT using ventilation data. The polynomial least square curve-fitting software program generated regression of \dot{V}_E on incremental exercise time to discern the point of abrupt nonlinear rise in \dot{V}_E ($AT\dot{V}_E$).

The second method involved four independent reviewers who were instructed to carefully follow written instructions during the AT selection process. Each blindly reviewed subjects' plots of $F_{E}O_2/F_{E}CO_2$ and $\dot{V}_E/\dot{V}O_2/\dot{V}_E/\dot{V}_E CO_2$ data for all four treadmill tests. AT was selected using previously described criteria.

The small number of exercise blood lactate samples per subject ($N=2$) in the present investigation limited determination of AT_{HLA} to the GXT time corresponding to an absolute blood lactate concentration ($4 \text{ mmol}\cdot\text{l}^{-1}$ HLa). An "idealized" lactate curve, derived from the literature (5, 12, 14), was formulated to allow determination of AT_{HLA} and subsequent validation of noninvasive AT estimates. The "idealized" curve, graphed on a clear sheet of acetate, was placed over separate plots of each subject's two exercise blood lactate values measured before (GXT_2) and after (GXT_4) training. The two plotted lactate data points served to "anchor" the "idealized" curve, allowing estimation of AT_{HLA} as the point of intersection of $4 \text{ mmol}\cdot\text{l}^{-1}$ on the y axis and GXT time on the x axis with the "idealized" curve. The GXT time on the x axis corresponding to the $4 \text{ mmol}\cdot\text{l}^{-1}$ lactate concentration (AT_{HLA}) was rounded to the nearest tenth of a minute. Those subjects whose exercise blood lactate values were too low ($< 2 \text{ mmol}\cdot\text{l}^{-1}$) ($N=2$) or whose exercise blood

lactate values decreased from the first to the second exercise sample (N=1) were omitted from this analysis leaving a total of nine subjects for whom AT_{HLA} could be determined.

Aerobic Training Program. The 8 wk aerobic training program began 2 d following completion of the submaximal exercise tests. The subjects walked/jogged outdoors $3 \text{ d} \cdot \text{wk}^{-1}$ at a pace that elicited 70% of their $\dot{V}O_{2\text{max}}$. Each training session consisted of a 5 min warm-up, a 30 min stimulus phase, and a 5 min cool down. All training sessions were supervised by the investigator. Subjects attended 78% of the exercise sessions. Exercise heart rate data collected during the exercise training sessions demonstrate that subjects were consistently (90%) exercising within their prescribed target heart rate range. Concurrently with the training, subjects consumed a hypocaloric diet ($1200\text{-}1400 \text{ kcal} \cdot \text{d}^{-1}$) and attended weekly nutrition education sessions. Subjects attended 87% of the nutrition education sessions.

Post-training Exercise Tests. Post-training exercise tests, utilizing procedures identical to those employed during the pre-training tests, were administered at the conclusion of the training program. A minimum of 48 h was interposed between the final training session and the post-training exercise tests.

Statistical Analysis. Spearman Rho correlation coefficients were determined to establish test-retest reliability of AT estimates using the AT-computer and AT-reviewer methods. Spearman Rho correlation coefficients were also utilized to provide estimates of validity for each noninvasive index when compared to the AT criterion, a $4 \text{ mmol}\cdot\text{l}^{-1}$ blood lactate concentration (AT_{HLA}). An ANOVA was performed to determine the % variance which could be attributed to the four reviewers versus that due to the day-to-day variability of the subjects, pre- and post-training. Dependent t-tests were performed to determine if significant differences existed between estimates of AT pre- to post-training. Dependent t-tests were also employed to evaluate a possible training effect on body weight, body fat, maximal oxygen uptake, resting heart rate, and resting and submaximal blood lactate. Significance was established at the 0.05 probability level.

Results

Reliability. Prior to determination of the reliability of AT estimates, the reproducibility of the underlying ventilatory and gas exchange data was determined through comparisons of ventilatory and gas exchange indices from data collected during two graded exercise tests (GXTs) performed pre- and post-training. All five variables (\dot{V} , $F_{\text{E}}\text{O}_2$, $F_{\text{E}}\text{CO}_2$,

$\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$) were shown to be reproducible (Range for $P=0.58-0.69$, $p<0.05$), prior to training, at the time in the GXT associated with the predicted AT. Post-training, however, only \dot{V} was shown to be reproducible ($P=0.79$, $p<0.05$).

Test-retest reliability of noninvasive AT estimates was then determined through the use of Spearman Rho correlations performed on the estimates of $AT_{\dot{V}_E}$ -computer (Figure 1), AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$ determined by each of the four reviewers (Tables 2 and 3), and on the means of the four reviewer's AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$ estimates for the four graded exercise tests (Figures 2 and 3). $AT_{\dot{V}_E}$ -computer demonstrated a high test-retest correlation coefficient in the pre-training state ($P=0.85$, $p<0.01$).

Insert Figure 1 Here.

Post-training, however, $AT_{\dot{V}_E}$ no longer demonstrated significant test-retest reliability. Evaluation of reliability (variability) among the four reviewers for all four GXTs revealed that 100% of the AT_{FEO_2} and 83% of the $AT_{\dot{V}_E/\dot{V}O_2}$ estimates were significantly correlated ($p<0.05$).

Insert Tables 2 and 3 Here.

In addition, the mean percent variation was calculated for duplicate AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ plots judged by the same reviewer to get an estimate of within-reviewer reliability. Assessments of duplicate plots by reviewers were found to fall within a 2 and a 10% variability range, i.e., acceptable limits for research and clinical purposes, respectively. Since there was some disagreement among all four reviewers in the estimate of AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$, the mean of the four estimates was chosen as the comparative measure of the AT for test-retest reliability analysis.

Insert Figures 2 and 3 Here.

The results indicated that both AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ demonstrated unreliability (large variability) in test-retest comparisons both at the pre- and post-training conditions. (AT_{FEO_2} $P=0.10$, $P=0.22$; $AT_{\dot{V}E/\dot{V}O_2}$ $P=0.15$, $P=0.70$).

Validity. Spearman Rho correlation coefficients were also utilized to provide estimates of validity for each noninvasive AT index in comparison with AT_{HLA} .

Insert Table 4 Here.

The AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$ estimates used for comparison with AT_{HLa} in Table 4 were determined as the mean of the 4 reviewers AT selections derived from GXT_1 and GXT_2 for pre-training values and GXT_3 and GXT_4 for post-training values. Table 4 reveals that only $AT_{\dot{V}_E}$ -computer, based on pre-training data, was shown to be significantly correlated with $AT_{\dot{V}_E}$. Post-training, none of the noninvasive AT indices correlated significantly with AT_{HLa} .

Effects of Training. Estimates of invasive and noninvasive AT were evaluated via dependent t-tests for significant differences pre- to post-training.

Insert Figure 4 Here.

No significant differences were demonstrated. Dependent t-tests were also used to evaluate possible effects of training on maximal oxygen uptake, resting heart rate, resting and submaximal HLa concentrations, and body weight and fat.

Insert Table 5 Here.

Table 5 reveals that only body weight, body fat, and $\dot{V}O_{2\max}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) were shown to be significantly different post-training. Body weight and fat decreased 6.5% and 8.3% respectively. Relative $\dot{V}O_{2\max}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) showed a 9.8% increase after training. However, no significant differences were demonstrated for $\dot{V}O_{2\max}$ ($\text{l}\cdot\text{min}^{-1}$), resting and exercise HLa concentrations, or resting heart rate.

Discussion

The major finding of this study is the lack of support for the methodology which utilizes multiple reviewers to increase the objectivity of noninvasive AT estimation. Evaluation of intra- and inter-reviewer variability revealed that the four reviewers were consistent within and among themselves for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ estimation. Test-retest analysis of the mean values among the reviewers for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$, however, revealed low correlation coefficients for both pre- and post-training data. Previous studies have shown considerable variation between replicate tests for noninvasive AT, resulting in a low test-retest correlation (3, 16). This variability has previously been attributed to changes in the AT of the subjects between the replicate tests

or the result of the subjective nature of the methods used to determine AT. In the present investigation, however, it appears that the variability observed between GXTs pre- or post-training is the result of day-to-day physiological variability of the subjects. For example, prior to training the percent variance attributable to the day-to-day variability of the subject's responses to graded exercise was 19% for AT_{FEO_2} and 60% for $AT_{\dot{V}E/\dot{V}O_2}$. Post-training data revealed a ~2 and a 1% day-to-day variance for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$, respectively. These results indicate that 8 wk of walk/jog training greatly decreased the variability of subject responses to graded exercise. In addition, assessment of duplicate data plots by each of the four reviewers yielded a 2-7% variance range for AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ values which is within acceptable limits for research and clinical purposes. Therefore, the major portion of the variance in noninvasive AT, based on gas exchange and ventilatory equivalent data, cannot be attributed to the intrinsic variability of the reviewers. Possible explanations for this physiologic variability include differences in the amount of sleep the night before each GXT, pre-GXT muscle glycogen stores, or pre-GXT anxiety levels. It can also be speculated that perhaps there is a certain amount of inherent instability in the ventilatory and

respiratory responses (upon which AT is based) of sedentary overweight women to ramp-type incremental exercise. It is interesting to note that analysis of test-retest reproducibility of the basic data underlying noninvasive AT estimation, compared at a point below predicted AT, revealed that the day-to-day physiological variability of the subjects was less for \dot{V} than for $F_{E}O_2$, $F_{E}CO_2$, $\dot{V}_E/\dot{V}O_2$, and $\dot{V}_E/\dot{V}CO_2$ in the pre-training state.

A secondary finding of importance is the demonstration of reliability and validity of $AT\dot{V}_E$ estimation via a two-component polynomial regression computer method in untrained individuals. It is puzzling, however, that $AT\dot{V}_E$ -computer method did not remain reliable or valid in comparison with AT_{HLA} for the same subjects following 8 wk of aerobic training. Perhaps the 8 weeks of dietary intervention significantly altered the preexercise concentration of muscle glycogen in the exercising muscles. A reduction in preexercise muscle glycogen concentration has been shown to have a significant effect on the metabolic and ventilatory response to subsequent exercise. For example, both resting and submaximal blood lactate concentration have been shown to be significantly lower during conditions of reduced muscle glycogen content compared with a control study (10). This, in turn, results in a shift of lactate AT to a

greater work rate during the glycogen depleted trial relative to the normal glycogen trial (11). Lower respiratory exchange ratios (R) have also been observed at every workrate in comparison with the normal glycogen condition (18), indicating an increased contribution of fat as a metabolic substrate. In addition, dietary-induced muscle glycogen depletion has been shown to affect resting and exercise minute ventilation (\dot{V}_E) values. Segal and Brooks (18) obtained lower resting \dot{V}_E values yet higher exercise \dot{V}_E values from subjects in the glycogen-depleted condition. A higher \dot{V}_E during graded exercise causes $AT\dot{V}_E$ to occur sooner than in the normal glycogen condition (11). Hughes, Turner, and Brooks (11) have previously demonstrated that a reduction in muscle glycogen prior to exercise causes a divergence between the anaerobic threshold as measured by nonlinear increases in \dot{V}_E and blood lactate. The AT values based on blood lactate nonlinearity occurred at a higher $\dot{V}O_2$ while the AT values based on \dot{V}_E nonlinearity occurred at a lower $\dot{V}O_2$ than in the normal glycogen state. Significant changes in AT based on blood lactate or \dot{V}_E nonlinearity were not observed pre- to post-training in this investigation. It is important to note, however, that the subjects in the present investigation continued to follow the hypocaloric diet up until the post-training graded exercise tests were performed.

If muscle glycogen concentration was lower after 8 wk of dietary intervention coupled with significant weight/fat loss, it could possibly explain the lack of significant correlation between AT_{HLA} and $AT_{\dot{V}_E}$ demonstrated post-training.

Another possible explanation is that in the untrained state minute ventilation (\dot{V}_E) and blood lactate concentration are linked more closely than gas exchange variables ($F_{E}O_2$, $F_{E}CO_2$, $\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$) and blood lactate. Training may somehow alter the respiratory control mechanisms responsible for this close linkage, thus altering the shape of the \dot{V}_E curve, making $AT_{\dot{V}_E}$ estimation more difficult. A poor correlation between $AT_{\dot{V}_E}$ and the AT criterion, $4 \text{ mmol} \cdot \text{L}^{-1}$ HLa concentration, would most likely result. A third possible explanation for this discrepancy between reproducibility of $AT_{\dot{V}_E}$ and \dot{V} involves the manner in which data for comparison; single data points occurring at a specific point in the GXT (\dot{V}) versus comparison of $AT_{\dot{V}_E}$ estimates based on computer regression analysis of data curves. Perhaps the \dot{V} curves were not stable enough post-training to allow reliable estimation of $AT_{\dot{V}_E}$ via computer. Instability of ventilatory data (\dot{V}) may be attributed to limitations in instrumentation. In this investigation, technicians recorded V data every 30 s during

each exercise test from the pneumotach LED display. Others have used computer controlled ventilatory sampling systems which are thought to allow a more precise measurement of ventilatory data based on a uniform number of respiratory cycles.

With the exception of $AT\dot{V}_E$ in the pre-training state, none of the AT estimates demonstrated validity in comparison with AT accepted as $4 \text{ mmol}\cdot\text{L}^{-1}$, pre- or post-training. This is in agreement with the results of a study conducted by Powers et al. (15) who also demonstrated low correlations between noninvasive and invasive AT. A recent study by Davis et al. (6) compared $AT\dot{V}_E/\dot{V}O_2$ to three lactate criterion ("breakpoint," $2 \text{ mmol}\cdot\text{L}^{-1}$, $4 \text{ mmol}\cdot\text{L}^{-1}$) for AT estimation. The results demonstrated that the ventilatory response consequent to metabolic acidosis during incremental exercise corresponds to the lactate "breakpoint" and not to a fixed, absolute lactate concentration of either 2 or $4 \text{ mmol}\cdot\text{L}^{-1}$. The use of the 2 or $4 \text{ mmol}\cdot\text{L}^{-1}$ criterion for AT determination resulted in large errors when compared to $AT\dot{V}_E/\dot{V}O_2$. One can speculate that perhaps the reviewer-selected $AT\dot{V}_{EO_2}$ and $AT\dot{V}_E/\dot{V}O_2$ values in this investigation would have demonstrated validity if lactate "breakpoint" instead of an absolute lactate value of $4 \text{ mmol}\cdot\text{L}^{-1}$ had been accepted as AT criterion. However, determination of lactate "breakpoint"

was not possible in this study due to lack of enough data points, (N=2 lactate samples) to establish a lactate curve for each subject.

Dependent t-tests revealed that training did not alter the invasive or noninvasive estimates of AT. This is in agreement with the results of a study by Skinner, Lemieux, and Taylor (20) which also indicated no change in AT after 8 wk of endurance training, 3 d•wk⁻¹ for 30-45 min at 65% $\dot{V}O_{2max}$. In contrast, both Davis et al. (4) and Sady et al. (17), employing training regimes similar to the one followed in the present investigation, found significant increases in AT. Davis et al. (4) examined sedentary middle-aged males before and after 9 wk of cycling 45 min•d⁻¹ 5 d•wk⁻¹ at an intensity above AT and found a 44% rise in AT expressed as $\dot{V}O_2$ (l•min⁻¹) and a 15% increase in AT expressed as $\dot{V}O_2$ (ml•kg⁻¹•min⁻¹). Sady et al. (17) trained 18 overweight college-aged women on a cycle ergometer 4 d•wk⁻¹ for 8 wk at either 80% $\dot{V}O_{2max}$ or 40% $\dot{V}O_{2max}$. Only those subjects who trained above the AT (80% $\dot{V}O_{2max}$) demonstrated a significant increase in AT. It appears that one must train above the level of AT in order to elicit changes in AT. This is supported by the results of a recent study by Gibbons et al. (8). Twenty-nine female college students trained on a treadmill for 20 min•d⁻¹, 4 d•wk⁻¹ for 8 wk. Subjects were

randomly assigned to one of three different intensity training groups. AT was shown to increase only in those subjects who trained at an intensity 40% above AT. The manner in which blood lactate was sampled and AT_{HLA} established in the present investigation limited the number of subjects for whom $\dot{V}O_2$ at AT_{HLA} could be determined prior to training ($N=6$, $\bar{x}=2.09$, $l \cdot \text{min}^{-1}$). Comparison of $\dot{V}O_2$ at 70% $\dot{V}O_{2\text{max}}$ with $\dot{V}O_2$ at AT_{HLA} (GXT_2) for these six subjects revealed that five would be exercising below AT_{HLA} while one would be exercising above AT_{HLA} when training at an intensity designed to elicit 70% $\dot{V}O_{2\text{max}}$. A similar analysis comparing $\dot{V}O_2$ at 70% $\dot{V}O_{2\text{max}}$ with $\dot{V}O_2$ occurring at $AT_{\dot{V}E}$ (GXT_2) demonstrated that five subjects would be training above while five subjects would be training below their respective anaerobic thresholds. $AT_{\dot{V}E}$ could not be determined for the remaining two subjects due to "indeterminate" data curves. Subjects training above either AT_{HLA} or $AT_{\dot{V}E}$ when training at 70% $\dot{V}O_{2\text{max}}$ did not demonstrate a significant increase in $\dot{V}O_2$ at either AT_{HLA} or $AT_{\dot{V}E}$ post-training.

However, the differences in initial fitness levels of the subjects examined here and those examined by Davis et al. (4) and Sady et al. (17) accounts at least in part for the fact that they detected significant increases in AT following training while the present study reported no changes in this

parameter. The subjects examined in this study while untrained could not be classified as low fit ($\bar{x}=2.37 \dot{V}O_{2max}$, $l \cdot \text{min}^{-1}$). Those examined by Davis et al. (4) ($\bar{x}=2.77 \dot{V}O_{2max}$, $l \cdot \text{min}^{-1}$) and Sady et al. (17) ($\bar{x}=2.16$, $\dot{V}O_{2max}$, $l \cdot \text{min}^{-1}$) would readily fit into the low fitness classification. The magnitude of increase in AT with training appears to depend on the individual's initial level of fitness as well as the intensity of the training program.

In conclusion, it appears that a given reviewer can unknowingly discriminate the same AT_{FEO_2} or $AT_{\dot{V}E/\dot{V}O_2}$ from duplicate data plots. It is also apparent that different reviewers can reliably discriminate the same AT_{FEO_2} or $AT_{\dot{V}E/\dot{V}O_2}$ based on visual inspection of gas exchange data plots. It is likely, therefore, that physiological variability of the subjects is the cause of the low test-retest reproducibility of AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ observed pre- and post-training. Neither AT_{FEO_2} nor $AT_{\dot{V}E/\dot{V}O_2}$ was shown to be valid in comparison with AT_{HLA} . $AT_{\dot{V}E}$, based on computer regression, however, was shown to be both reliable and valid in comparison with AT_{HLA} in the pre-training state. Further research should be directed toward determining whether AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ are highly correlated with AT_{HLA} when selected via computer regression method rather than by multiple reviewers. Furthermore, the results of the present

investigation revealed that this training program does not provide sufficient stimulus to produce significant changes in $\dot{V}O_{2\max}$ ($L \cdot \text{min}^{-1}$) or the anaerobic threshold in originally sedentary, overweight women.

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TABLE 1. Physical characteristics of subjects

n=12	Age (yr)	Weight (kg)	Body Fat (%)	$\dot{V}O_{2max}$ (L·min ⁻¹)(ml·kg ⁻¹ ·min ⁻¹)	
\bar{X}	23.6	73.3	32.2	2.4	33.4
±SEM	±1.7	±4.7	±1.2	±0.1	±1.8

$\dot{V}O_{2max}$, maximal oxygen consumption

TABLE 2. Intercorrelations between reviewers' ratings for all four GXT's using AT₁ p₁:0.2 expressed as GXT time

	GXT ₁			GXT ₂			GXT ₃			GXT ₄		
	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃
R ₂	0.71			0.87			0.85			0.76		
R ₃	0.92	0.90		0.90	0.98		0.87	0.83		0.95	0.70	
R ₄	0.89	0.83	0.83	0.92	0.97	0.97	0.97	0.85	0.88	0.95	0.91	0.87
	n=10			n=11			n=9			n=10		

All Spearman Rho values shown are statistically significant at $P \leq 0.05$; those ≥ 0.83 are significant at $p \leq 0.01$.

GXT₁, pre-training maximal treadmill test
 GXT₂, pre-training submaximal treadmill test
 GXT₃, post-training maximal treadmill test
 GXT₄, post-training submaximal treadmill test

TABLE 3. Intercorrelations between reviewers' ratings for all four GXT's using $\Delta\dot{V}_E/\dot{V}O_2$ expressed as GXT time

	GXT ₁			GXT ₂			GXT ₃			GXT ₄		
	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃	R ₁	R ₂	R ₃
R ₁	0.59			0.96			0.88			0.80		
R ₂	0.58	0.60		0.91	0.94		0.99	0.86		0.90	0.93	
R ₃	0.96	0.50	0.61	0.94	0.86	0.77	0.99	0.86	1.00	0.97	0.67	0.82
	n=10			n=11			n=9			n=10		

All Spearman Rho values shown are statistically significant at $P \leq 0.05$, those ≥ 0.83 are significant at $P \leq 0.01$.

GXT₁, pre-training maximal treadmill test
 GXT₂, pre-training submaximal treadmill test
 GXT₃, post-training maximal treadmill test
 GXT₄, post-training submaximal treadmill test

TABLE 4. Validity estimates for noninvasive indicators of AT in overweight women

Comparison	Pre-Training Test	Post-Training Test
AT_{HLa} vs $AT\dot{V}E$	0.94 df=5	0.59 df=8
AT_{HLa} vs AT_{FEO_2}	0.41 df=6	0.37 df=7
AT_{HLa} vs $AT\dot{V}E/\dot{V}O_2$	0.71 df=6	0.36 df=6

a = Spearman Rho correlation coefficient significant at $p < 0.01$

See text for special AT symbol clarification and explanation of origin of pre-training and post-training values.

TABLE 5. Effect of 8 wk training program on weight, % fat, and various fitness variables.

Variable	Begin	8-wk	t-ratio
Weight (kg)	73.25±4.7	68.52±4.4	7.72*
Body Fat (%)	32.23±1.2	29.55±1.2	7.10*
$\dot{V}O_{2\max}$ ($l \cdot \text{min}^{-1}$)	2.37±0.1	2.45±0.1	-1.18
$\dot{V}O_{2\max}$ ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	33.43±1.8	36.71±1.6	-2.68*
Resting heart rate ($\text{bts} \cdot \text{min}^{-1}$)	85.00±4.1	88.00±3.7	-0.91
Resting HLa ($\text{mmol} \cdot \text{l}^{-1}$)	0.88±0.2	0.68±0.1	1.51
HLa ₁ vs HLa ₃ ($\text{mmol} \cdot \text{l}^{-1}$)	2.14±0.3	1.98±0.2	0.64
HLa ₂ vs HLa ₄ ($\text{mmol} \cdot \text{l}^{-1}$)	2.82±0.4	3.13±0.3	-1.45

* significant at ($p < 0.05$)

Values are means \pm SEM

HLa, blood lactate concentration

$\dot{V}O_{2\max}$, maximal oxygen consumption

HLa₁, first blood sample taken during GXT₂ pre-training

HLa₂, second blood sample taken during GXT₂ pre-training

HLa₃, first blood sample taken during GXT₄ post-training

HLa₄, second blood sample taken during GXT₄ post-training

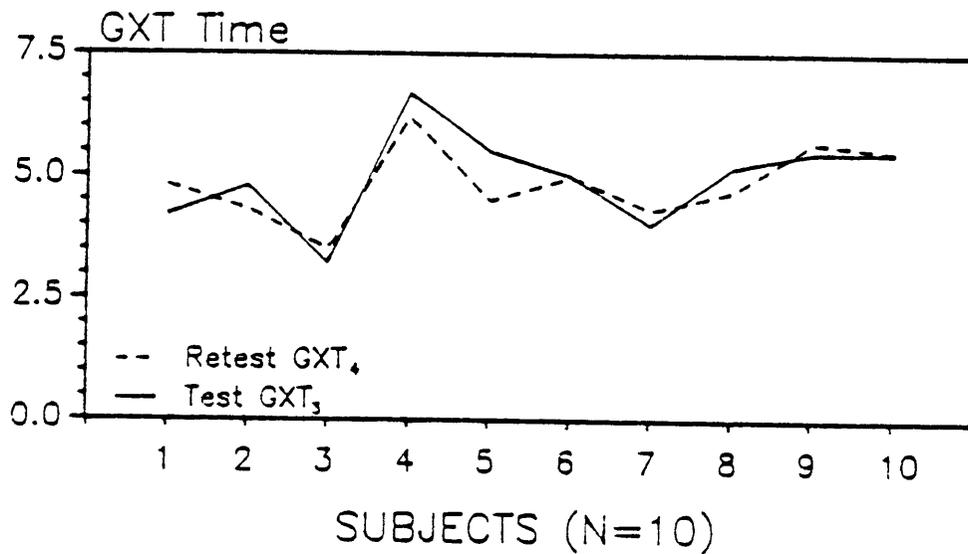
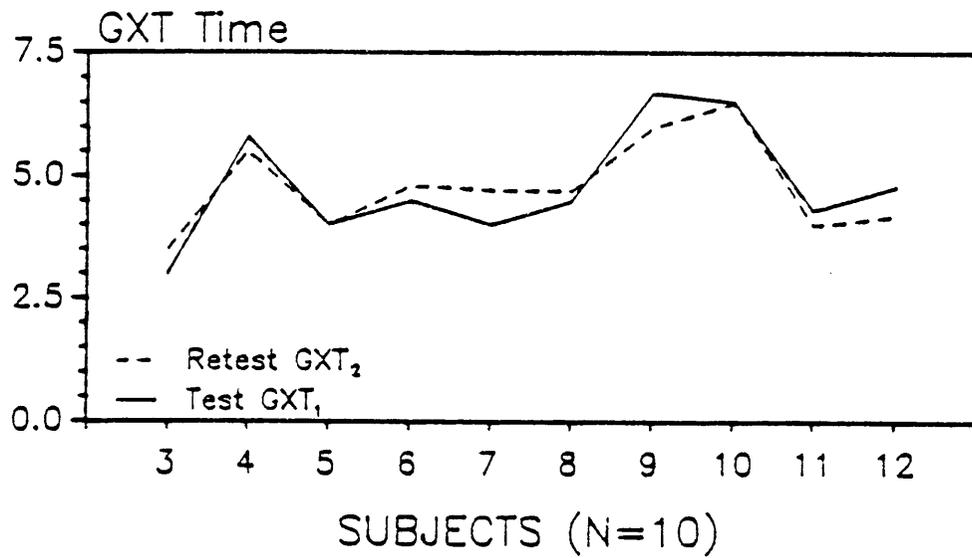


Figure 1. Spearman Rho Test-Retest Reliability Estimate for $AT_{\dot{V}_E}$ - Computer Method ($P=.85$)

Spearman Rho Test-Retest Reliability Estimate for $AT_{\dot{V}_E}$ - Computer Method ($P=.55$)

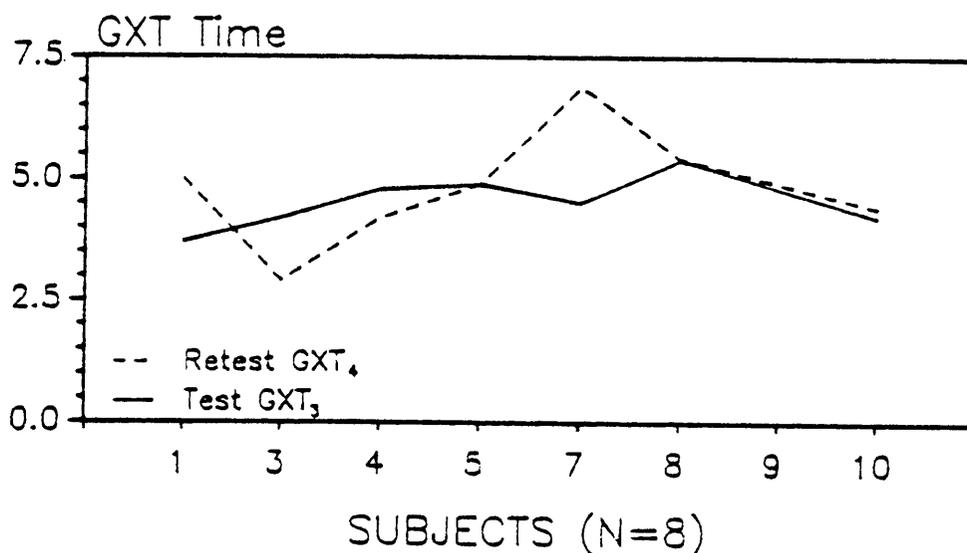
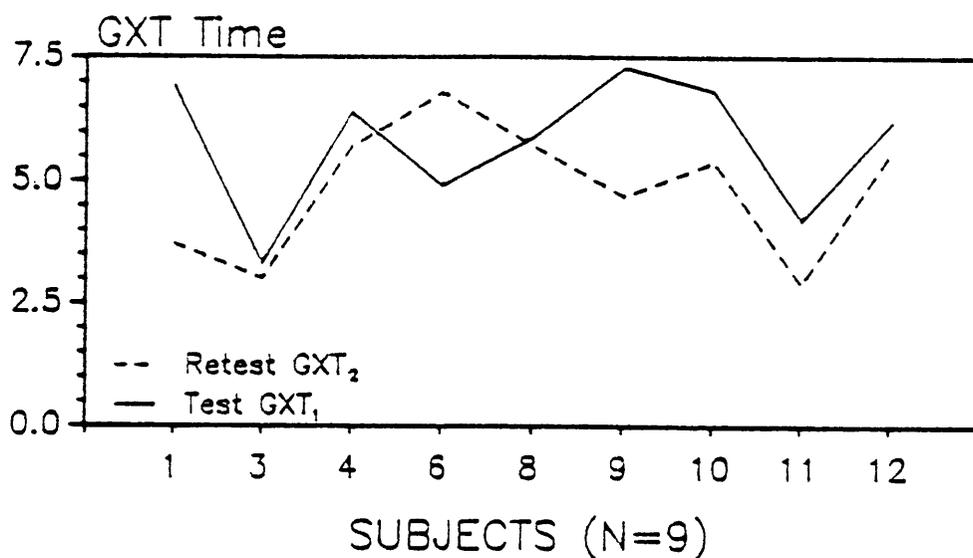


Figure 2. Spearman Rho Test-Retest Reliability Estimate for $AT_{F_{E}O_2}$ - Reviewer Method
($P=.10$)

Spearman Rho Test-Retest Reliability Estimate for $AT_{F_{E}O_2}$ - Reviewer Method
($P=.22$)

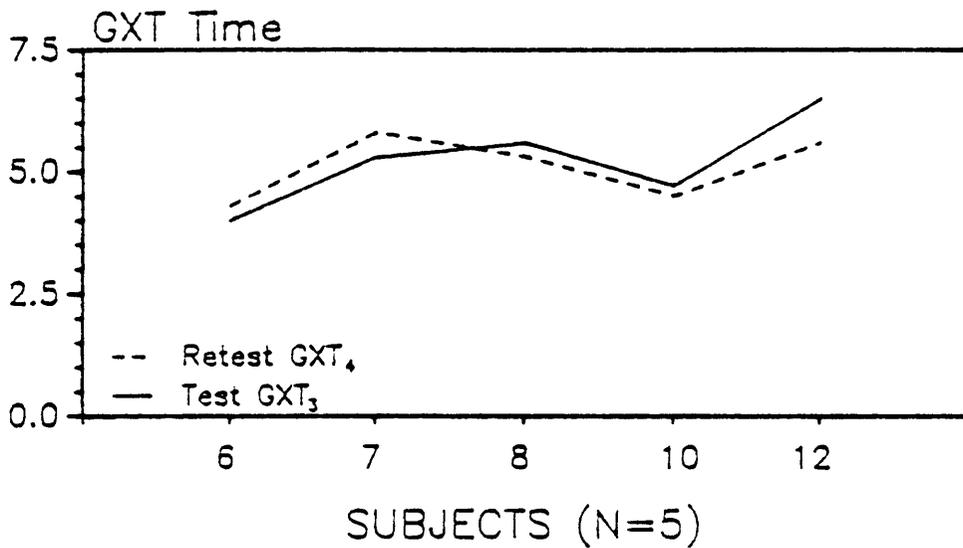
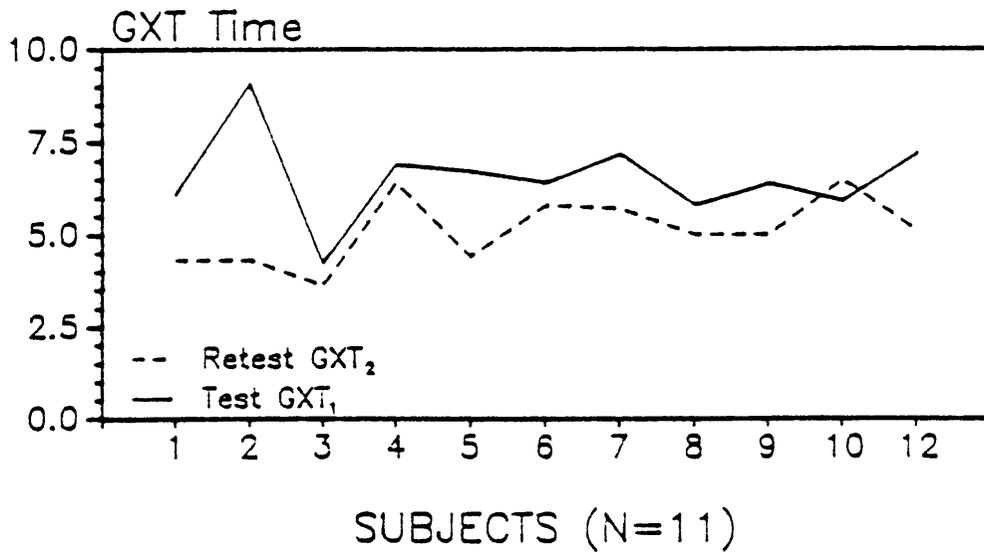


Figure 3. Spearman Rho Test-Retest Reliability Estimate for $AT_{\dot{V}_E/\dot{V}O_2}$ - Reviewer Method
($P=.15$)

Spearman Rho Test-Retest Reliability Estimate for $AT_{\dot{V}_E/\dot{V}O_2}$ - Reviewer Method
($P=.70$)

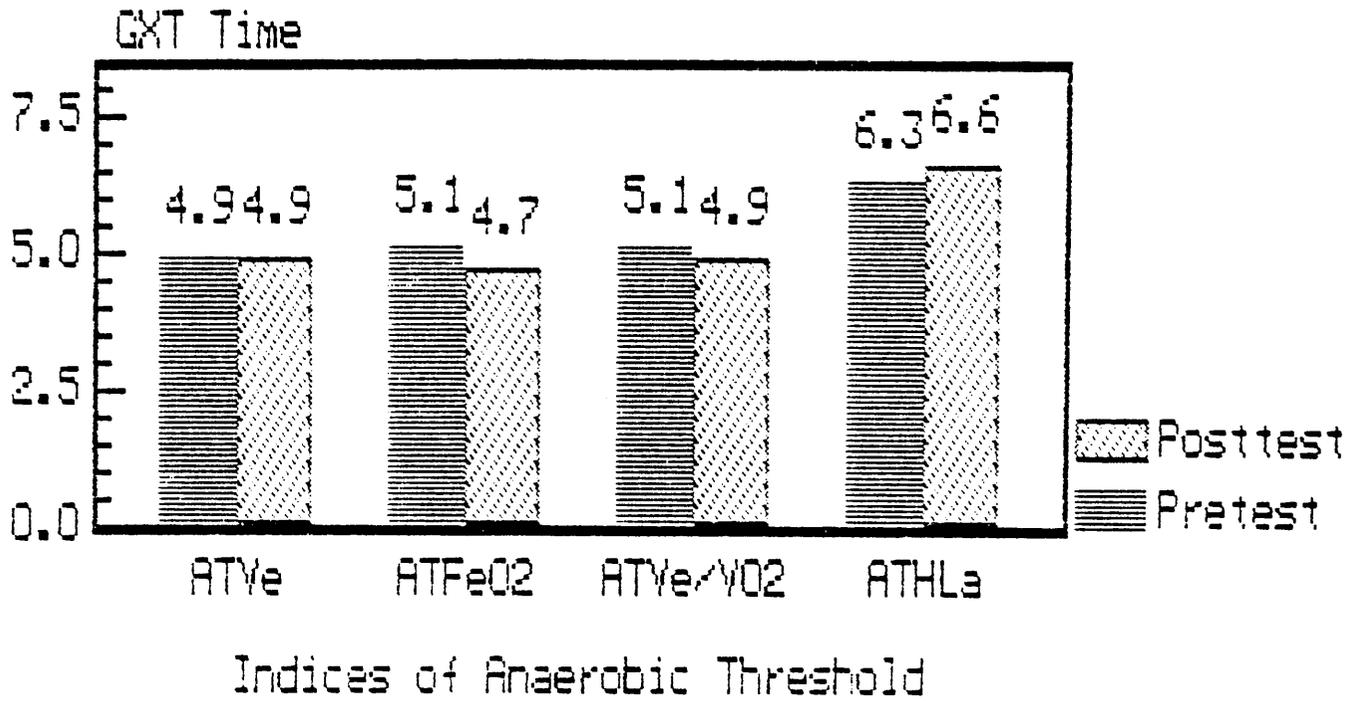


Figure 4. Training Effect on Anaerobic Threshold

Chapter IV

SUMMARY AND RECOMMENDATIONS FOR FUTURE RESEARCH

Summary

Hypotheses and Background. This investigation was primarily conducted to evaluate the accuracy and reproducibility of AT estimation from noninvasive indices by four independent reviewers and a computerized polynomial regression program against the AT criterion, represented by a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration. A secondary purpose was to examine the effects of a short-term aerobic training program on the AT determined from invasive and noninvasive measures in originally sedentary, overweight women.

Metabolic and blood lactate responses were evaluated in twelve sedentary, overweight women who underwent 8 wks of aerobic training coupled with dietary intervention. The aerobic training consisted of an outdoor walk/jog program conducted $3 \text{ d}\cdot\text{wk}^{-1}$ at $70\% \dot{V}O_{2\text{max}}$. Concurrently with the training, subjects consumed a hypocaloric diet (i.e., $1200\text{-}1400 \text{ kcal}\cdot\text{d}^{-1}$) designed to promote a $1 \text{ kg}\cdot\text{wk}^{-1}$ weight loss.

Before and after the 8 wk training program, subjects underwent two incremental treadmill tests. The first graded

exercise test (GXT_1) required the subject to exercise to exhaustion. The resultant ventilatory and gas exchange data, collected via open circuit spirometry, was utilized in the determination of each subjects' exercise training prescription and times for fingertip blood sampling during the subsequent exercise test (GXT_2).

Within 3 d of GXT_1 , GXT_2 was administered to all subjects. The blood lactate and ventilatory/gas exchange data collected during this test was used to determine invasive and noninvasive AT values, respectively. The validity of noninvasive AT estimates was determined in comparison with an accepted AT criterion measure, blood lactate concentration of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$. Furthermore, the protocol for GXT_2 remained identical to GXT_1 until the second fingertip blood sample was taken to allow determination of test-retest reliability of the ventilatory and gas exchange indices upon which noninvasive AT is based. All blood samples were immediately analyzed for lactate concentration utilizing the YSI Scientific Model 23L lactate analyzer. Estimates of GXT time at AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$ were made by four independent reviewers and determination of GXT time at $AT_{\dot{V}_E}$ was made through the use of a computerized two-component polynomial regression method (Warne, 1980). The resulting AT values were then compared to the GXT time corresponding to

a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration. Other investigators from this laboratory have previously utilized this computerized regression program to determine AT (Herbert, Sebolt, Bradley, Moore, & Robbins, 1982; Jones, 1984; Moore, 1983). However, none of these investigators attempted to validate this computerized regression method by comparison with accepted blood lactate indicators of AT. In the present investigation, a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration was used to represent the anaerobic threshold in an attempt to eliminate the subjectivity involved in determination of AT as either an increase above resting lactate values (Caiozzo et al., 1982) or the point of lactate "breakaway" when graphed as a function of exercise time (Davis et al., 1983). Moreover, a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ lactic acid level seems to be associated with the highest exercise intensity level that an individual can sustain for prolonged periods of time (Sjodin & Jacobs, 1981). Hence, the $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa level may be considered to be an important predictor of endurance performance (Farrell et al., 1979; Sjodin & Jacobs, 1981).

Spearman Rho correlation coefficients were determined to identify relationships between variables. Dependent t-test were conducted to examine the differences pre- to post-training in blood lactate concentration, AT values, body

weight, body fat, maximal oxygen consumption, and resting heart rate values. All tests were considered significant at the 0.05 probability level. SAS computer programs were used to conduct statistical analyses.

Results of Reliability Hypotheses. The results of this study showed that prior to training all of the noninvasive indices of AT (\dot{V} , $F_{E}O_2$, $F_{E}CO_2$, $\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$) were reproducible, demonstrating significant test-retest reliability when measured at the predicted AT point (0.59, 0.58, 0.69, 0.64, 0.58, $p < 0.05$). Yet, after training, only \dot{V} remained reproducible ($P = 0.79$, $p < 0.05$) when measured at the same predicted AT time. In addition, $AT\dot{V}_E$ values, estimated via the computer method, were shown to be significantly correlated ($P = 0.85$, $p < 0.01$), thus, reproducible from test to test in the pre-training state. Post-training, however, the correlation was no longer significant ($P = 0.55$, $p < 0.05$).

Test-retest reliability was also determined for the reviewer method of AT estimation. The reviewer-selected AT_{FEO_2} values were shown to be more reliable than estimation of $AT\dot{V}_E/\dot{V}O_2$ by reviewers (100% of AT_{FEO_2} values significant at the 0.05 probability level vs 83% of $AT\dot{V}_E/\dot{V}O_2$ values). (However this is most likely a function of the number of "indeterminate" graphs for each method.) Intra-reviewer reliability was assessed by including duplicate graphs ($n = 8$)

mixed in among the other coded data graphs (n=96) given to each reviewer for AT estimation.

Results of Validity Hypothesis. Validity coefficients were determined by individually correlating each noninvasive AT value with the criterion measure, AT_{HLA} , for each subject (Appendix F). Only one noninvasive AT estimation method, AT_{VE} -computer, derived from pre-training data, demonstrated a high correlation coefficient with AT_{HLA} ($P=0.94$, $p<0.01$). After the training treatment, none of the noninvasive AT estimation methods significantly correlated with AT_{HLA} .

CONCLUSIONS

From the findings of this investigation the following conclusions are noted:

1. The method of AT_{VE} estimation, from pre-training data, via a two-component polynomial regression appears to be both highly reliable and valid when tested against the AT criterion of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate.

2. The estimation of AT_{FEO_2} and $AT_{VE}/\dot{V}O_2$ by four independent reviewers appears to be reproducible yet not valid when tested against this AT criterion, a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration.

3. The 8 wk training program failed to alter the AT derived from either blood lactate or ventilatory/gas exchange data.

4. The lack of a significant decrease in either resting or submaximal blood lactate concentrations, pre- to post-training, demonstrates that the aerobic training program was not of sufficient intensity/duration to produce a training effect.

5. In addition, the significant increase in relative $\dot{V}O_{2\max}$, without a significant increase in absolute $\dot{V}O_{2\max}$, suggests that it is a function of weight/fat loss resulting from the dietary intervention rather than an increase in aerobic capacity due to the training program.

IMPLICATIONS

The results of this study contribute information regarding the reliability and validity of two currently popular methodologies of noninvasive AT estimation. The high correlation between $AT_{\dot{V}E}$, estimated using the computer regression program, and AT_{HLA} only in the pre-training state, may have been due to the small number of subjects. On the other hand, the possibility exists that this high correlation reflects a close relationship that is altered by physical training. Further research, using a greater number of subjects, is needed to establish whether or not $AT_{\dot{V}E}$ can be used to accurately and reliably predict AT_{HLA} . The low correlations between AT_{FEO_2} , $AT_{\dot{V}E}/\dot{V}O_2$ and AT_{HLA} , despite evidence of inter- and intra-reviewer reliability, leads to

the conclusion that multiple reviewers can provide reliable yet invalid estimates of noninvasive AT in comparison with AT criterion of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ HLa concentrations. Therefore, the results of this investigation do not support the future use of the multiple reviewer method of noninvasive AT estimation from gas exchange data.

The combination of aerobic training and dietary intervention used in this study was sufficient to produce a significant increase in relative $\dot{V}O_{2\text{max}}$ as well as significant decreases in body fat and weight in this small sample of originally sedentary, overweight women. These results support the future use of this particular training/diet program for weight/fat loss in a similar subject sample. However, an increase in relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) coupled with weight/fat loss without a significant increase in absolute $\dot{V}O_{2\text{max}}$ ($\text{L}\cdot\text{min}^{-1}$) demonstrates that it is possible to increase exercise capacity without developing aerobic fitness.

Measurement of AT in the Laboratory Setting. Evidence from this study indicates a poor correlation between noninvasive AT and AT_{HLa} except for $AT_{\dot{V}E}$ in the pre-training state. This may possibly be due to the small number of blood samples taken during exercise. It is suggested, therefore, that future researchers sample blood for lactate

concentration every workload. This would allow estimation of AT as the "breakpoint" in the lactate curve which has been previously shown to correlate more highly with noninvasive AT than absolute blood lactate concentrations of 2 or 4 $\text{m}\cdot\text{mol}\cdot\text{L}^{-1}$ (Davis et al., 1983). The ramp-type protocol used in this investigation (30 s stages) does not make fingertip sampling every workload a practical means of obtaining data for plotting a lactate curve. Fingertip sampling involves pre-warming the finger for 30 s- 45 s prior to puncture. In addition, fingertip sampling is somewhat painful and annoying for the subject who continues to keep pace with the treadmill during sampling. It is recommended that frequent blood sampling be accomplished through the use of an indwelling catheter.

Use of this computerized polynomial regression method in future research is recommended to resolve the question of whether or not it provides an accurate and reliable estimation of noninvasive AT. Others (Green et al., 1983; Orr et al., 1982) have utilized multisegment linear computer algorithms for a similar purpose. Orr et al. (1982) demonstrated good correlation of $\text{AT}\dot{V}_E$ -computer with $\text{AT}\dot{V}_E$ -reviewer (0.94). This indicates that both the computer algorithm and reviewers are evaluating the data for $\text{AT}\dot{V}_E$ in the same manner. However Green et al. (1983), using the

computer algorithm developed by Orr et al. (1982), failed to demonstrate a relationship between $AT\dot{V}_E$ and blood lactate AT. In theory, the polynomial regression computer method utilized in the present investigation appears to be better suited to accurately select $AT\dot{V}_E$ due to the polynomial nature of the \dot{V}_E response to exercise.

Practical Application of the AT. The appearance of an AT during incremental exercise indicates insufficient oxygen supply to the working muscles. The impaired cardiac output of cardiac patients is a common contributor to early onset of exercise-induced metabolic acidosis. It is not surprising to find that the AT is currently being used in the diagnosis and treatment of cardiac patients (Wasserman, 1984). The practice of prescribing exercise intensity via an arbitrary percentage of maximal heart rate or $\dot{V}O_{2max}$ accounts for the differences between individuals whose maximum heart rate or oxygen consumption values differ, yet ignores differences between individuals at submaximum exercise levels. Basing exercise intensity prescriptions on the patient's $\dot{V}O_{2max}$ and the percent of $\dot{V}O_{2max}$ at which a patient can sustain aerobic work (AT), avoids setting target heart rates too high in relationship to the level of metabolic acidosis. A patient whose exercise prescription is set above his AT may experience early onset of fatigue during rehabilitative

exercise sessions, possibly causing poor adherence or even attrition from the program.

In addition, this investigator speculates that use of the AT in addition to $\dot{V}O_{2max}$ in establishment of exercise intensity prescriptions may potentially increase adherence of obese individuals to an exercise program. An AT-based exercise prescription may enable obese individuals to continue exercising for the prolonged periods needed to foster weight/fat loss. Exercise of moderate intensity is probably best for weight reduction because of its fat mobilizing effects. More intense, brief exercise causes the accumulation of lactate which is known to inhibit the mobilization of free fatty acids from adipose tissue.

The AT can also be employed to effectively evaluate submaximal fitness which is of special importance to those subjects for whom maximal testing is difficult (i.e., cardiac patients, obese individuals). Evaluation of the AT allows determination of the effectiveness of the training program on submaximal fitness despite lack of an apparent increase in $\dot{V}O_{2max}$.

RECOMMENDATIONS FOR FUTURE RESEARCH

It is recommended that future AT research investigations utilize an indwelling catheter to allow blood lactate sampling at each stage of an incremental exercise test.

Frequent blood sampling would allow the investigator to determine lactate AT from a "breakpoint" in the lactate curve when graphed as a function of exercise time. This would clearly be an improvement over the estimation of AT_{HLA} , accepted as $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$, via use of an "idealized" blood lactate curve and two blood lactate values per subject used in the present investigation.

Another potential improvement over the current technique would involve basing each subject's exercise training intensity prescription on their AT_{HLA} value instead of an arbitrary percentage of their $\dot{V}O_{2\max}$. This would avoid the problem encountered in the present investigation of subjects exercising above or below their AT_{HLA} value at the prescribed $70\% \dot{V}O_{2\max}$ exercise intensity. Standardization of exercise intensity for training across subjects is mandatory for meaningful interpretation of pre- to post-training alterations.

Use of the reviewer method to determine $AT_{\dot{V}E}$ would allow determination of whether the reviewer method is a valid method for AT estimation based on ventilatory data. In the present investigation, the reviewer method was shown to be reliable yet not valid in comparison with the AT criterion, blood lactate concentration. However, the reviewers were

only allowed to select AT values (AT_{FEO_2} , $AT_{\dot{V}E/\dot{V}O_2}$) from graphs of gas exchange data.

Therefore, it is recommended that a future study, designed to assess the reliability and validity of noninvasive AT estimation, utilize both the computer and reviewer methods to select $AT_{\dot{V}E}$ values. Then the $AT_{\dot{V}E}$ -computer and $AT_{\dot{V}E}$ -reviewer values could be compared with each other and against AT_{HLa} values determined from the "breakpoint" in blood lactate curves. The results of such a study may provide the additional evidence needed to justify incorporation of either or both of these AT estimation methodologies into future research investigations in place of blood lactate sampling. This would be of value since blood sampling is an expensive and frequently painful procedure.

Another recommendation would be to further investigate the effects of aerobic training on the AT. The present investigation could not effectively distinguish between effects of the exercise training and dietary intervention on the relative $\dot{V}O_{2max}$, weight, and body fat changes. An investigation in which subjects were randomly assigned to a control group, an exercise group, and a diet group (randomized groups, pretest-posttest design) could shed some light on the separate effects of training and dietary

intervention on $\dot{V}O_{2\max}$, blood lactate levels, and estimates of AT.

The training program utilized in the present study was of insufficient duration and/or intensity to produce a significant training effect on AT values, absolute $\dot{V}O_{2\max}$, resting heart rate, or resting and submaximal blood lactate values. A well-controlled study is needed in which groups of overweight or obese women are trained at different intensities for a longer duration than the present investigation. For example, subjects could be monitored while they walk/jog on motor-driven treadmills at exercise intensities eliciting 60%, 75%, or 85% $\dot{V}O_{2\max}$ for 45 minutes $3 \text{ d}\cdot\text{wk}^{-1}$ for twelve weeks. An alternative study would involve assessment of the effects of training above or below one's AT level. All subjects would first undergo a maximal exercise test for the purpose of collecting metabolic and blood lactate samples to aid in determination of AT. Subjects would then be randomly assigned to train at an exercise intensity above or below their respective AT levels. The results of such a study would hopefully answer the question of whether training overweight or obese women above or below their AT results in differing effects on such factors as adherence to the exercise program, weight and fat

loss, increases in aerobic capacity, and increases in AT levels.

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APPENDIX A
DETAILED METHODOLOGY

METHODOLOGY

Introduction

A one-group pretest-posttest design (Ary, Jacobs, & Razavieh, 1979) was used in this investigation to assess the reliability and validity of anaerobic threshold determination via two currently popular methods of "objectifying" noninvasive AT determination. Metabolic and blood lactate responses were evaluated in twelve sedentary, overweight females before and after an 8 wk experimental period in which they performed aerobic exercise training 3 d•wk⁻¹ and consumed a hypocaloric diet. Data collection required the subjects to undergo two incremental treadmill tests, pre- and post-training.

Subject Selection and Screening

Twelve females were selected from a pool of volunteers who had responded to a public advertisement. The criteria for inclusion into the sample were:

1. females, aged 18-35 yrs;
2. sedentary, defined as not currently engaged in a 3 d•wk⁻¹ endurance activity;
3. overweight, defined as greater than 22% body fat;
4. free from medical contraindications to maximal exercise testing and walk/jog endurance training.

Important physical characteristics are presented in Appendix B.

Each subject completed a detailed medical history questionnaire (Appendix C) which included questions concerning physical activity participation. All subjects selected were non-smokers. None had participated in a 3 d•wk⁻¹ conditioning program in the 6 mos immediately prior to this investigation.

Prior to participation, each subject was informed of the nature of the experiment and its inherent risks. Each gave verbal and written consent (Appendix C).

Resting blood pressure was measured and an investigator trained in body composition assessment recorded each subject's height, weight, and skinfold measurements on the screening form (Appendix C). Each subject was weighed on the same scale to the nearest 0.5 kg each week during the study. The scale was checked for calibration accuracy prior to weekly weigh-ins.

Determination of Body Composition. Skinfold measurements were taken with a Harpenden caliper (John Bull, LTD) at three sites: triceps, thigh, and abdomen. The average of three successive measurements at each site was recorded to the nearest 0.1 mm. Body composition was then

determined using equations developed by Durnin and Womersley in 1974.

This preliminary screening of prospective subjects was done to ensure that subjects met all criteria for inclusion in the study.

Experimental Protocol

Orientation and Pre-testing Instruction. Prior to exercise testing, subjects participated in a laboratory orientation session to become familiarized with the data collection equipment and procedures. This was done in an attempt to minimize subject anxiety and habituation effects in the exercise test data.

Pre-exercise test instructions (Appendix C) were fully explained; instructing subjects to abstain from eating, exercise, and consumption of caffeine, alcohol, nicotine, and certain medications for the 3 h period preceding each exercise test. Each subject received a written copy of these instructions.

Maximal Oxygen Uptake Exercise Test. A maximal graded exercise test was first administered to collect noninvasive metabolic data needed for determination of each subject's exercise training prescription and determination of two exercise blood lactate sampling times.

Pre-testing Preparation of Subjects. Upon arrival at the laboratory each subject was weighed to the nearest 0.5 kg on a physician's scale. Electrodes were placed in the CM₅ lead configuration on the subject's chest. Subjects were then allowed to rest in the supine position on a padded table directly adjacent to the treadmill. After approximately 5 min, resting heart rate and blood pressure were taken in the supine and then the standing position. The subject was then asked to stand on the treadmill (Quinton Models 24-72 or 55) and breathe through a Daniel's two-way valve fitted with a flexible, rubber mouthpiece for at least 1 min prior to testing. A noseclip was used to ensure occlusion of the nasal airway.

Treadmill Protocol. An approximation of a ramp treadmill protocol (Appendix C) was used in which the increments resembled the Balke clinical test protocol. Each subject began walking at a speed of 3.4 m.p.h. at 0% grade (3.6 METS). After 2 min, the speed and/or was increased every 30 s in increments corresponding to an overall increase of 0.5 METS/stage. Subjects were allowed to run to keep pace with the treadmill and encouraged to continue until exhaustion. Subjects were not allowed to grasp the treadmill handrail during the exercise test to avoid falsely increasing the estimated exercise capacity.

Data Collection. Open circuit spirometry methods were utilized for collection of metabolic data throughout the test. Minute equivalents of ventilation (\dot{V}_E , BTPS, $L \cdot \text{min}^{-1}$) were continuously measured by a digital Pneumotach or P-C dry gas meter. Measurements were recorded every 30 s. After passing through a dessicant (Aquasorb®), fractions of expired oxygen and carbon dioxide ($F_{E\text{O}_2}$, $F_{E\text{CO}_2}$) were measured continuously by rapid-response electronic gas analyzers and recorded at 15 s intervals. As with any system that uses a mixing chamber and a drying tube, there is a temporal misalignment between measurements of ventilation and the corresponding gas exchange. Therefore, a 15 s phase delay was accepted between \dot{V} measurement and stabilization of $F_{E\text{O}_2}$ and $F_{E\text{CO}_2}$ readings.

Heart rate was continuously monitored on an oscilloscope and recorded using an electrocardiograph (Hewlett-Packard model 1500B) at rest (supine and standing), during the last 10 s of each 30 s treadmill stage, as well as every minute post-exercise.

Blood pressure was taken with the subject at rest (supine and standing), and every odd minute during exercise and recovery phases.

Borg's (Noble et al., 1983) category-ratio scale (Appendix C) was used to assess each subject's rating of perceived exertion (RPE) every minute throughout the test.

Following termination of the test, the subject rested in the supine position on a padded table directly adjacent to the treadmill while blood pressure and heart rate monitoring continued until both returned to resting levels.

$\dot{V}O_2$ max Determination. The $\dot{V}O_2$ max for each subject was considered to be the highest oxygen uptake observed within the last minute of the test.

Maximal Test Determination. The subject was determined to have performed maximally if at least two of the three following criteria were attained (LaFontaine et al., 1981):

1. Heart rate at the end of the test was $\geq 90\%$ of age-adjusted predicted HR_{max} ($220 - \text{age}$).
2. The respiratory exchange ratio (R) reached or exceeded 1.0 toward the end of the test.
3. Oxygen consumption ($\dot{V}O_2$) plateaued (i.e., increase of less than $150 \text{ ml} \cdot \text{min}^{-1}$) or declined during the final stages of the test despite increased speed and/or grade.

Determination of Exercise Training Prescription. Each subject's exercise prescription was set at a heart rate value corresponding to 70% of subjects' $\dot{V}O_2$ max. Seventy percent of each subject's $\dot{V}O_2$ max value was calculated. The

investigator then reviewed the heart rate and oxygen consumption data collected during each treadmill stage. The recorded heart rate occurring at the time 70% $\dot{V}O_2$ max was achieved was selected to serve as the training target heart rate. Exercise prescriptions allowed a range of 5 bts above and below the target heart rate.

Determination of Noninvasive Anaerobic Threshold

Values. For the purposes of this investigation, the anaerobic threshold was accepted as:

$AT\dot{V}_E$: The point beyond which there occurred a non-linear rise in \dot{V}_E when plotted versus incremental exercise time (Wasserman et al., 1973; Davis et al., 1976).

AT_{FEO_2} : The point at which an increase in F_{EO_2} occurred without a corresponding decrease in F_{ECO_2} when plotted versus incremental exercise time (Withers et al., 1981).

$AT\dot{V}_E/\dot{V}O_2$: The point at which a systematic increase in $\dot{V}_E/\dot{V}O_2$ occurred without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ when plotted versus incremental exercise time (Caiozzo et al., 1982).

After completion of pre-training testing, a microcomputer (Apple II-48k) was used to objectively and graphically display the ventilatory (\dot{V}_E), gas fraction (F_{EO_2} , F_{ECO_2}), and ventilatory equivalent ($\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$) data versus incremental exercise time.

Determination of Anaerobic Threshold via Computing Regression Method. A two-component polynomial least square curve-fitting technique (Warne, 1980) was used to produce regression models of \dot{V}_E on exercise time to aid in the objective determination of the "breakaway" point (Appendix E).

1. Using the Apple II keyboard, enter bivariate data points (\dot{V}_E vs. incremental exercise time). Determine the lowest order polynomial which best fits the data (i.e., the highest r , lowest $SE \cdot y$). Print the polynomial plot of \dot{V}_E values versus incremental exercise time.

2. From this graph, visualize the point at which \dot{V}_E becomes non-linear. Determine the number of bivariate points which constitute the linear segment of the curve. Add the bivariate point above that level and record this value on the data recording form.

3. Enter this new data set for regression analysis by punching the return key the required number of times to include the exact number of bivariate data points just recorded on the data recording form. Each new data set in the regression should include one less bivariate data point.

4. Record the correlation coefficient and standard error for each new data set on the data recording form.

Determining the Best Regression Line.

1. Determine the most appropriate linear regression line. This line should consist of the greatest number of data points which gives the highest r and lowest $SE \cdot y$ values. Record the number of points included in the line on the data recording form.

2. From the line selected in step 1, select two representative points and record and x and y coordinates for each. These will be designated as $1x$ and $1y$ and $2x$ and $2y$. Record these coordinates on the data recording form.

3. Refer to the previously printed second degree polynomial plot. On this graph, impose the coordinates $1x$ and $1y$ and $2x$ and $2y$.

4. Draw a line connecting the points plotted using $1x$ and $1y$ and $2x$ and $2y$. This is the best linear regression.

5. The point on the x axis which corresponds to the intersection of the linear regression line and the second degree polynomial curve is considered the anaerobic threshold (expressed as an incremental exercise time value).

Determination of the Anaerobic Threshold via Reviewer Method. Determination of the AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ was performed by four independent, outside reviewers. Reviewers were volunteers selected from students pursuing Master's degrees in Exercise Physiology. Each reviewer independently reviewed computer-generated graphs of gas fraction (F_{EO_2} ,

$F_{E}CO_2$) and ventilatory equivalent ($\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$) data plotted versus incremental exercise time. Each page contained either a subject's $F_{E}O_2$, graph printed directly above the corresponding $F_{E}CO_2$ graph or a subject's $\dot{V}_E/\dot{V}O_2$ graph printed above the corresponding $\dot{V}_E/\dot{V}CO_2$ graph to aid in the examination of curve relationships. The graphs were independently coded by the investigator to allow for a blind review. In addition, the selection of $AT_{F_{E}O_2}$ and $AT_{\dot{V}_E/\dot{V}O_2}$ from the graphs were made independent of any knowledge of the blood HLa changes.

The methods of AT selection were carefully explained to each reviewer and written instructions were provided (Appendix E). Reviewers were encouraged to follow the instructions carefully and to work independently. Each reviewer's packet contained twelve subject's graphs of gas fraction and ventilatory equivalent data for each of the four treadmill tests (n=96). In addition, each reviewer also unknowingly assessed a total of 8 duplicate data plots. The reviewers indicated the chosen AT's by drawing a vertical line down through the two data curves to intersect the bottom graph's x axis. Selected AT's were recorded as the incremental exercise time intersected on the x axis (Appendix G). These AT times were rounded to the nearest 30 s to correspond to treadmill stage duration and minute ventilation

collection intervals. The AT times were then mathematically converted from min sec to minutes with seconds rounded to 1/10th of a minute (min.sec).

Determination of Fingertip Blood Sampling Times. The $AT\dot{V}_E$ and AT_{FEO_2} for each subject, visually determined by two investigators from the pre-training maximal exercise test data, were used to determine when two fingertip blood samples should be taken during the second exercise test. If a clear "breakaway" in \dot{V}_E versus exercise time relationship was not discernable, an increase in F_{EO_2} , without a corresponding decrease in F_{ECO_2} was employed to aid in AT determination. An average AT time was calculated and used to set blood sampling times one minute pre- and one minute post-AT time for each subject. These sampling times were not recalculated post-training in order to facilitate determination of a possible training effect on submaximal HLa concentration. During the second exercise test (GXT_2), the treadmill protocol remained identical to the first exercise test (GXT_1) until the post-AT blood sample was taken. For the purposes of this investigation, a blood lactate concentration of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ was accepted as the criterion of onset of metabolic acidosis, or anaerobic threshold.

Submaximal Exercise Test. A submaximal graded exercise test (GXT_2) was administered within three days of the maximal

test, pre- and post-training, for the purpose of validating, through comparison with blood lactate concentration, the noninvasively-determined AT. The treadmill protocol remained identical to GXT₁ until the post-AT blood sample was drawn to allow for determination of test-retest reliability of noninvasive AT estimates. After collection of the second fingertip blood sample (1 min post-AT), the endurance phase of the GXT began. Speed and grade were changed to elicit 70% of subjects' $\dot{V}O_{2max}$ for a maximum of 20 min.

A baseline blood sample (5 ml) was drawn approximately 2 min before the test via venipuncture of an antecubital vein. Fingertip blood samples (200 ul), taken at two predetermined times during the graded portion of GXT₂, were used to assess validity of noninvasive AT estimation against 4 m•mol•L⁻¹ HLa level for each subject. All blood samples were immediately analyzed for lactate concentration using a YSI Scientific model 23L lactate analyzer. The measurement of lactate was achieved by monitoring the concentration of hydrogen peroxide produced as a result of the catalytic action L-Lactate oxidase and flavin adenine dinucleotide (FAD) on oxygen and L-Lactate. The hydrogen peroxide produced comes into contact with the platinum anode where it undergoes oxidation, yielding a current which is linearly proportional to the concentration of lactate in the sample.

Procedures for Collection of Blood Samples. Resting venous blood samples were drawn in the following manner:

1. Subject in supine resting position on padded table adjacent to the treadmill.
2. Approximately 2 min prior to testing, a registered nurse located the vein, cleaned the skin with an alcohol pad, applied a tourniquet to the upper arm, and drew a 5 ml blood sample via venipuncture of an antecubital vein.
3. After withdrawal of the blood via a syringe, a sterile guaze pad was placed over the puncture site and the subject was asked to apply compression by bending their forearm up toward their shoulder. Subjects were observed for signs of poor reaction to blood sampling (i.e., dizziness, paleness, etc.).
4. Prior to exercise testing, a bandaid was placed over the puncture site.

Exercise Capillary Blood Samples. Fingertip blood samples were collected during testing while the subject continued to walk/run on the treadmill. The same finger was used for all blood sampling.

1. Approximately 60 s prior to sampling, the subject's hand was placed in a cup of warm water to increase blood flow in an attempt to make sampling faster, easier, and less painful.

2. Approximately 20 s prior to sampling, the subject's hand was removed from the cup of warm water and carefully dried with a towel to avoid dilution of the blood samples.

3. Approximately 5 s prior to sampling time, the investigator made two consecutive pricks in the tip of the subject's middle finger with an automatic lancet device (Autoclix).

4. The use of a gentle downward massaging action on the middle finger aided in blood collection.

5. Blood (200 ul) was collected on a sterile square of parafilm. Approximately 5 s elapsed from the initial finger-pricks to completion of blood sample collection.

6. The blood sample was immediately taken to the lactate analyzer for analysis.

7. The subject was given a sterile guaze pad to hold between fingertip and thumb to apply compression over the fingerpricks. A bandaid was applied to fingertip at the conclusion of the exercise test.

Exercise Training Treatment

The 8 wk aerobic training program began 2 d after completion of GXT₂. The subjects walked/jogged outdoors 3 d•wk⁻¹ at a pace that elicited heart rates corresponding to 70% $\dot{V}O_{2max}$. Each exercise session consisted of a 5 min warmup, a 30 min stimulus phase, and a 5 min cool down. During the first exercise training session, subjects were taught how to accurately assess their heart rates via palpation of the radial artery. All training sessions were supervised by the investigator. The investigator and other exercise leaders frequently monitored pulse-taking to ensure that each subject was exercising at her exercise prescription. An attendance chart was provided by the investigator for the subjects to record each session's resting, peak exercise, and recovery heart rates. It was recommended that subjects gradually progress their training duration to achieve 30 consecutive minutes of activity at their respective exercise prescriptions within the first week. Upon completion of the 8 wk training treatment, subjects underwent maximal and submaximal exercise tests identical to those given pre-training. The purpose of these tests was to elicit data for evaluation of potential training effects.

Nutrition Education and Diet Program

Concurrent with the aerobic training, subjects were requested to follow either a 1200 or 1400 kcal·d⁻¹ diet. This diet, based on the diabetic exchange list diet (American Diabetes Association, Inc., 1976), was designed to promote a 1 kg·wk⁻¹ weight loss. Nutrition education sessions were held weekly to provide subjects with information regarding behavior modification techniques as well as the nutritional and caloric content of various foods. A graduate student in the VPI & SU human nutrition training program examined each subject's daily food intake records for compliance to the diet. Attendance and body weight were recorded at each session.

Instrumentation and Sampling of Dependent Measures

Blood Lactate Determination. A model 23L lactate analyzer from YSI Scientific Instrument Co., Inc. (Yellow Springs, Ohio) was used to analyze subject blood samples for lactate concentration via measurement of hydrogen peroxide. This analysis was performed in the following manner:

Calibration Procedures

1. Set the RUN/STANDBY switch to STANDBY.
2. Depress the CLEAR button for 5 s.
3. Set the RUN/STANDBY switch to RUN.
4. Press and release the CLEAR button.

5. Zero the LED numerical display when the ZERO/INJECT light is lit.
6. Rinse the syringe tip twice with distilled water.
7. Rinse the syringe tip once with $5.0 \text{ m}\cdot\text{mol}\cdot\text{l}^{-1}$ standard solution.
8. Fill the syringe tip with $5.0 \text{ m}\cdot\text{mol}\cdot\text{l}^{-1}$ of the standard solution and inject it into the analyzer through the injection port.
9. Press the CALIBRATE button when the LED display shows a number
10. Adjust the LED numerical display to read 5.0 by turning the Calibrate knob.
11. Watch the LED numerical display for the next 30 s to check for drift. If the value drifts more than $0.1 \text{ m}\cdot\text{mol}\cdot\text{l}^{-1}$ in 10 s, check for adequacy of stirring and possible leakback from the sampling chamber.
12. Repeat steps 4-11 until the zero value is between -0.1 and 0.2 and the calibration value is $5.0 \pm 0.1 \text{ m}\cdot\text{mol}\cdot\text{l}^{-1}$.
13. Press and release the CLEAR button.

Linearity Check

1. After the analyzer has been calibrated according to the steps above for a $5 \text{ m}\cdot\text{mol}\cdot\text{l}^{-1}$ level,

calibrate for linearity using a $15 \text{ m}\cdot\text{mol}\cdot^{-1}$ standard solution. At step 10 adjust LED numerical readout to 15.0 ($14.4 - 15.6 \text{ m}\cdot\text{mol}\cdot^{-1}$) using the CALIBRATION knob.

2. Repeat steps 4-11 using the $15 \text{ m}\cdot\text{mol}\cdot^{-1}$ solution to ensure that LED numerical readout will be 15.0 ($14.4 - 15.6 \text{ m}\cdot\text{mol}\cdot^{-1}$). If it is not 15.0, repeat steps 4-11 three more times. If LED numerical readout is still not 15.0 change the membrane and begin calibration process again with the $5 \text{ m}\cdot\text{mol}\cdot^{-1}$ standard solution.

The lactate analyzer was calibrated immediately prior to each subject and after every third sample measurement.

Reliability Estimate. The YSI lactate analyzer is linear within the range of $0-15.0 \text{ m}\cdot\text{mol}\cdot^{-1}$. Comparison of YSI 23L versus photoenzymatic LDH-NAD methods yields a correlation coefficient of $r = 0.997$, standard error of estimate $S_{y\cdot x} = 0.33$.

Sample Measurement Procedures. All samples were analyzed immediately. Two samples from each specimen (venous and capillary) were analyzed consecutively, whenever possible, in the following manner:

1. Rinse syringepet twice with the sample.

2. Fill syringe with sample and inject it into the analyzer. (Samples were drawn up into the syringe slowly and steady to avoid air bubbles which can cause significant errors in the form of low lactate readings.)
3. When the word "READ" is illuminated, record the sample value.
4. Press and release the CLEAR button.
5. Start again at step 2 if enough unclotted blood remains to allow duplicate sample measurement.
6. After sampling measurement is completed on each subject's specimen, immediately rinse the syringe twice with distilled water.

HLA data are presented in Appendix F.

Minute Ventilation Determination. Open circuit spirometry methods were utilized for measurement of expired ventilation. Subjects breathed through a Daniel's two-way valve fitted with a rubber mouthpiece for the duration of each test. The expiration port of the Daniel's valve was attached to a 3.0 L mixing chamber using flexible low resistance tubing (3 ft - I.D.=1.5 in). The flow sensor of a digital pneumotachograph (Hewlett-Packard model 47303A) was placed just upstream from the inspiratory port of the mixing chamber. Ventilations (\dot{V}_E , BTPS, $L \cdot \text{min}^{-1}$) were continuously sampled directly from the mixing chamber. Every 30 s

measurements were recorded from the visual LED display on the pneumotach. Prior to each day's testing, the pneumotach was calibrated with repeated injections of air from a 3.0 L volumetric syringe.

For some subjects inspired ventilation instead of expired ventilation was measured using a Parkinson-Cowan P-4 dry gas meter connected to the inspired side of the breathing valve. It was calibrated in the same manner as the pneumotach.

A modular instrument computer (MINC-11, Digital Equip. Corp., Maynard, MA) was utilized to calculate minute ventilation (\dot{V}_E , STPD), respiratory exchange ratio (R), absolute and relative oxygen consumption ($\dot{V}O_2$), and expired carbon dioxide ($\dot{V}CO_2$).

Respiratory Gas Fraction Determination. Expired gases were continuously sampled directly from the mixing chamber through inlet tubing (2 ft - I.D.=3 mm) by rapid-response electronic analyzers (Beckman OM-11, LB-2) for fractions of expired oxygen and carbon dioxide, respectively. After passing through a dessicant (Aquasorb[®]), measurements of F_{EO_2} and F_{ECO_2} were recorded every 15 s from the LED display on the gas analyzers. A 15 s phase delay was accepted between \dot{V} measurement and stabilization of F_{EO_2} and F_{ECO_2} readings.

The gas analyzers were calibrated with standard reference gases and room air immediately before and after each treadmill test and if analyzer drift occurred over the test period, gas fractions were corrected accordingly. Two reference gas mixtures previously verified by Haldane analysis were used in the calibration process.

After each subject's exercise test, the respiratory apparatus was dismantled and washed in hot, soapy water. The dessicant (Aquasorb[®]) was checked and replaced as needed.

Rating of Perceived Exertion Determination. Borg's (1983) category-ratio scale (Appendix C) was used to assess each subject's rating of perceived exertion (RPE) every minute throughout each test. Prior to testing, the scale was carefully explained. During testing, the subjects were instructed to point to the number on the RPE scale which correlated with their current feelings of effort. A value of zero was denoted as feeling no fatigue and a value of 10 as feeling almost maximal effort.

Heart Rate and Blood Pressure Determination. Throughout each test, the electrocardiographic signal was continuously monitored on an oscilloscope (Hewlett-Packard Model 7803B). Heart rate was recorded electrocardiographically via a CM₅ lead to determine heart rate at rest (supine and standing), during the last 10 s of each 30 s treadmill stage, and every

minute post-exercise until heart rate returned to resting level. The maximum heart rate (HR_{max}) was accepted as the highest rate observed.

Blood pressure was measured by auscultation with a mercury manometer and stethoscope with the subject at rest (supine and standing) and every odd minute during exercise and recovery phases. Phase I Korotkoff sounds were accepted as the systolic blood pressure value and phase V, the diastolic blood pressure value.

Estimation of Body Composition. Skinfold measurements were taken with Harpenden skinfold calipers (John Bull, LTD) at three sites: triceps, thigh, and abdomen. The average of three successive measurements at each site was recorded to the nearest 0.1 mm. Body composition was then determined using equations developed by Durnin and Womersley in 1974. Each subject was weighed on the same scale to the nearest 0.5 kg each week during the study. The scale was checked for calibration accuracy prior to weekly weigh-ins.

Determination of the Reliability of Dependent Measures.

Prior to the comparison of AT values determined using either the AT-computer or the AT-reviewer method, test-retest reliability of the measurement of basic ventilatory and gas exchange data (\dot{V} , $\dot{V}O_2$, $\dot{V}CO_2$, F_{EO_2} , F_{ECO_2}) was determined via the Spearman Rho correlation coefficient, pre- and

post-training. The ventilatory and gas exchange variables were compared at two points in time, below the predicted AT time and at the predicted AT time (Appendix F).

Determination of the Validity of Dependent Measures.

An "idealized" blood lactate curve (Appendix D), derived from the literature (Davis & Gass, 1981; Hurley et al., 1984; Kindermann et al., 1979), was employed in the validation of noninvasive AT estimates. The blood lactate data from these three studies, each testing different numbers and types of subjects on either the cycle ergometer or treadmill, was averaged and subsequently used to plot what is referred to in this investigation as the "idealized" lactate curve. The formulation of an "idealized" lactate curve provided the means to allow determination of invasive AT values (GXT time at $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$) for each subject pre- and post-training. A blood lactate concentration of $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ served as the criterion threshold in the present investigation. Separate plots were made of each individual's two fingertip blood lactate values measured before (GXT₂) and after (GXT₄) training. The "idealized" lactate curve, graphed on a sheet of clear acetate, was then placed directly over the X axis of each subject's blood lactate plot. Identical scaling was used for both the "idealized" lactate curve and the individual lactate plots. The investigator then moved the

"idealized" curve to either the left or right, keeping it directly over the X axis of the individual plot, until the two lactate data points fell on a portion of the "idealized" curve. Thus, the two blood lactate data points served to "anchor" the "idealized" curve allowing estimation of the GXT time corresponding to a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration. The investigator used a clear "T" square ruler to measure over from the $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ blood lactate concentration on the y axis of the "idealized" curve to the point of intersection with the "anchored" "idealized" lactate curve. The investigator then drew a line directly down from this point of intersection to the x axis of the individual lactate plot. The GXT time at this intersection was rounded to the nearest tenth of a minute and recorded as the GXT time corresponding to a $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ lactate concentration. The exercise $\dot{V}\text{O}_2$ corresponding to the GXT time at $4 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$ (rounded to the nearest 30 s) was then determined from review of each subject's treadmill test data sheet. Those subjects whose blood lactate values were too low ($< 2 \text{ m}\cdot\text{mol}\cdot\text{L}^{-1}$) ($n=2$) or whose blood lactate values decreased from the first to the second value ($n=1$) were omitted from this analysis.

Statistical Analysis for Assessment of Data

Analysis of the data were performed with the aid of a computer utilizing the Statistical Analysis System (SAS).

In all statistical analyses, the 0.05 level of significance was used. In order to examine the research hypotheses, the data analysis was divided into the following sections:

Reliability. To estimate the reproducibility of the AT-computer method (HO_1), Spearman Rho correlation coefficients were computed for mean $AT\dot{V}_E$ from GXT₁ - GXT₂ data (pre-training) and mean $AT\dot{V}_E$ from GXT₃ - GXT₄ data (post-training).

To estimate the reproducibility (objectivity) of the AT-reviewer method (HO_2), Spearman Rho correlation coefficients were separately computed pre- and post-training for AT_{FEO_2} and $AT\dot{V}_E/\dot{V}O_2$. Since the four reviewers varied in their choice of AT, the mean AT_{FEO_2} and $AT\dot{V}_E/\dot{V}O_2$ values for the four reviewers were used in this analysis.

The percentage of graphs judged to be "indeterminate" by the four reviewers for the four GXT's was shown to be 14% for AT_{FEO_2} and 10% for $AT\dot{V}_E/\dot{V}O_2$.

Inter-reviewer Reliability. Spearman Rho correlation coefficients were also determined between all four reviewers for each GXT for both AT_{FEO_2} and $AT\dot{V}_E/\dot{V}O_2$ (Appendix F). The percentage of Spearman Rho correlation coefficients significant at the 0.01 and 0.05 probability levels for each method were 88% and 12% for AT_{FEO_2} and 66% and 17% for $AT\dot{V}_E/\dot{V}O_2$, respectively. Twelve Spearman Rho correlation

coefficients were higher than 0.90 for AT_{FEO_2} while eleven were above 0.90 for $AT_{\dot{V}E/\dot{V}O_2}$.

Intra-reviewer Reliability. The percent variation was calculated for duplicate graphs judged by the same reviewer to get an estimate of intra-reviewer reliability. The mean percent variations across four GXTs are presented below:

AT_{FEO_2}	$AT_{\dot{V}E/\dot{V}O_2}$
$R_1 = 7.7\%$	$R_1 = 4.5\%$
$R_2 = 3.9\%$	$R_2 = 7.2\%$
$R_3 = 2.1\%$	$R_3 = 4.2\%$
$R_4 = 2.4\%$	$R_4 = 2.3\%$

Validity. Calculation of Spearman Rho correlation coefficients provided an estimate of validity for each noninvasive AT index ($AT_{\dot{V}E}$, AT_{FEO_2} , $AT_{\dot{V}E/\dot{V}O_2}$) in comparison with the criterion measure of AT used in this investigation, AT_{HLA} .

Effects of Training. Dependent t-tests were utilized to evaluate the estimates of AT and other variables for the effects of training. Differences were considered significant at the 0.05 probability level.

Research Design

Design. A one-group pretest-posttest design (Ary et al., 1979) was used.

External Validity. The experimental findings from this investigation may be generalizable only to subjects

possessing the same characteristics as the subject sample (volunteer sedentary, overweight females, 18-35 yrs).

Internal Validity. Internal validity is concerned with whether the experimental treatment (aerobic training) really brought about a change in the dependent variable (anaerobic threshold). Control of several extraneous variables contributed to the internal validity of this design.

1. History: Specific events, other than the experimental treatment (aerobic training), may occur between the first and second measurements of the subjects' responses to exercise testing to produce changes in the dependent variable (anaerobic threshold). This was controlled by administering the maximal and submaximal exercise tests, pre- and post-training, within 48 hr of each other.

2. Pretesting: Exposure to the pretest may affect the subject's performance on a second test due to the practice effect. This was controlled through familiarization of subjects with testing procedures and equipment prior to the initial exercise test.

3. Measuring instruments: Changes in the measuring instruments or the reviewers used may produce changes in the obtained measures. Identical treadmill protocols were used for all exercise tests, pre- and post-training. Also, the same four reviewers were utilized to visually determine

AT_{FEO_2} and $AT_{\dot{V}E/\dot{V}O_2}$ from computer-generated graphs. Each of the four reviewers received coded photocopies of the graphs (n=104) at the same time to avoid a practice effect in AT determination pre- to post-training.

The dietary intervention may have confounded the claim that the observable results can be attributed to the manipulation of the independent variable (aerobic fitness level) via the exercise treatment rather than to uncontrolled variance.

APPENDIX B
SUBJECT CHARACTERISTICS

Appendix B
Subject Characteristics

Subject	Age (yr)	Height (cm)	Weight (kg)	Predicted Body Fat (%)	$\dot{V}O_2\text{max}$	
					(L·min ⁻¹)	(ml·kg ⁻¹ ·min ⁻¹)
1	22	169.5	69.9	27.2	2.2	32.2
2	22	166.5	74.7	33.1	2.5	34.9
3	35	152.0	118.4	42.6	2.4	20.0
4	22	171.3	73.7	31.7	2.8	39.3
5	34	163.0	84.2	35.5	2.1	24.6
6	21	159.0	56.4	29.1	2.3	41.0
7	19	161.3	79.0	33.6	2.4	38.0
8	18	175.3	71.7	34.0	2.2	33.1
9	30	171.5	65.8	28.0	2.5	41.0
10	20	163.8	65.4	32.2	2.5	41.2
11	20	161.0	63.1	29.1	2.5	40.6
12	20	158.0	59.7	30.7	2.0	32.0
\bar{X}	23.6	164.4	73.3	32.2	2.4	33.4
SEM	1.7	1.9	4.7	1.2	0.1	1.8

APPENDIX C
TEST ADMINISTRATION INFORMATION

Informed Consent

HUMAN PERFORMANCE LABORATORY

Division of Health, Physical Education and Recreation
Virginia Polytechnic Institute and State University

INFORMED CONSENT

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by the personnel of the Human Performance Laboratory of the Division of Health, Physical Education and Recreation of Virginia Polytechnic Institute and State University.

Title of Study: Metabolic vs. Hormonal Indices Contributing to Perceived Exertion Before and After a Training Program for Overweight College Women.

PURPOSES: To investigate the relationship between metabolic factors and perceived exertion before and after training in overweight women.

I voluntarily agree to participate in this testing program. It is my understanding that my participation will include:

Pre and Post Program Testing to include:

1. An exercise test requiring your maximum effort, conducted by qualified personnel at the Exercise Physiology Lab before the training program.
2. An endurance exercise test at 70% $\dot{V}O_2$ max for 20 minutes, conducted by the above, before and after the training program.
3. Body composition will be analyzed by measurement of skinfolds and circumferences at three sites on your body before and after the training program.
4. Blood lactic acid and plasma hormone levels will be analyzed from a 5ml blood sample taken via venipuncture, at the Exercise Physiology Lab before and after your pre- and post-endurance exercise test. Two other blood

samples will be taken during the endurance test by finger prick (.2ml each).

Weekly:

1. Attendance at eight weeks of exercise and nutrition education classes will be required. Nutrition classes are once per week and exercise classes are 3 times per week.

I understand that participation in this experiment may produce certain discomforts and risks. These discomforts and risks include:

Fatigue and muscle soreness may be transiently experienced as a result of the exercise test or exercise classes. Temporary discomfort will be experienced due to needle puncture with blood drawing.

Certain personal benefits may be expected from participation in this experiment. These include:

I will receive information regarding proper nutrition and/or exercise procedures for healthy, effective weight loss. In addition I will be informed of my dietary profile, body composition, and exercise capacity at the end of the program.

I understand that many data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statements and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Scientific inquiry is indispensable to the advancement of knowledge. Your participation in this experiment provides the investigator the opportunity to conduct meaningful scientific observations designed to make significant educational contribution.

If you would like to receive the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

_____ I request a copy of the results of this study.

Date _____ Time _____ a.m./p.m.

Participant Signature _____

Witness _____
HPL Personnel

Project Director Dr. Janet Walburg Telephone 961-7545

HPER Human Subjects Chairman Dr. Don Sebolt

Telephone 961-5104

Dr. Charles Waring, Chairman, International Review Board for Research Involving Human Subjects. Phone 961-5283.

Appendix C

Detailed Medical History Questionnaire

Name _____ Date _____

Occupation _____

Age _____ Sex _____

Campus Address _____

Home Address _____

Local Phone _____ Home Phone _____

Family Physician _____ City _____

MEDICAL HISTORY/CHD RISK SCREENING

1. Indicate nature of condition* for male members of immediate family.

Personal _____
 Grandfather _____
 Maternal _____
 Paternal _____
 Father _____
 Brother(s) _____
 Uncle(s) _____

*Coronary artery disease, angina pectoris, coronary thrombosis, rheumatic fever, cardiac enlargement, valvular heart disease, arrhythmia, other.

2. Have you ever experienced any of the following? Please check the circumstances in which they occur.

	at rest	exertion	cold weather	emotion
chest pain	_____	_____	_____	_____
chest pressure	_____	_____	_____	_____
discomfort/pain in jaw	_____	_____	_____	_____
discomfort/pain in teeth	_____	_____	_____	_____

discomfort/pain in throat _____
 discomfort/pain in elbow _____
 discomfort/pain in wrist _____
 palpitations/skipped beats _____

3. Have you ever had an exercise or fitness evaluation?
If yes, please explain.
4. Are you taking any medications on a regular basis? Yes No
5. If yes, please list any and all medications you are taking (both prescription and non-prescription).

Name of Medication	Dosage	Doses per Day
_____	_____	_____
_____	_____	_____

EXERCISE/ACTIVITY HISTORY

1. Are you currently involved in a regular exercise program (3 days/week)?
2. Do you regularly walk or run one or more miles continuously?

If yes, average number of miles per workout or day:
 _____ miles. What is your average time per mile?
 ____:____ (minutes:seconds)

3. Please check the following sports/activities in which you have participated over the past 6 months.

Activity	Days/Week	Min/Day	INTENSITY		
			Light	Moderate	Vigorous
Basketball	_____	_____	_____	_____	_____
Calisthenics	_____	_____	_____	_____	_____
Dancing	_____	_____	_____	_____	_____
Chopping Wood	_____	_____	_____	_____	_____
Golf (without cart)	_____	_____	_____	_____	_____

Handball	_____	_____	_____
Mt. climbing/ Hiking	_____	_____	_____
Racquetball	_____	_____	_____
Swimming	_____	_____	_____
Tennis	_____	_____	_____
Weight Training	_____	_____	_____
Other	_____	_____	_____
Other	_____	_____	_____

SCREENING MEASUREMENTS

NAME _____

DATE _____

AGE _____

HEIGHT (to nearest 1.0 cm) _____

WEIGHT (to nearest 0.5 kg) _____

SKINFOLD MEASUREMENTS

TRICEPS (mm) _____

THIGH (mm) _____

ABDOMEN (mm) _____

PREDICTED BODY FAT (%) _____

PRE-TESTING INSTRUCTIONS

1. Subjects should come dressed in running attire.
2. Subjects should get a good night's sleep (7 hours) prior to test day.
3. Subjects should abstain from eating for 2-3 hours prior to testing.
4. Subjects should abstain from any medications that could alter heart rate or pulmonary responses on the test day. Subjects should abstain from smoking for 8 hours prior to testing.
5. Subjects should abstain from consuming any beverages that contain caffeine or alcohol in the 8 hours prior to testing.

THE CATEGORY-RATIO SCALE OF PERCEIVED EXERTION

0	NOTHING AT ALL	
0.5	VERY, VERY WEAK	(JUST NOTICEABLE)
1	VERY WEAK	
2	WEAK	(LIGHT)
3	MODERATE	
4	SOMEWHAT STRONG	
5	STRONG	(HEAVY)
6		
7	VERY STRONG	
8		
9		
10	VERY, VERY STRONG	(ALMOST MAX)
	MAXIMAL	

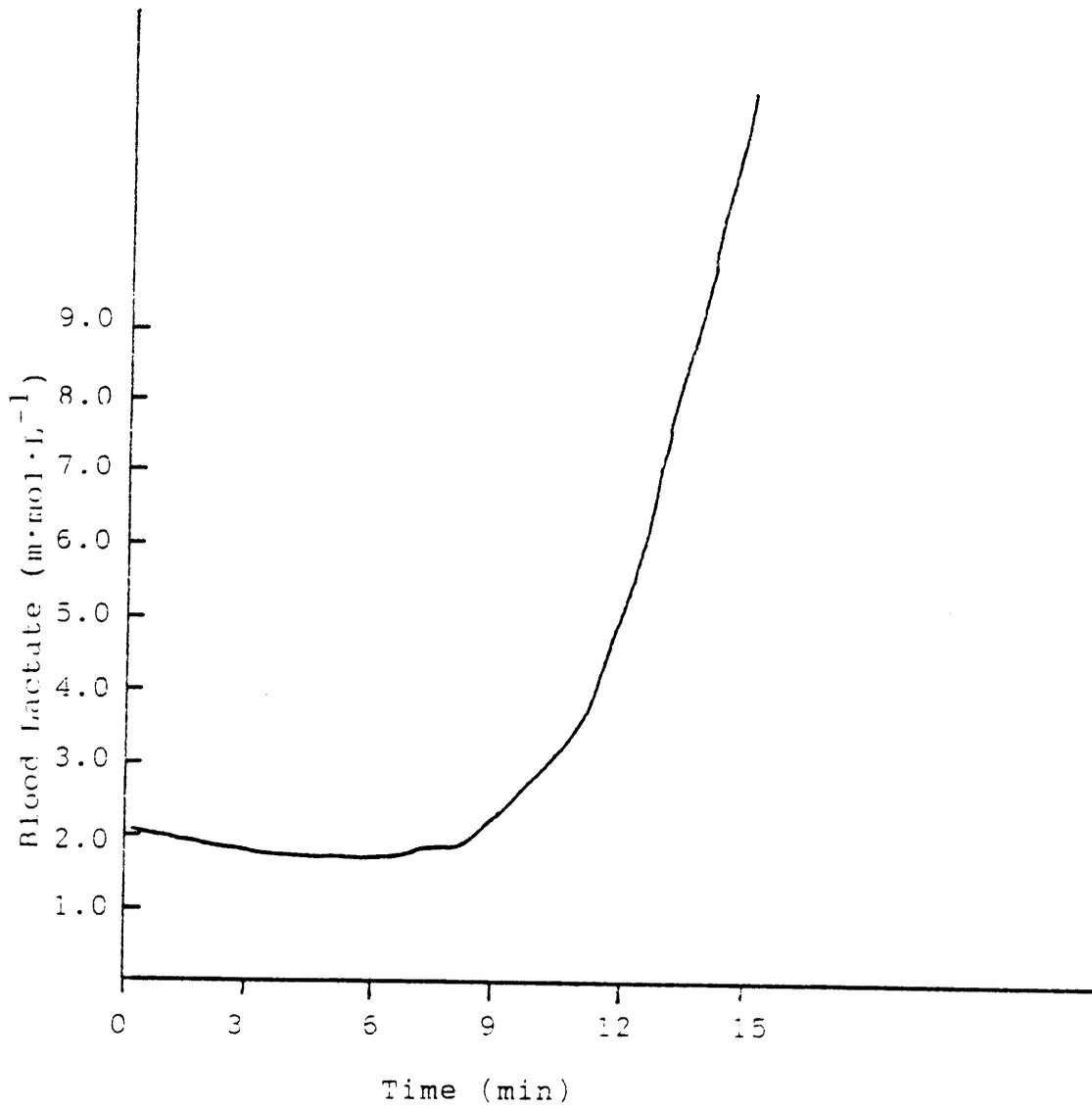
TREADMILL PROTOCOL

TEST TIME	SPEED (m.p.h.)	GRADE (%)	METS	STAGE DURATION
0:00-2:00	3.4	0	3.6	2:00
2:00-2:30	3.4	2.5	4.8	:30
2:30-3:00	3.4	3.5	5.2	:30
3:00-3:30	3.4	5.0	5.9	:30
3:30-4:00	3.4	6.5	6.6	:30
4:00-4:30	3.4	7.5	7.1	:30
4:30-5:00	3.4	9.0	7.8	:30
5:00-5:30	3.4	10.0	8.3	:30
5:30-6:00	3.4	11.5	8.9	:30
6:00-6:30	3.4	12.5	9.5	:30
6:30-7:00	3.4	14.0	10.2	:30
7:00-7:30	3.4	15.0	10.6	:30
7:30-8:00	3.4	16.0	11.0	:30
8:00-8:30	3.4	17.5	11.8	:30
8:30-9:00	3.4	19.0	12.5	:30
9:00-9:30	3.4	20.0	12.9	:30
9:30-10:00	3.4	21.0	13.4	:30
10:00-10:30	3.4	22.0	13.9	:30
10:30-11:00	4.0	18.0	14.3	:30
11:00-11:30	4.0	19.0	15.0	:30
11:30-12:00	4.0	20.0	15.7	:30

12:00-12:30	4.0	21.5	16.3	:30
12:30-13:00	4.0	23.0	17.0	:30
13:00-13:30	4.0	25.0	17.6	:30
13:30-14:00	4.0	26.0	18.3	:30

APPENDIX D
"IDEALIZED" BLOOD LACTATE CURVE

"IDEALIZED" BLOOD LACTATE CURVE



APPENDIX E
DETERMINATION OF NONINVASIVE ANAEROBIC THRESHOLDS
VIA COMPUTER REGRESSION AND REVIEWER METHODS

Procedure for Anaerobic Threshold Determination
(AT_VE) Via a Computerized Two-Component Regression Analysis

Part A. To Plot Graphs

Step 1. Insert Curve Fitter disk into disk drive and boot.

2. Run Curfit Program

3. Follow prompting statements:

: Read format file name? <None>	Return
: Input standards X, Y pairs <Y:N>	Y
: Error Bars (Y:N)	N
: Disk, Keyboard or Sensor input	K

4. Enter the paired observations of GXT time and minute ventilation for each minute. These will be the x and y variables respectively.

5. Follow prompting statements:

: Type Control Z now to save raw data.	Interchange X
and Y data (Y:N)	Control Z
: Write standard file name	"Your Label"
: Write fitted file name	Return
: Write unknowns	Return
: Write format file name	Return
: Write picture file name	Return
: Erase graph (Y:N)	Y
: Read format file name	Return
: Input same standards (Y:N)	N
: X, Y pairs (Y:N)	Y
: Error bars (Y:N)	N
: Disk, Keyboard or Sensor input	D
: Read file name	"Your Label"/Return
: First Point to be used (1:1000)	1
: Interval between points (1:1000)	1
: Type Control Z now to save raw data.	
Interchange X, Y	N
: X scale factor	1
: X offset	0
: X log scale	N

NOTE: Enter Control G to display graph and Control T to display text.

- | | |
|---|----|
| : Y scale factor | 1 |
| : Y offset | 0 |
| : Y log scale | N |
| : List standards (Y:N) | Y |
| : Plot standards | Y |
| : Points averaging (0:10) | 0 |
| : Points smoothing (0:10) | 0 |
| : Next symbol (2:4) | 2 |
| : Interpolation of least squares fitting (I:L:N) | L |
| : No. of points on curve (25:931) | 50 |
| : Linear, geometric, exponential, or polynomial least squares | P |
| : Degree of polynomial (2:6) | 2 |
6. Record the correlation coefficient (r) and the standard error ($SE \cdot y$) on data recording form.
7. Type Control G to view graph.
8. Type Control T to return to text and then following prompting statements:
- | | |
|----------------------------|---------------------|
| : Satisfactory (Y:N) | Y |
| : Input unknowns | N |
| : Write standard file name | Return |
| : Write fitted file name | Return |
| : Write unknowns file name | Return |
| : Write formal file name | Return |
| : Write picture file name | "Your Label"/Return |
9. Type Control G to view graph.
10. From the graph displayed on the screen, visualize the point at which \dot{V} becomes non-linear. Determine the number of bivariate points which constitute the linear segment of the displayed curve. Add the bivariate point above that level and record this value on the data recording form.
11. Type Control Text and follow prompting statements:
- | | |
|----------------------------|--------|
| : Erase graph (Y:N) | Y |
| : Read format file name | Return |
| : Same standards (Y:N) | N |
| : X, Y Pairs (Y:N) | Y |
| : Erase bars (Y:N) | N |
| : Disk, Keyboard or Sensor | K |

To enter a new data set for regression analysis, keep punching the return key the required number of times to include the exact number of bivariate data points just recorded on the data recording form. Type 9999 to terminate data entry.

12. Follow prompting statements:

: Type Control Z now to save raw data.	
Interchange X:Y data (Y:N)	N
: X scale factor	1
: X offset	0
: X log (Y:N)	N
: Y scale factor	1
: Y offset	0
: Y log (Y:N)	N
: List standards (Y:N)	Y
: Plot standards (Y:N)	Y
: No. points averaging	0
: No. points smoothing	0
: Next symbol (2:4)	2
: Interpolation or least squares fitting (I:L)	L
: No. of points on curve	50
: Linear, geometric, exponential or polynomial least squares	L

13. Record correlation coefficient and standard error on data recording form.

14. Follow prompting statements:

: Satisfactory (Y:N)	Y
: Input known values (Y:N)	N
: Write standards file	Return
: Write fitted file name	Return
: Write unknowns file name	Return
: Write format file name	Return
: Write picture file name	"Your Label"/Return
: Erase graph (Y:N)	Y

NOTE: Repeat steps 10-14 as needed deleting the last 2 bivariate points.

Part B. To Print Graphs

Step 1. Press Control Key.

2. Type RUN MX-80 Graphics Dump.

Set up printing format from Enhanced MX-80 graphics menu.

3. Print all graphs.

Part C. To Determine Anaerobic Threshold

Step 1. Determine the Best Regression Line

- a. Determine the most appropriate linear regression line to describe the lower end of the \dot{V}_e -Met bivariate data distribution. This line should consist of the greatest number of data points which gives the highest (r) and lowest (SE·y) values. Record the number of points included in the line of your data sheet.
 - b. Refer to the printed plot which corresponds to the regression line selected in step 5a. From this regression line select two representative points and record the x and y coordinates for each. These will be designated as lx and ly and 2x and 2y. Record these coordinates on your data sheet.
2. a. Refer to the second degree polynomial plot produced in step 2. On this graph impose the coordinates lx and ly and 2x and 2y.
b. Draw a line connecting the points plotted using lx and ly and 2x and 2y. This is the best linear regression for lower end of the \dot{V}_E -GXT time distribution.
3. Anaerobic Threshold Determination
 - a. The point on the x axis which corresponds to the intersection of the linear regression line and the second degree polynomial curve produced in step 6b is considered the anaerobic threshold GXT time level. Record this point on your data sheet.

Determination of AT_{FEO_2} and $AT_{\dot{V}_E/\dot{V}O_2}$
via Four Independent Reviewers

Instructions to Reviewers

Please take your time and study each page carefully. Use only the rules stated below for selection of the anaerobic threshold.

Please do not ask anyone else's opinion as that would defeat the purpose of using four independent reviewers.

STEPS TO FOLLOW IN ANAEROBIC THRESHOLD SELECTION

1. Look at the lower right hand corner of each page for either a I or a II. Divide the graph pages into two piles; one for I pages and one for II pages. This will help you avoid possible mix-ups when applying the corresponding anaerobic threshold definitions.
2. Note that on all graph pages with a I in the lower right hand corner, a F_{EO_2} graph appears directly above a F_{ECO_2} graph.
3. Note that on all graph pages with a II in the lower right hand corner, a $\dot{V}_E/\dot{V}O_2$ graph appears directly above a $\dot{V}_E/\dot{V}CO_2$ graph.
4. Use definition one to select the anaerobic threshold on all graph pages with a I in lower right hand corner.
5. Use definition two to select the anaerobic threshold on all graph pages with a II in lower right hand corner.

IMPORTANT NOTE: WHEN APPLYING THESE DEFINITIONS, YOU EVALUATE BOTH GRAPHS ON THE PAGE IN RELATIONSHIP TO EACH OTHER.

DEFINITION ONE: THE ANAEROBIC THRESHOLD OCCURS WHERE AN INCREASE IN F_{EO_2} (TOP GRAPH) CURVE IS SEEN WITHOUT A CORRESPONDING DECREASE IN F_{ECO_2} (BOTTOM GRAPH).

DEFINITION TWO: THE ANAEROBIC THRESHOLD OCCURS WHERE A SYSTEMATIC INCREASE IN THE $\dot{V}_E/\dot{V}O_2$ CURVE (TOP) IS SEEN WITHOUT A CONCOMMITANT INCREASE IN $\dot{V}_E/\dot{V}CO_2$ (BOTTOM).

6. Indicate your selection of the anaerobic threshold (AT) by drawing a vertical line through both graph curves on a page and writing AT at the point where the vertical line intersects the x axis of the bottom graph.
7. IMPORTANT NOTE: ON ANY PAGE WHERE YOU FEEL THAT AT SELECTION CANNOT BE MADE DUE TO THE CURVES NOT CORRESPONDING TO THE ASSOCIATED DEFINITION, PLEASE WRITE "INDETERMINATE" AND BRIEFLY WHY ON THE BOTTOM OF THAT PAGE.

If you have any questions, please feel free to call me. Thank you for your time and effort. I will gain valuable information from the anaerobic thresholds you select.

APPENDIX F
DATA TABLES

TABLE 6. Test-retest Spearman Rho correlation coefficients for basic ventilatory and gas exchange data

Time of Comparison	Trials	\dot{V}	$F_{E}O_2$	Variables $F_{E}CO_2$	$\dot{V}_{E}/\dot{V}O_2$	$\dot{V}_{E}/\dot{V}CO_2$
Below predicted AT point	Pre-T	0.76 ^a n=12	0.50 n=12	0.62 ^b n=12	0.62 ^b n=12	0.42 n=12
	Post-T	0.83 ^a n=11	0.30 n=11	0.42 n=11	0.32 n=11	0.49 n=11
At the predicted AT point	Pre-T	0.59 ^b n=12	0.58 ^b n=12	0.69 ^b n=12	0.64 ^b n=12	0.58 ^b n=12
	Post-T	0.79 ^b n=9	0.50 n=11	0.58 n=11	0.43 n=9	0.44 n=9

a denotes significant at $p < 0.01$

b denotes significant at $p < 0.05$

\dot{V} , minute ventilation; $F_{E}O_2$, fraction of expired oxygen; $F_{E}CO_2$, fraction of expired carbon dioxide; $\dot{V}_{E}/\dot{V}O_2$, ventilatory equivalent for oxygen; $\dot{V}_{E}/\dot{V}CO_2$, ventilatory equivalent for carbon dioxide.

Appendix F
 TABLE 7. Blood lactate Concentrations pre- to post-training.

Subject	Resting Illa (mmol·l ⁻¹)		Pre-AT Illa (mmol·l ⁻¹)		Post-AT Illa (mmol·l ⁻¹)	
	Pre-T	Post-T	Pre-T	Post-T	Pre-T	Post-T
1	1.3	0.7	2.4	1.7	3.1	3.0
2	0.4	0.6	0.9	0.9	0.8	1.2
3	2.5	1.2	3.9	2.4	4.6	4.9
4	0.5	0.6	2.2	2.0	3.8	3.7
5	0.5	0.6	1.8	2.1	2.9	3.1
6	0.7	0.5	1.2	2.7	1.6	3.6
7	1.0	0.7	1.8	1.9	2.2	3.3
8	0.4	0.4	1.6	1.8	1.4	2.2
9	0.4	0.8	2.4	3.1	3.8	4.1
10	0.5	0.3	1.6	1.5	3.0	3.3
11	1.2	1.2	1.6	1.1	1.9	1.5
12	1.1	0.5	4.3	2.5	4.7	3.7
\bar{X}	0.9	0.7	2.1	2.0	2.8	3.1
SEM	0.2	0.1	0.3	0.2	0.4	0.3

* significant ($p < 0.05$)
 represents average of trials per sample

Appendix F

TABLE 8. Effect of 8 wk training program on weight, body fat, resting heart rate, and maximal oxygen consumption.

Subject	Weight (kg)		Predicted Body Fat (%)		$\dot{V}O_{max}$ ($L \cdot min^{-1}$)		$\dot{V}O_{max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)		Resting Heart Rate ($bt \cdot s \cdot min^{-1}$)	
	pre	post	pre	post	pre	post	pre	post	pre	post
1	69.9	66.4	27.2	27.3	2.2	2.5	32.1	38.0	75	96
2	71.7	66.0	33.1	30.7	2.5	2.8	34.9	43.0	90	94
3	118.4	112.4	42.6	40.8	2.4	2.5	20.0	22.6	83	68
4	73.7	68.0	31.7	28.0	2.8	2.5	39.3	35.3	56	68
5	84.2	78.5	35.5	31.0	2.1	2.7	24.6	34.7	88	83
6	56.4	53.9	29.1	25.8	2.3	2.2	41.0	41.0	94	88
7	79.0	69.1	33.6	32.2	2.4	2.6	29.8	38.0	94	100
8	71.7	67.3	34.0	29.0	2.2	2.2	31.2	33.1	75	94
9	65.8	62.4	28.0	25.3	2.5	2.6	37.3	41.0	88	98
10	65.4	60.6	32.2	29.1	2.5	2.5	38.1	41.2	84	75
11	63.1	61.2	29.1	26.7	2.5	2.5	38.9	40.6	78	84
12	59.7	56.4	30.7	29.7	2.0	1.8	34.0	32.0	115	108
\bar{X}	73.3	68.5*	32.2	29.6*	2.37	2.45	33.4	36.7*	85.00	88.00
SEM	4.7	4.4	1.2	1.2	0.1	0.1	1.8	1.6	4.08	3.68

* significantly different from the pre-training responses ($p < 0.05$)

Table 9. Effect of 8 wk training program on invasive and noninvasive estimates of the anaerobic threshold.

	AT_{HLA}	$AT_{\dot{V}E}$	AT_{FEO_2}	$AT_{\dot{V}E/\dot{V}O_2}$
t-ratio	-0.93	-0.12	-0.94	-0.76
df	6	8	8	7

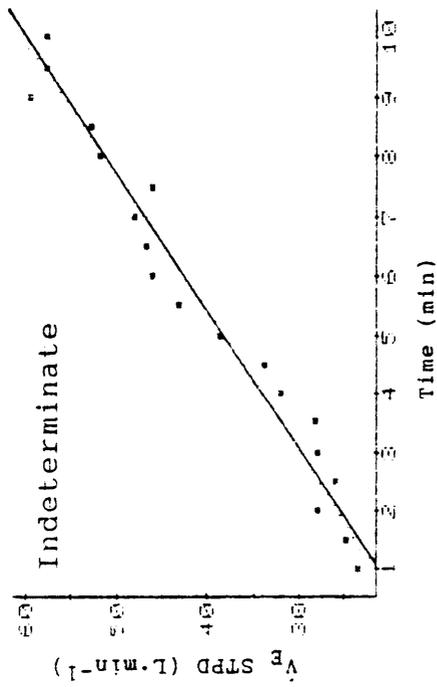
t required for significance at $p < 0.05$ for df 6-8 is 2.45-2.31

APPENDIX G
INDIVIDUAL ANAEROBIC THRESHOLD ($AT_{\dot{V}E}$) FIGURES

Subject 1

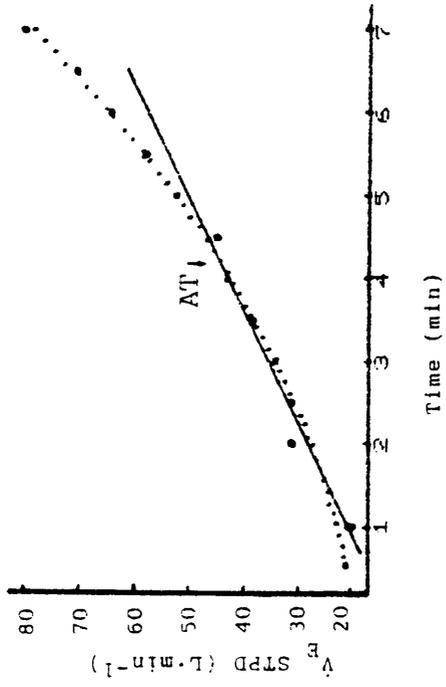
Pre-training

GXT₁

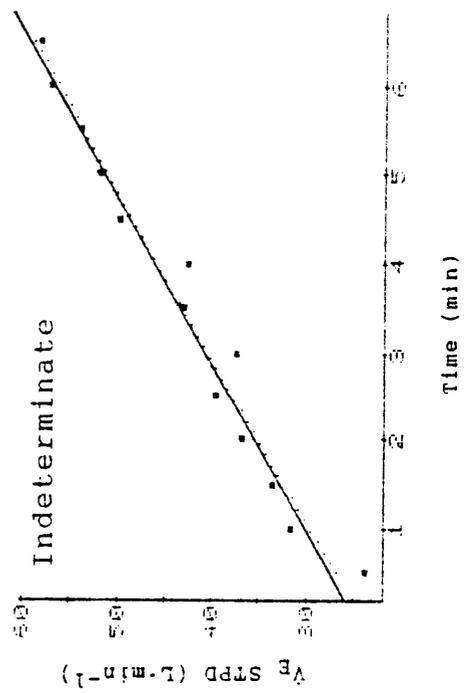


Post-training

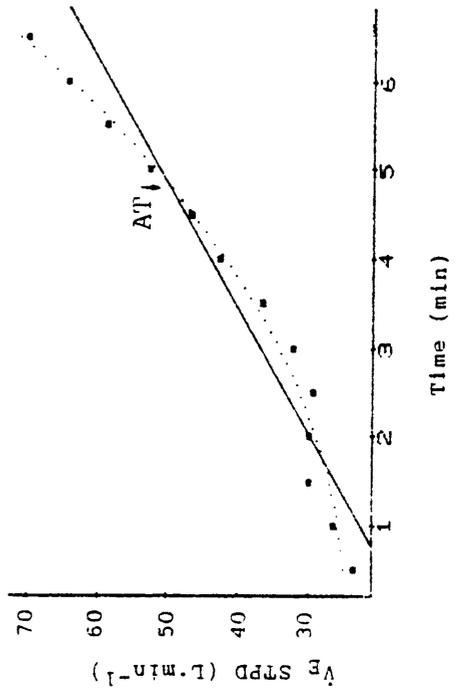
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GXT₂



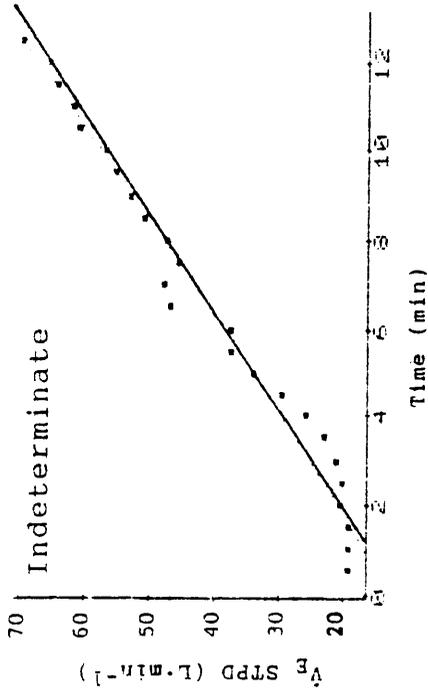
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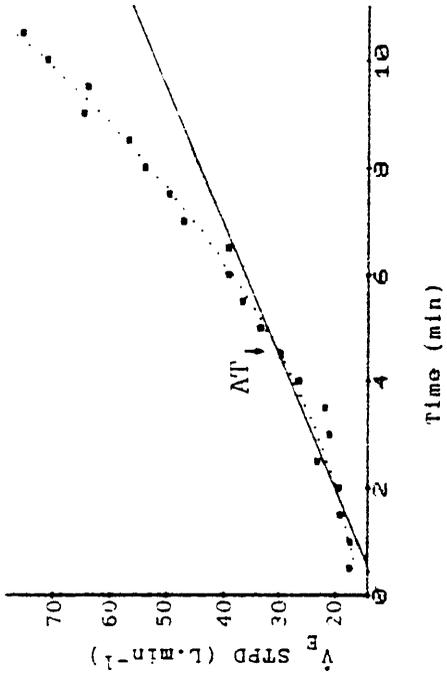
Pre-training

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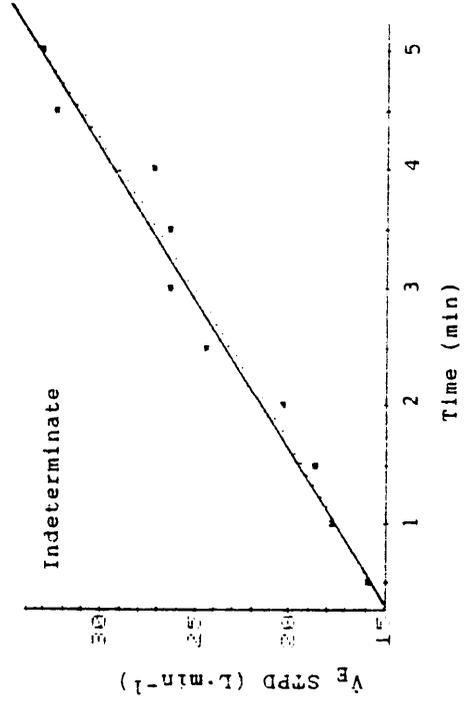


Post-training

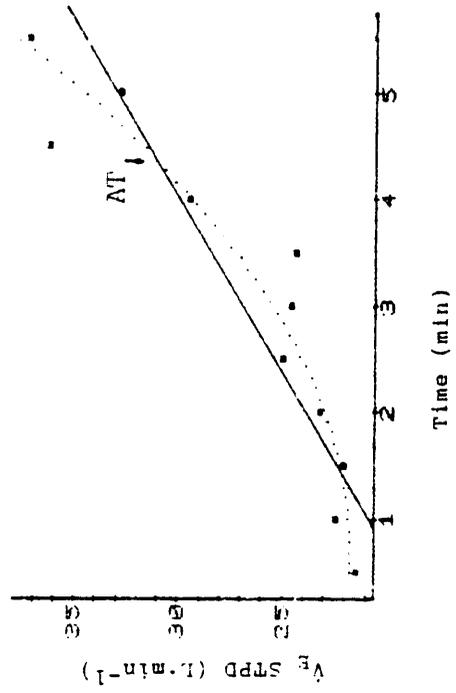
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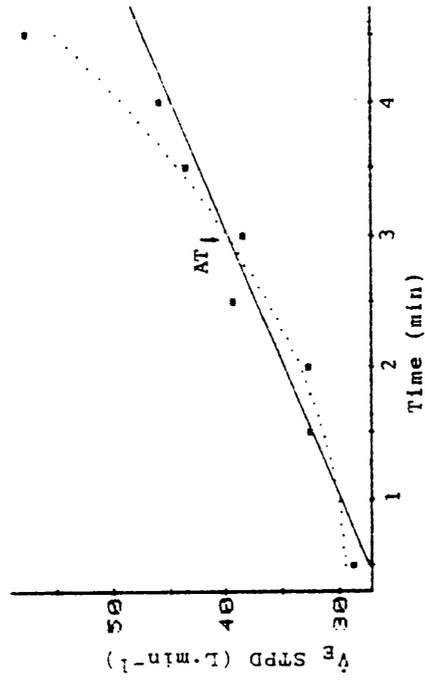
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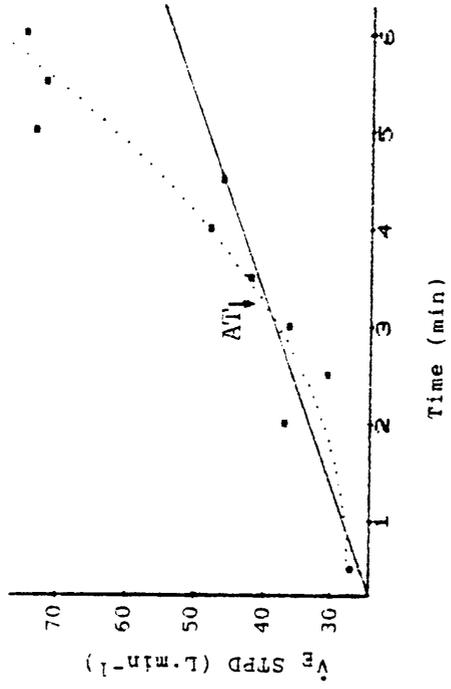
Pre-training

GXT₁

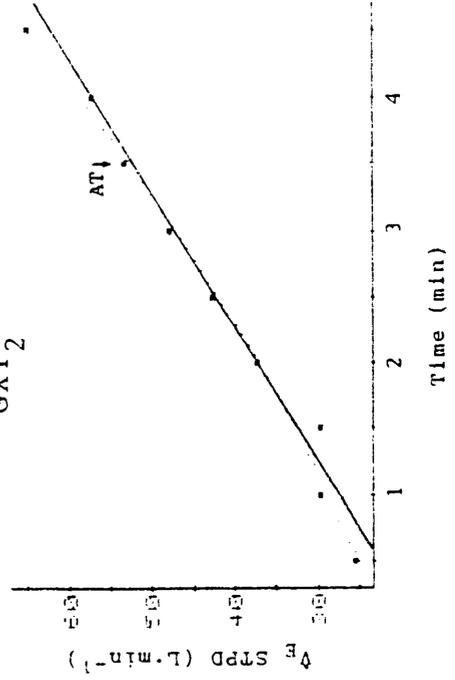


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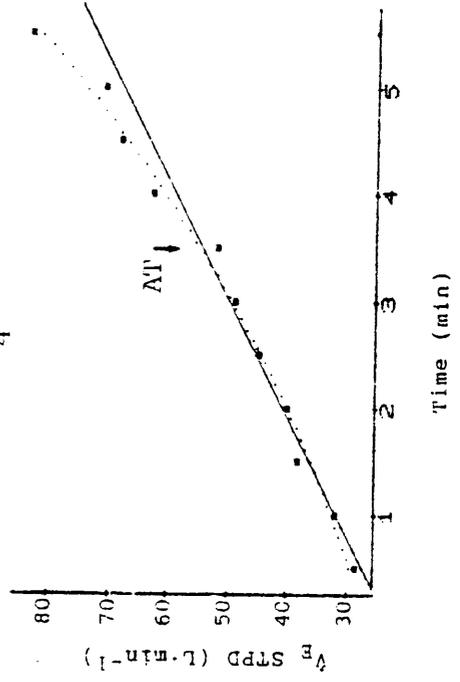
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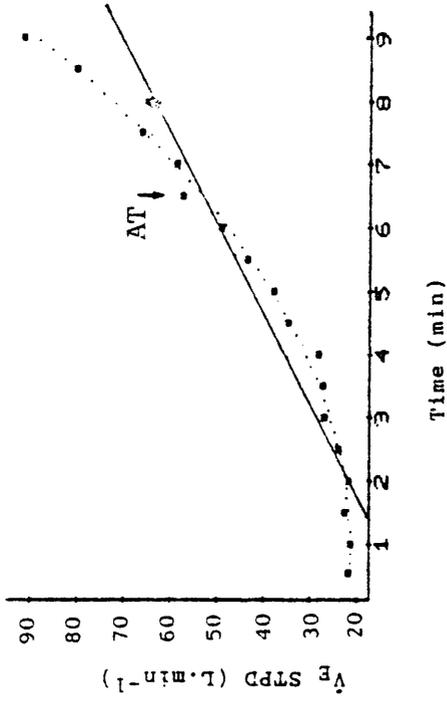
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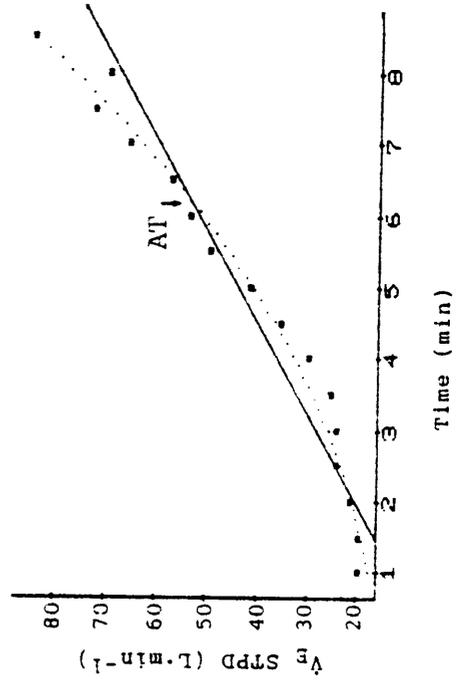
Subject 4

21C

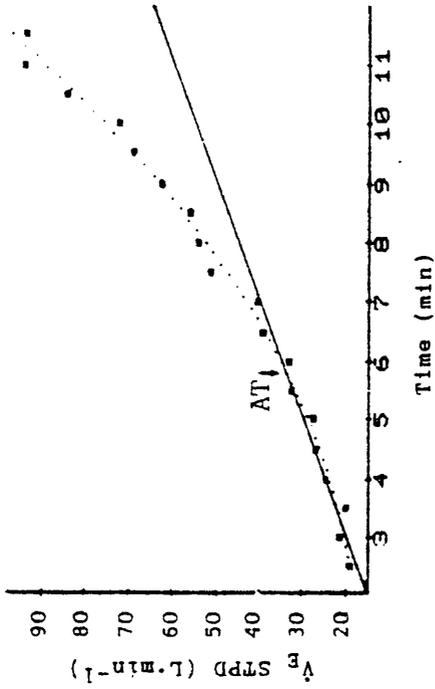
Post-training
GXT 3



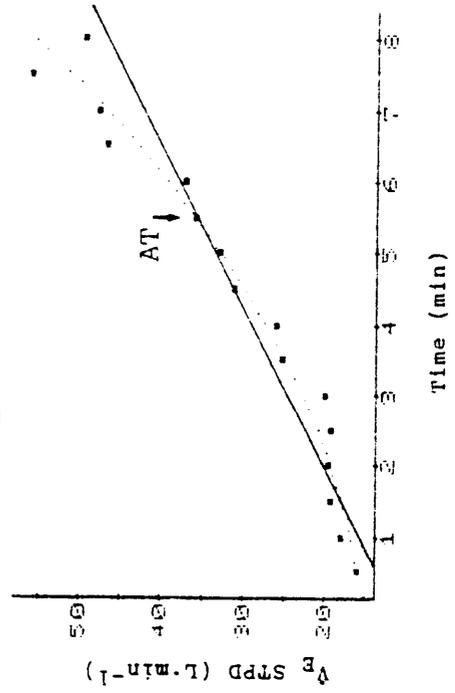
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Pre-training
GXT 1



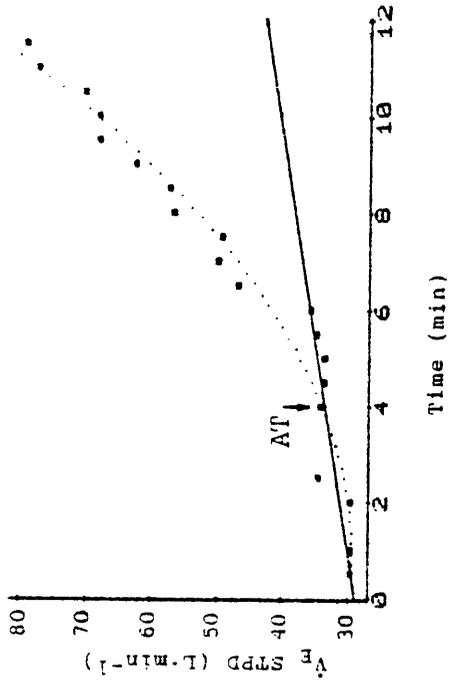
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Subject 5

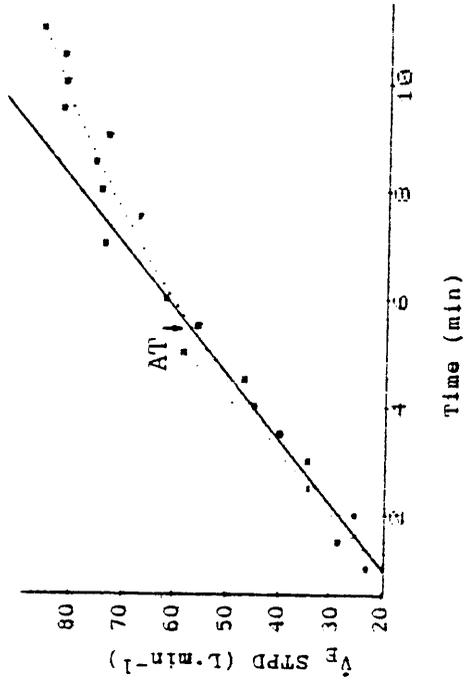
Pre-training

GXT 1

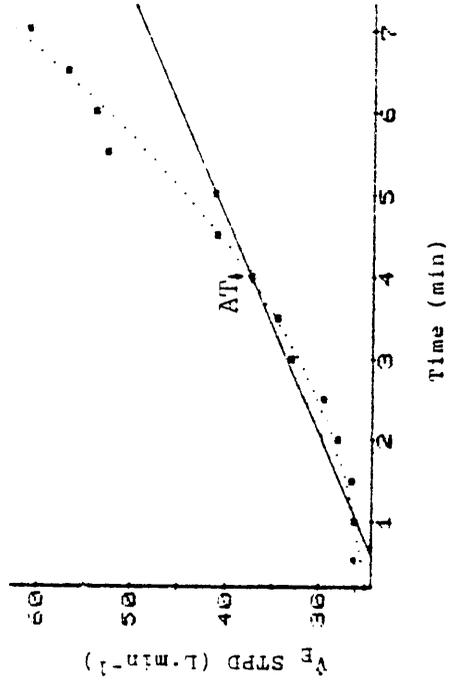


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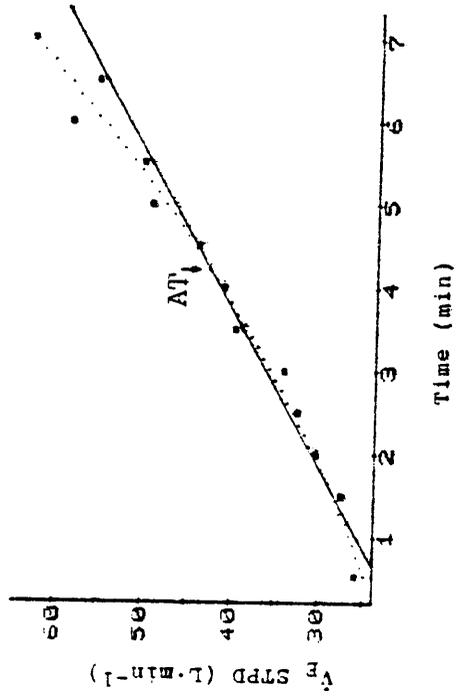
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GXT 2



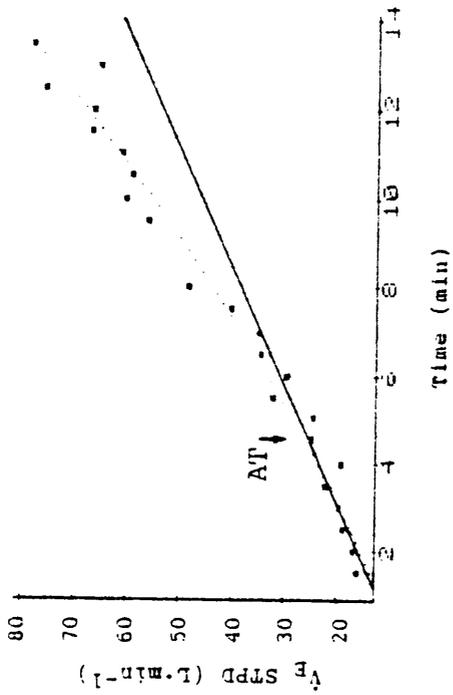
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Subject 6

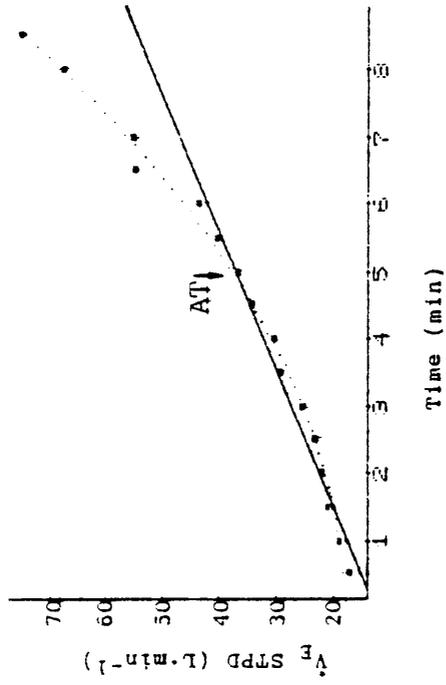
Pre-training

GXT₁

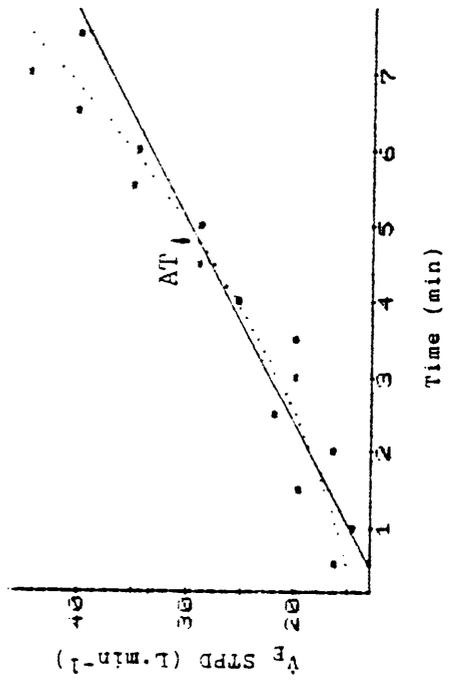


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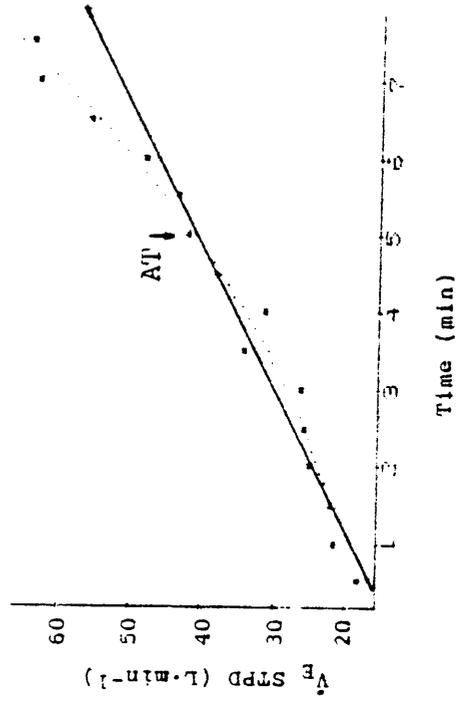
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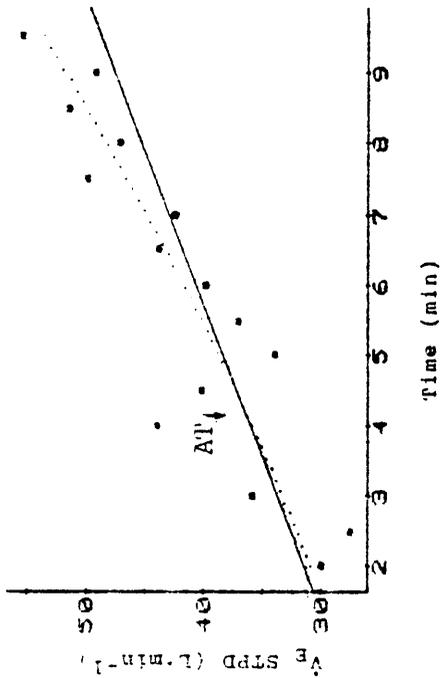


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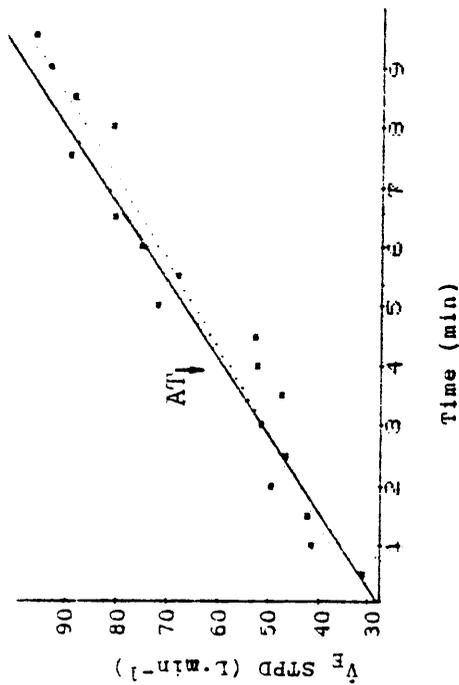


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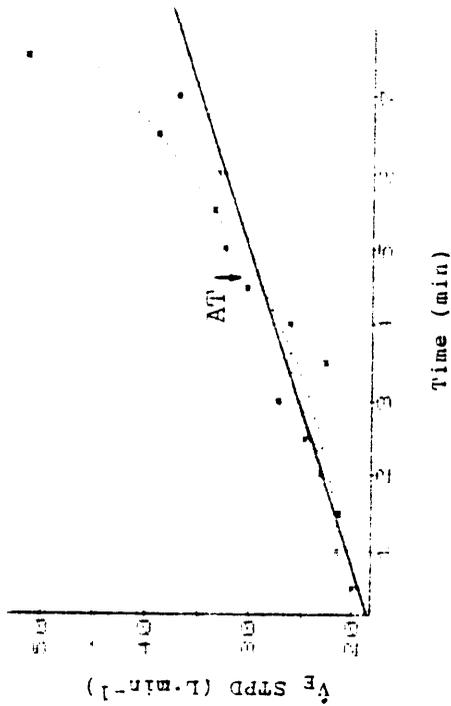
Pre-training
GXT₁



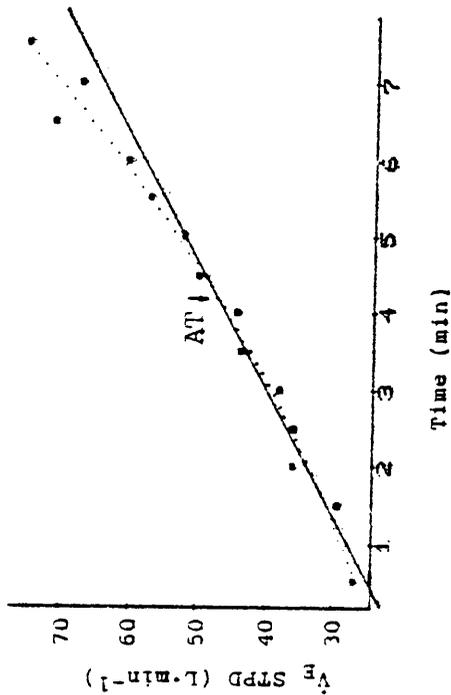
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GXT₂



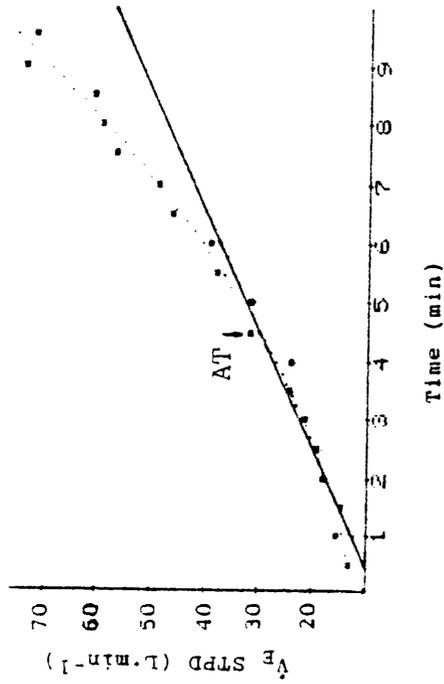
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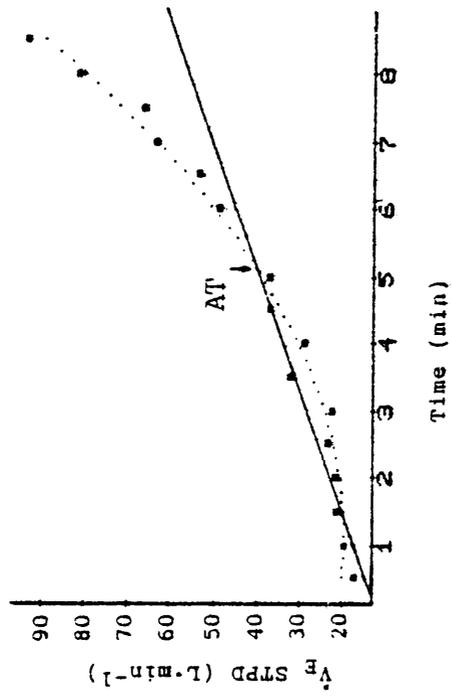
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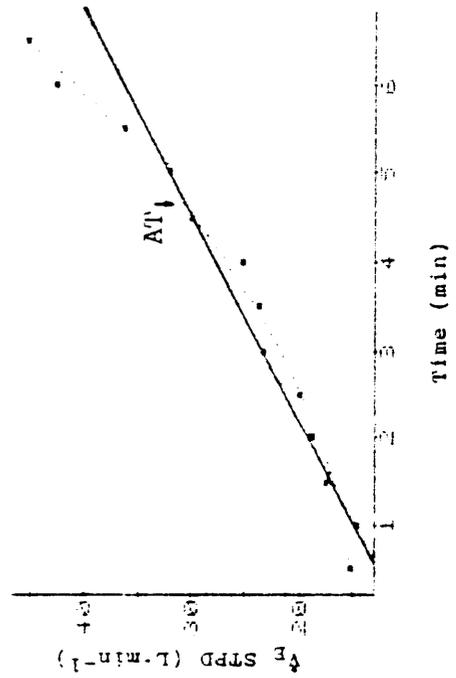


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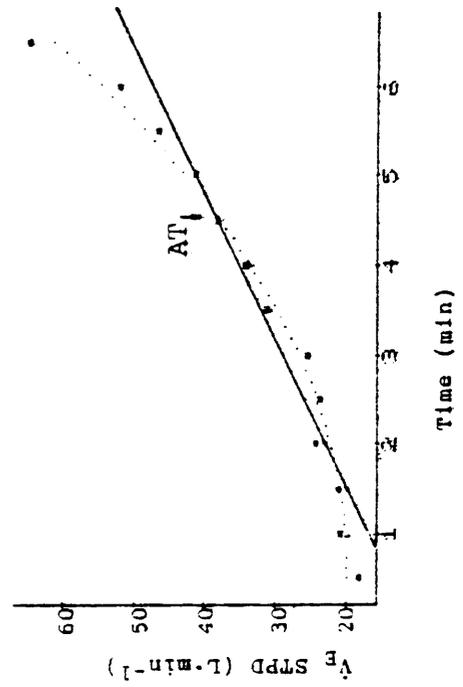
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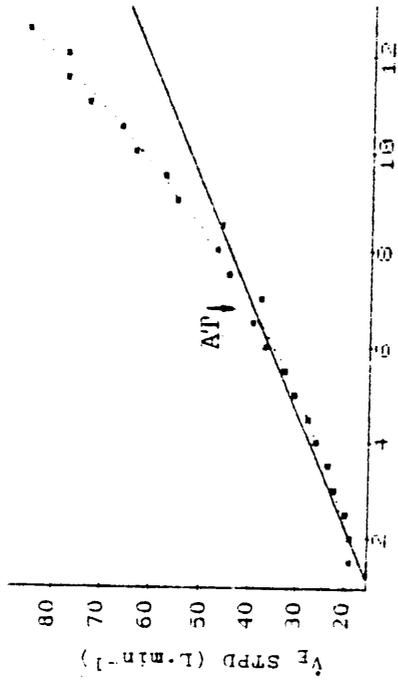


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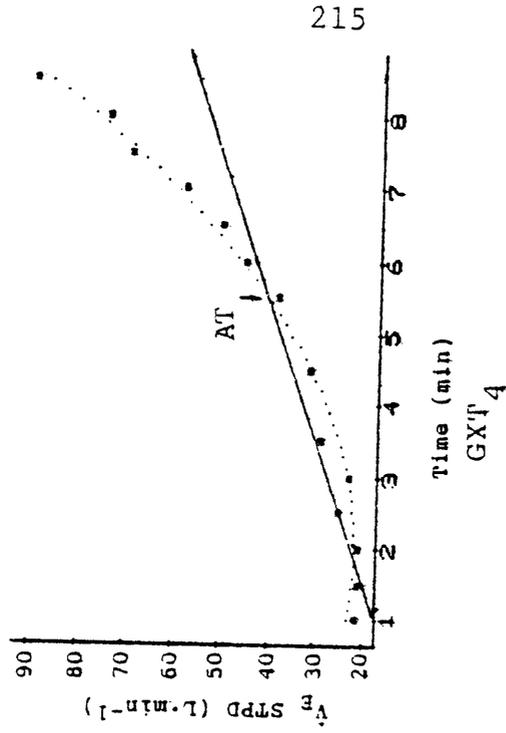


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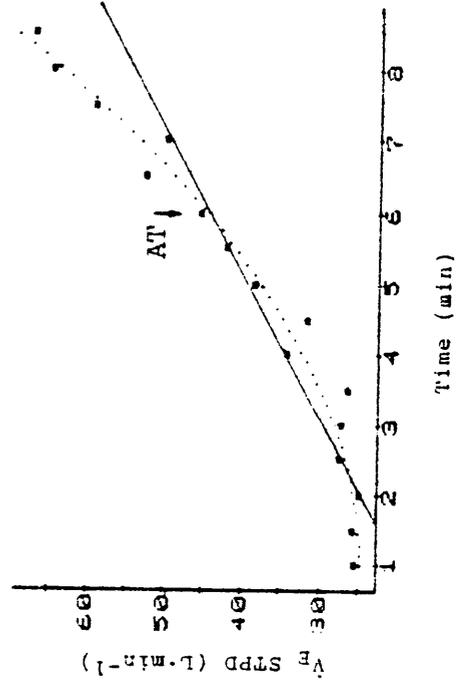
Pre-training
GXT₁



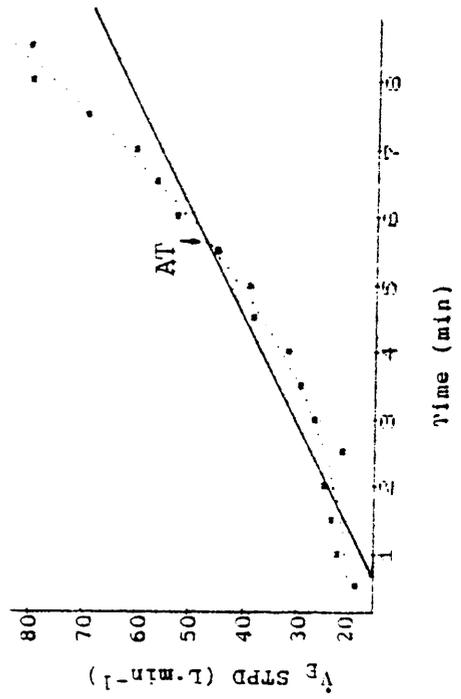
Post-training
GXT₃



GXT₂



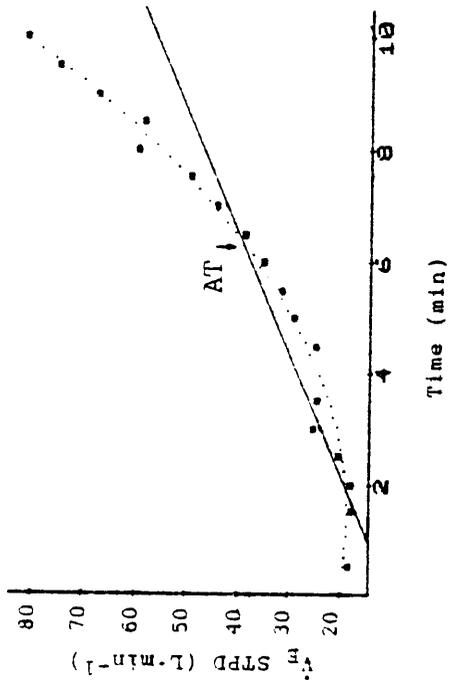
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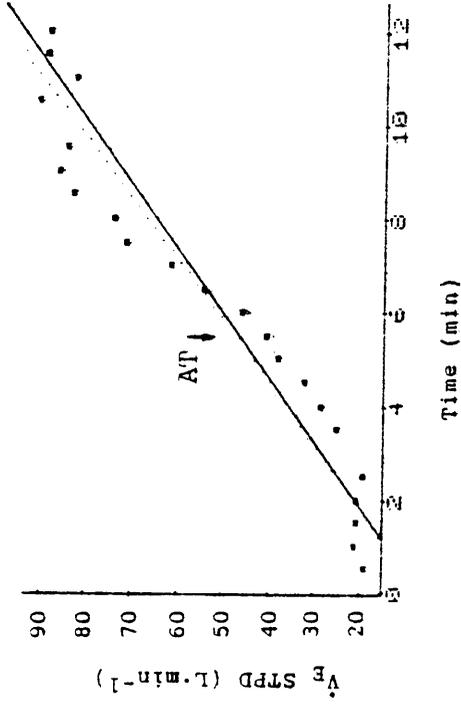
Pre-training

GXT 1

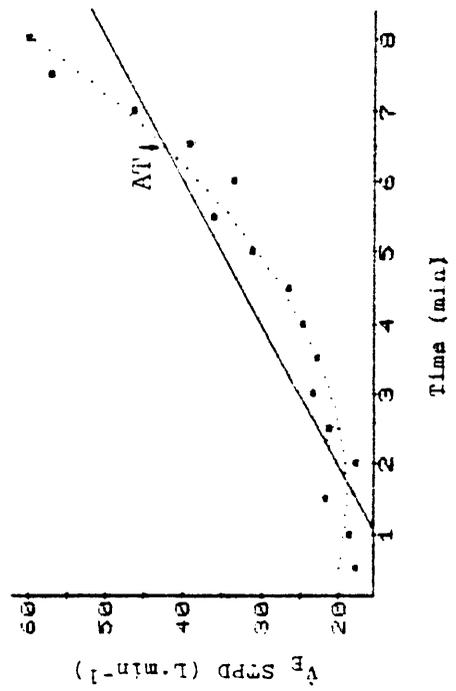


post-training

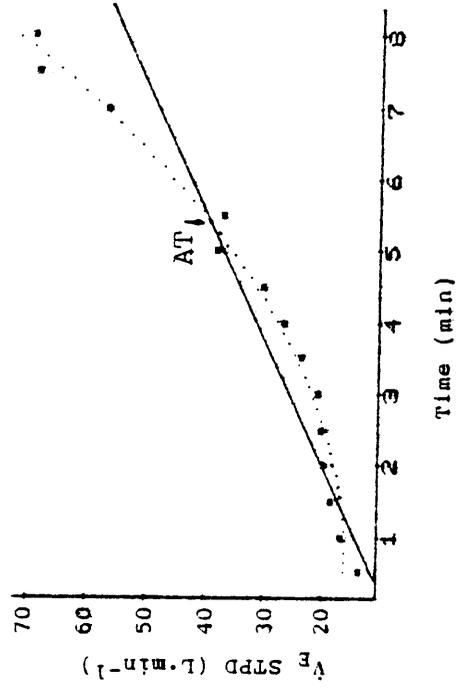
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GXT 2



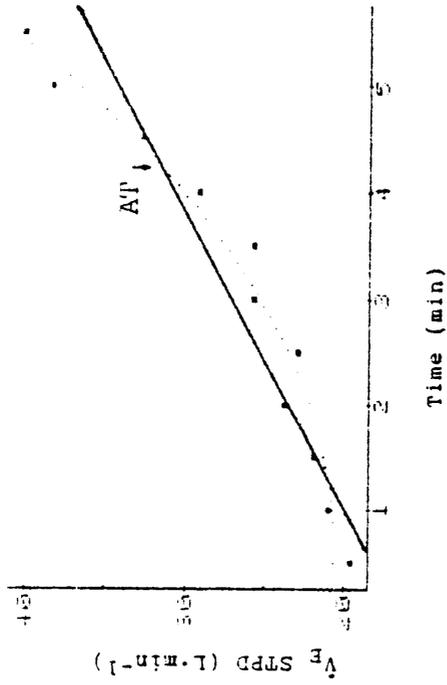
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Subject 11

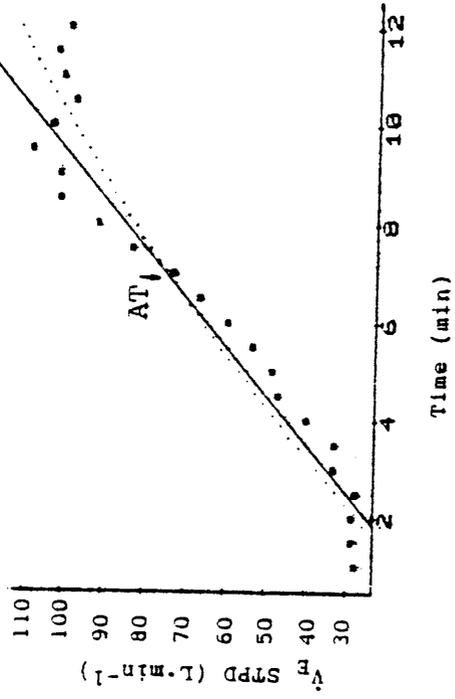
Pre-training

GXT₁

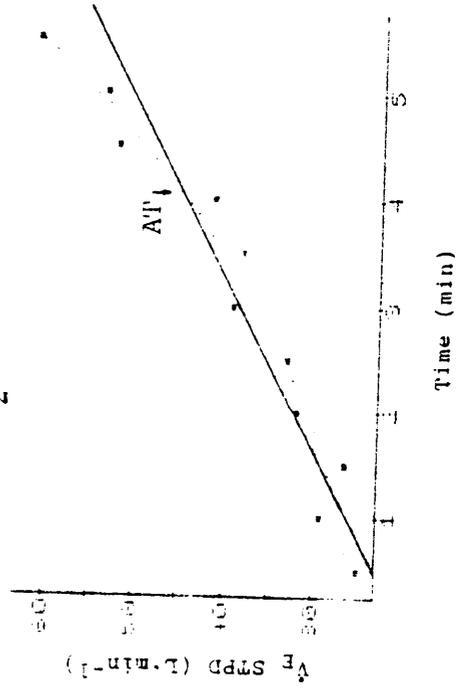


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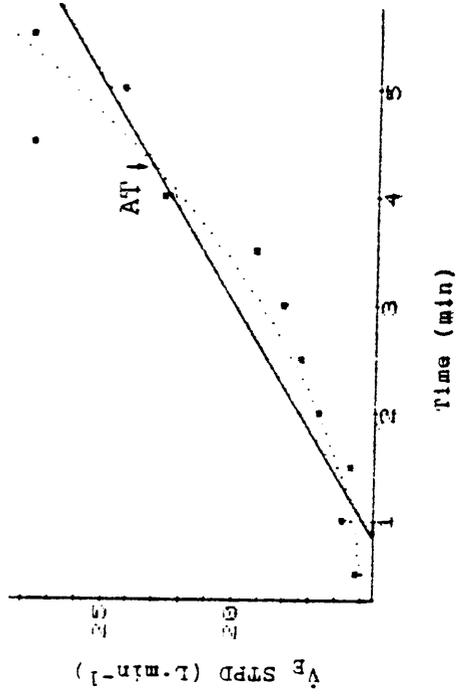
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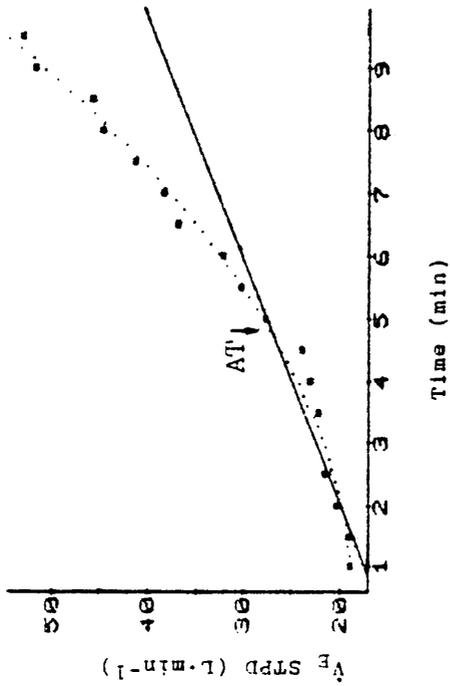


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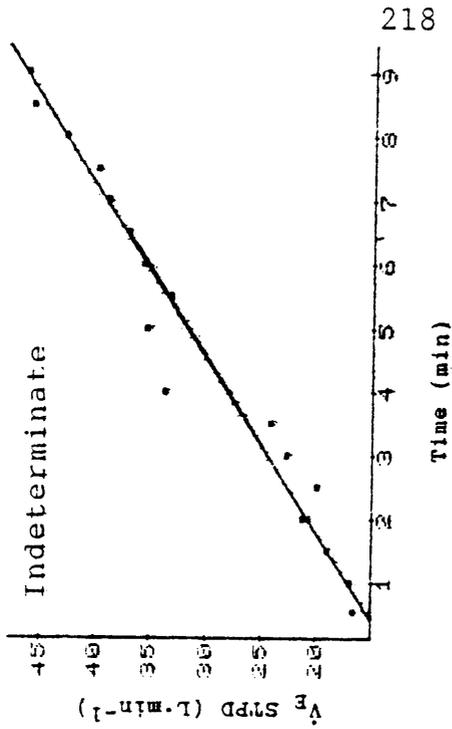


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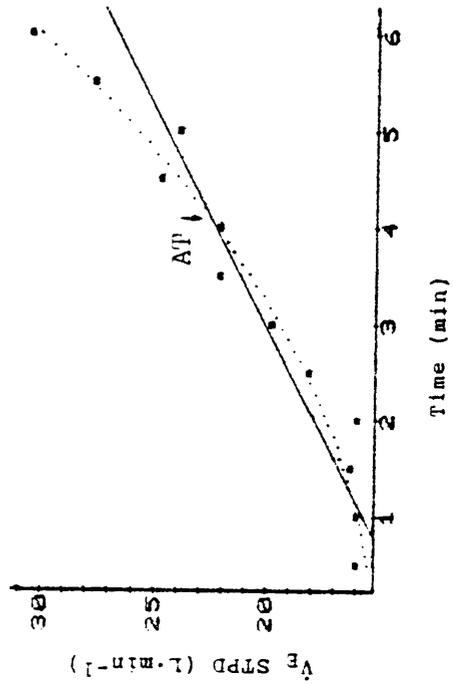
Pre-training
GXT₁



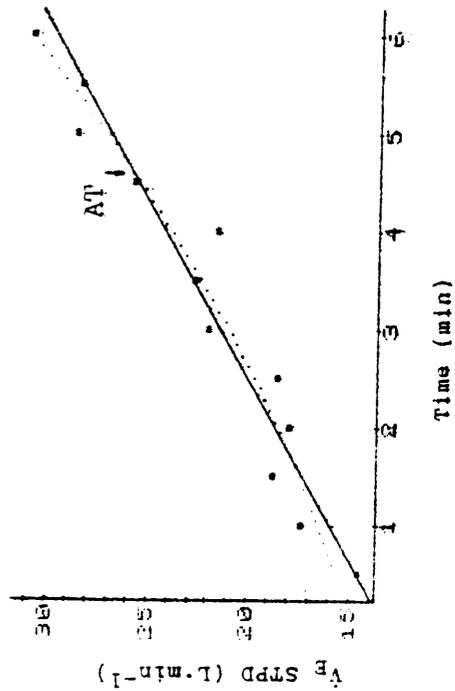
Post-training
GXT₃



GXT₂

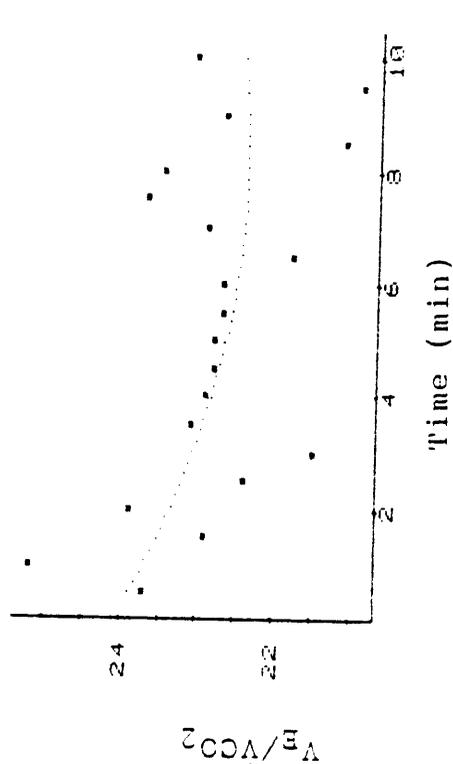
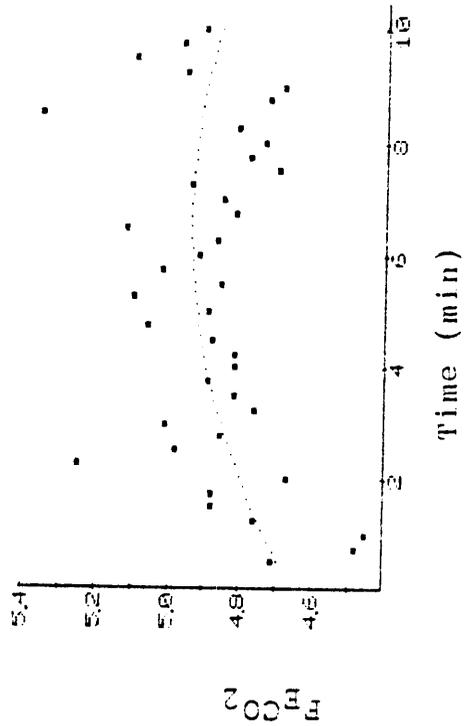
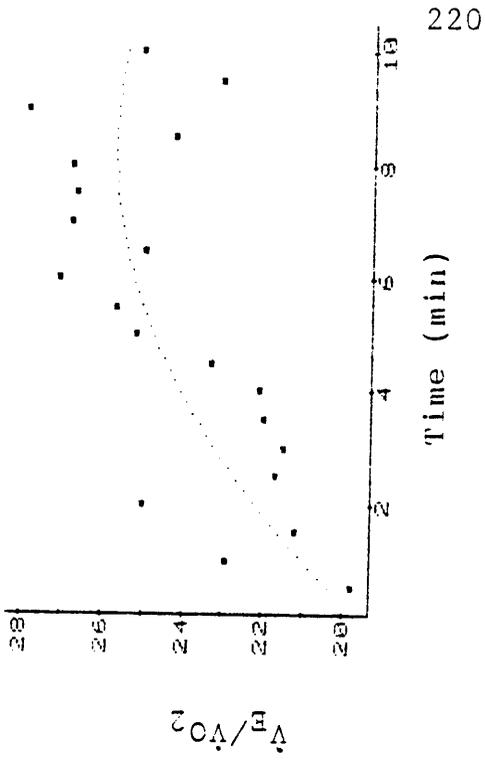
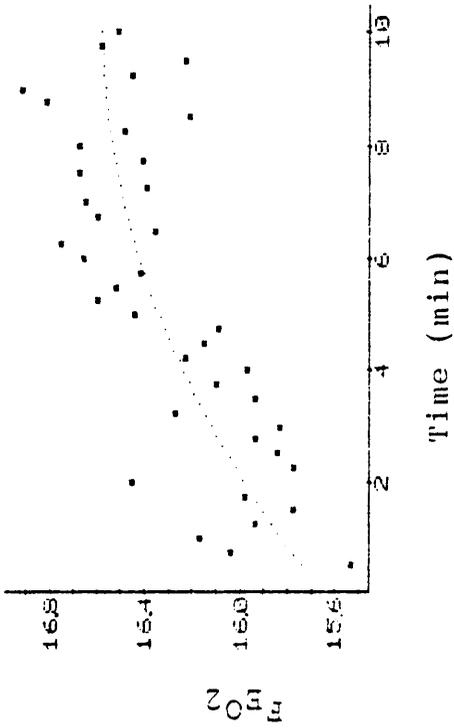


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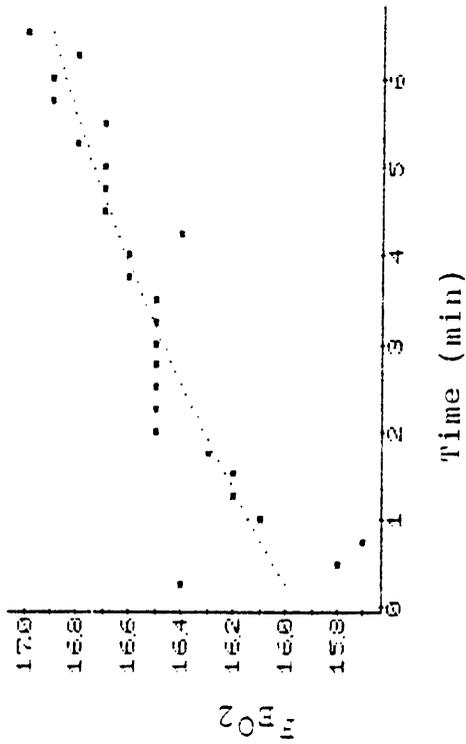


APPENDIX H
INDIVIDUALIZED GAS FRACTION AND
VENTILATION EQUIVALENT DATA CURVES

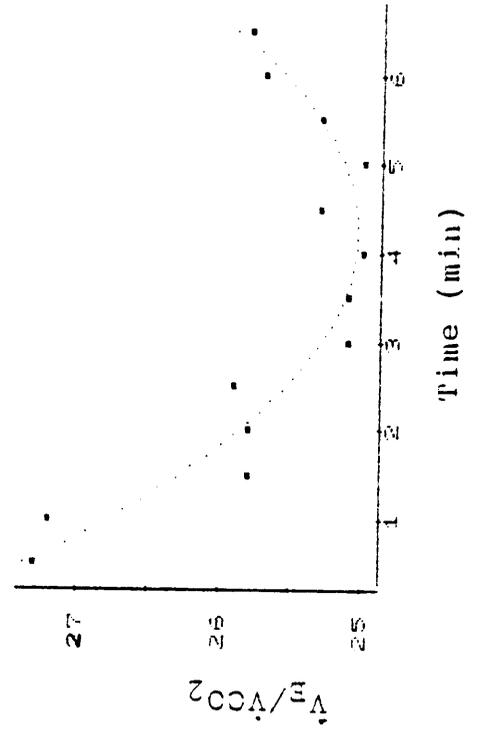
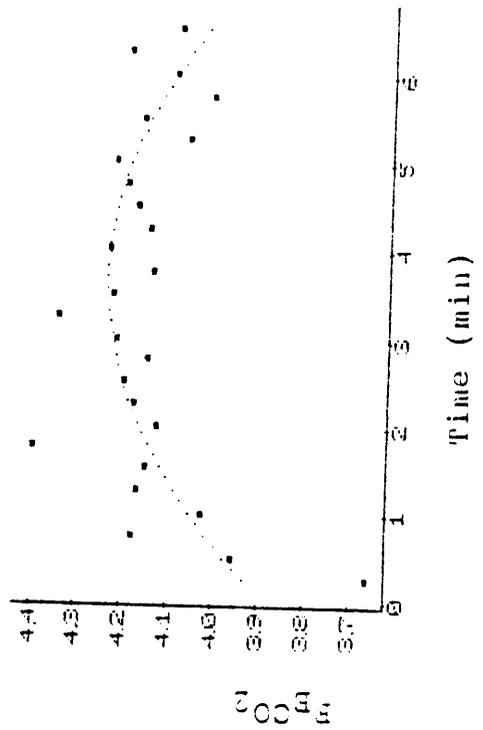
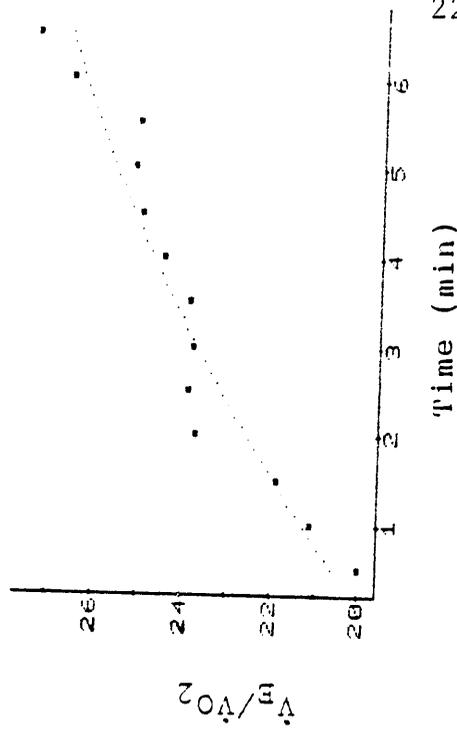
Subject 1
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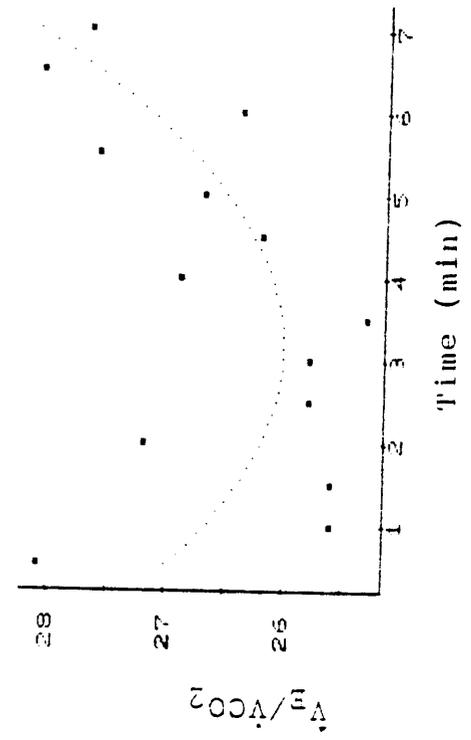
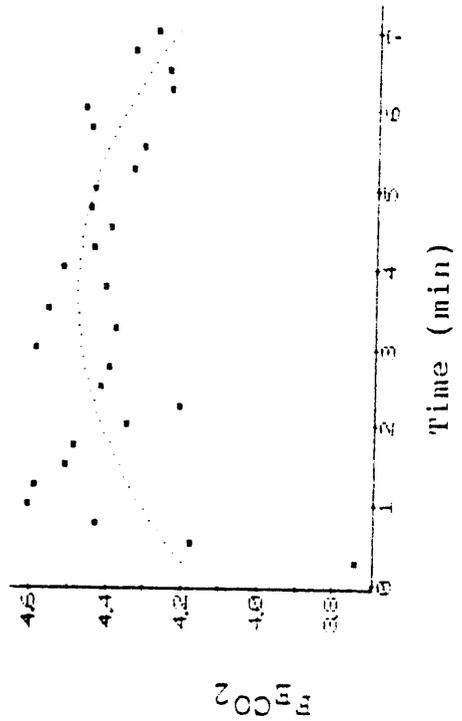
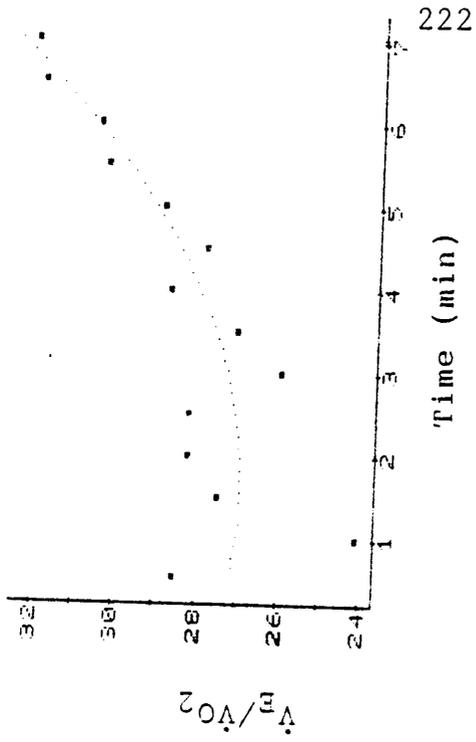
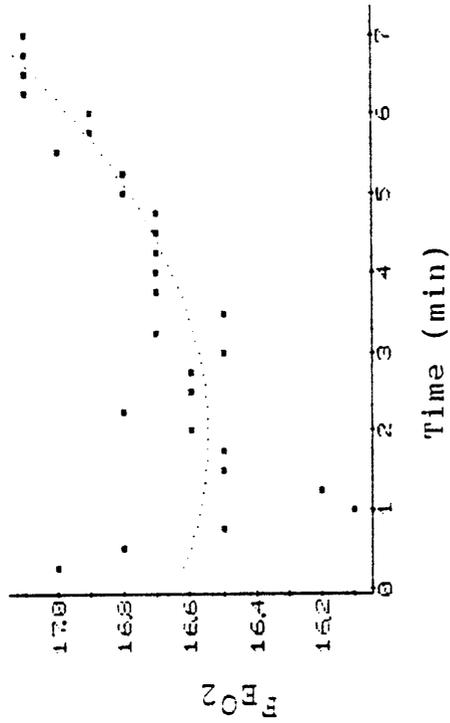
Subject 1
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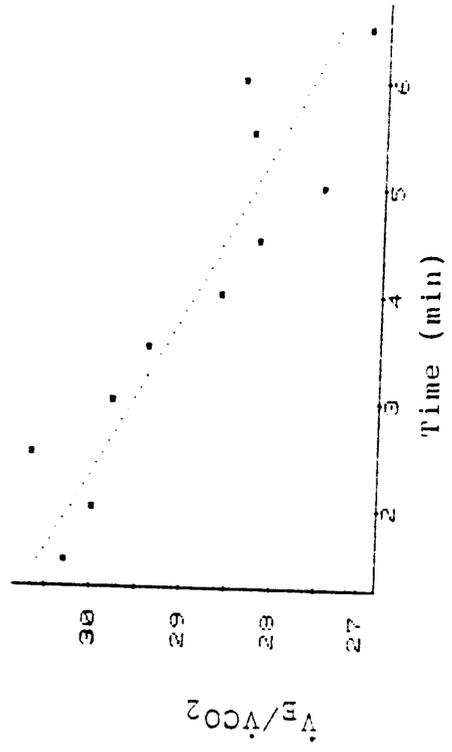
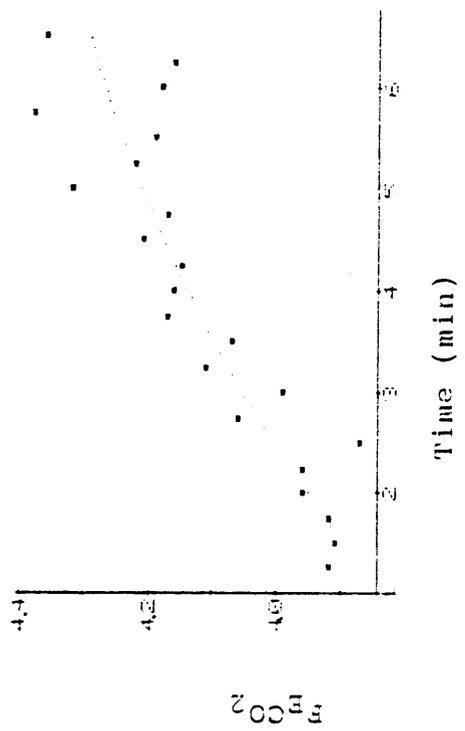
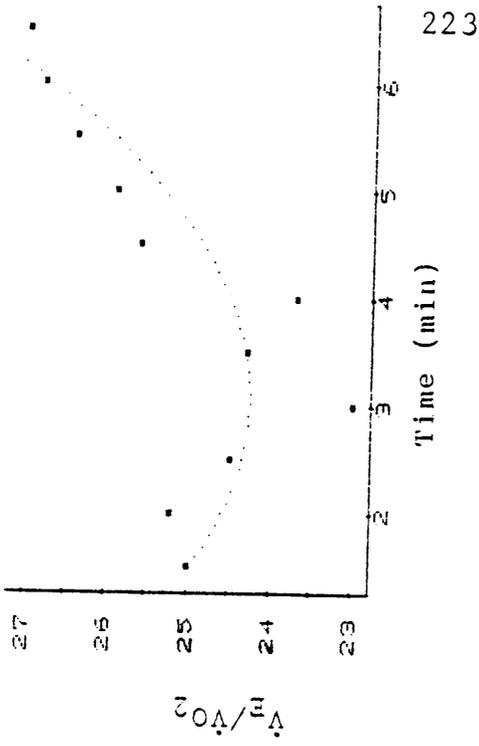
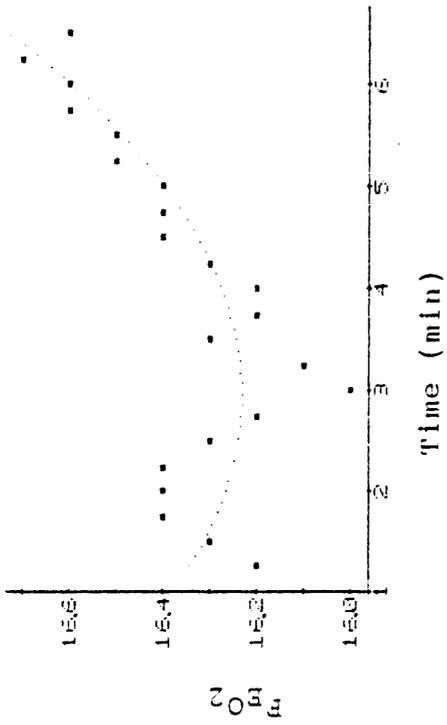
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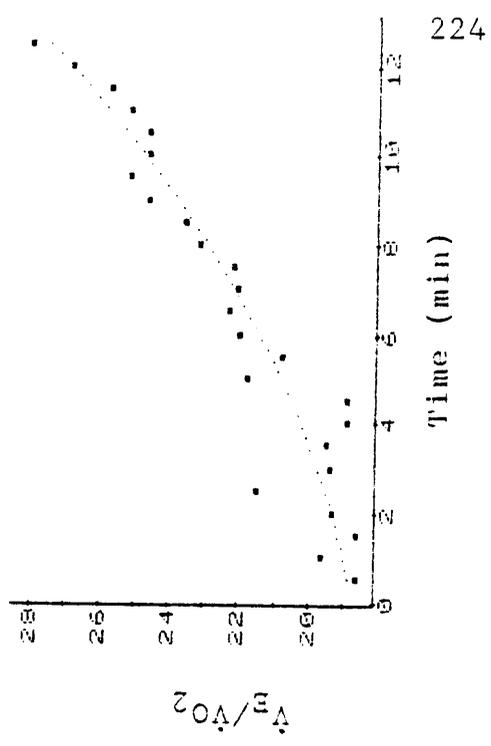
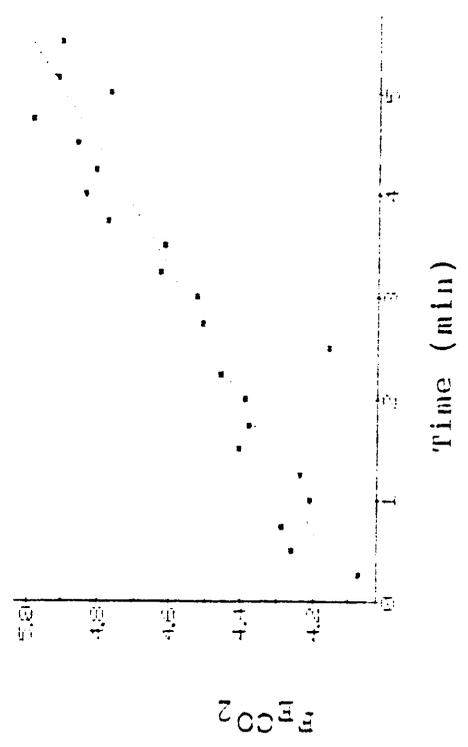
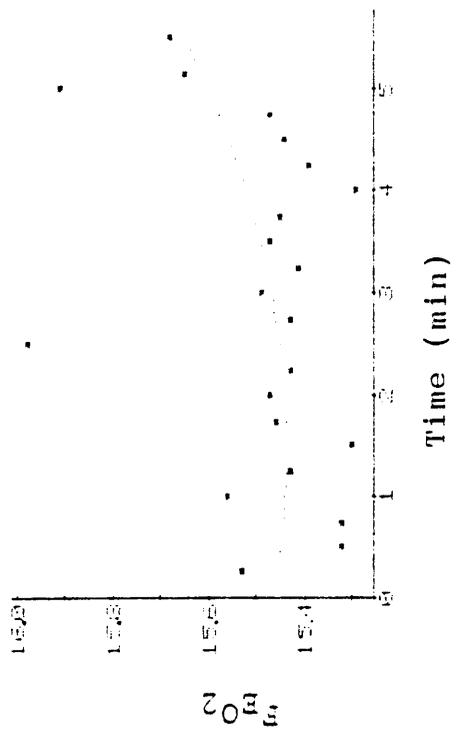
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GXT3



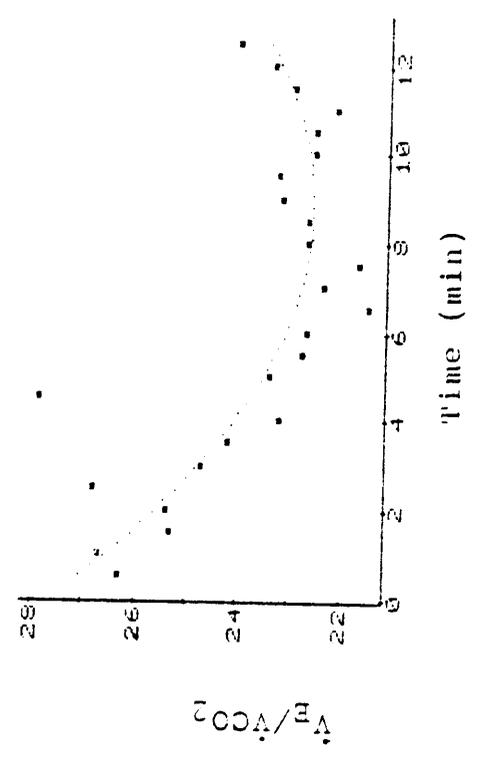
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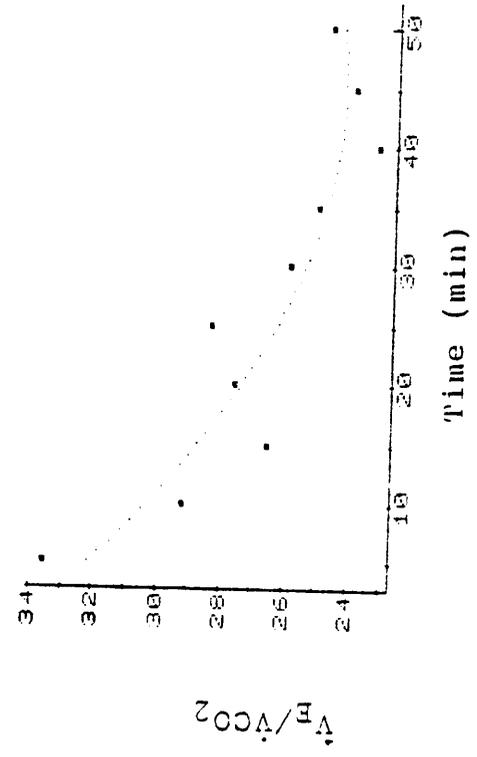
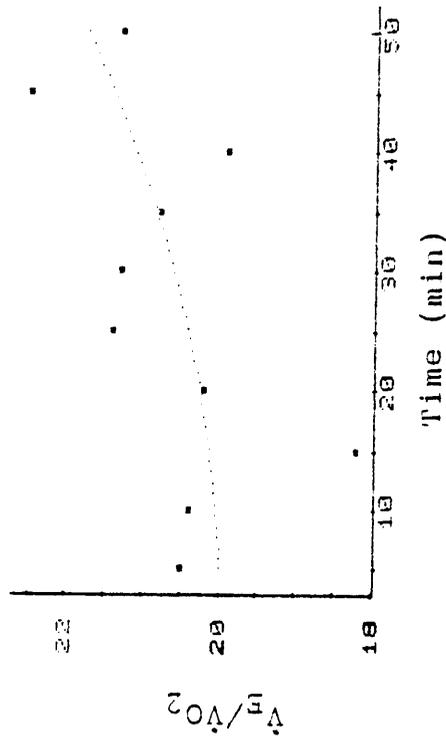
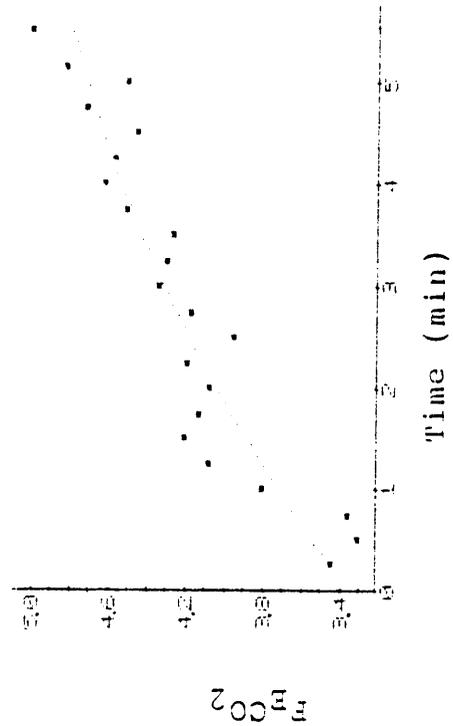
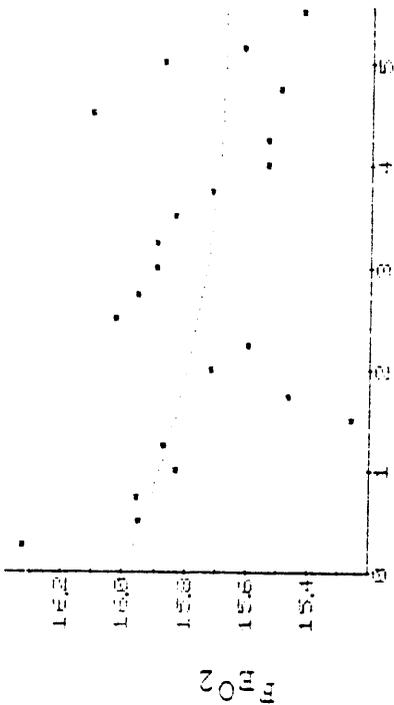
Subject 2
GXT₁



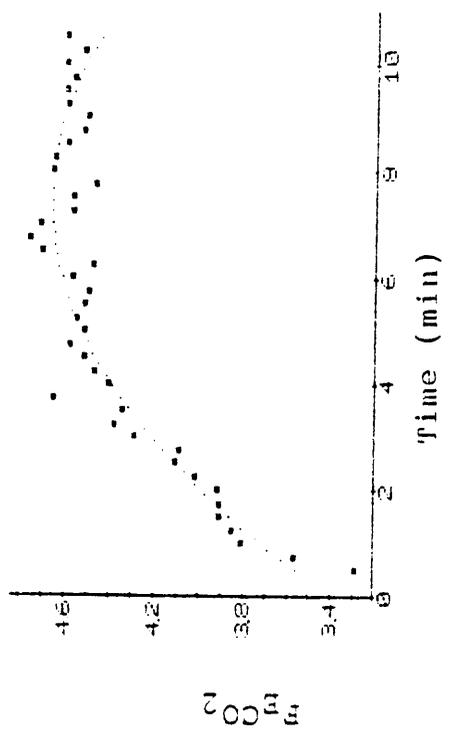
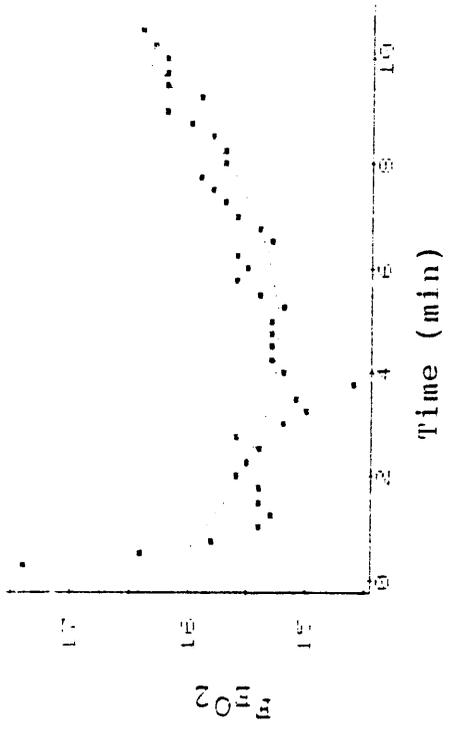
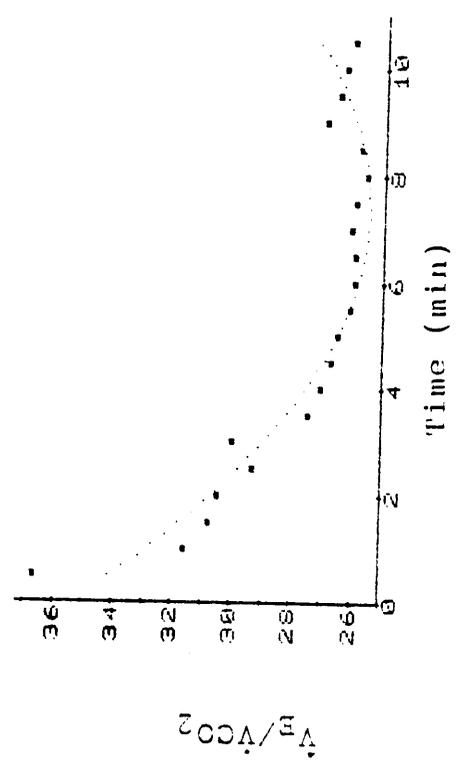
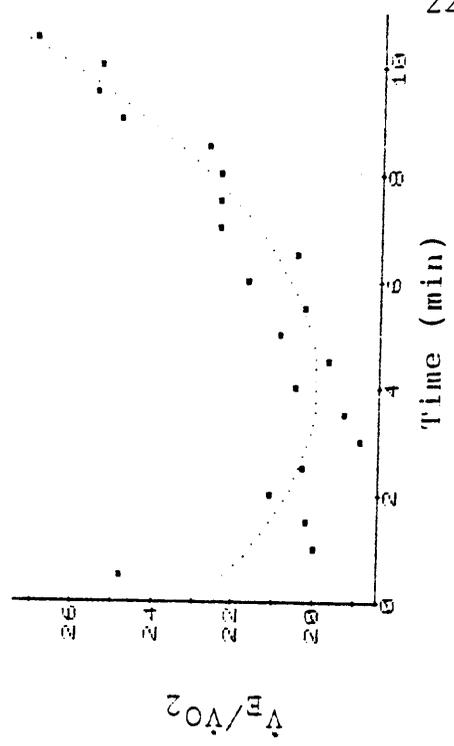
224



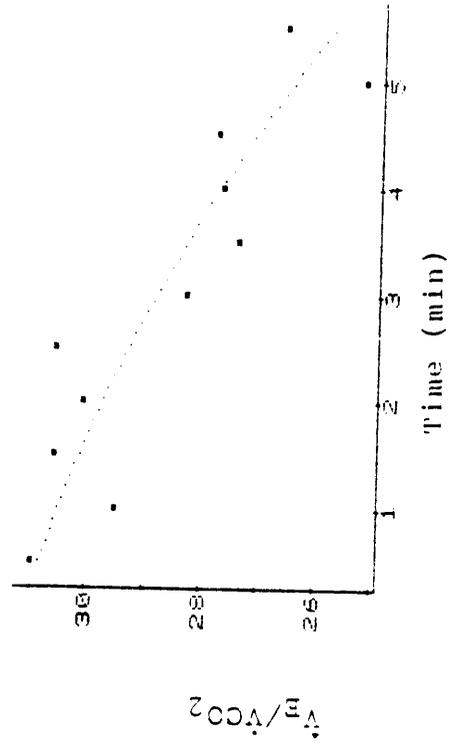
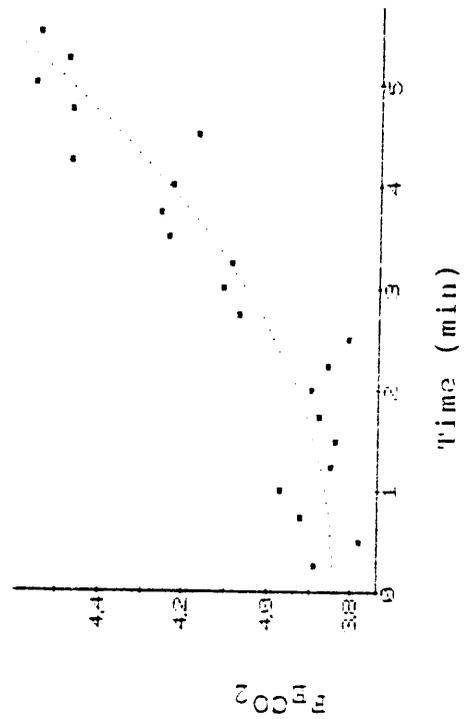
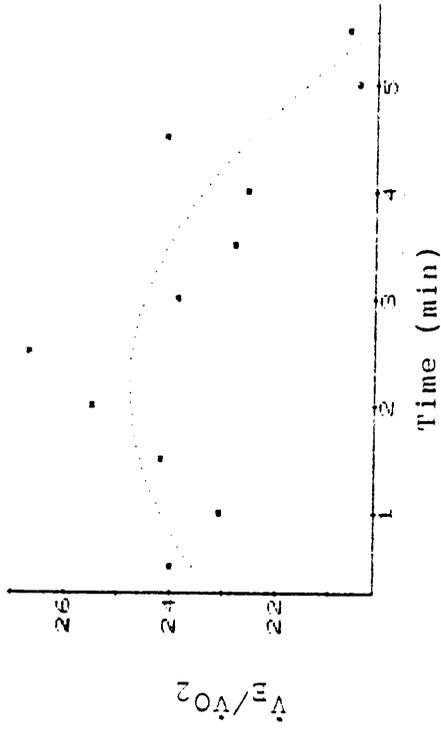
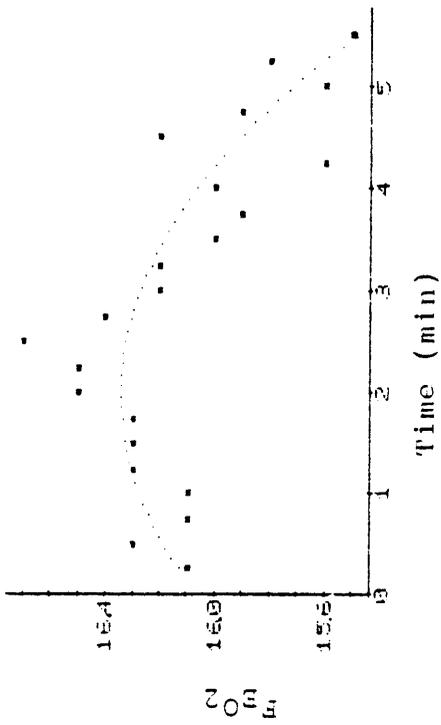
Subject 2
GXT2



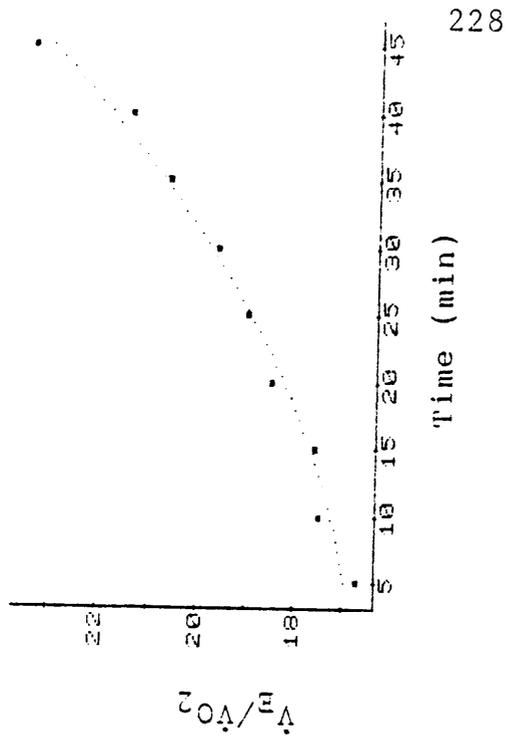
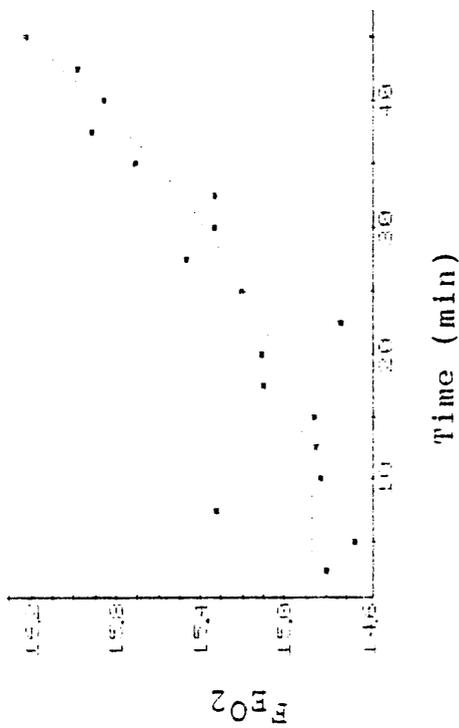
Subject 2
GXT3



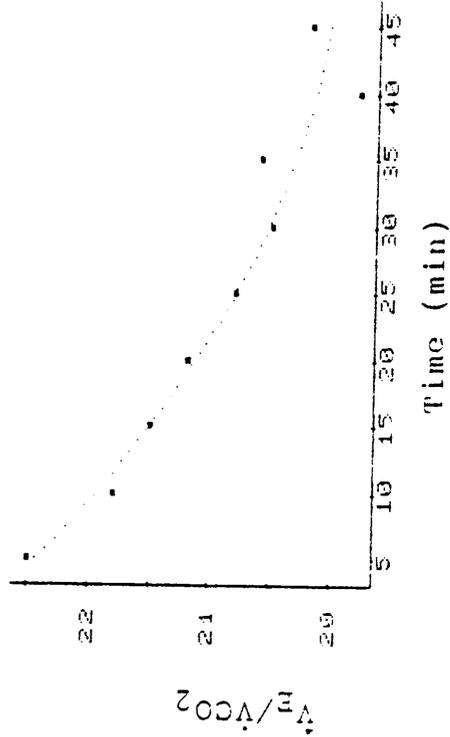
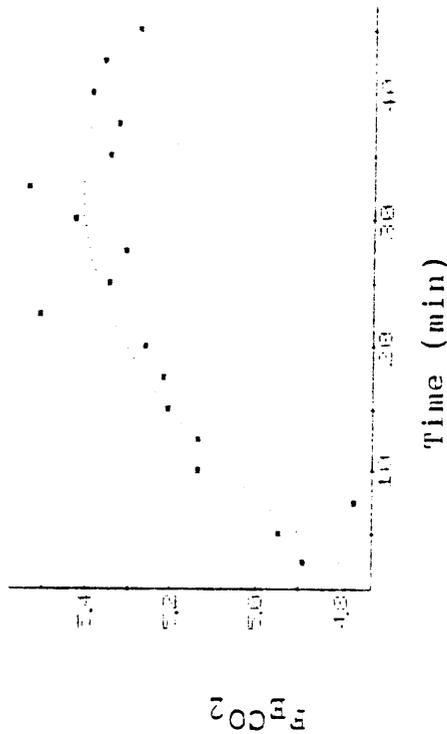
Subject 2
GXT₄



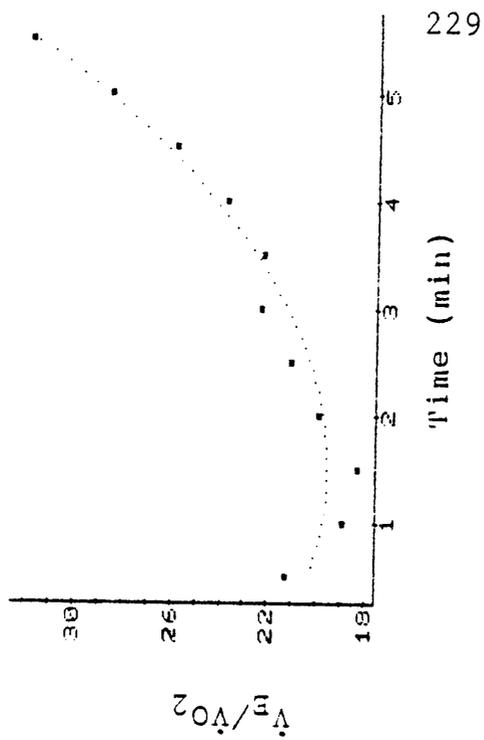
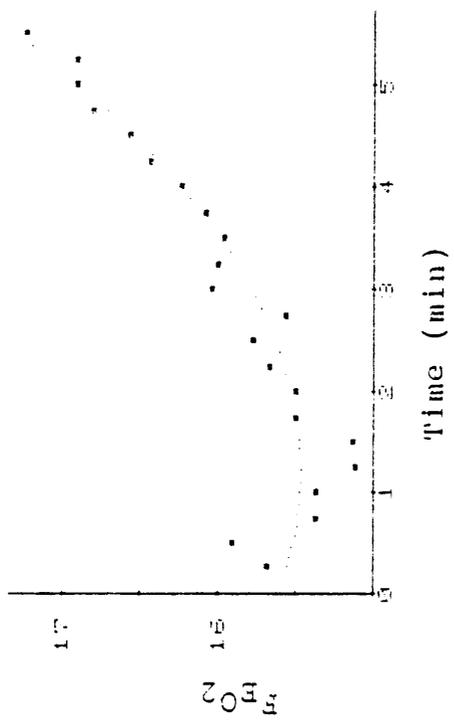
Subject 3
GXT1



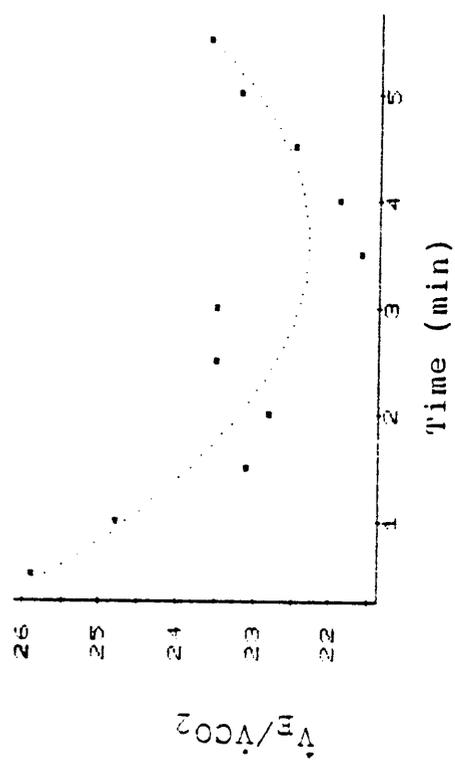
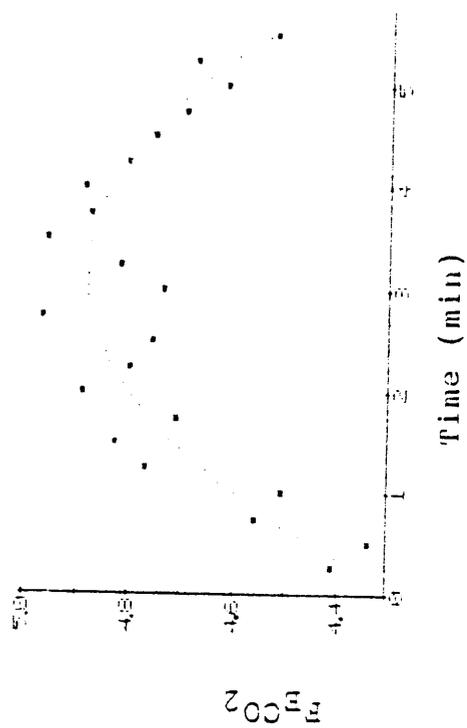
228



Subject 3
GXT2

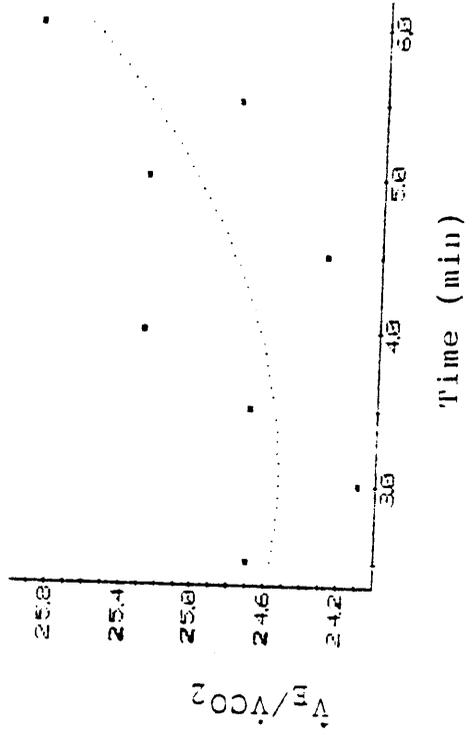
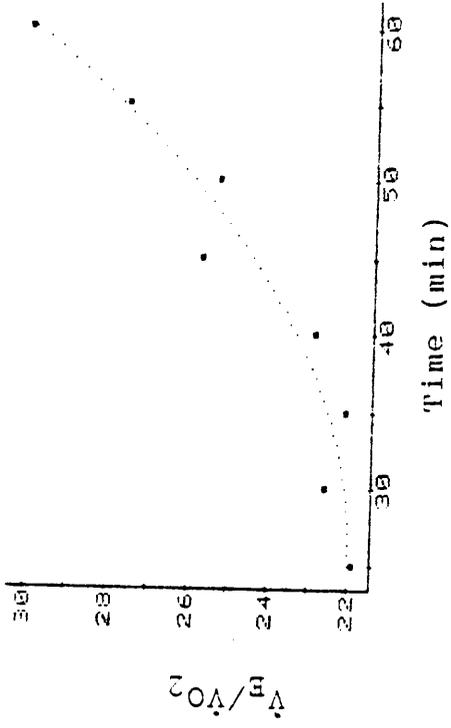
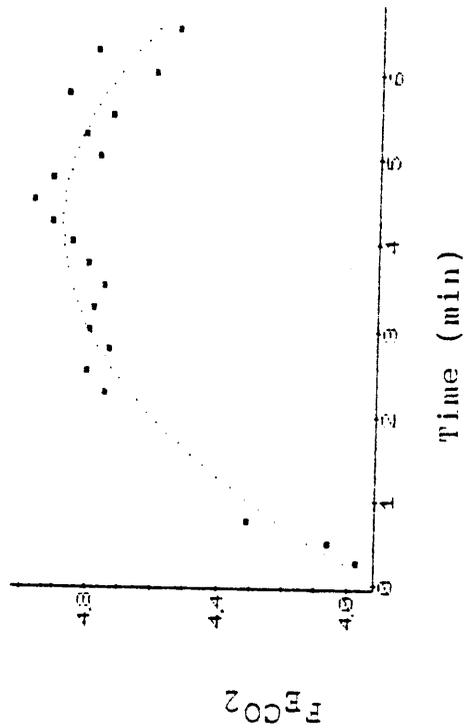
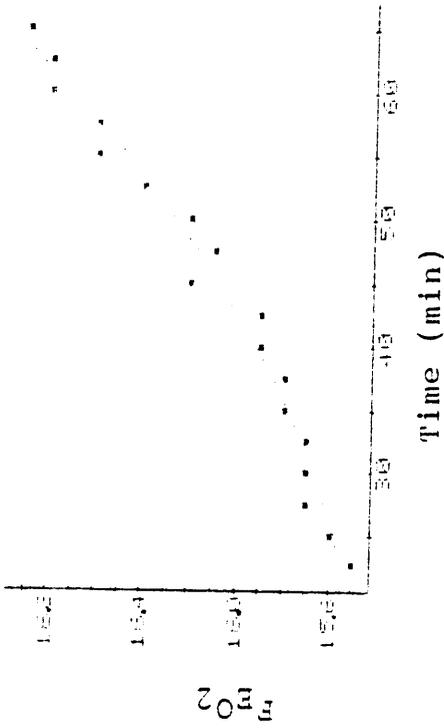


229

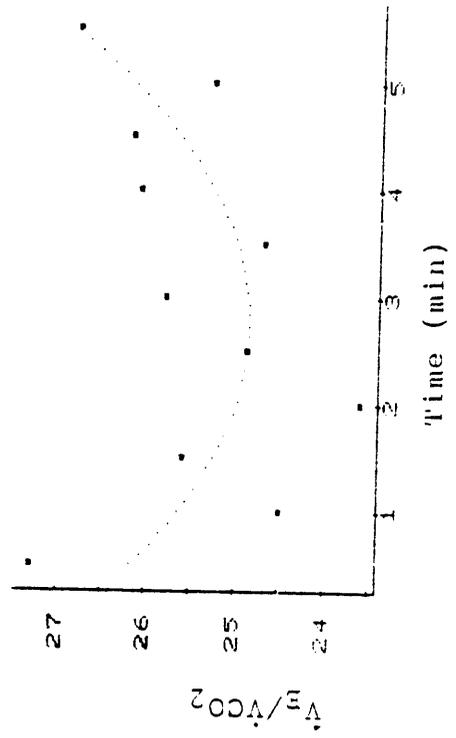
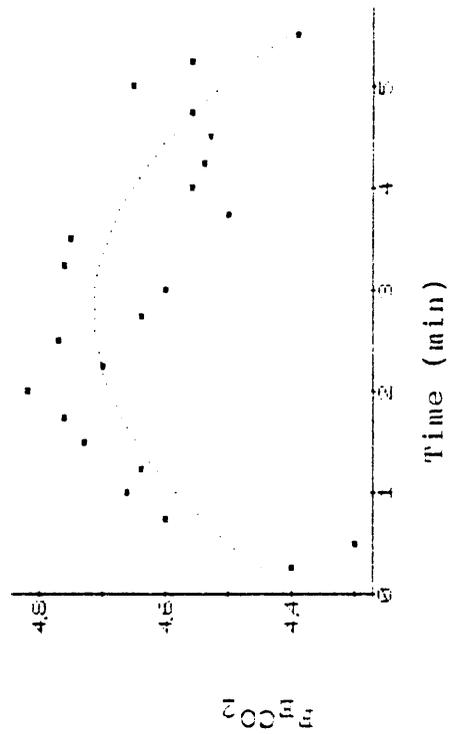
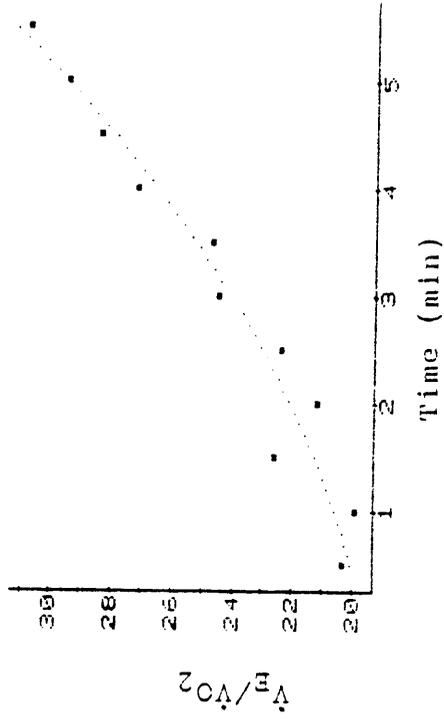
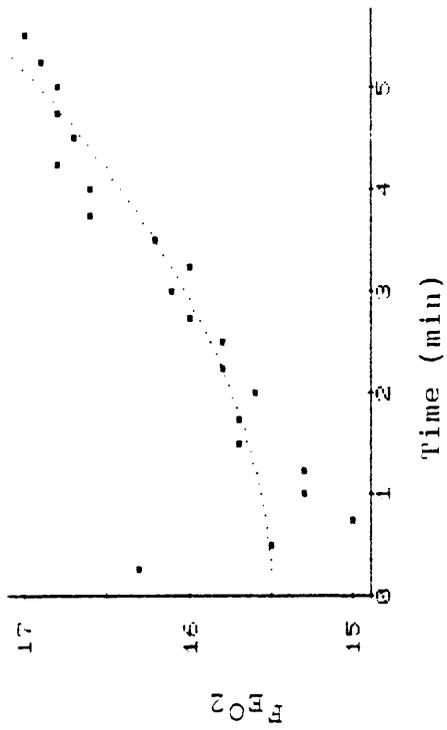


Subject 3
GXI3

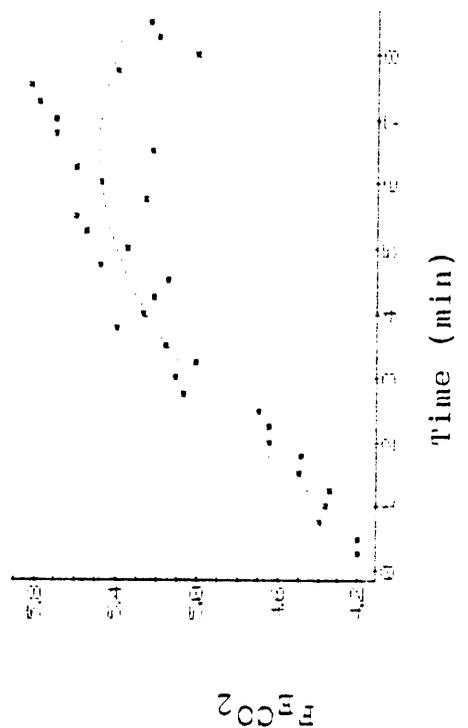
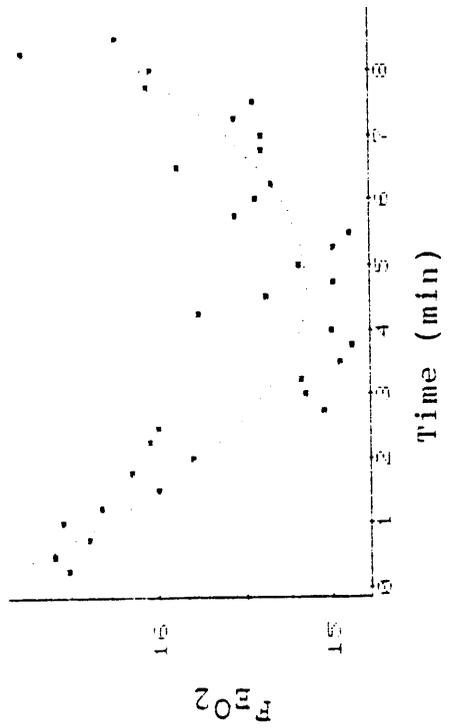
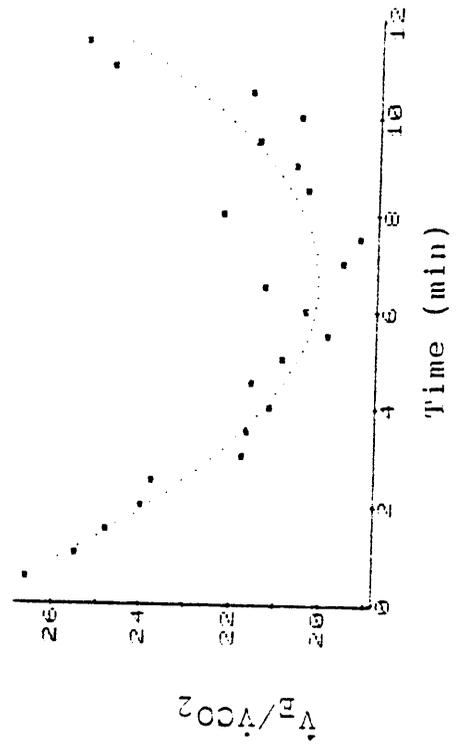
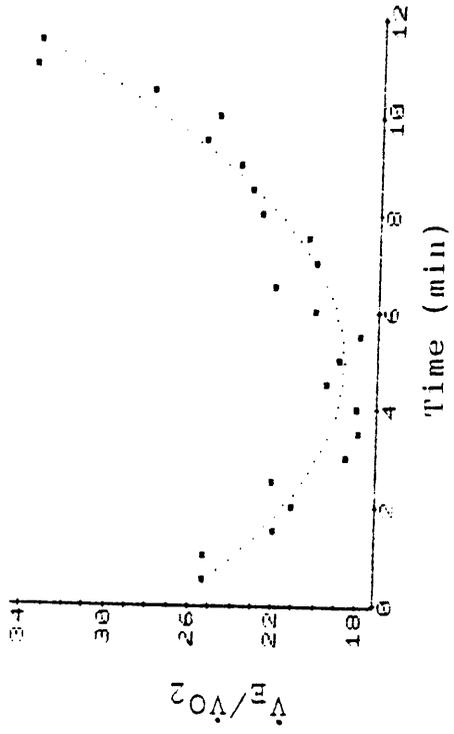
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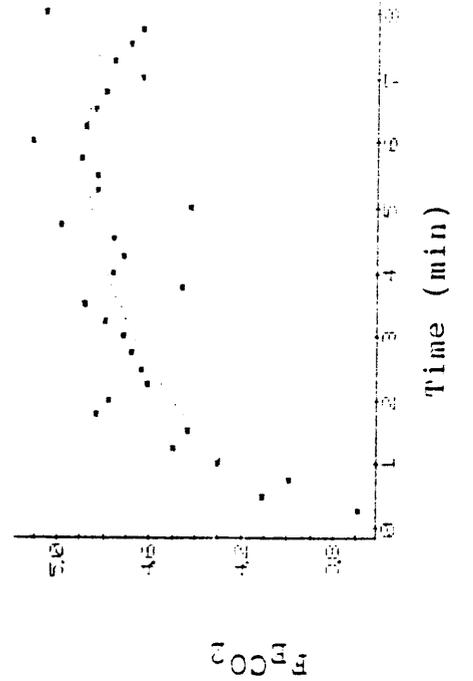
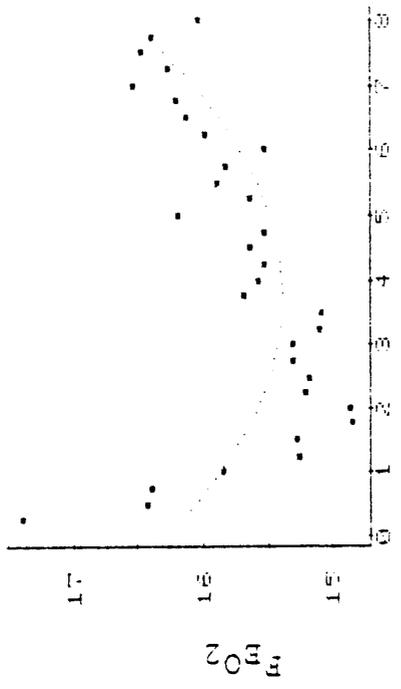
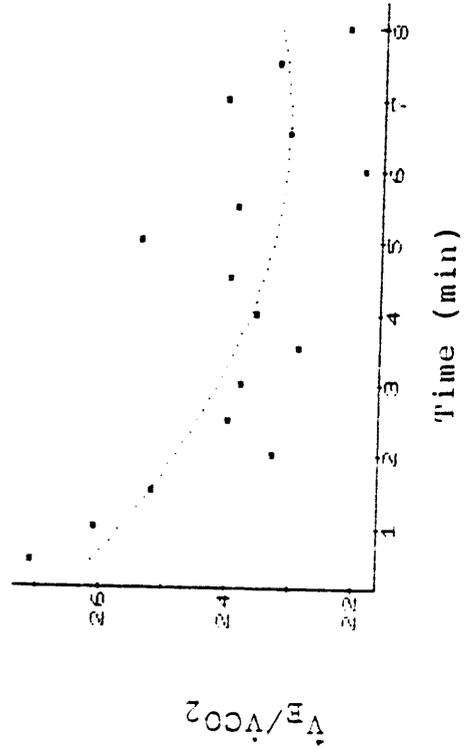
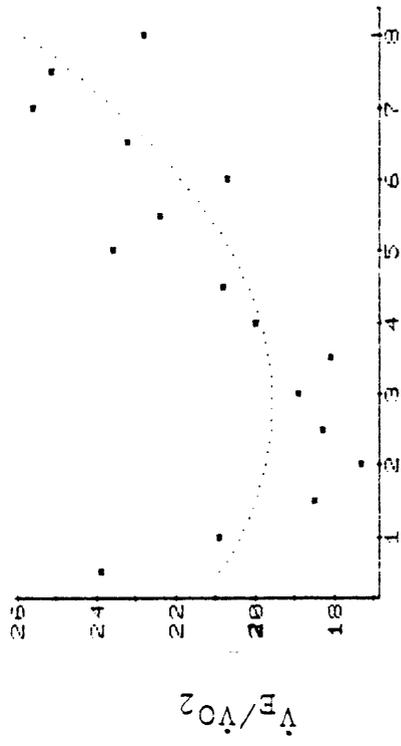
Subject 3
GXT4



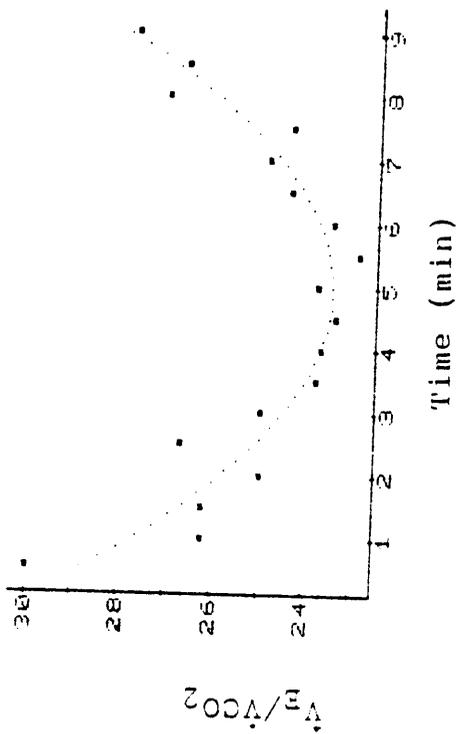
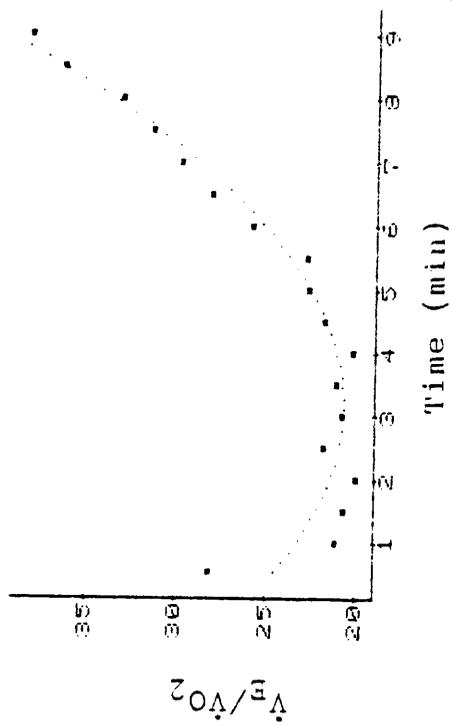
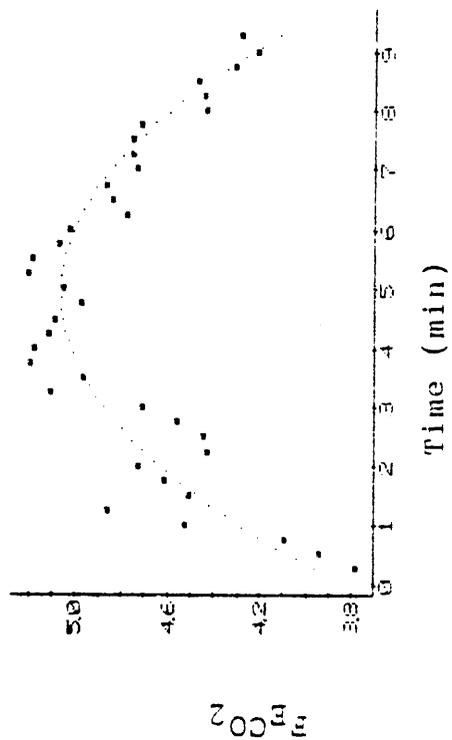
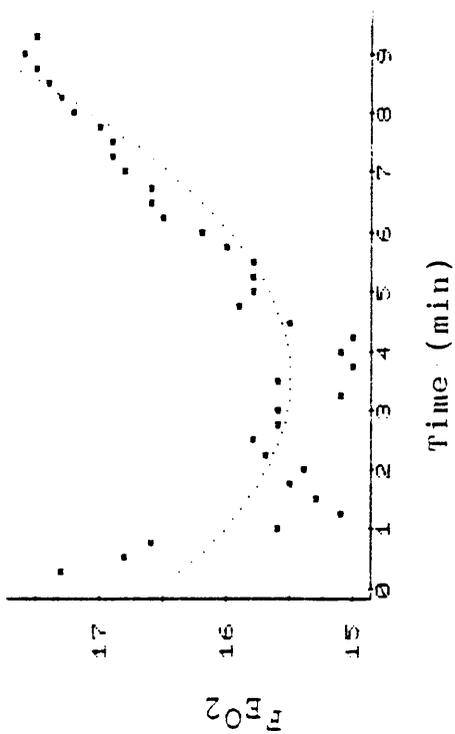
Subject 4
GXT₁



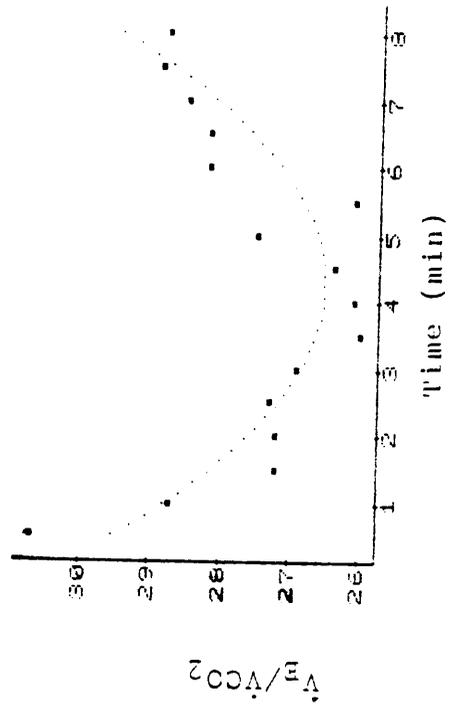
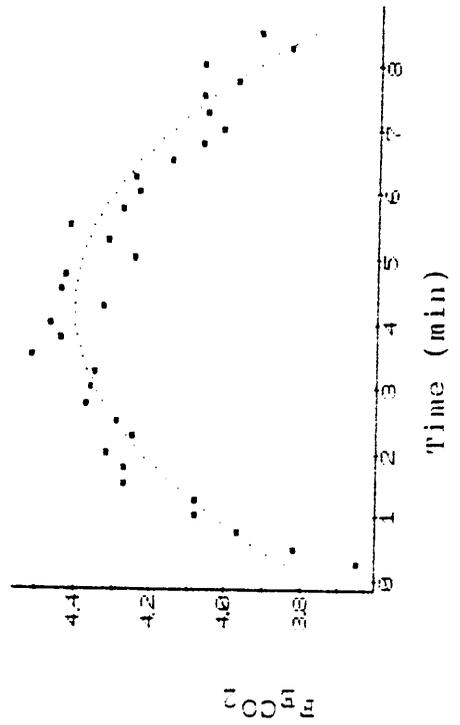
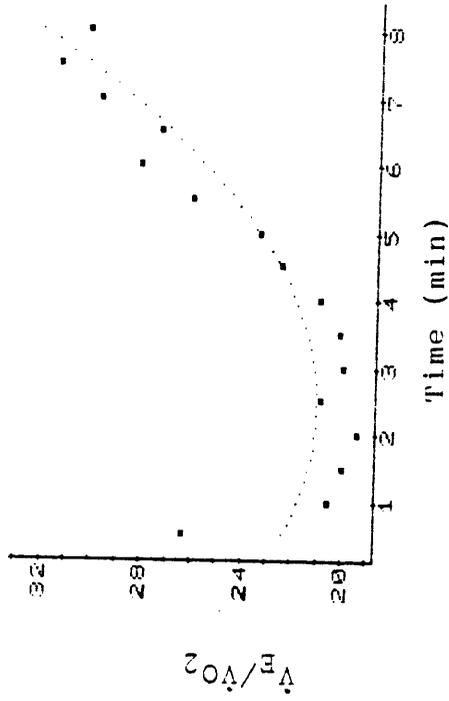
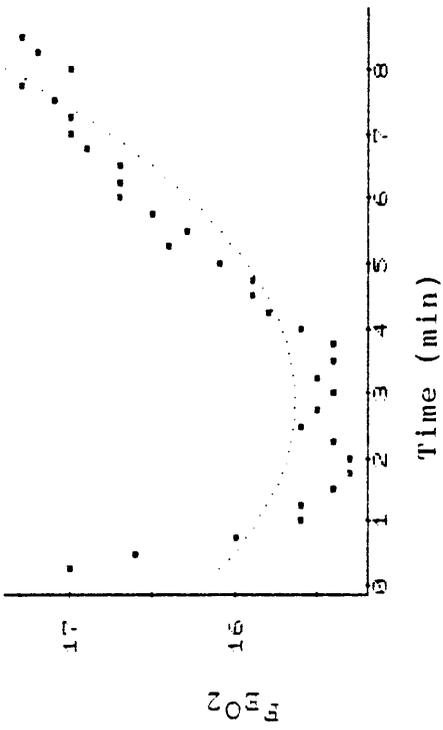
Subject 4
GXT'2



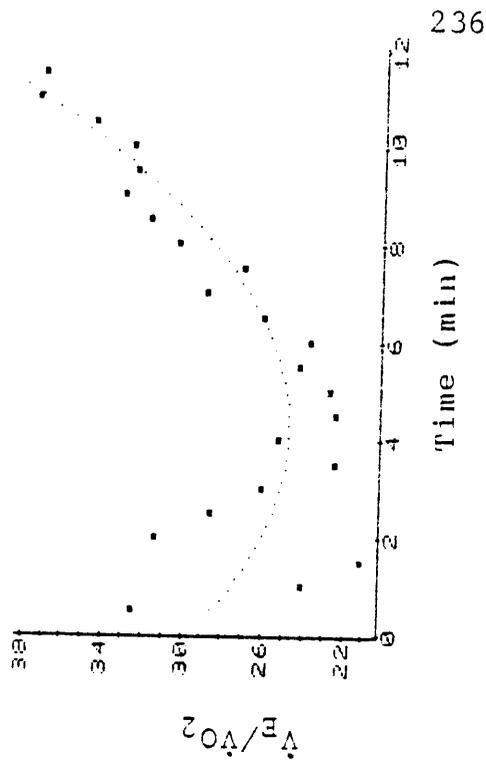
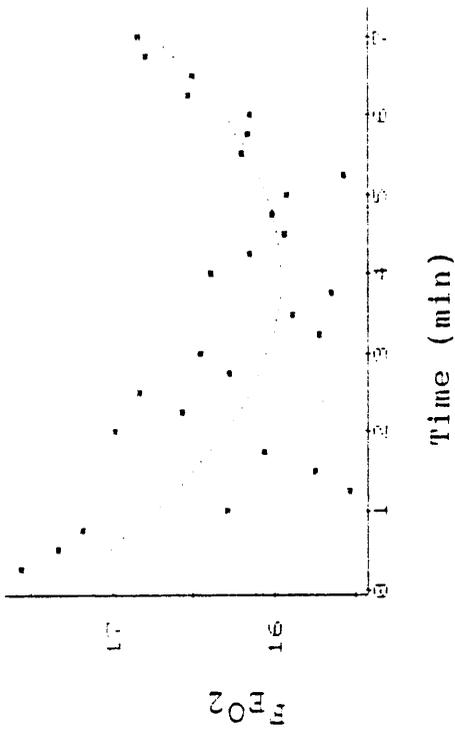
Subject 4
GXT3



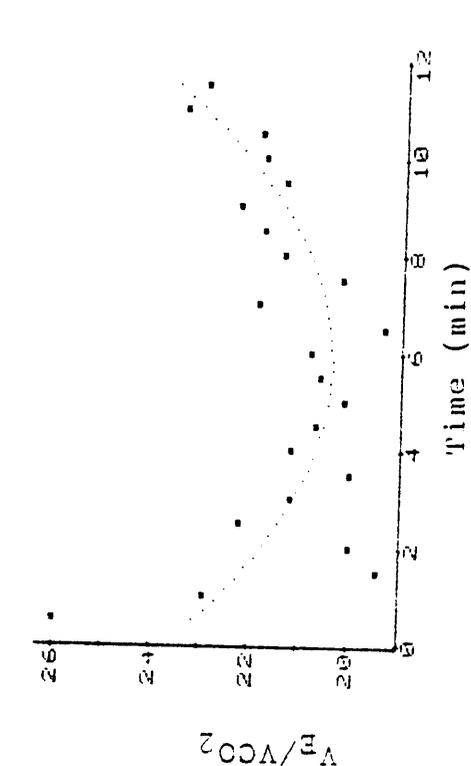
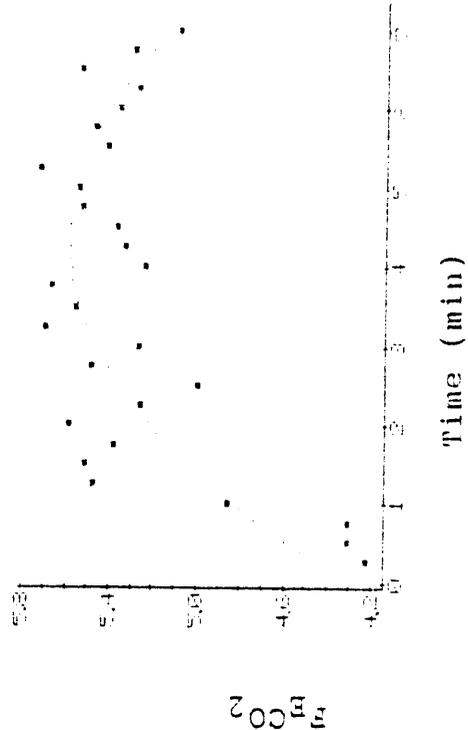
Subject 4
GXT₄



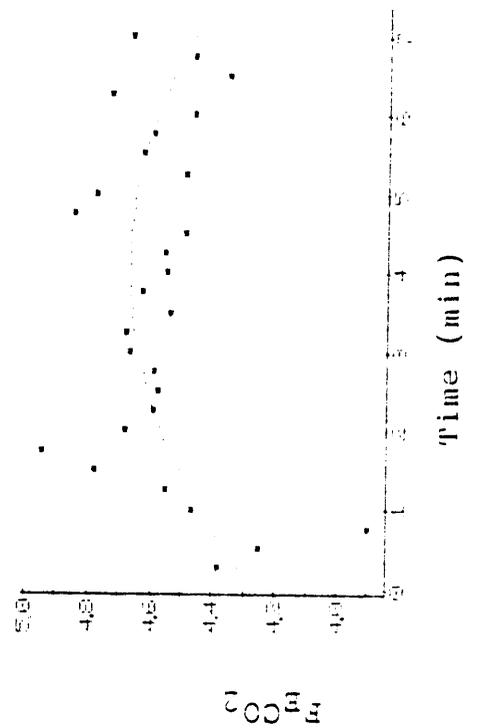
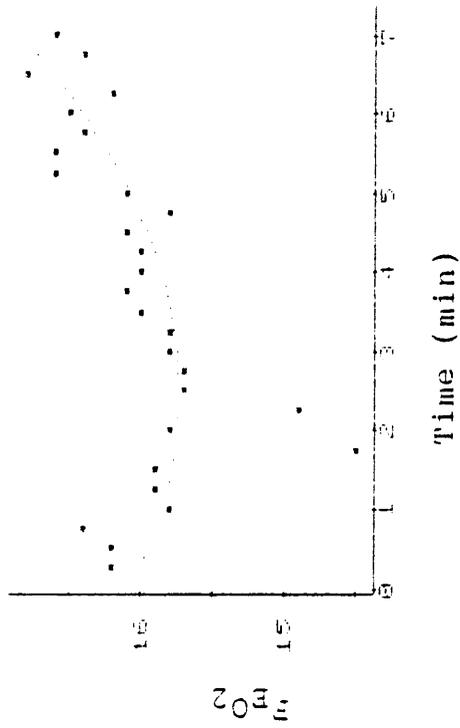
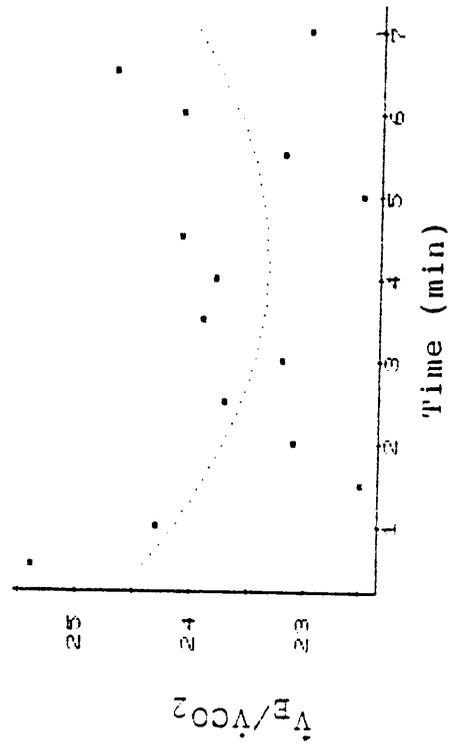
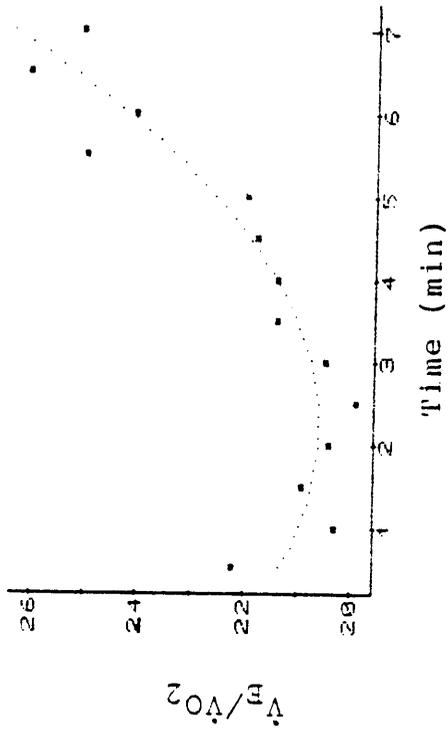
Subject 5
GXT1



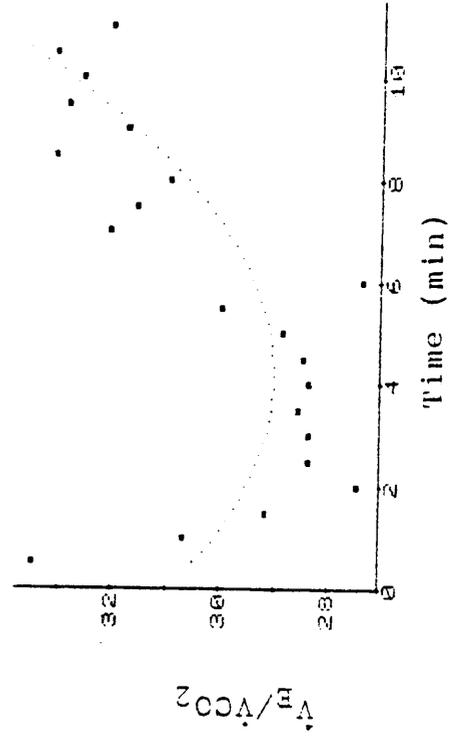
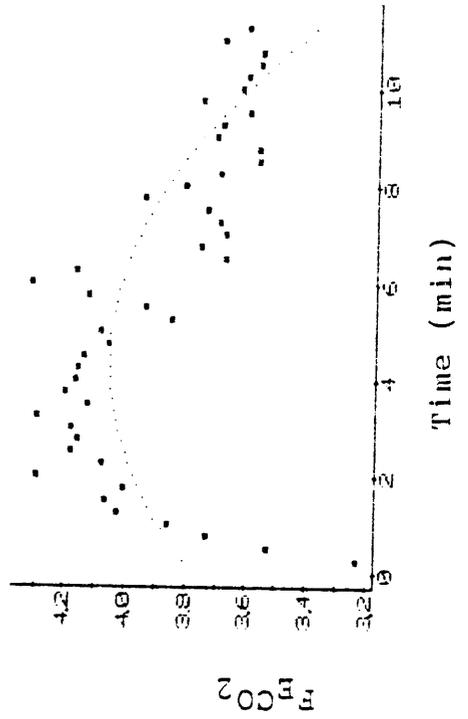
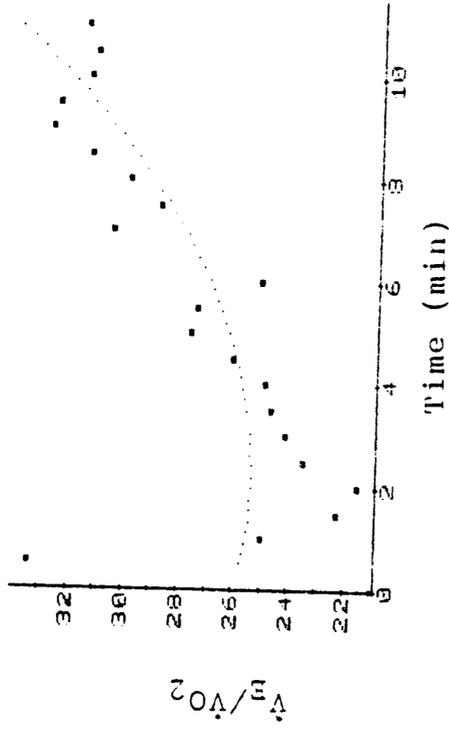
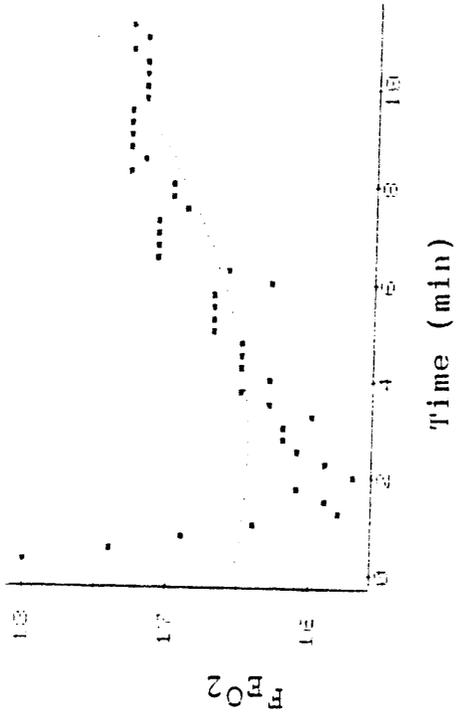
236



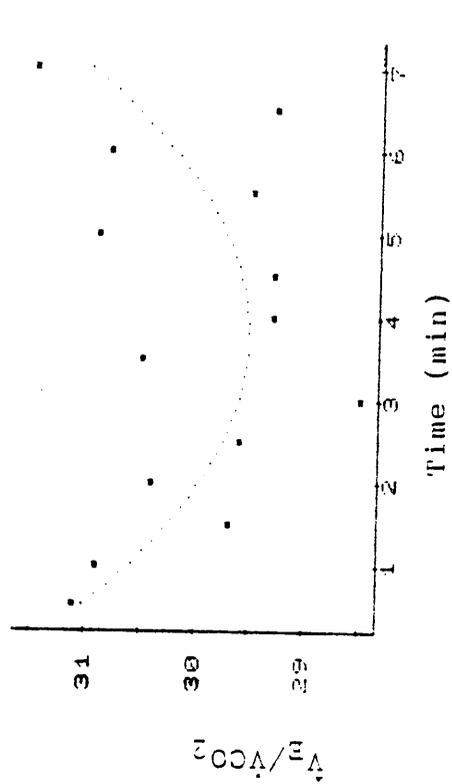
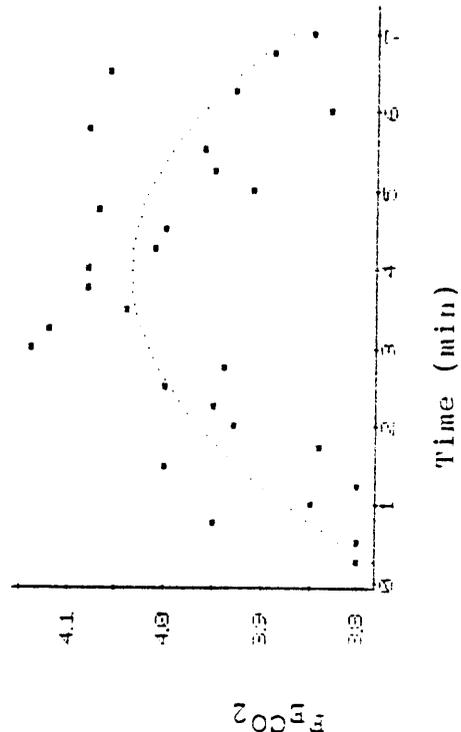
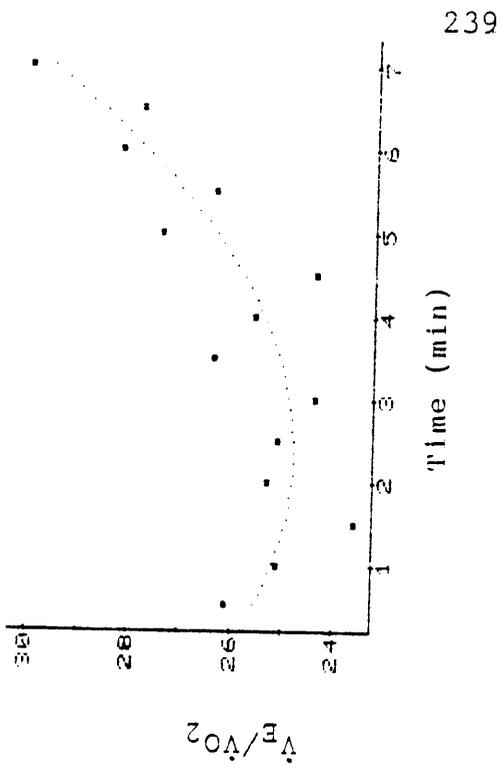
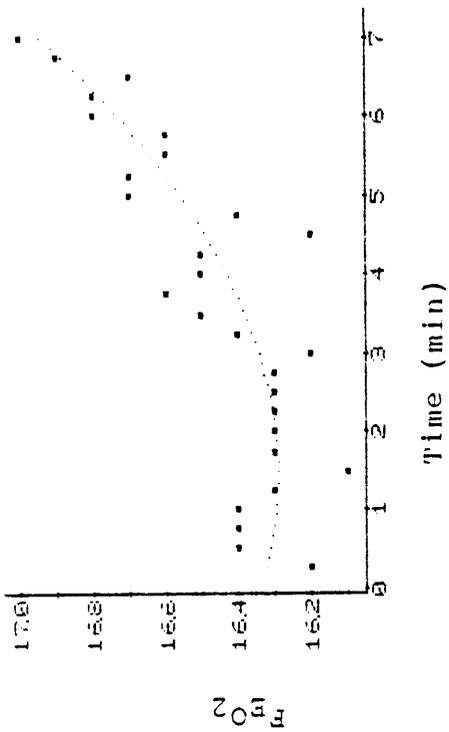
Subject 5
GXT2



Subject 5
GXT3

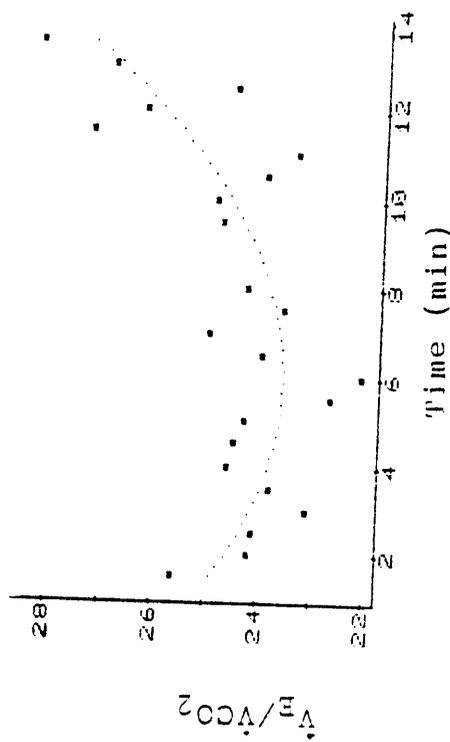
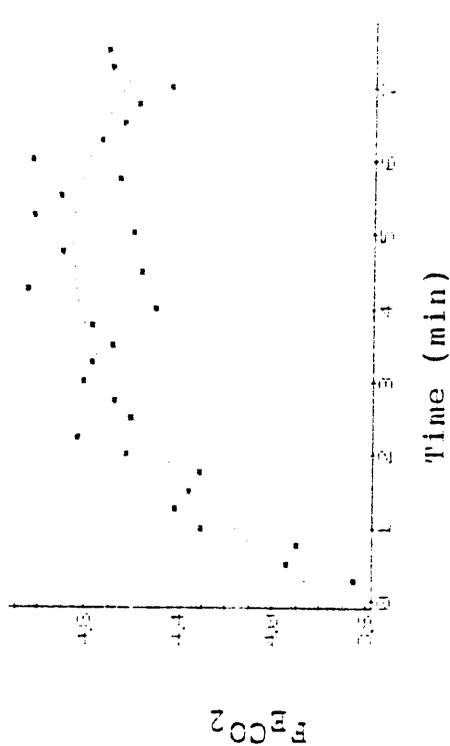
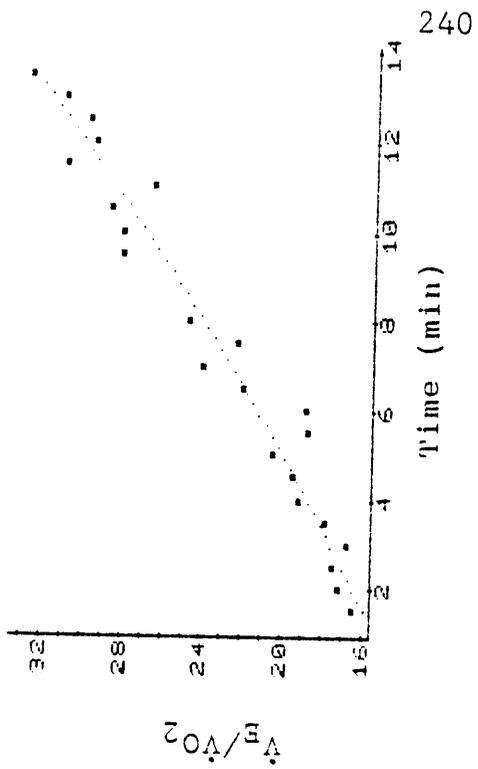
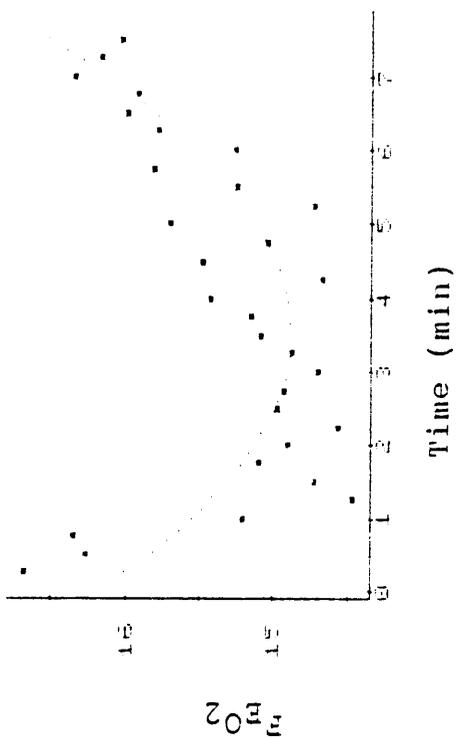


Subject 5
GX14

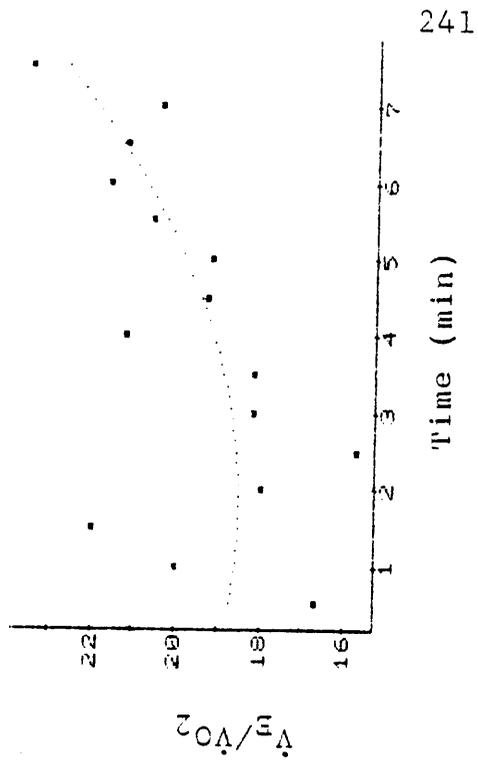
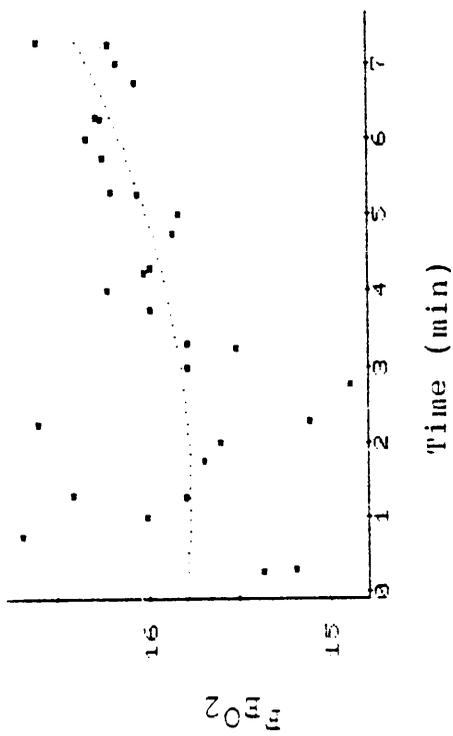


239

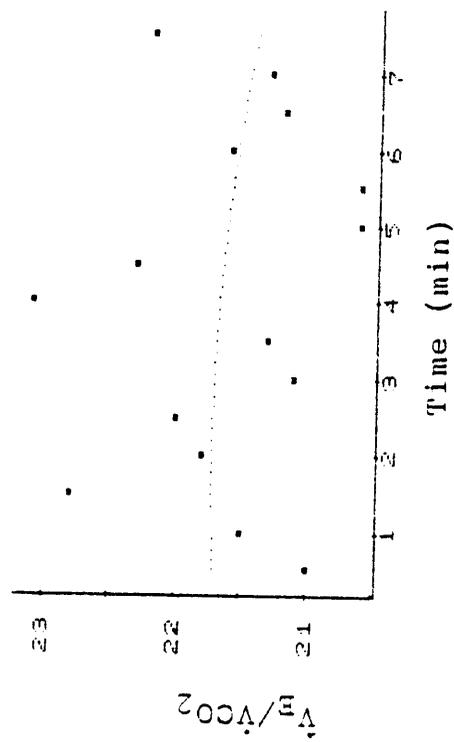
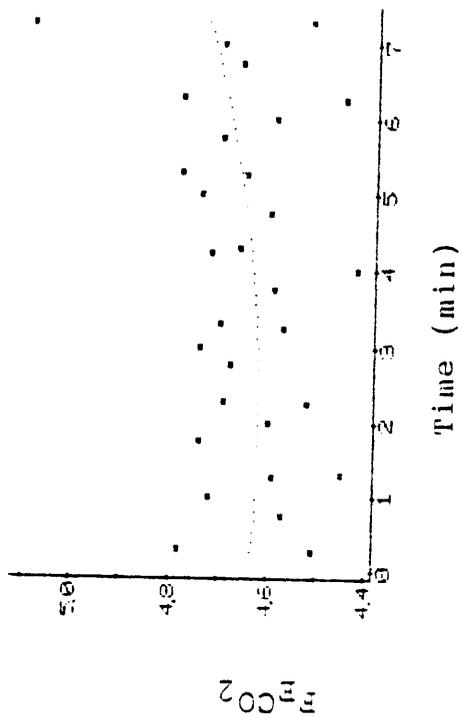
Subject 6
GXT I



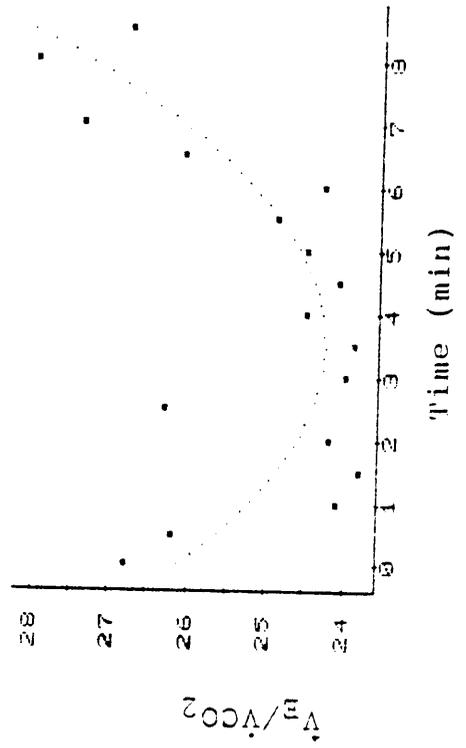
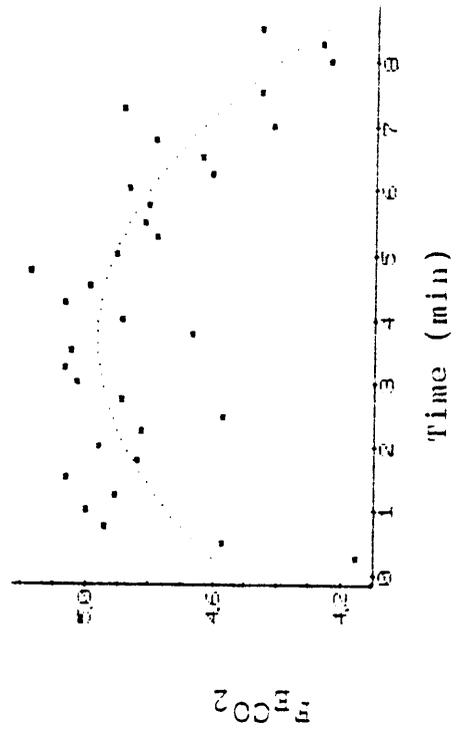
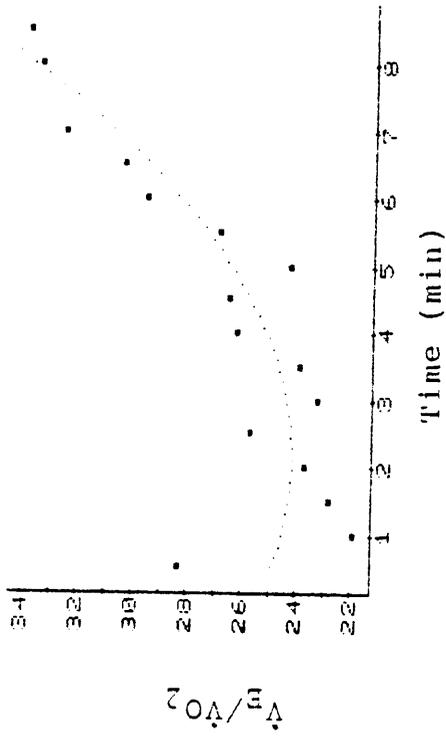
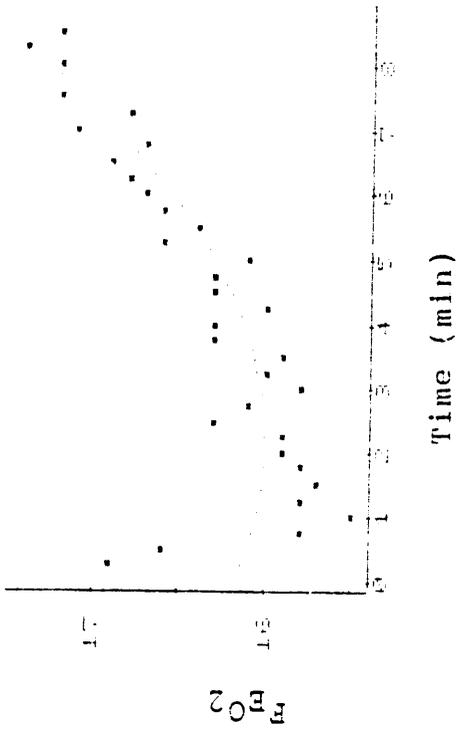
Subject 6
GXT2



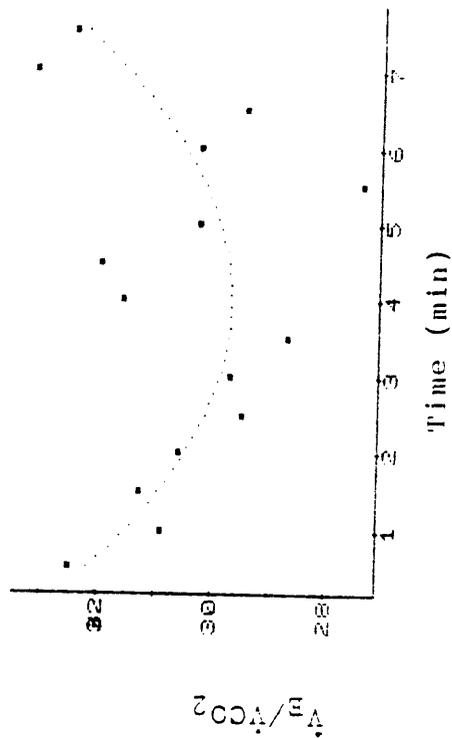
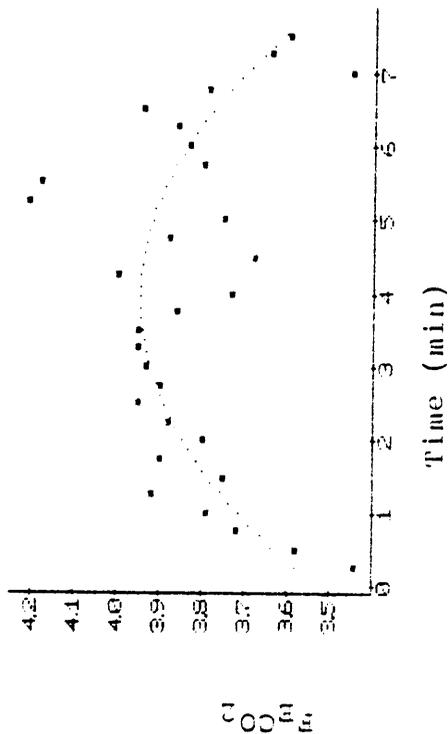
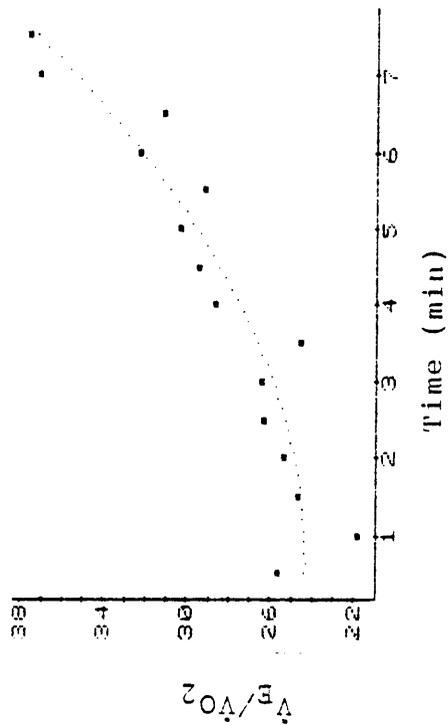
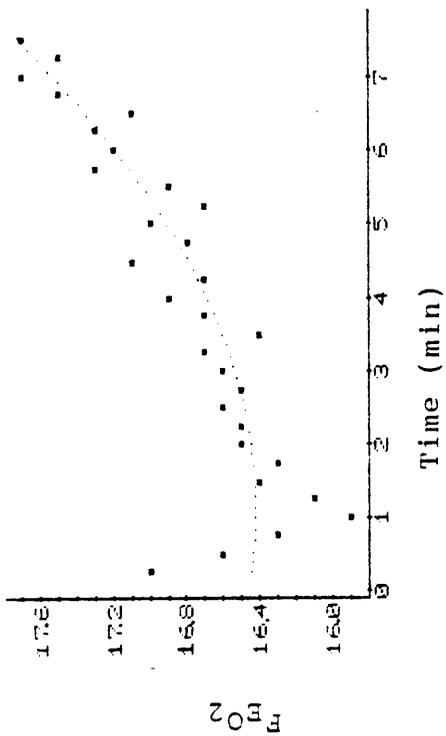
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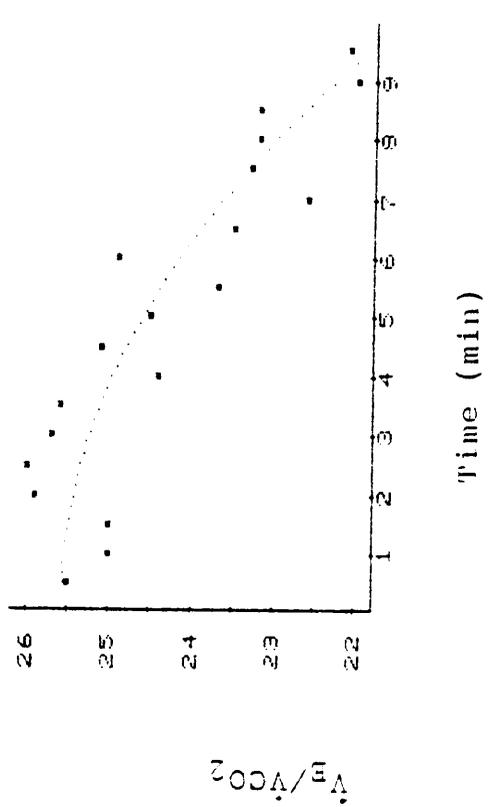
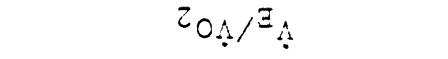
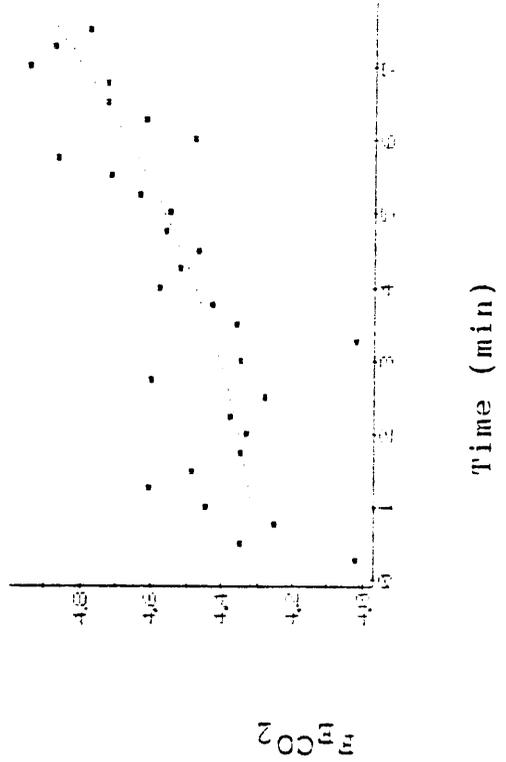
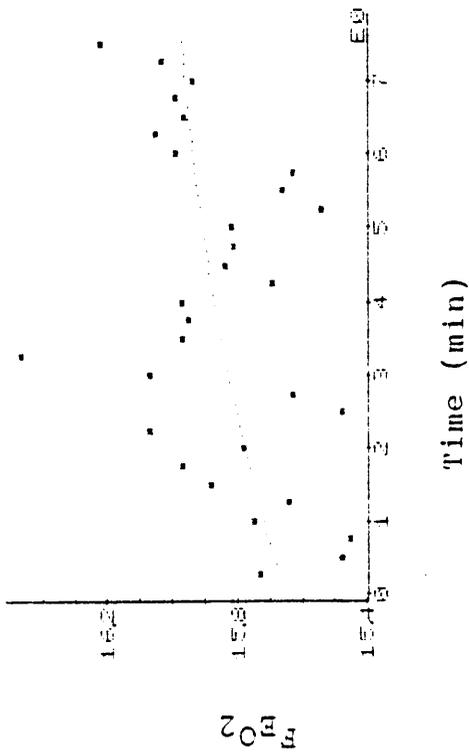
Subject 6
GXT3



Subject 6
GXT14

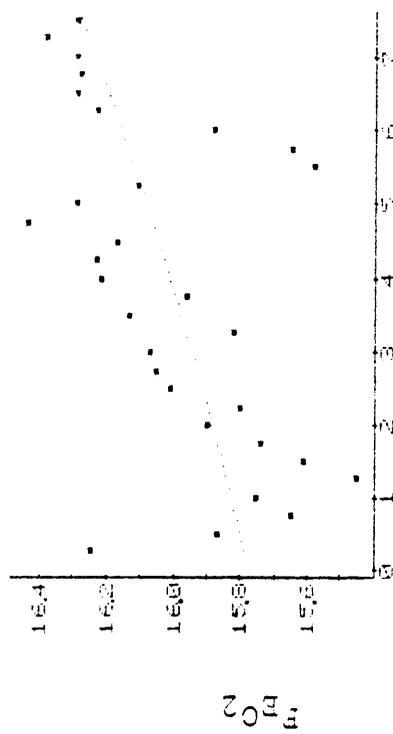


Subject 7
GXT1

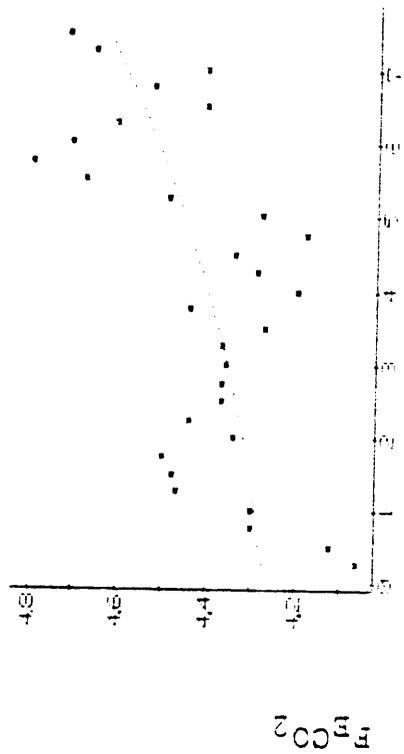


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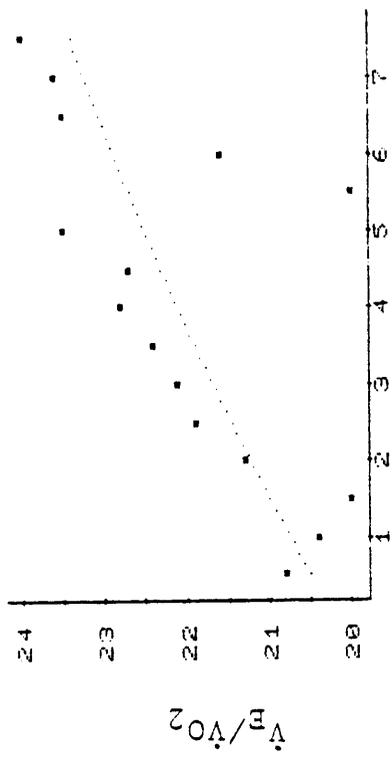
Subject 7
GXT2



Time (min)

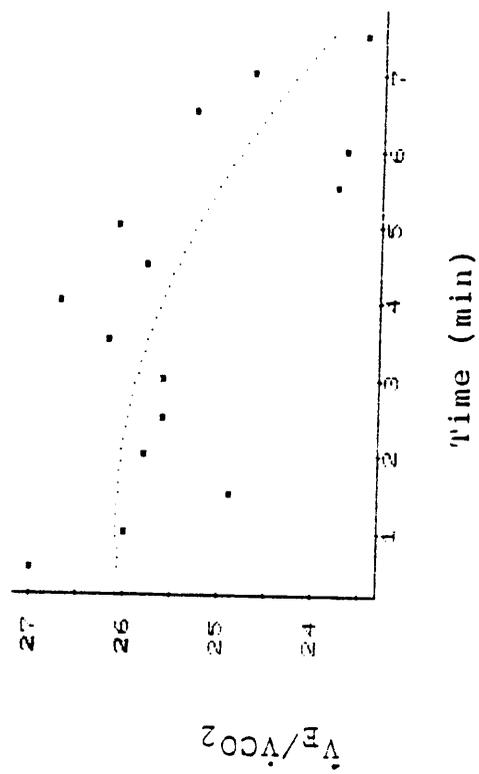


Time (min)



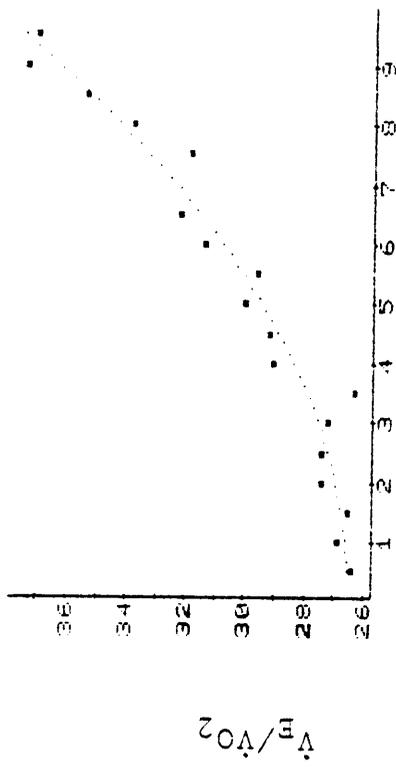
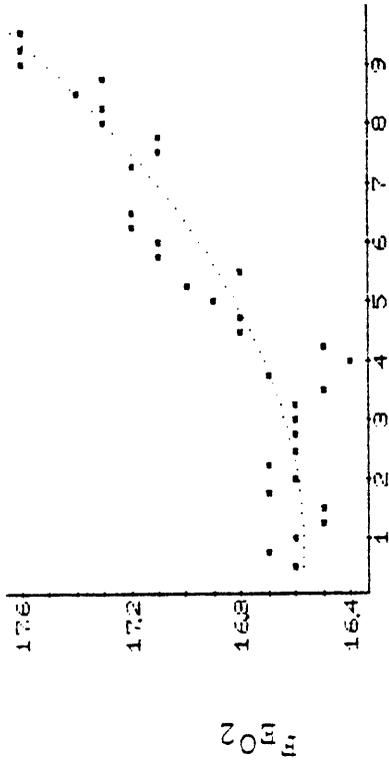
Time (min)

245



Time (min)

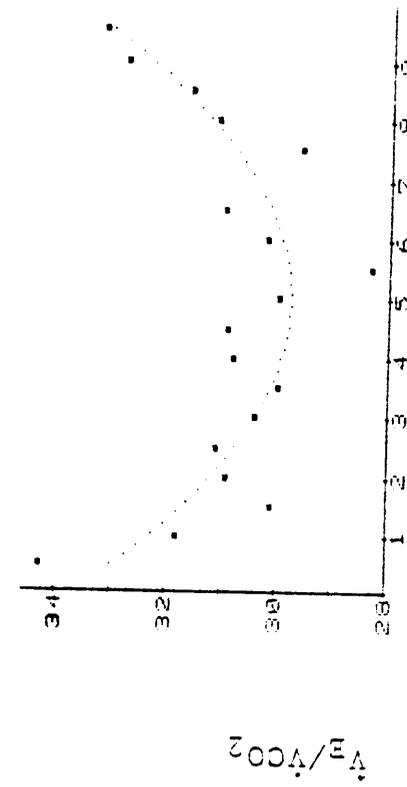
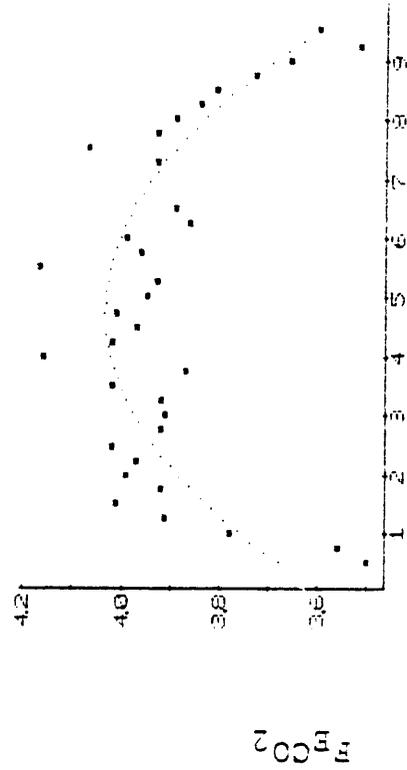
Subject 7
GXT3



Time (min)

Time (min)

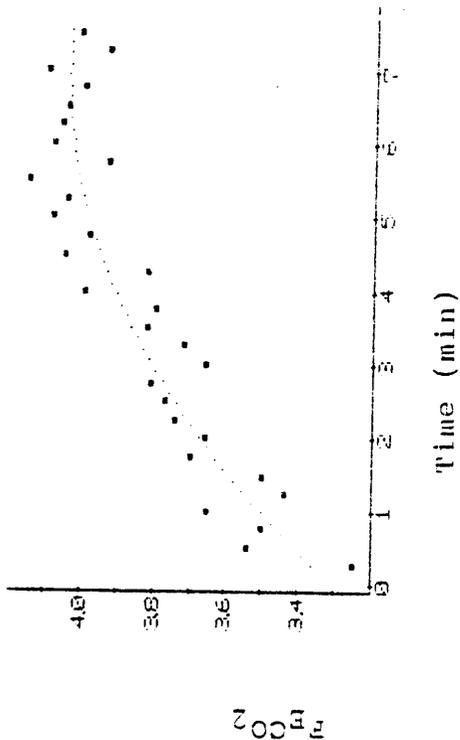
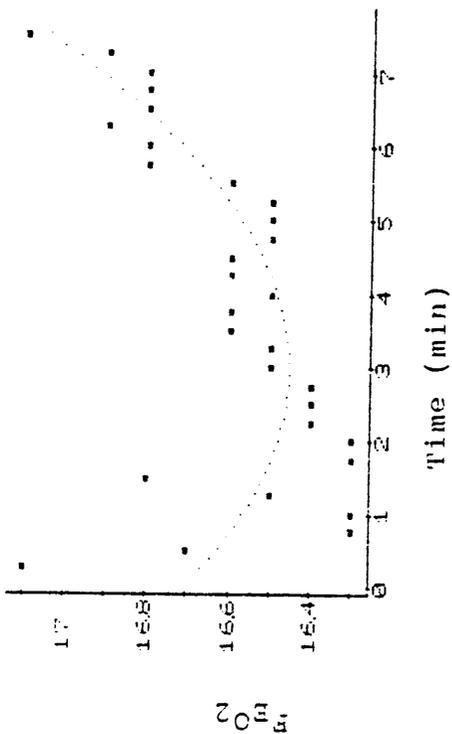
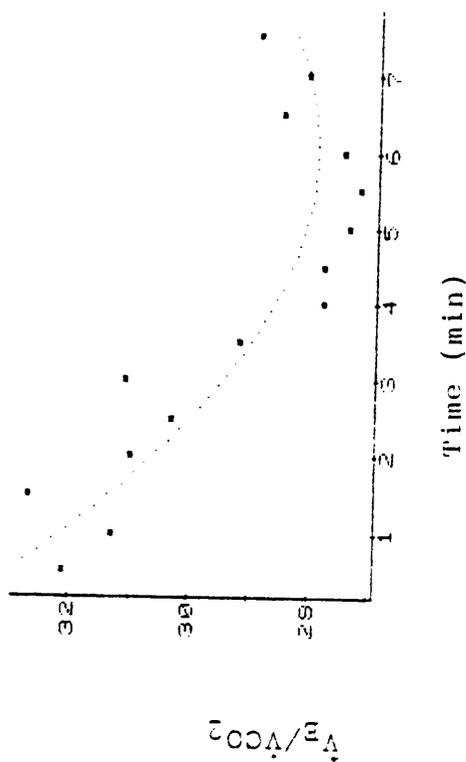
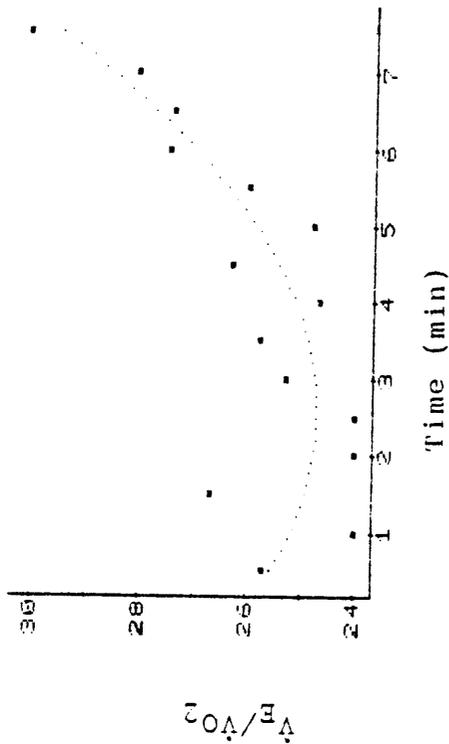
246



Time (min)

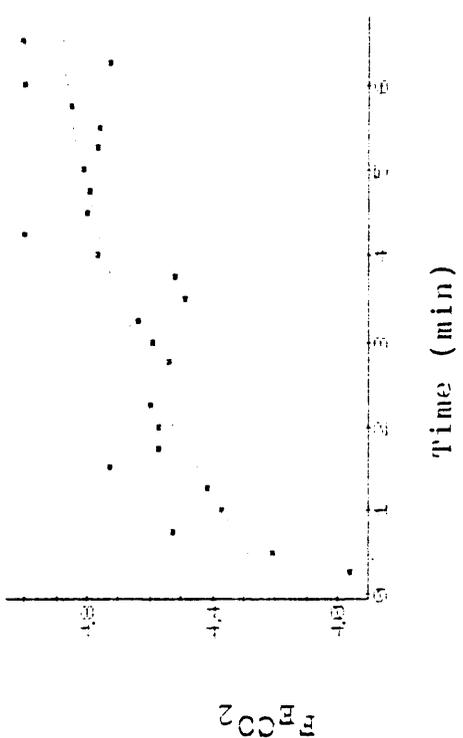
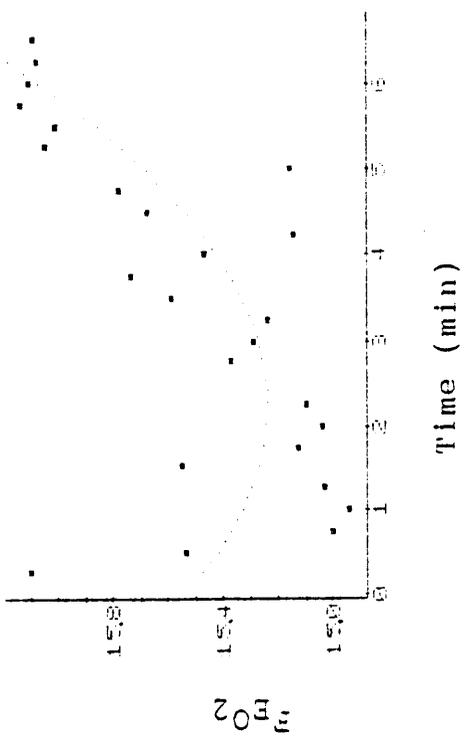
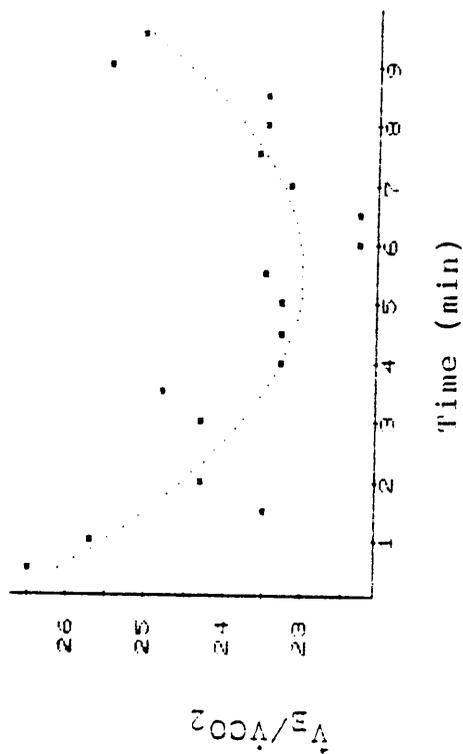
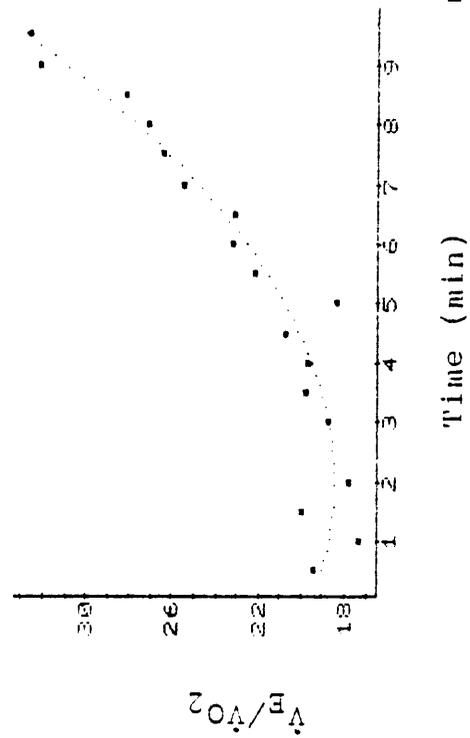
Time (min)

Subject 7
GXT₄

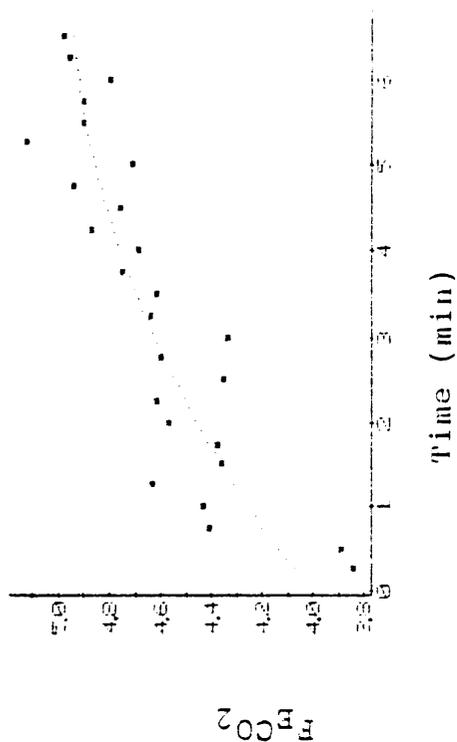
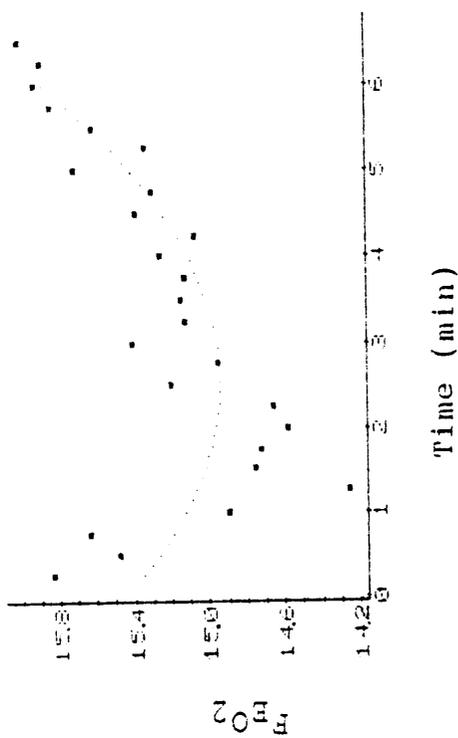
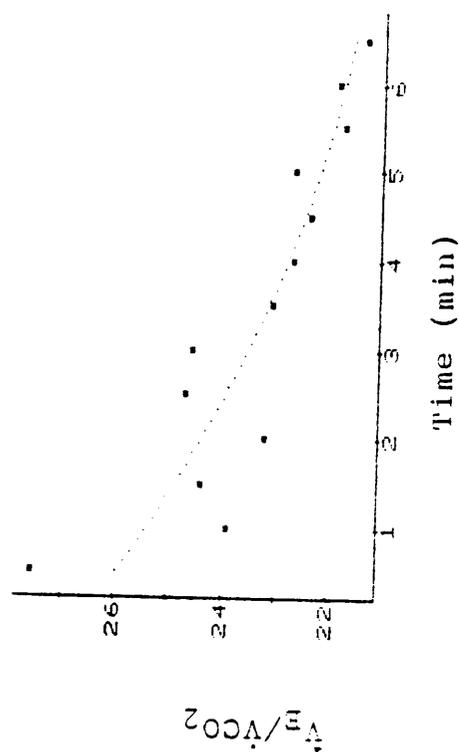
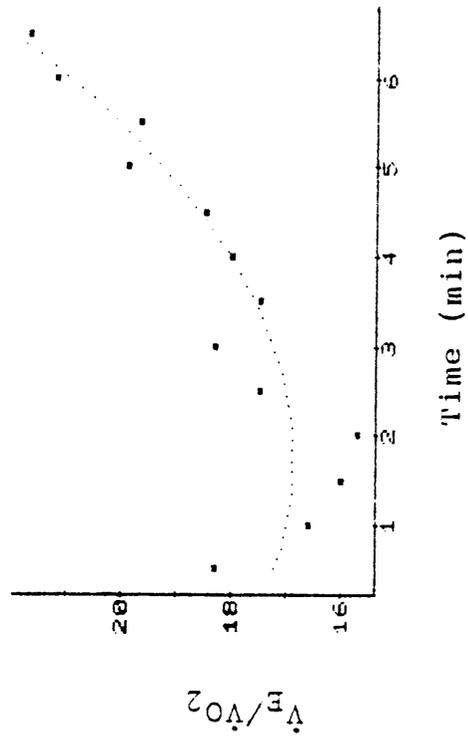


Subject 8
GXT1

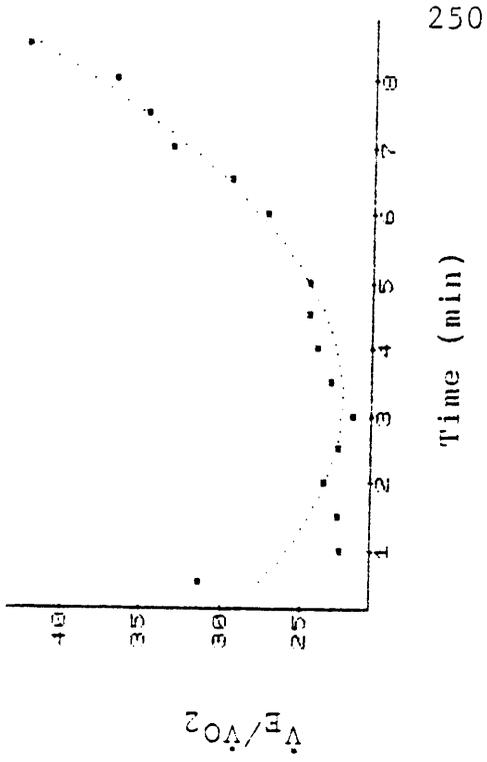
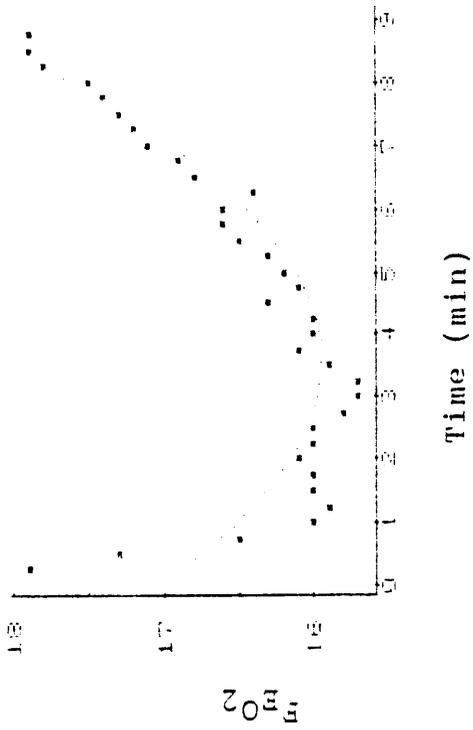
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Subject 8
GXT2

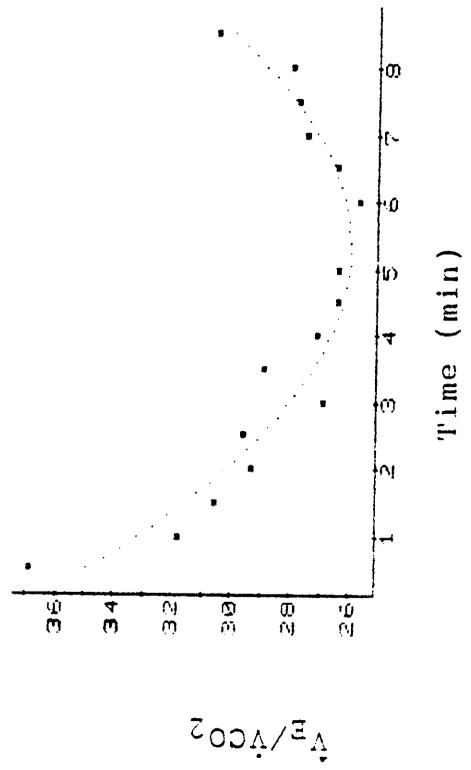
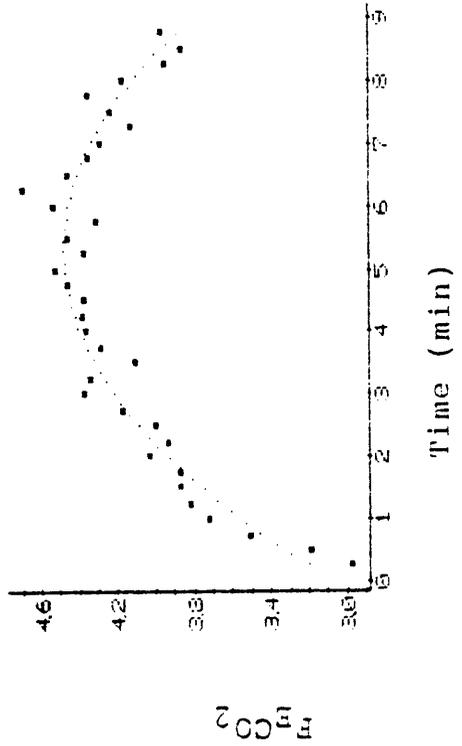


Subject 8
GXT3



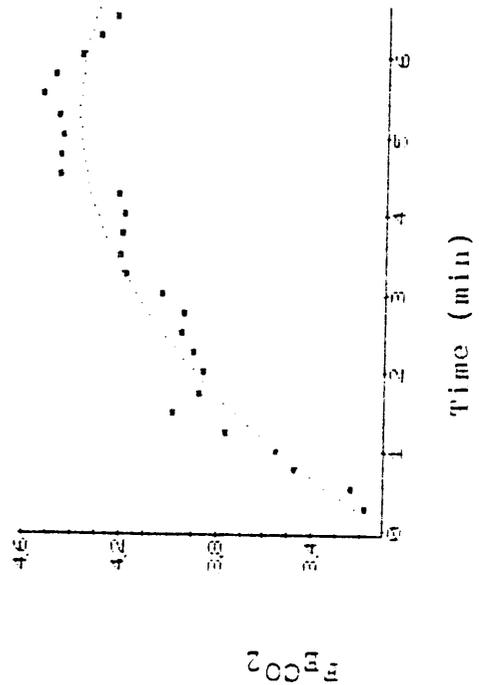
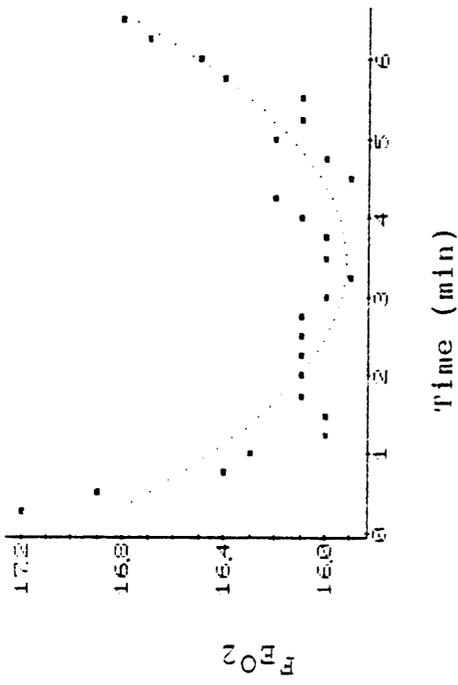
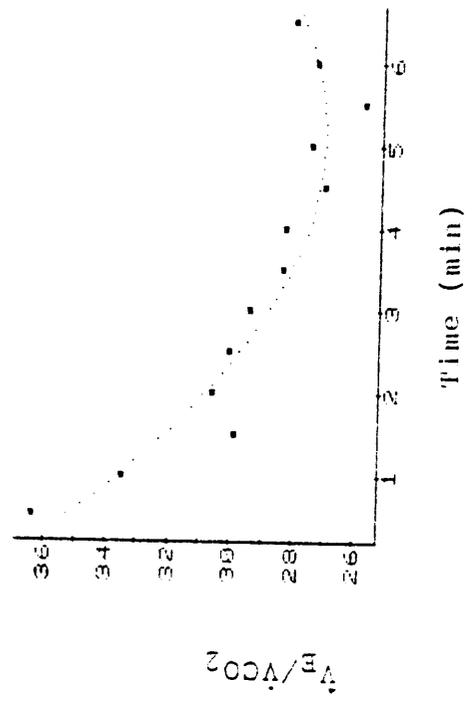
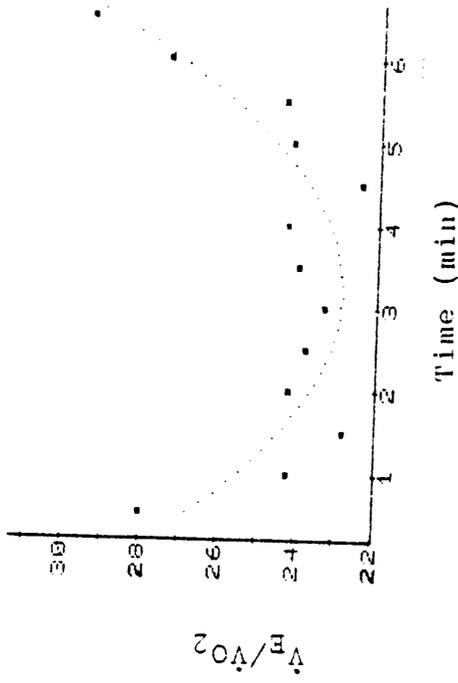
Time (min)

250

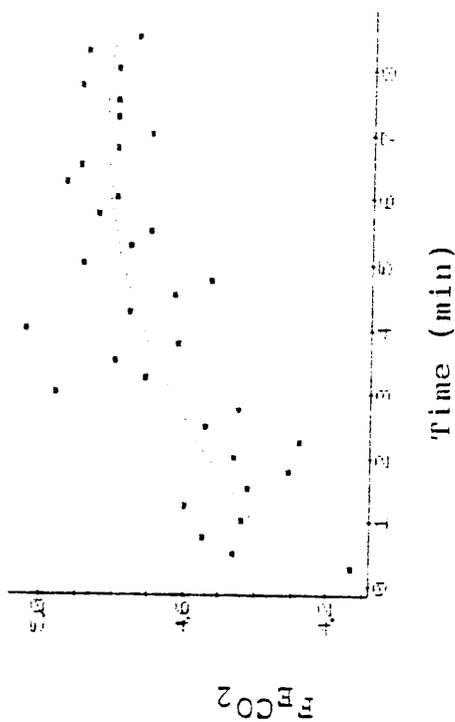
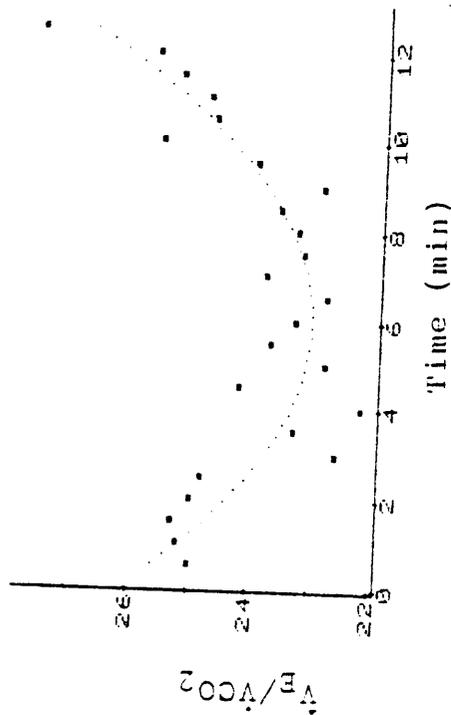
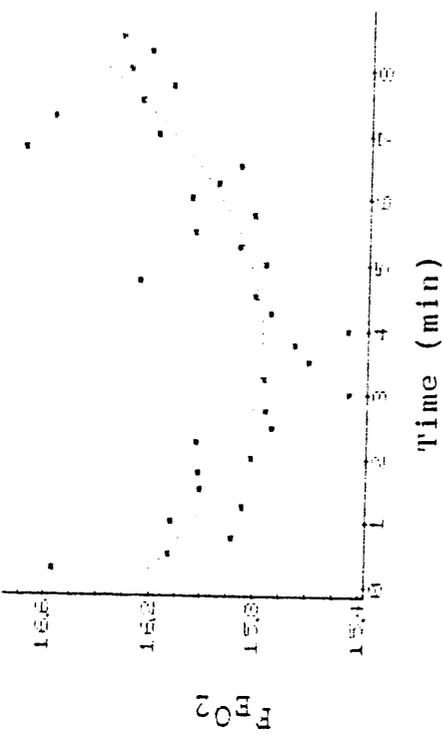


Time (min)

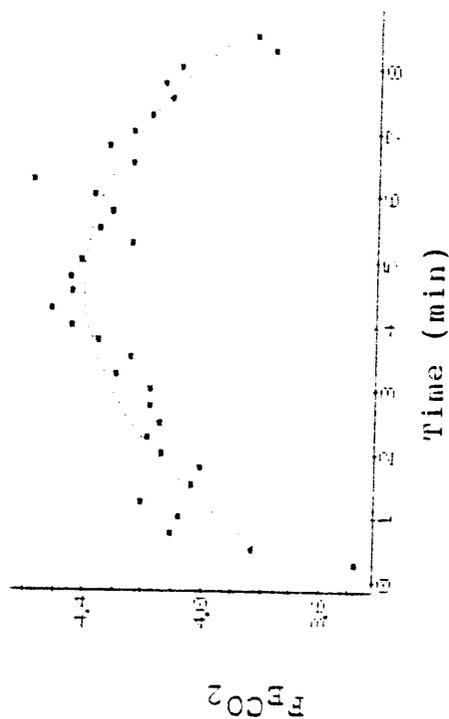
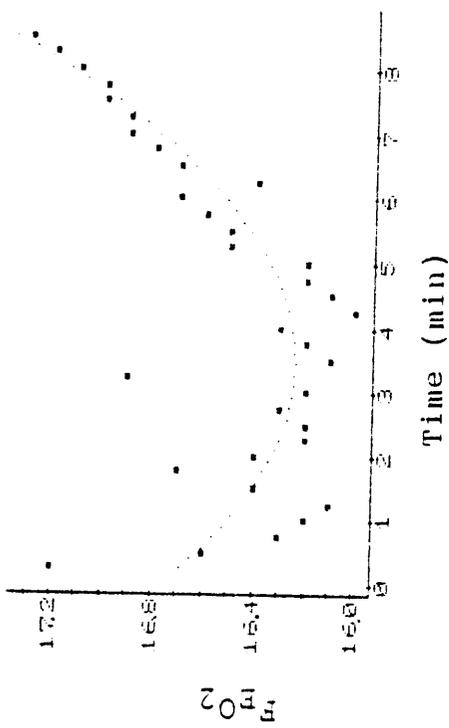
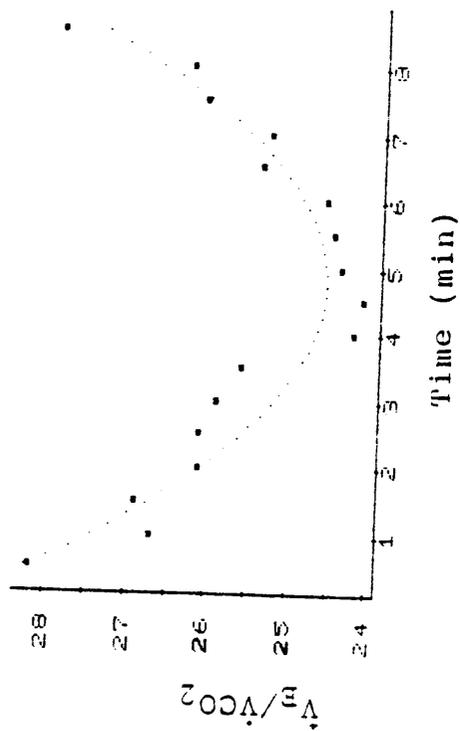
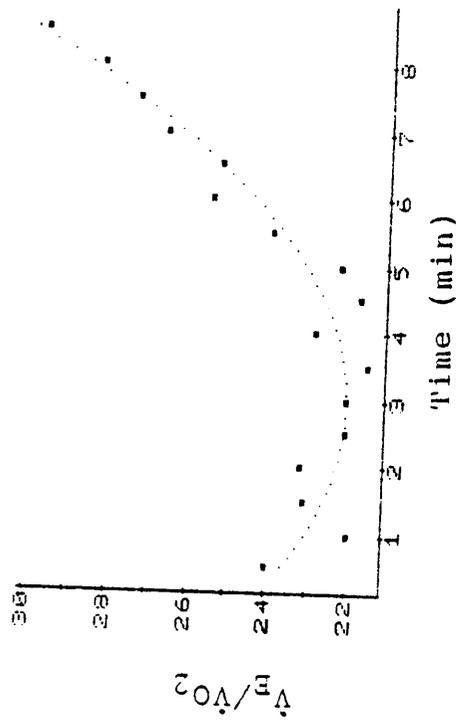
Subject 8
GXT₁



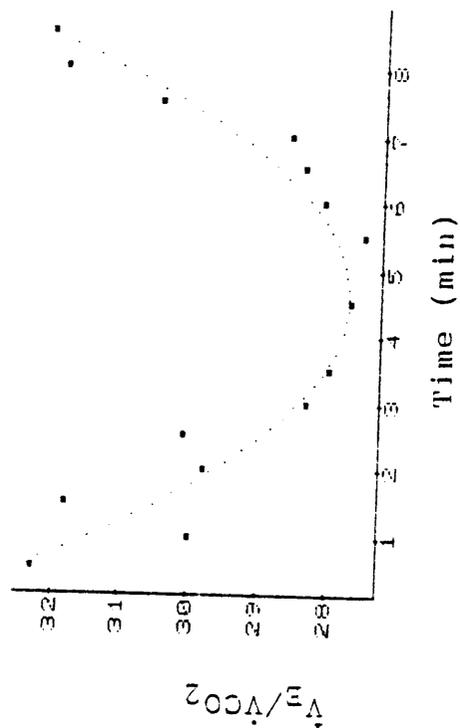
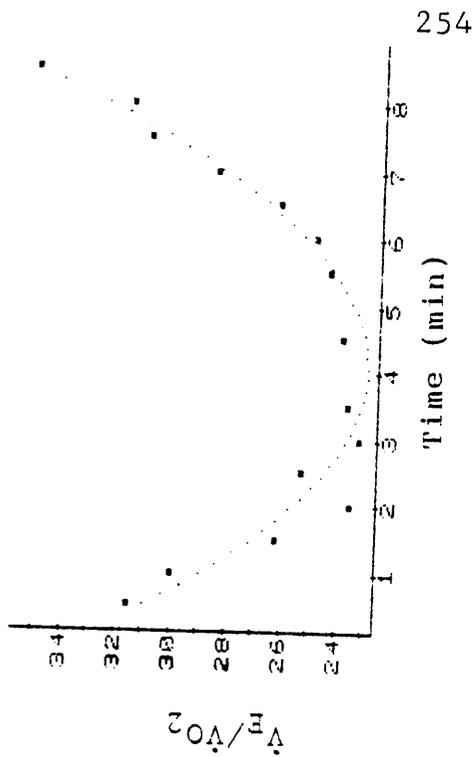
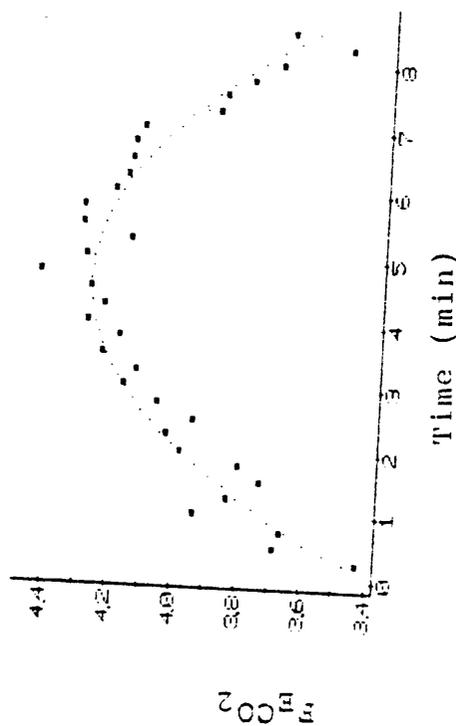
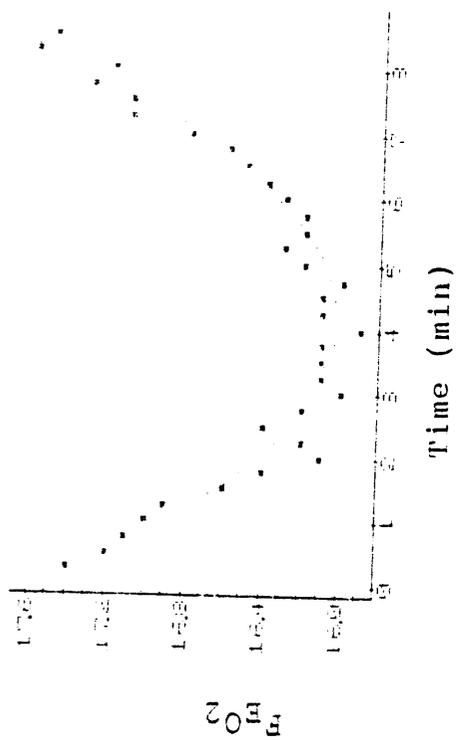
Subject 9
GXT I



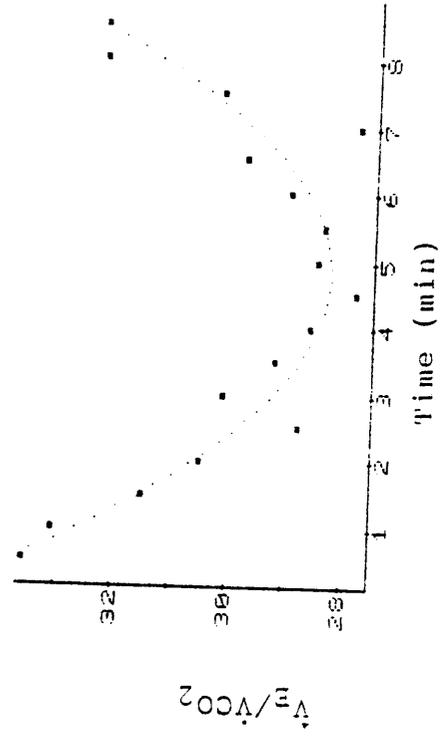
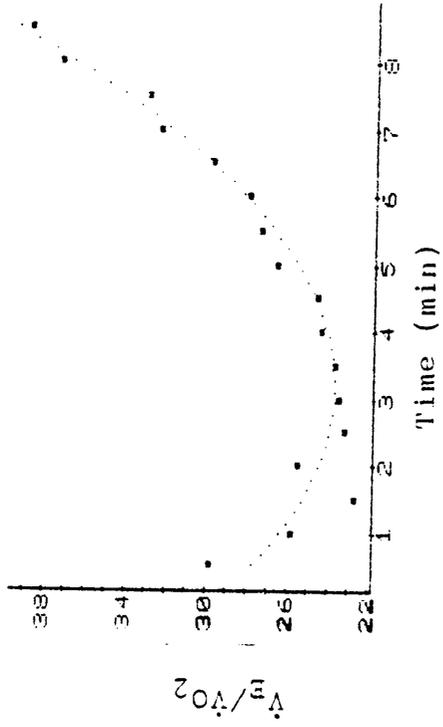
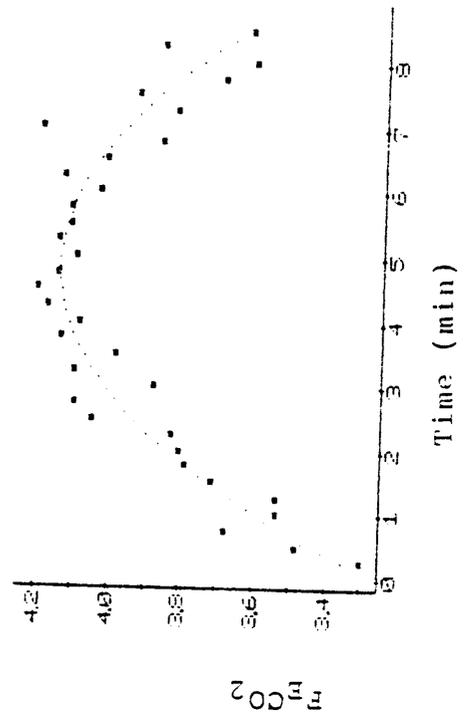
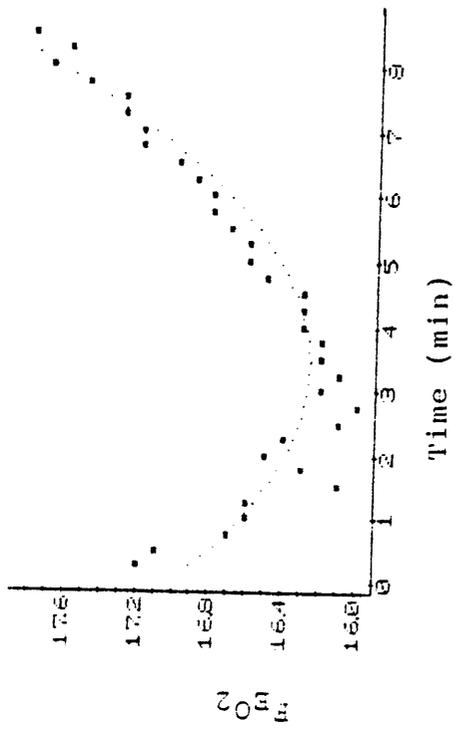
Subject 9
GXT'2



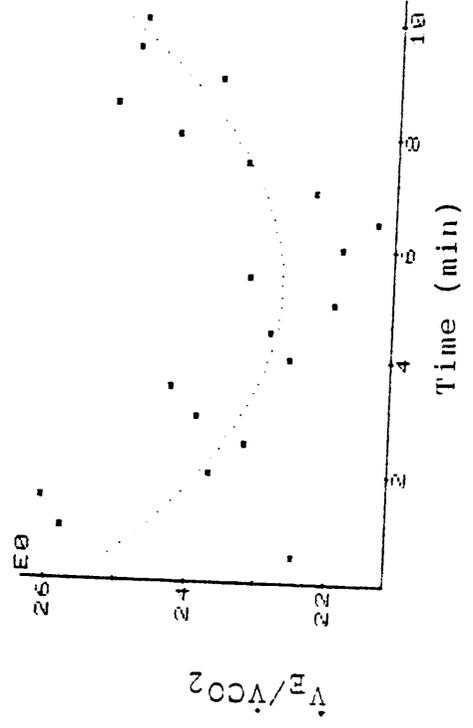
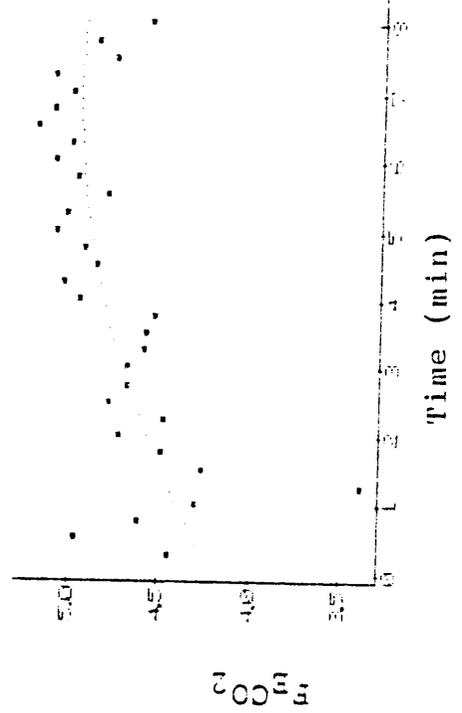
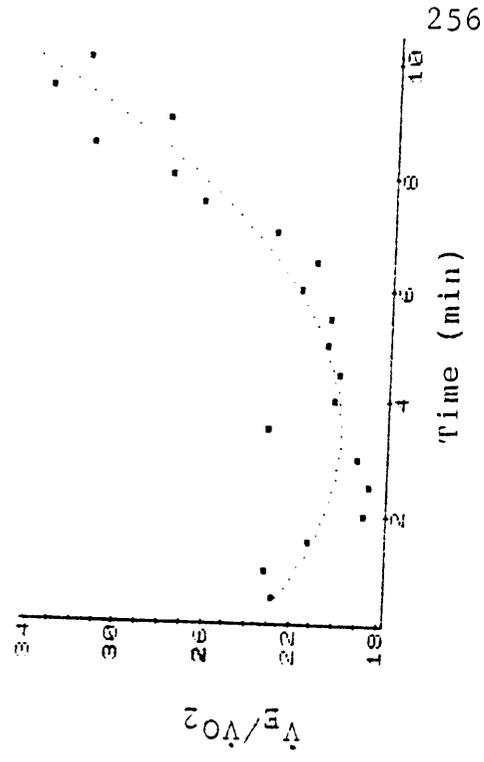
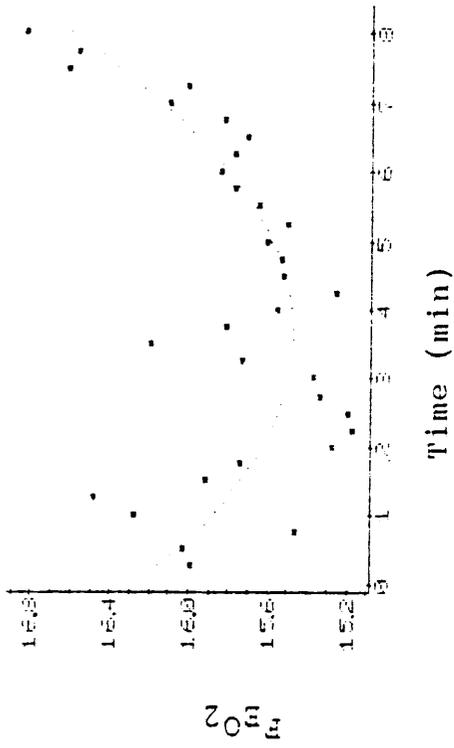
Subject 9
GXT3



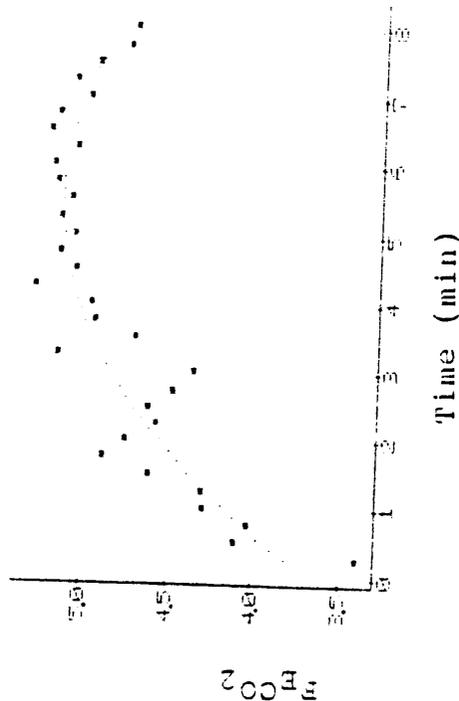
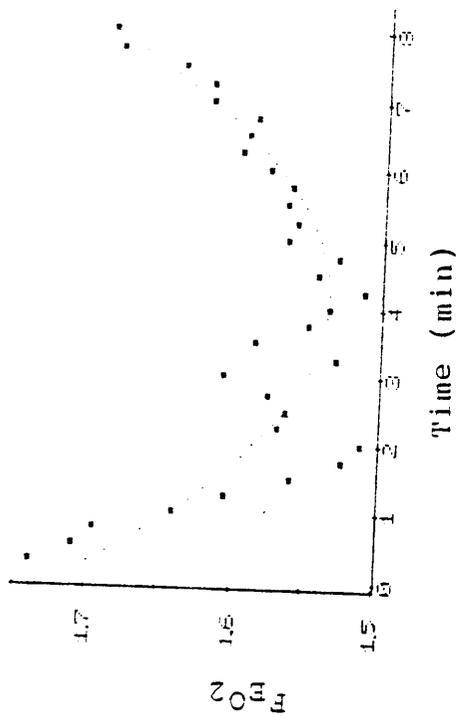
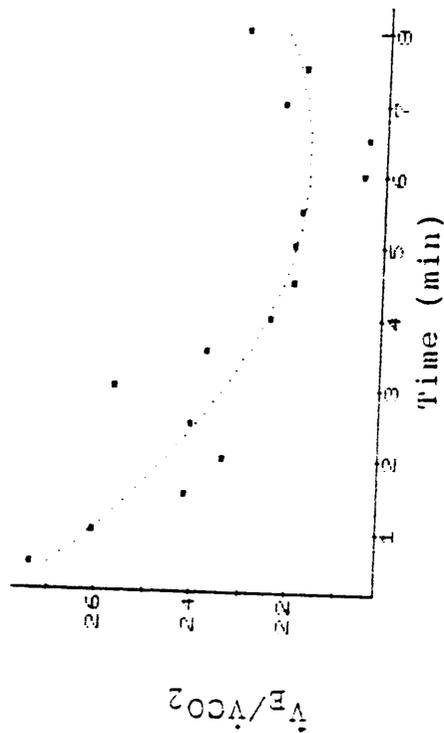
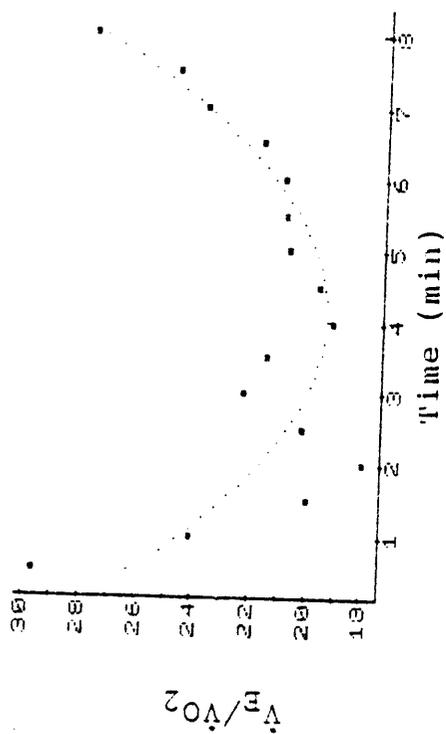
Subject 9
GXT4



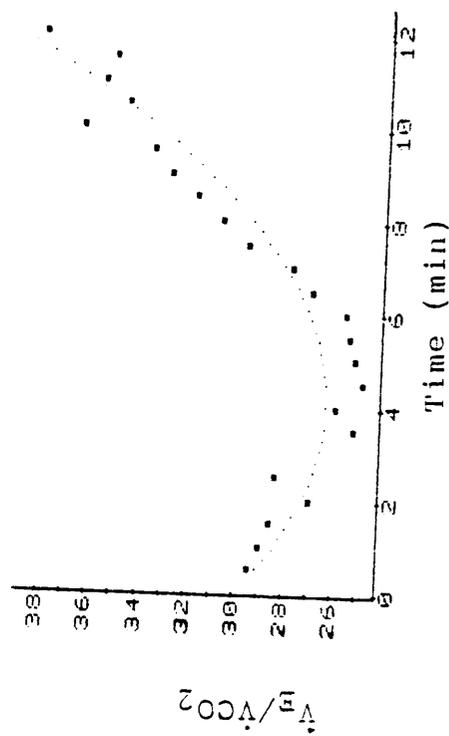
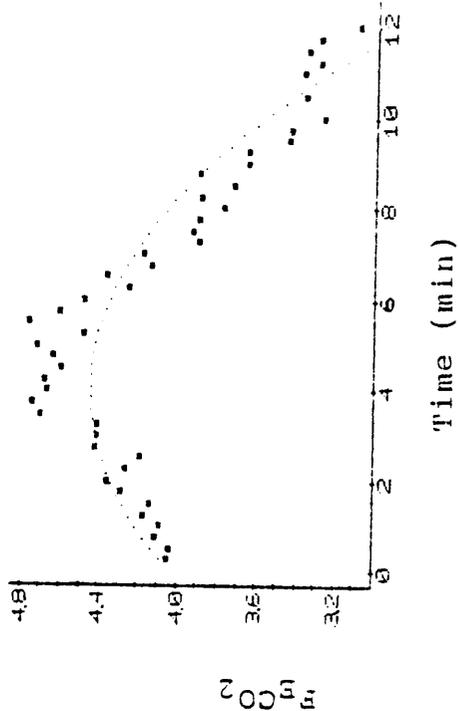
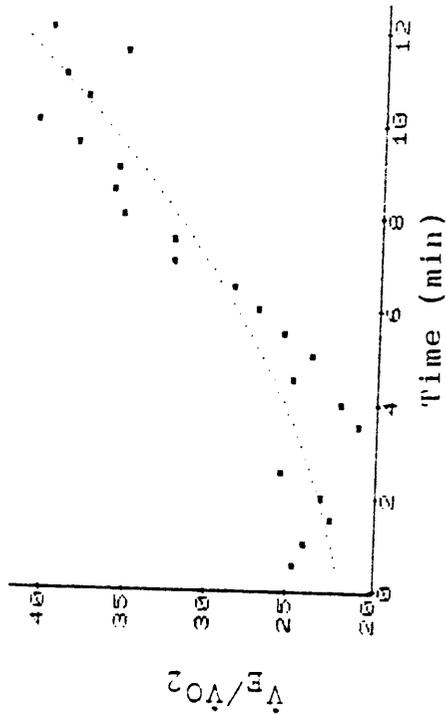
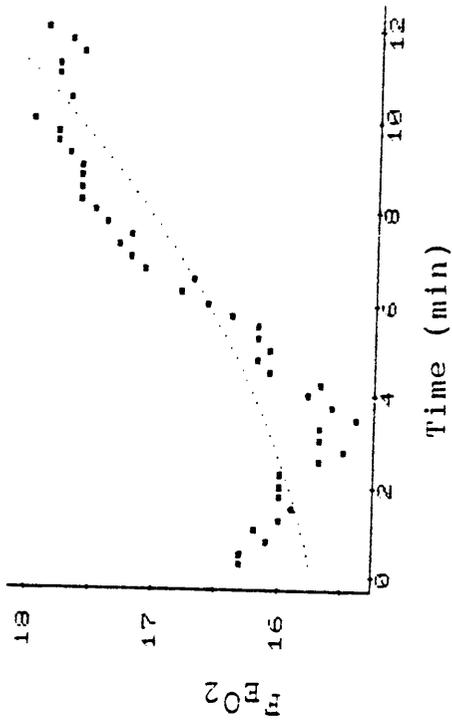
Subject 10
GXT1



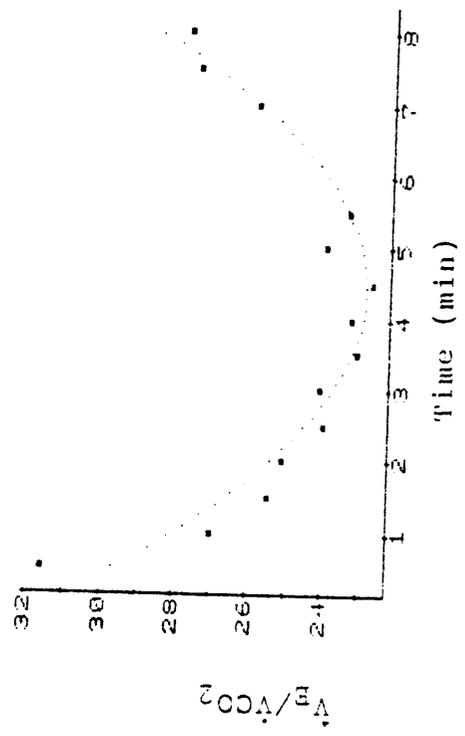
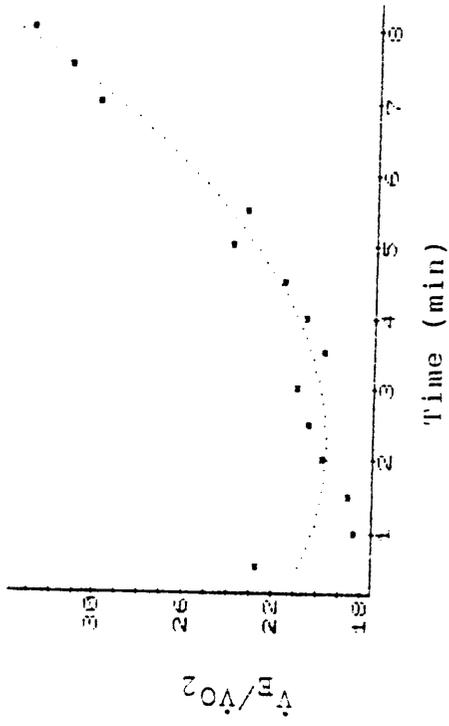
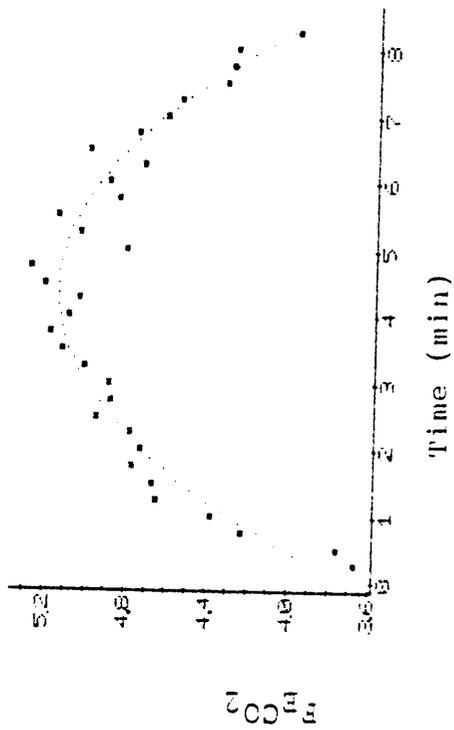
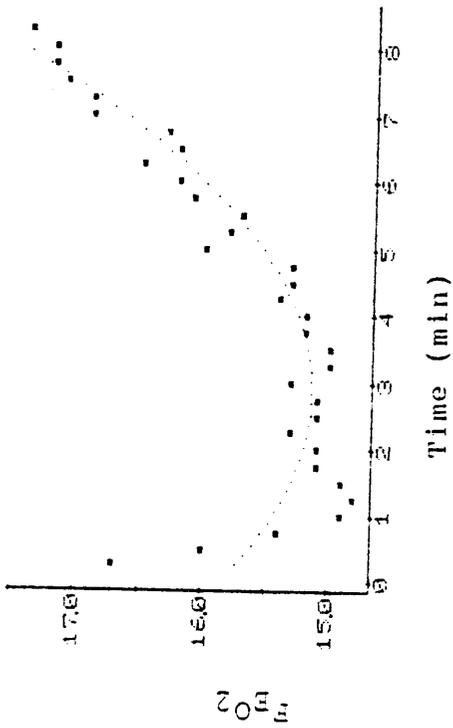
Subject 10
GXT2



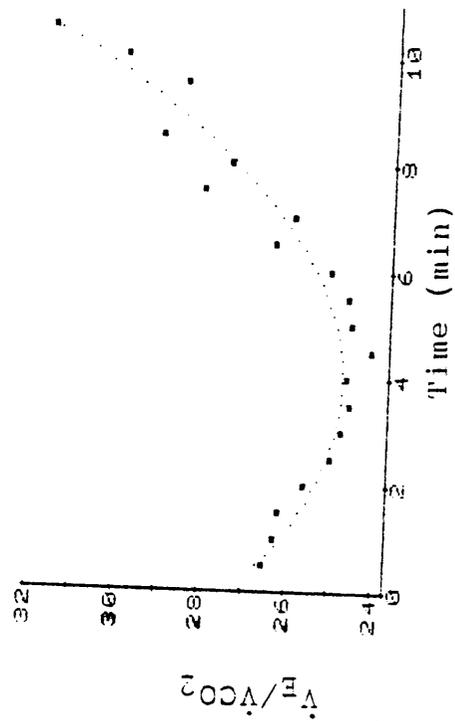
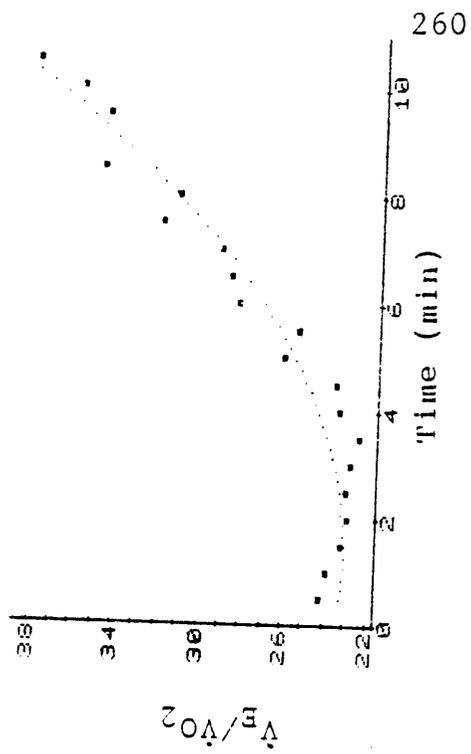
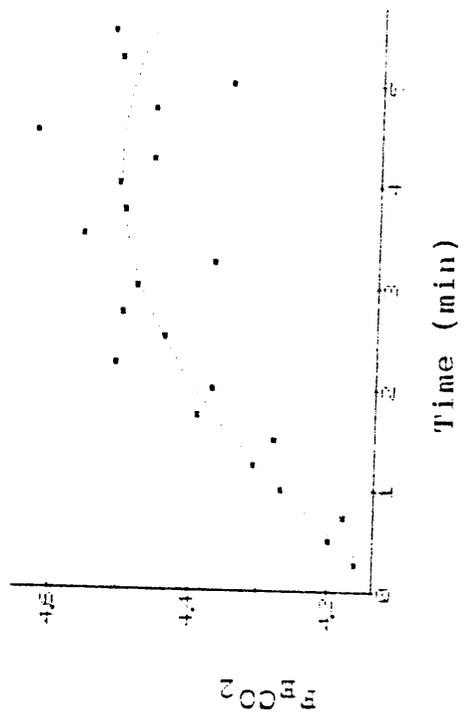
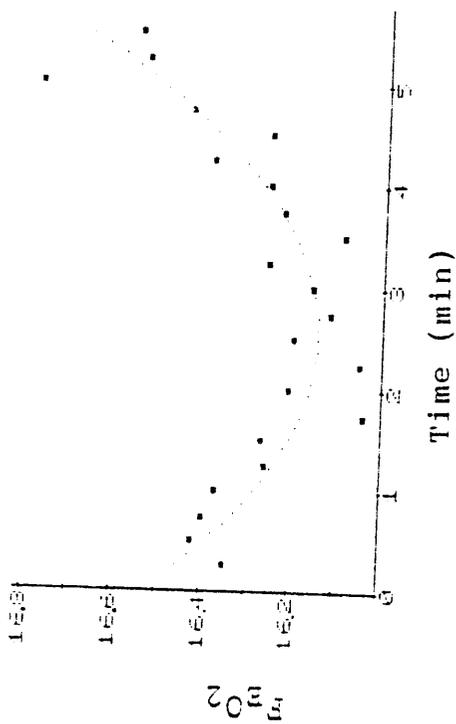
Subject 10
GXT3



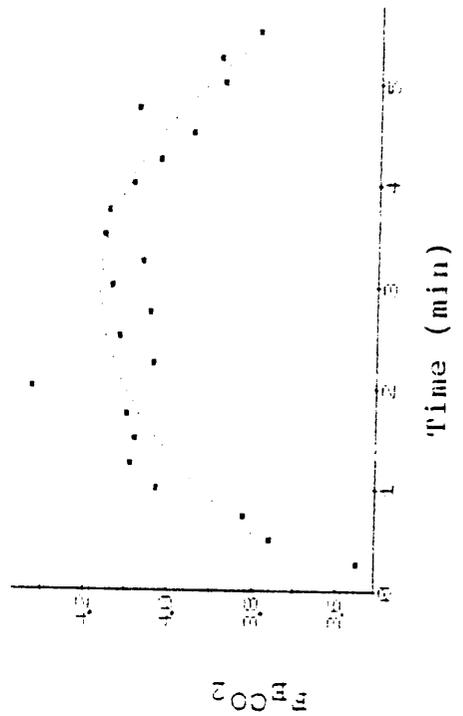
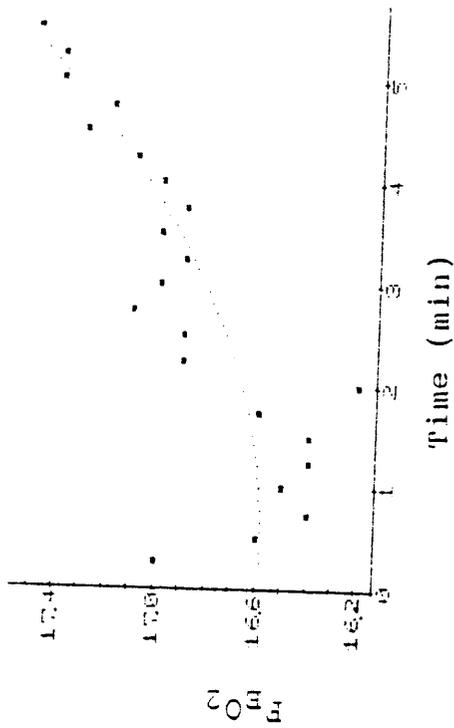
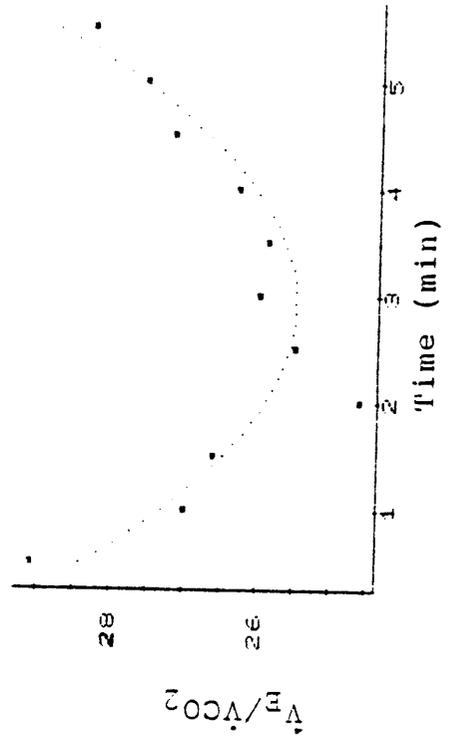
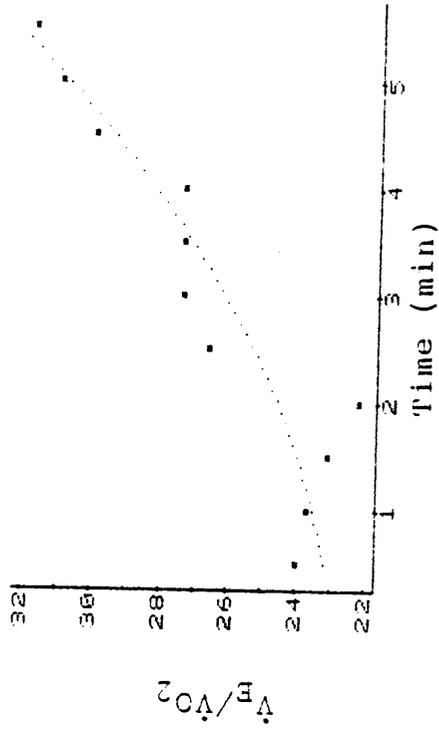
Subject 10
GXT4



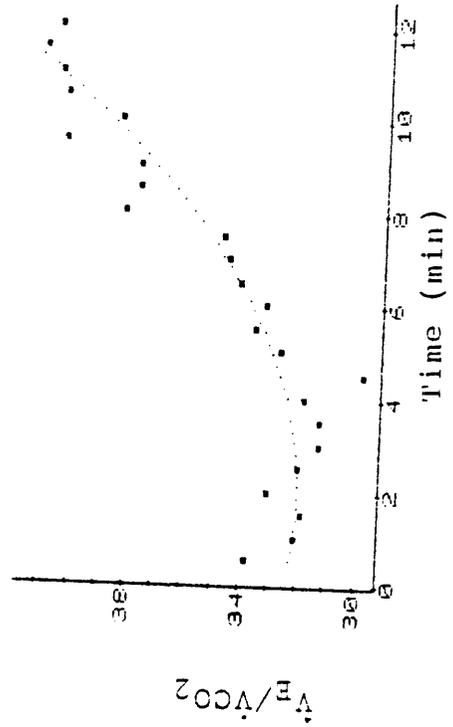
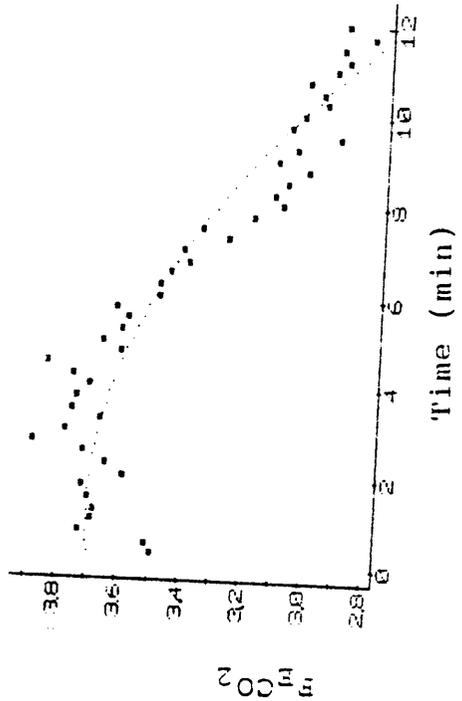
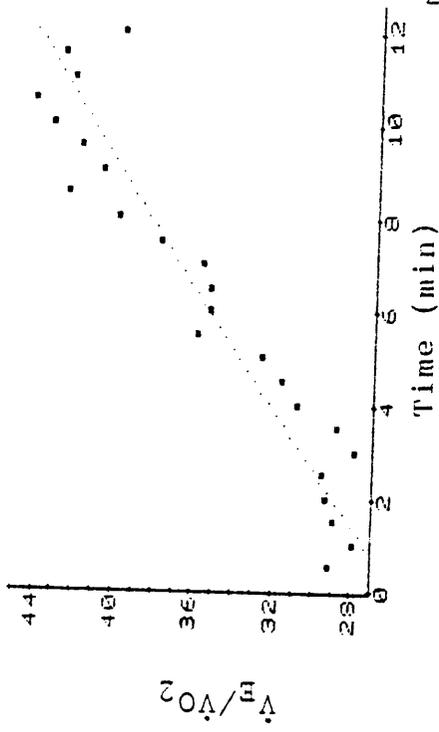
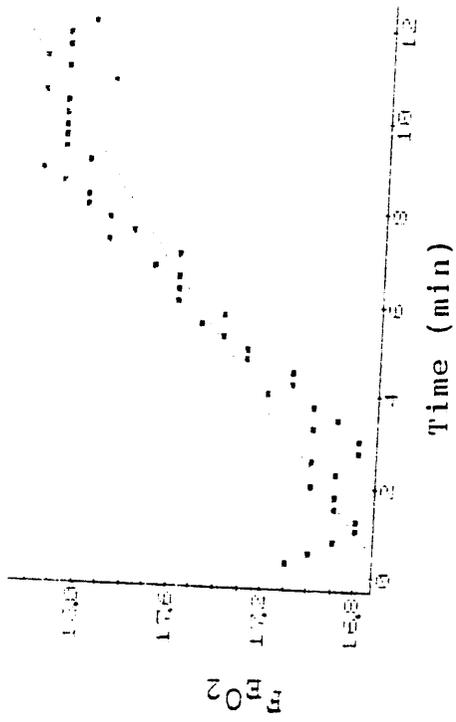
Subject 11
GXT1



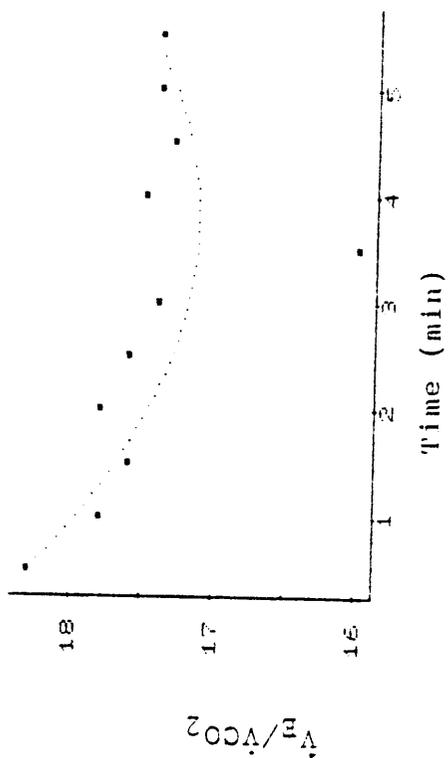
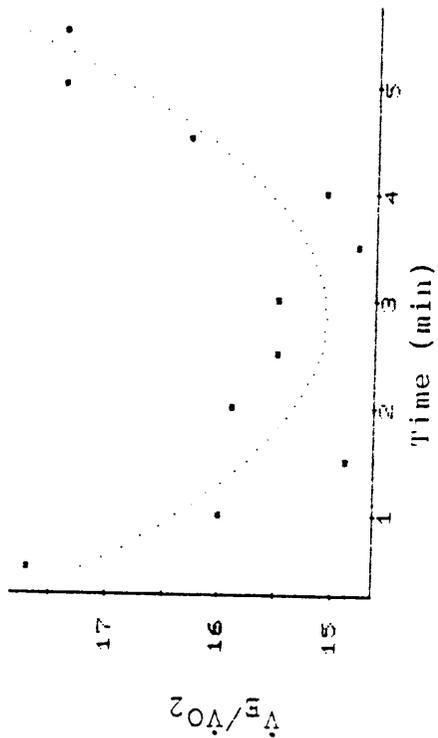
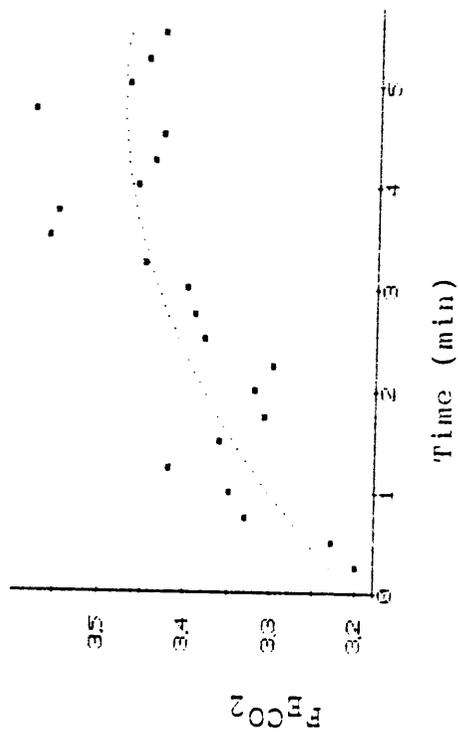
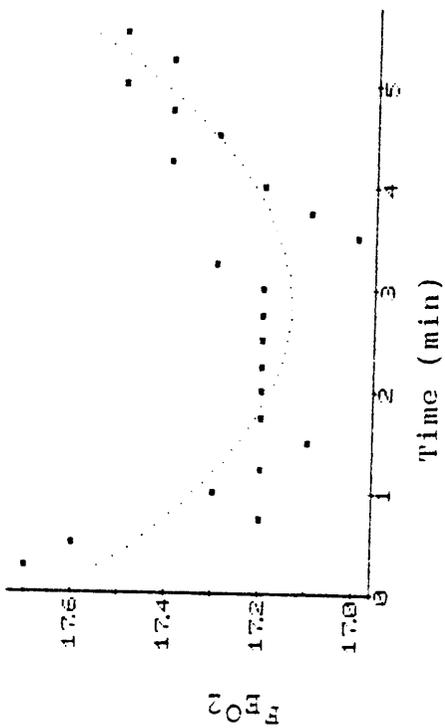
Subject 11
GXT'2



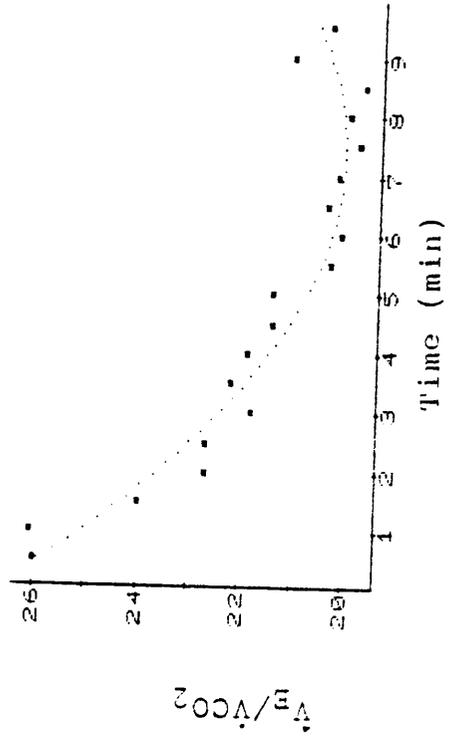
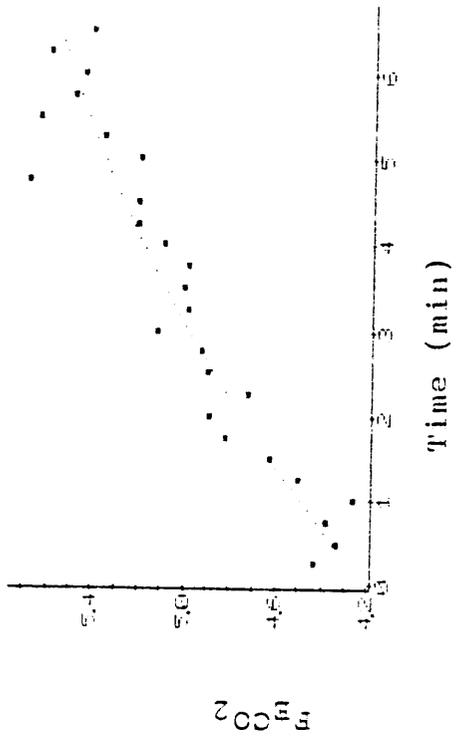
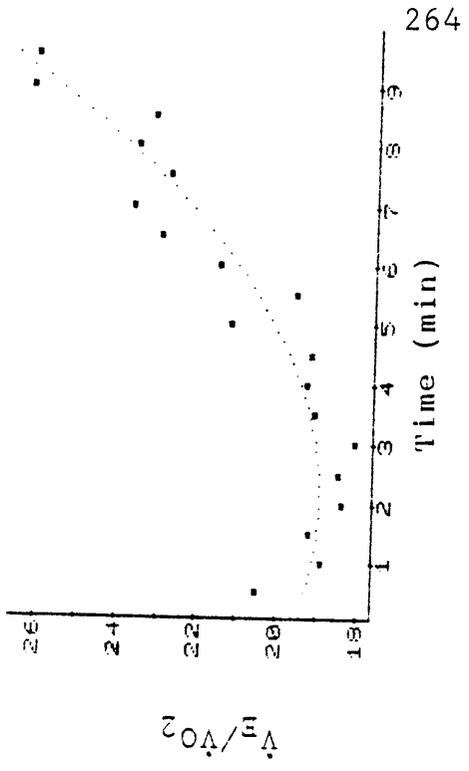
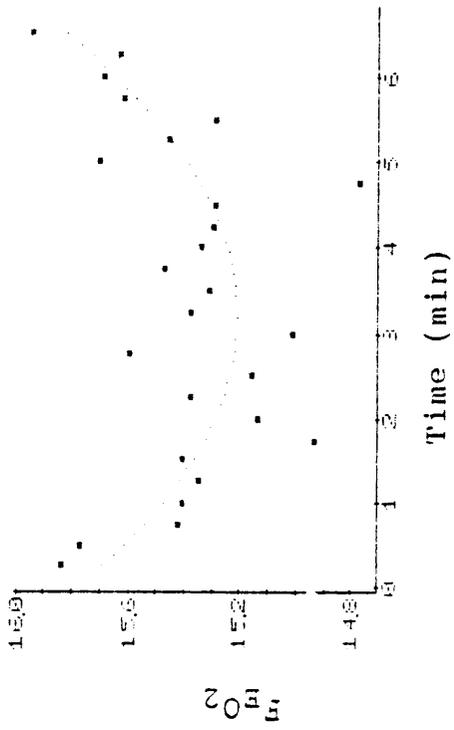
Subject 11
GXT3



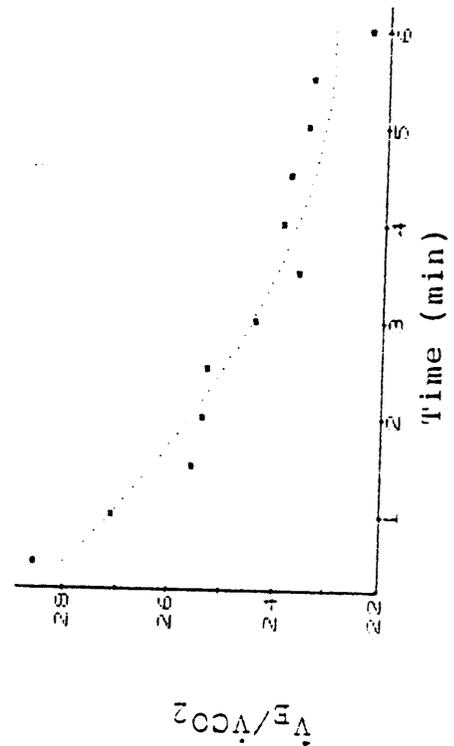
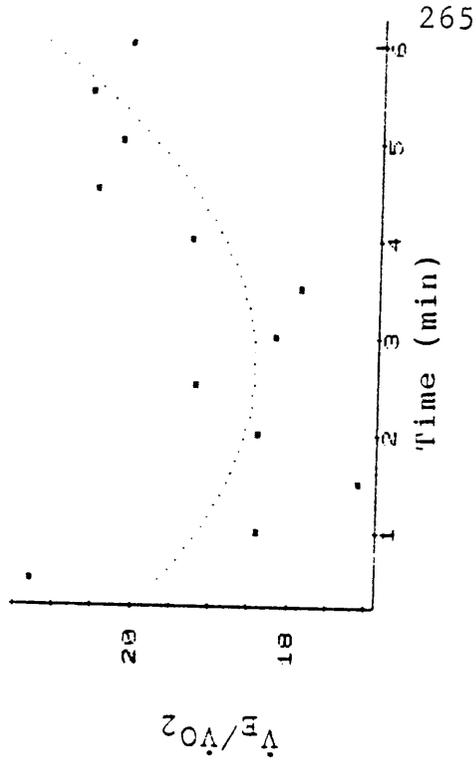
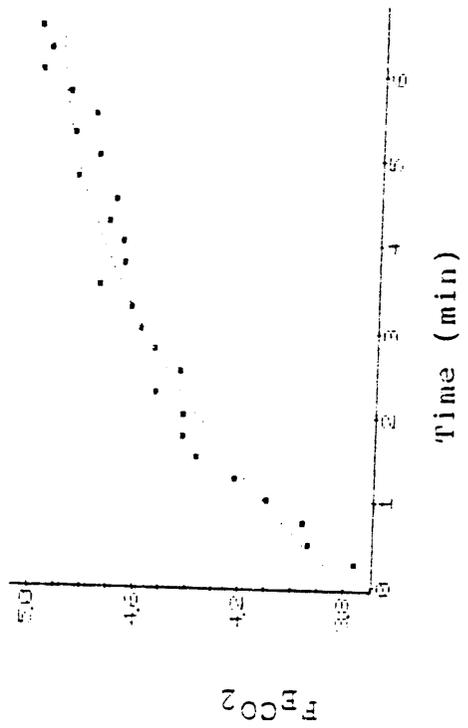
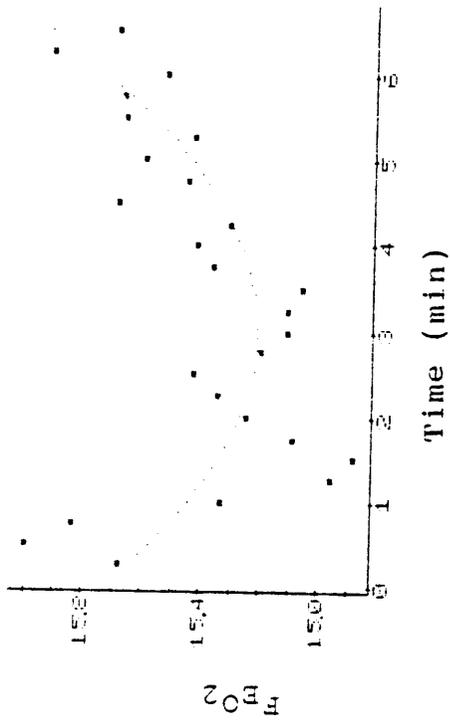
Subject 11
GXT₄



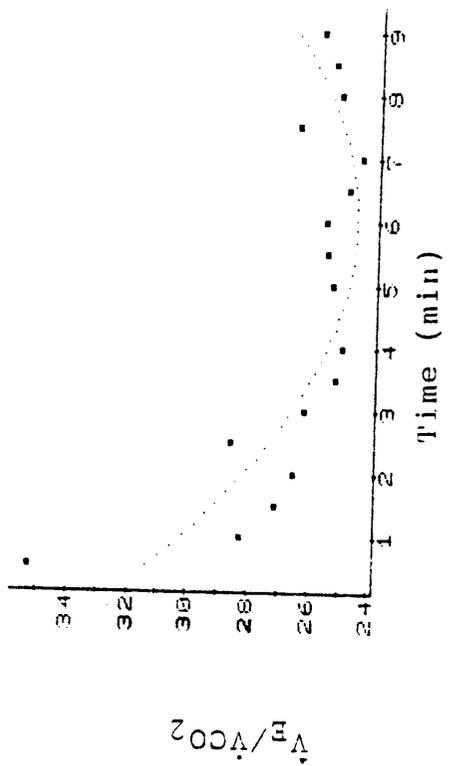
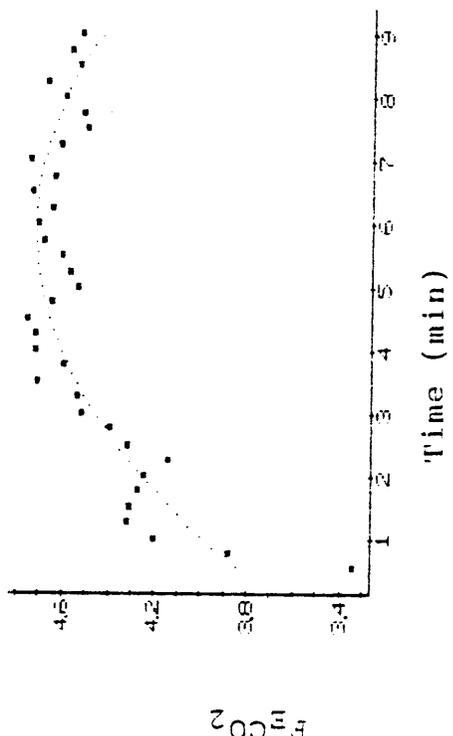
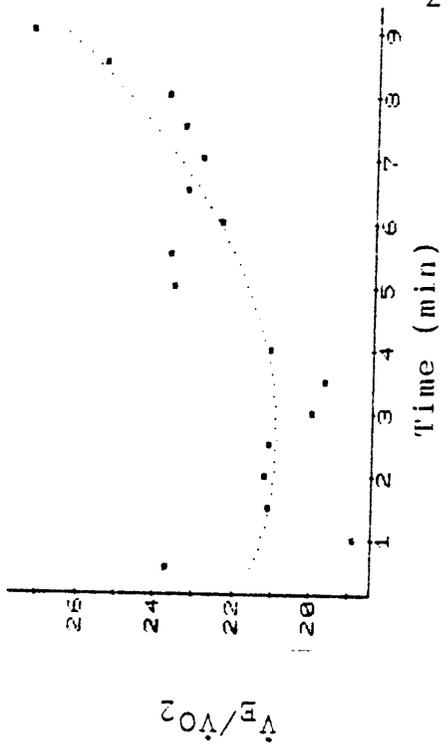
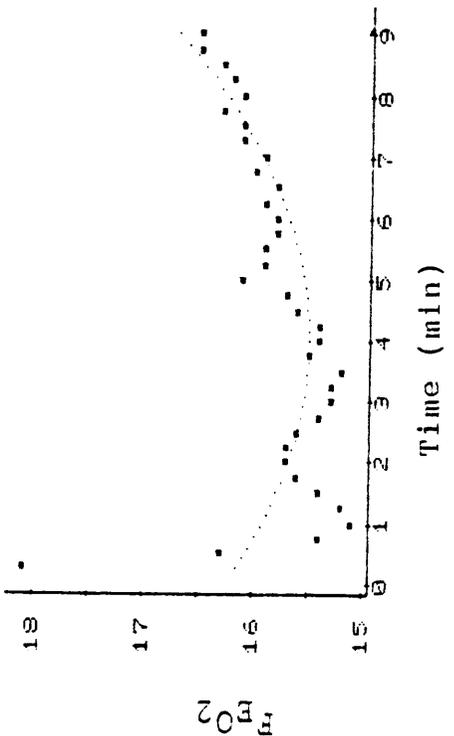
Subject 12
GXT I



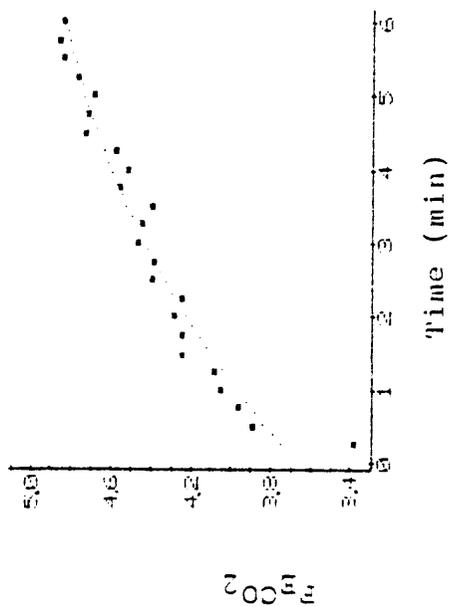
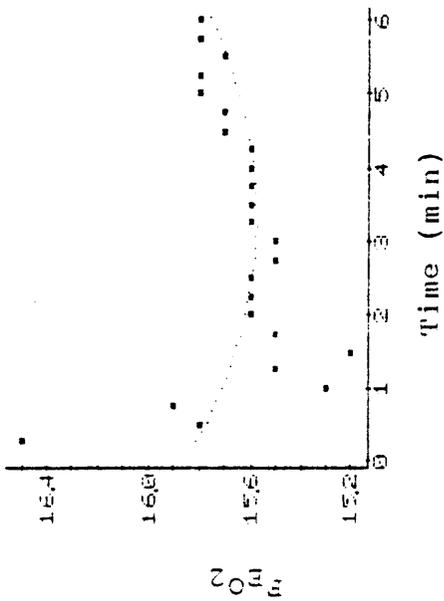
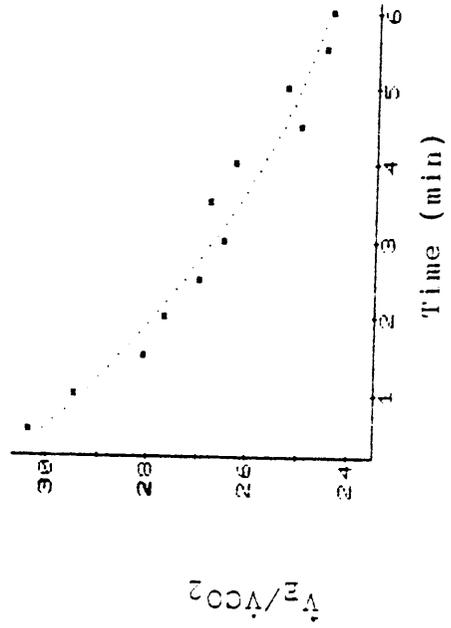
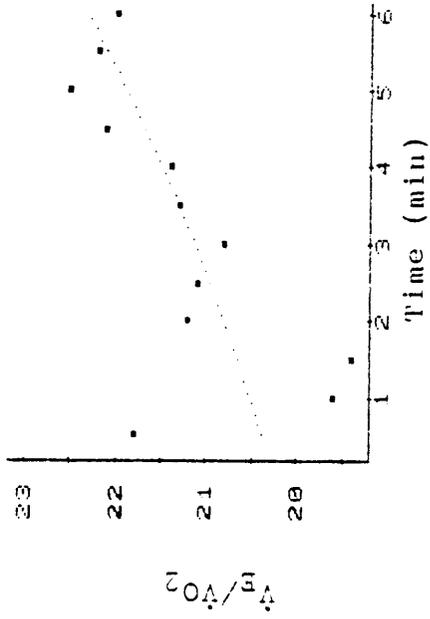
Subject 12
GXT2



Subject 12
GXT3



Subject 12
GXT₄



**The vita has been removed from
the scanned document**