

THE RELATIONSHIP OF INDIVIDUAL ANAEROBIC THRESHOLDS TO TOTAL, ALACTIC,
AND LACTIC OXYGEN DEBTS AFTER A SET TREADMILL RUN

by

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ABSTRACT

Anaerobic threshold speed (V_{TAM}) was determined for 20 male university students using a continuous treadmill protocol. The onset of anaerobiosis was determined by analyzing excess CO_2 elimination. The following week, all subjects ran at the V_{TAM} median speed of 7.25 miles per hour for 10 minutes. Recovery oxygen consumption was monitored after this run. Application of double exponential equations by computer and subsequent integration, calculated Total, Alactic, and Lactic Oxygen Debts. Subjects who ran above their V_{TAM} (group L- V_{TAM}) had significantly ($p < .05$) higher total, lactic and alactic debts than those subjects who ran below their V_{TAM} (group H- V_{TAM}). The total debt showed a significant ($p < .05$) negative correlation ($r = -.77$) to V_{TAM} in group L- V_{TAM} . This appears to be due to the increasing lactic debt, that was also significantly ($p < .05$) negatively correlated ($r = -.73$) to V_{TAM} . Group H- V_{TAM} did not exhibit this characteristic. This study demonstrates that V_{TAM} is a critical factor in determining oxygen debt and therefore, work above this point results in the onset of metabolic acidosis, which may limit the optimal running speed for a given distance.

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CHAPTER 1

STATEMENT OF THE PROBLEM

Introduction

Anaerobic metabolism and its effect on recovery was first investigated by Hill, Long and Lupton (1924). At that time, such work level terms as moderate and severe were used to describe the exercise that produced either immediate or extended recovery oxygen uptake situations. Since this classic investigation, researchers have labelled the oxygen recovery curve (Margarita et al., 1933), quantified the oxygen recovery curve (Henry & DeMoor, 1950), and attempted to explain the oxygen recovery curve (Huckabee, 1958b). However, no one has examined oxygen recovery curves with respect to individual onset of anaerobic metabolism.

Although some anaerobic metabolic activity continually occurs during exercise (Graham, 1978), only when lactate diffuses into venous blood flow from the muscle tissues is it possible to easily monitor anaerobic activity. This increase in venous lactate production appears to be the result of the imbalance between lactate production and oxidation in the muscle tissues (Graham, 1978). Lactate in the venous circulatory system has been the standard for examining work intensity since Margarita et al. (1933) first examined phases of lactate production and removal. Results of these earlier investigations (Margarita et al., 1933; Margarita & Edwards, 1934b) and more recent research (Hermansen & Stensvold, 1972) demonstrate the following:

1. there is a greater lactate concentration for higher work intensity,
2. lactate levels stabilize for all but the very extreme levels of steady-state work, and
3. long recovery periods are associated with high lactate levels.

These early investigations imply that there is a point in the transition from aerobic to anaerobic work intensity where anaerobic metabolism starts to play a very important role. This increased role is marked by the appearance of lactate in the blood in excess of resting levels. Initial investigation (Issekutz & Rodahl, 1961; Wasserman et al., 1964; Naimark et al., 1964; Wasserman & McIlroy, 1964) monitored this phenomenon and associated cardiovascular-respiratory changes. This resulted in non-invasive procedures involving measurement of respiratory gas exchange to determine the onset of metabolic acidosis.

The term "Anaerobic Threshold" (AT) which has become synonymous with the onset of metabolic acidosis, was originally defined by Wasserman et al. (1964) as ". . . the work level (oxygen uptake) above which the subject develops metabolic acidosis." This definition is convenient in that it is the limit of predominately aerobic energy production (as determined by normal blood lactate levels) but misleading in that no detectable anaerobic activity has occurred (again as determined by blood lactate level). Most studies use Wasserman's definition of the AT but some define the AT as the point where blood lactate production just exceeds its removal (Davis et al., 1976).

The study of the role of the AT and its relationship to human performance has only just begun. This phenomenon has been used for testing hospital patients (Wasserman & Whipp, 1975), athletes (Costill, 1970), and models of hypernea (Whipp, 1977). However, the role of the onset

of anaerobic metabolism and its relationship to recovery analysis has not yet been investigated.

Statement of the Problem

The purpose of this study was to investigate the effects of individual Anaerobic Threshold Levels on the accumulation of an Oxygen Debt for a set treadmill run.

Hypotheses

1. Subjects with an Anaerobic Threshold speed (V_{TAM}) above the speed of the treadmill (group H- V_{TAM}) have a lower Total Oxygen Debt than those with a V_{TAM} below the speed of the treadmill (group L- V_{TAM}).
2. Group H- V_{TAM} has a lower Alactacid Debt than group L- V_{TAM} .
3. Group H- V_{TAM} has a lower Lactacid Debt than group L- V_{TAM} .
4. Group H- V_{TAM} has a lower ratio of Lactacid Debt to Alactacid Debt than group L- V_{TAM} .
5. Group H- V_{TAM} has a lower Total Recovery Time than group L- V_{TAM} .
6. Within group L- V_{TAM} , Total Oxygen Debt is a decreasing function of V_{TAM} .
7. Within group L- V_{TAM} , Alactacid Debt is a decreasing function of V_{TAM} .
8. Within group L- V_{TAM} , Lactacid Debt is a decreasing function of V_{TAM} .
9. Within group L- V_{TAM} , the ratio of Lactacid Debt and Alactacid Debt is a decreasing function of V_{TAM} .

10. Within group $L-V_{TAM}$, Total Recovery time is a decreasing function of V_{TAM} .
11. Within group $H-V_{TAM}$, Total Oxygen Debt and V_{TAM} are unrelated.
12. Within group $H-V_{TAM}$, Alactacid Debt and V_{TAM} are unrelated.
13. Within group $H-V_{TAM}$, the Lactacid Debt and V_{TAM} are unrelated.
14. Within group $H-V_{TAM}$, the ratio of Lactacid Debt to Alactacid Debt and V_{TAM} are unrelated.
15. Within group $H-V_{TAM}$, Total Recovery time and V_{TAM} are unrelated.

Rationale

The Anaerobic Threshold is the threshold of appearance of venous lactate as a result of anaerobic metabolism. As defined, V_{TAM} is the running speed above which these metabolites start to increase. Therefore, a subject running at a speed above his V_{TAM} will have a larger contribution of anaerobic metabolism as an energy supply, relative to a subject running at a speed below his V_{TAM} .

This suggests that Alactacid and Lactacid Debts will be larger for the subject running at a speed above his V_{TAM} . If this is true, this implies a larger Total Oxygen Debt and a longer Recovery Time.

The difference between the treadmill speed and V_{TAM} (for the subjects running faster than their V_{TAM}) should be reflected in a larger contribution of anaerobic metabolism to their activity. This suggests that the components of Oxygen Debt and V_{TAM} will be inversely related. Furthermore, it is speculated that this relationship will be approximately linear.

This relationship is not expected for those subjects running below their V_{TAM} .

Assumption

That an oxygen consumption during recovery after exercise can be described by a double exponential equation.

Delimitations

This study is delimited by:

1. the sample type (college age males),
2. the sample size,
3. the sample's fitness levels,
4. the speed of the treadmill at which the subjects will run, and
5. the length of the time each subject is on the treadmill before recovery.

Limitations

This study's results are limited by:

1. data collection capabilities of the Beckman Metabolic Measurement Cart and the Hewlett Packard Data Acquisition system interfaced with it,
2. the method of Anaerobic Threshold determination, and
3. the individuals' metabolic responses to the protocols.

Definitions

1. Alactacid Oxygen Debt (Alactic Debt): the Oxygen Debt incurred as a result of decreased body oxygen stores and high energy phosphate compounds (the fast exponential recovery phase).
2. Anaerobic Threshold: the point above which excess CO_2 production

is associated with elevated blood Lactate levels as a result of anaerobic metabolism.

3. Excess Lactate (XL): as calculated by Huckabee (1958a) is

$$XL = (L_n - L_o) - (P_n - P_o)(L_o / P_o)$$

where:

L_n = concentration of blood lactate at time of sampling

L_o = concentration of blood lactate at rest

P_n = concentration of blood pyruvate at time of sampling

P_o = concentration of blood pyruvate at rest

4. Excess CO_2 (Exc CO_2): is the excess of carbon dioxide in expired air (Issekutz & Rodahl, 1961) expressed as:

$$ExcCO_2 = \dot{V}CO_2 - \dot{V}O_2 \Delta RQ$$

where:

CO_2 is the total expired carbon dioxide

O_2 is the total expired oxygen

ΔRQ is the resting RQ (determined by Issekutz & Rodahl as .75).

5. Lactacid Oxygen Debt (Lactic Debt): the oxygen debt in excess of Alactic Debt due to lactate oxidation and higher metabolic rate (the slow exponential recovery phase).
6. Total Oxygen Debt: the total oxygen required during recovery in excess of resting.
7. Total Recovery Time: the time from cessation of exercise until oxygen consumption returns to resting.
8. V_{TAM} : the treadmill speed corresponding to the Anaerobic Threshold.

CHAPTER 2

REVIEW OF THE LITERATURE

I. Historical Considerations

Early research of anaerobic metabolism and its relationship to physical activity investigated oxygen uptake curves after exercise. The first major study was completed by Hill, Long and Lupton (1924). In this investigation, the first attempt at quantifying exercise exertion with regard to recovery time and blood lactate accumulation was made. The distinction between moderate and severe exercise and the possible role of lactate in determining oxygen consumption after exercise was investigated. R. Margaria (Margaria et al., 1933; Margaria & Edwards, 1934a, b) further researched oxygen consumption after exercise and developed recovery oxygen concepts that are still popular today. These studies supported a two-phase recovery system where the initial fast recovery component was termed the 'alactacid debt' and the later, slower recovery component was designated the 'lactacid debt'. The concept that the fast repayment phase repays fast energy supply systems and the slow repayment phase corresponds to blood lactate removal was hypothesized.

Berg (1947) applied a single exponential equation to oxygen and carbon dioxide recovery curves after exercise, as suggested earlier (Hill et al., 1924). This equation varied dependence between individuals and conditions, and Berg advanced no possible interpretation

for these results. Later, Henry and DeMoor (1950) applied double exponential equations to the oxygen recovery curve. This double exponential equation fit the data points extremely well and provided a mathematical way to separate the oxygen recovery curve into alactacid and lactacid components, as first suggested by Margaria et al. (1933). The following year, Henry (1951) postulated that the alactacid component is an essential phase of any physical exertion due to equilibria processes that the exponential function predicts. This added support to his double exponential model.

Subsequently, Henry and DeMoor (1956) investigated the effect of varying work intensity on a bicycle ergometer with regard to oxygen debt. Their findings demonstrated that increased work intensity produced a larger oxygen debt and recovery lag. Double exponential equations accurately described all recovery curves and showed an increasing lactacid component with increasing intensity.

It is from this model, developed by Margaria et al. (1933) and Henry and DeMoor (1950), that current recovery curve analysis is based.

II. Anaerobic Metabolism and Oxygen Debt

In 1958, W. E. Huckabee produced a series of articles (Huckabee, 1958a, b, c) that attempted to explain anaerobic metabolism and its relationship to oxygen recovery curves. He proposed, based on lactate: pyruvate equilibria relationships, that oxygen debt is directly related to Excess Lactate (XL) production. Excess lactate traced the oxygen recovery curve exactly and could be used as a predictor for total oxygen debt. This was in direct contrast to the concept of a two-phase recovery curve as supported by Margaria et al. (1933) and Henry and

DeMoor (1950).

This finding was subsequently investigated by other researchers. Knuttgen (1962), using four different work rates on a bicycle ergometer, observed that past a "critical level of work," there was a rapid increase in oxygen debt. This increase in debt was paralleled by rises in lactate and excess lactate. However, this parallel rise underestimated the oxygen debt. At the lower levels of work, where the fast component of recovery was dominant, the level of lactate or excess lactate could not predict the oxygen debt.

Margarita et al. (1963) also observed no increase in venous lactate until a specific exercise intensity. However, below this work intensity, an oxygen debt is still measurable and possibly attributable to the alactacid component. This study once again demonstrated the possible existence of a two-phase oxygen debt payment.

An editorial in the Annals of Internal Medicine by R. E. Olson (1963) cautioned the use of "Excess Lactate" as a means of signifying anaerobiosis. The assumption that the lactate/pyruvate ratio reflects the DPNH/DPN ratio in the cell and that the latter reflects aerobic capacity at a given time is disputed. The presence of at least two DPNH/DPN pools, with at least one membrane (mitochondrial) separating hydrogen ions, suggests that the lactate/pyruvate ratio may not reflect mitochondrial deficiency as much as limited hydrogen transfer from the cytoplasm to the mitochondria.

Wasserman et al. (1965) also challenged Huckabee's debt explanation, as it directly conflicted with their concept of anaerobic threshold (Wasserman & McIlroy, 1964; Wasserman et al., 1964). Their findings support the concepts of Knuttgen (1962) and Margarita et al. (1963). This was especially true at the low work levels (subthreshold) where

Wasserman et al. (1965) found excess lactate to predict less than ten percent of oxygen debt.

Thomas et al. (1965) also investigated the excess lactate concept. In a repetition of Huckabee's original works (with slight procedural modifications), Thomas failed to demonstrate the correlations for excess lactate that Huckabee presented. The investigation did reconfirm the role of lactate and pyruvate as indicators of anaerobic activity, however, made no attempt to further delineate the mechanisms.

Research next advanced to the analysis of the compartments of oxygen debt. Piiper et al. (1968), utilizing intact dog gastrocnemius muscle, observed a decreased concentration of high energy phosphates (specifically creatine phosphate) and oxygenated myoglobin during stimulated exercise. They hypothesized that the return to resting values is accomplished during the alactacid phase of recovery (as mathematically predicted by Henry [1951]).

DiPrampero and Margaria (1968) conducted a similar experiment with in vivo dog gastrocnemius muscle. Their analysis showed a decreasing creatine phosphate concentration with increased exercise intensity while the concentration of ATP and ADP at steady-state remained constant. This decrease and the linear relationship between exercise oxygen uptake at steady-state and alactacid oxygen debt (reflecting phosphagen resynthesis half reaction time) supports creatine phosphate's resynthesis role in alactacid oxygen debt.

Cerretelli (1969) continued this line of investigation to determine energy equivalents for the lactate and alactic phases (associated with decreased phosphagen stores). The linear equation for energy expenditure for these two phases, based on modern hypotheses, does not

significantly differ from that calculated by Margaria et al. (1934).

This fact supports the two component oxygen debt hypothesis empirically.

Direct evidence of creatine phosphate synthesis after exercise was investigated by Piiper and Spiller (1970). Their data shows almost complete phosphagen resynthesis in the first two minutes of recovery. During this time period, the alactacid oxygen recovery component appears to explain the resynthesis of phosphagen. Total oxygen debt over-predicted high energy phosphate resynthesis.

In 1970, a very extensive investigation into oxygen debt and sub-maximal exercise was completed by Knuttgen. Oxygen debt was observed after steady-state work performed at varying maximal oxygen uptake percentages and at different work durations. In his discussion, he stressed a two-phase recovery system as it accurately described all the results. The slow component increased exponentially, and the fast component increased linearly after an initial basal rate as work intensity increased. Only the slow component increased as the time duration increased at a constant work load. This did not correlate with exercise blood lactate levels, therefore adding more doubt to the postulate that the slow component is related only to blood lactate levels. Knuttgen (1970) suggested that other factors, such as hormones, body temperature, and electrolyte changes might also be considered.

A comparison of steady-state and nonsteady-state exercise, employing different time durations for a constant workload was researched by Whipp et al. (1970). Their conclusions suggested that the difference seen in efficiency between short and longer term exercise related to oxygen debt, could be compared to:

1. delay of alactic debt repayment during steady-state, and
2. lactate accumulation during the second and third minutes of

exercise due to delay in oxygen supply.

They did not attempt to separate the recovery curve into two components.

A similar experiment by McMiken (1976) demonstrated work intensity to be a key in predicting oxygen debt ($r=0.83$). He also showed that steady-state and nonsteady-state debt were quite similar, but did not imply that the previous interpretation of Whipp et al. (1970) was incorrect.

Current analysis of recovery has centered on supramaximal work (Katch, 1972; diPrampo et al., 1973; Freund & Gendry, 1978; Roberts & Morton, 1978). The main thrust of this work has been the examination of the alactacid component of recovery. The delay of an exponential return to basal level was seen in diPrampo's (1973) work which suggested a delayed return of metabolism to normal (perhaps an anaerobic recovery phase as suggested by Cerretelli et al. [1974]). Roberts and Morton (1978) demonstrated the reliability of this technique to determine maximum alactic debt but so far only a few pieces of a very large puzzle have been found.

III. Anaerobic Threshold

Since Margaria et al. (1933) monitored lactate build-up in the blood, most investigation has involved the time course of lactate during and after exercise. However, in 1962, Knuttgen (1962) noticed a "critical level of work" where lactate first appears in the blood. This level of work had been seen before (Huckabee, 1958), but this investigation appears to be the first to attach significance to it.

Issekutz and Rodahl (1961) observed a lactate breakaway using the change in the Respiratory Quotient and Excess CO_2 . This was the first

attempt at noninvasively following lactate levels in the blood (a correlation of 0.918 for lactate and Excess CO_2 was found).

Wyndham et al. (1962) speculated that the onset of metabolic acidosis limited prolonged physical activity. Their investigation demonstrated that the "trained" individual had a higher tolerance to this anaerobic onset. These ideas were novel, but the use of Huckabee's (1958a) Excess Lactate concept in determining the start of anaerobiosis detracted from their study.

Williams et al. (1962) investigated the anaerobic threshold and heat stress. Anaerobic activity appeared to increase at about 1.4 to 1.5 liters per minute of oxygen uptake above resting in the hot environment (about 95°F) and at about 1.6 to 2.2 liters per minute of oxygen uptake above resting in the comfortable environment (about 70°F). This suggests that the anaerobic metabolism mechanism is initiated faster at the higher temperature. Excess lactate determined the onset of anaerobic activity therefore the results must be interpreted with caution.

In 1964, three studies from Karlman Wasserman's laboratory in Palo Alto, California, introduced the concept of the "Anaerobic Threshold" (AT). The AT was originally defined by Wasserman et al. (1964) as ". . . the work level (oxygen uptake) above which the subject develops metabolic acidosis." The AT and its relationship to varying levels of work rate was investigated by Wasserman et al. (1964). Their findings reiterated Wyndham et al.'s (1962) postulate, that activity may be prolonged for workloads at or below the AT.

The continual observation of blood lactate may be inconvenient, so Naimark et al. (1964) investigated respiratory exchange variables concomitant with lactate and bicarbonate changes. When lactate and

bicarbonate were compared to the Respiratory Quotient and Excess CO₂ production (as similarly done by Issekutz & Rodahl [1961]) identical time of change was observed and a correlation (r) of 0.98 was calculated for excess CO₂ and bicarbonate concentration. Trained athletes did not exhibit an increase in the Respiratory Quotient until a much higher work level (oxygen uptake) compared to that of the untrained, suggesting a delay in onset of anaerobic activity. Their bike protocol for detecting the AT proved very reliable when checked the following day.

The third investigation (Wasserman & McIlroy, 1964) utilized breath-by-breath analysis of end-tidal gas concentrations to calculate the Respiratory Quotient to determine the AT for patients in a hospital. AT monitoring procedures were administered to determine some forms of cardiovascular deficiency. This provided an added measure of safety to testing procedures not available under maximal stress test conditions.

Wyndham et al. (1965) also investigated the AT comparing normal and hospital patients. Although excess lactate was used to determine the point of onset of anaerobic metabolism, the relationship between maximum oxygen uptake and the AT was introduced. The use of a 'percent' of maximum oxygen uptake would allow comparisons of normal and cardiac patients, however, exclusion of work-load would limit:

1. absolute improvement recognition after rehabilitation, and
2. comparisons to other studies with subjects of different maximal oxygen uptakes.

The result of investigation of the AT and its application to hospital patients was the review of muscular exercise analysis in normal and disease situations (Wasserman & Whipp, 1975). Results of previous investigation, especially relating to the AT, and its application to

patient diagnosis was reviewed.

Bouhuys et al. (1966) conducted an exhaustive analysis of the relationships between blood lactate and excess CO_2 , respiratory quotient, blood pH and bicarbonate. Although significant correlations ($p < .001$) existed between lactate and all variables, the highest correlation was to excess CO_2 ($r=.796$). As with all predictors, there were discrepancies which led these researchers to conclude that the best index of metabolic acidosis is blood lactate. However, they do agree that the respiratory quotient and excess CO_2 are good indicators of 'change' in blood lactate levels.

A study relating AT and training was conducted by Williams et al. (1967). This investigation showed that daily exercise of four hours, continuing from four to six weeks, increases the percent of maximum oxygen uptake at which anaerobiosis occurred (maximum oxygen uptake also increased). Analysis of anaerobiosis consisted of excess lactate determination therefore limiting interpretation of the results.

The following year, Williams et al. (1968) conducted a similar, but more extensive study on endurance trained males. The results showed that blood lactate and excess lactate levels were significantly different when determined after the sixth and sixtieth minute of exercise. A more important finding was the difference in the AT as predicted by lactate and excess lactate (this difference was significant according to the authors, but no confidence limits were given). This again casts doubt on the use of excess lactate as previously discussed.

Wasserman et al. (1967) investigated work intensity and duration of exercise on a bicycle ergometer related to the AT. The concepts of "pay-as-you-go" (aerobic) and "credit" (anaerobic) energy supply terms

relating to exercise could explain the different metabolic responses. Increased oxygen debt, as a result of increased work intensity showed an increased debt proportion due to lactate oxygen equivalents. At a sub-threshold (moderate intensity as described by Wells et al. [1957]) work intensity, activity could be maintained by all subjects for a minimum fifty minute period. However, only four of ten subjects completed the heavy work intensity (just above the AT) for the fifty minute period. No one completed the fifty minutes at the very heavy work load. This suggests that the AT is the limit of aerobic work capacity and that activity above this work level requires an increasing amount of anaerobic energy supply. The finite limits of energy production from the pyruvate: lactate system and its feedback on aerobic energy supply (Wenger & Reed, 1976) appears to restrict prolonged activity.

Nagle et al. (1970) investigated lactate accumulation during runs of 64 to 89 percent of maximum oxygen uptake. The untrained subjects demonstrated higher blood lactate levels for the same work intensity (as also shown by Wasserman et al. [1967]) as compared to the trained subjects. This again suggests that the AT may limit muscular activity as a result of anaerobiosis.

Costill (1970) was the first investigator to apply the AT to distance running. As might be predicted from previous studies, increased distance produced decreased steady-state blood lactate levels. The limited energy supply of anaerobic metabolism and discomfort from tissue acidosis (Simonson, 1971) probably determines the optimal running speed for a given race and therefore blood lactate level.

The relationship of work intensity above the AT to oxygen uptake time course to steady-state was investigated by Whipp and Wasserman

(1972). A slower rise to oxygen uptake steady-state was observed in all instances for work intensity above the AT. It was also shown that the trained individual had a higher level of work intensity before anaerobic energy supply started.

More recently, Whipp et al. (1979) utilized first order kinetics to demonstrate the oxygen uptake rise to steady-state at subthreshold exercise (time constant about 45 seconds). However, depending on the type of suprathreshold exercise (ramp, sinusoid, or square wave) a two-component rise to steady-state was only occasionally noted. Discrepancies between individuals suggested that a two-component or delayed rise is not necessary for suprathreshold activity.

The concept of continued anaerobic activity and removal of lactate during submaximal exercise was reviewed by Hermansen and Stensvold (1972). They failed to show lactate production for individuals at a work intensity below 60-80 percent maximum oxygen uptake (normally termed the AT). However, observations during exercise, where blood lactate is produced, showed that the major area for lactate oxidation is in skeletal muscle. Graham (1978), in a comprehensive review of lactate changes during exercise, showed that lactate may be produced in muscle but does not necessarily diffuse into the blood, thereby masking anaerobic activity. This suggests lactate's continual oxidation in muscle (active or inactive fibers).

Wasserman et al. (1973) summarized breath-by-breath non-invasive techniques for detecting the AT. A most significant result of the study was that for non-invasive AT determination, a one minute work increment rate was found to be optimal for showing the change in metabolism at the start of anaerobiosis. This permits stress testing duration

to be kept at a minimum. Reproducibility of the AT was shown to be exact over one hour, four hour, one week and nine month intervals.

Breath-by-breath analysis (Wasserman et al. [1973]) also solved a major problem of AT detection. Hyperventilation may obscure the AT when observing expired volume or CO_2 . Observation of end-tidal oxygen and carbon dioxide eliminates the chance of misinterpretation.

Koyal et al. (1978) researched the feasibility of using oxygen pulse in determining the AT. In a very restricted sample (maximum oxygen uptake less than 3.75 liters per minute), they observed that the curvilinear plateauing of oxygen pulse coincided with the AT. The leveling of oxygen pulse indicates impending maximum oxygen uptake.

An important change in testing technique of the AT was researched by Volkov et al. (1975). He utilized the Excess CO_2 (ExcCO_2) concept of Missekutz and Rodahl (1961) to determine the AT during a speed increase treadmill protocol. The result was a running speed (V_{TAM}) at which the AT occurred. Volkov et al. (1975) suggested that this AT speed could be used as an index of running efficiency.

Application of the AT to distance running was investigated by Weiser et al. (1978), Sucec (1979) and Farrell et al. (1979). Sucec (1979) used the AT, expressed as milliliters oxygen per kilogram per minute, in a regression equation to predict one and two mile times (the other regression factors were percent body fat, running efficiency, oxygen debt, lean body weight, and maximum oxygen uptake). Correlations of 0.98 and 0.91 were calculated for the one and two mile times respectively.

Farrell et al. (1979) determined the running speed at which the "onset of plasma lactate accumulation" (OPLA) occurred. This speed and

mean marathon speed were correlated significantly at $r=0.98$. Mean marathon pace was 0.28 miles per hour above the mean treadmill pace at OPLA. Weiser et al. (1978) also applied this concept and showed a correlation of 0.92 ($p < 0.001$) for 3.2 kilometer race pace and treadmill speed at the AT.

Wasserman's continued basic AT research (Wasserman et al. 1975) demonstrated the involvement of the carotid body in metabolic responses during exercise. The carotid body appears to be the mediator for ventilatory change for suprathreshold activity to maintain blood homeostasis. This respiratory compensation is the major factor stabilizing metabolic acidosis. These findings also support the use of non-invasive measurements for AT determination.

Whipp's model (Whipp, 1977) of hyperpnea during exercise also used the AT as a division of exercise intensity for determining ventilatory control. The carotid bodies seem to be the main mediators of control for suprathreshold work intensity. Subthreshold mediation of hyperpnea seems to be much more complex (as reviewed by Whipp [1977]).

AT determination for three different modes of exercise was analyzed by Davis et al. (1976). Similar results for maximal oxygen uptake and percent of maximum at the AT were obtained for treadmill walk/run exercise and cycling. Significantly lower results were obtained in all instances for arm cranking. Their results also show that the AT is reproducible for their three exercise protocols.

Wiswell et al. (1979) also investigated the relationship between the AT calculated for a bicycle ergometer and a treadmill. For each modality, oxygen uptake at the AT and the maximum oxygen uptake were significantly correlated, but the AT (expressed as a percent of maximum

oxygen uptake) for each exercise was significantly different ($p < 0.01$). This suggests that, similar to maximal oxygen uptake (Strømme et al., 1977; McKay & Bannister, 1976), the AT may be exercise specific, as suggested by Davis et al. (1976). However, Stamford et al. (1978) showed that for one- versus two-legged cycling, the AT occurs at the same relative percent of maximum O_2 uptake, though at a lower absolute work load. Maximum oxygen uptake was lower for one-legged cycling. These findings suggest that the AT and maximum oxygen uptake may be influenced differently under the same conditions.

MacDougall (1977) investigated the different training techniques of athletes and their AT. His observation suggests that the longer distance trained athletes have a higher AT. He also illustrates a hypothetical example of two identical athletes, except for different ATs, who run a marathon. The higher AT individual would have a significant advantage in supplying aerobic energy requirements that could be equated to distance advantage if constant stress to the cardiovascular system was maintained.

Comparison of sprinters and endurance runners are described by Roberts et al. (1979). The sprinters exhibited:

1. lower maximum oxygen uptake,
2. lower AT (percent maximum oxygen uptake),
3. lower oxygen uptake at the AT,
4. lower muscular capillarization, and
5. higher percent fast twitch fiber composition.

These data suggest that the sprinters have lower endurance capacity, especially when considering the AT and capillarization. However, comparison of the AT and muscle fiber composition by Green et al. (1979)

showed no correlation between the two, thereby limiting conclusions about AT and muscle fiber composition. No muscular sites were reported in this abstract.

Davis et al. (1979a, b) investigated the effect of endurance cycle ergometry training on maximum oxygen uptake and the AT for middle-aged men. Both significantly increased over the nine week program. Again this suggests increased AT with endurance type training.

Weltman et al. (1978) tested the use of the AT as a measure of submaximal fitness. They observed that the individuals with a high oxygen uptake at metabolic acidosis had:

1. a faster rate to steady-state oxygen consumption,
2. a lower steady-state oxygen uptake, and
3. a faster return to resting values after exercise.

These findings are in agreement to those of Whipp and Wasserman (1972).

The use of heart rate as a training index was investigated by Katch et al. (1978). These researchers suggest that the use of the relative percent of maximum heart rate concept is not a good training concept when considering whether or not an individual is producing lactate. Individuals appear to have different heart rates corresponding to their AT, therefore caution is stressed until the AT has been determined.

Patton et al. (1979) also investigated heart rate and its relationship to the AT. Their data suggest that heart rate does not vary between individuals (trained or untrained) at their AT. However, their heart rates at the AT were higher compared to Wyndham et al. (1965) and Wasserman et al. (1973).

Ivy et al. (1979) examined the effect of substrate availability

on the AT. Increased blood glucose had no effect on the AT. Increased blood free fatty acids significantly lowered the AT and absolute blood lactate. This has definite implications in calculation of the AT and Ivy suggests that standards should be adopted for AT determination.

Pendergast et al. (1979) compared performance on an arm crank exercise between kayakers and sedentaries. Kayakers were observed to have higher absolute ATs but not relative ATs (they also had higher maximum oxygen uptake). Their methodology of determining the AT consisted of separated exercises at different work loads for different time durations. This resulted in an early lactate release for some sub-threshold exercises that lasted for more than thirty seconds. They suggest that this lactate release may be used with the AT to evaluate muscle training.

Kindermann et al. (1979) analyzed the aerobic-anaerobic transition with reference to the AT and blood lactate concentrations. They suggest three categories for this transition as a result of their investigations. This classification system includes:

- i. The Aerobic Threshold (blood lactate less than 2 mmol/l, similar to the AT)
- ii. The Aerobic-anaerobic Transition (blood lactate 2 to 4 mmol/l)
- iii. The Anaerobic Threshold (greater than 4 mmol/l blood lactate).

These investigators found that under conditions of the Aerobic Threshold, activity could be maintained for at least two hours. The Aerobic-anaerobic Transition period of activity could be maintained for one hour. For conditions of the 'Anaerobic Threshold' only periods of exercise less than an hour in duration could be maintained.

The AT has been applied to many diverse testing situations. The

majority seem to be concerned with very specific but unrelated areas of application. Research of the AT is needed to explain the conclusions of these studies. This would allow applied research to progress with a sound knowledge base.

CHAPTER 3

METHODS AND PROCEDURES

Subjects

Twenty male subjects were selected for this study from volunteers from the University of British Columbia student population. The participants were subjectively chosen (from analysis of their activity patterns) to achieve a continuum of Anaerobic Threshold (AT) levels.

Testing Procedures

The testing format consisted of two sessions. During the first week, height, weight, anaerobic threshold speed (V_{TAM}) and maximum oxygen consumption ($\dot{V}O_2$ max) were determined for each subject. Resting oxygen uptake ($\dot{V}O_2$ rest) and recovery oxygen uptake after the set treadmill run were determined during the second week.

$\dot{V}O_2$ max and V_{TAM} were determined using a continuous treadmill protocol. As a warm-up, each subject walked on the treadmill at 3.5 miles per hour for ten minutes. Subsequently, the treadmill was set at 4.0 miles per hour and then increased one-half mile per hour at the end of each minute until volitional fatigue.

Heart rate was monitored by direct ECG utilizing an Avionics 4000 cardiograph with oscilloscope and ST depression computer and display. Expired gases were continuously sampled and analyzed by a Beckman Metabolic Measurement Cart (BMMC) interfaced into a Hewlett Packard 3052A

Data Acquisition system for fifteen second determination of respiratory gas exchange variables.

Maximum oxygen consumption was determined by averaging the highest four consecutive fifteen second oxygen uptake values. The anaerobic threshold speed was determined by examining excess CO_2 elimination (Volkov et al., 1975). The determination of the AT was consistent with the definition by Wasserman et al. (1964). In order to dichotomize the subjects into a low and high V_{TAM} grouping a median speed (V_{MED}) for the twenty V_{TAM} scores was calculated as 7.25 miles per hour.

Resting oxygen consumption was determined during a ten minute sitting period at the start of the week 2 assessment. Each subject then walked on the treadmill at 3.5 miles per hour for five minutes. After the warm-up, each subject ran at V_{MED} for ten minutes, during which time expired gases were continuously monitored by the aforementioned systems.

At the completion of the ten minute run, each subject sat until he either returned to his $\dot{V}\text{O}_2$ rest (minimum time sitting was set at twenty minutes) or had recovered for thirty minutes.

Data Analysis

The last four oxygen uptake values were averaged during the median speed run to determine time zero oxygen uptake for recovery. This value, plus each fifteen second value of oxygen uptake during recovery were fit with a double exponential equation of the form:

$$\dot{V}\text{O}_2 = a_1 e^{-\alpha_1 t} + a_2 e^{-\alpha_2 t} + a_3$$

where $\dot{V}\text{O}_2$ = oxygen consumption at time 't'

a_1 = alactic linear parameter

α_1 = alactic non-linear parameter

a_2 = lactic linear parameter

α_2 = lactic non-linear parameter

a_3 = asymptotic $\dot{V}O_2$ rest

The U.B.C. Computing Centre's program P:3R (Dixon & Brown, 1979) was utilized for this computation. Initial non-linear parameters were based on Henry and DeMoor (1950).

Each equation was integrated over thirty minutes to determine total oxygen debt, alactic oxygen debt, and lactic oxygen debt (see Appendix A). Each individual's lactic oxygen debt was divided by the alactic oxygen debt to give the lactic:alactic ratio (ratio debt).

Time to resting oxygen uptake (recovery time) after exercise was determined by observation of four consecutive recovery readings equivalent to each subject's initial resting oxygen uptake.

Total oxygen debt and ratio debt for subjects with anaerobic threshold speeds above (group H- V_{TAM}) and below (group L- V_{TAM}) the median speed were analyzed for difference between means by the U.B.C. Computing Centre program MULTIVAR (Finn, 1977). Significant ($p < .05$) multivariate F (Hotelling T^2) was followed by univariate F to determine where significance occurred. Alactic oxygen debt and lactic oxygen debt were similarly analyzed.

Correlation coefficients (r) were calculated for the oxygen debt variables and V_{TAM} for the H- V_{TAM} and L- V_{TAM} groups by the U.B.C. Computing Centre program SIMCORT (Le, 1979). Each coefficient was analyzed for significance from zero.

CHAPTER 4

RESULTS AND DISCUSSION

Results

The twenty subjects were tested, as per the schedule of week 1. One subject (RR) was added to the twenty after the median speed determination to attempt to further complete the AT continuum between 5 and 6.5 miles per hour. Their results are summarized in Table 1. Individual anaerobic threshold curves and determination of V_{TAM} appear in Appendix B.

Oxygen uptake curves during recovery in week 2 appear in Appendix C. Subject J0 experienced coughing during the recovery period therefore his recovery data are not included in this study.

Double exponential equations calculated for each subject appear in Table 2. These functions are represented as a continuous line on each recovery curve in Appendix C. The computer program P:3R did not produce a resting asymptotic value for subjects ML, BV, RF and DW. This appears to be due to insufficient data points at base line oxygen consumption. Therefore, a lower limit for this baseline was set as 0.225 liters of oxygen per minute. This value appears to be a limit for $\dot{V}O_2$ rest. The resulting equations are included in Table 2 and Table 3.

The computer program could not initially calculate a double exponential equation for subject GS. Observation of his recovery curve data points showed that three points (at 1.0, 1.24 and 1.50 minutes) seem to deviate from the regular pattern seen in all subjects. Removal

TABLE 1

Individual Subject Characteristics

Subject	Age (years)	Height (cm)	Weight (kg)	V _{TAM} (mph)	$\dot{V}O_2$ max (ml/kg/min.)
CN	22	175.6	77.9	4.0	42.6
ML	25	174.0	82.7	5.0	46.6
RR	24	188.4	95.5	6.0	52.2
TB	21	179.5	71.1	6.5	46.3
DG	24	174.0	60.5	6.5	59.2
DD	24	179.1	75.5	6.5	49.1
BV	21	184.5	88.5	6.5	67.9
GS	22	188.7	99.4	6.5	46.6
DA	23	175.6	77.9	7.0	58.5
RF	24	166.1	62.5	7.0	55.4
HB	20	170.2	65.0	7.0	50.0
AB	21	181.1	74.5	7.5	62.5
AO	26	174.9	63.4	7.5	58.6
GT	24	176.7	78.4	7.5	57.0
DH	21	179.3	69.3	8.0	57.4
JL	22	177.5	67.1	8.0	64.8
JO	24	180.8	79.0	8.0	69.2
DM	23	174.9	63.3	8.5	76.2
DW	24	178.6	79.8	8.5	52.0
SP	21	182.1	68.0	10.5	68.0
JH	23	176.4	71.0	11.0	72.7
\bar{X}	22.8	178.0	74.8	7.3	57.8
SD	1.60	5.37	10.57	1.5	9.4

TABLE 2

Individual Double Exponential Equations
for Subjects in Group L- V_{TAM}

V_{TAM}	Subject	Exponential Equation	VO_2 rest (l/min.)
4.0	CN	$3.269e^{-1.076t} + 1.221e^{-.168t} + .441$.317
5.0	ML	$2.923e^{-1.140t} + .181e^{-.097t} + .225$.273
6.0	RR	$3.472e^{-1.169t} + .151e^{-.075t} + .265$.320
6.5	TB	$2.453e^{-1.510t} + .391e^{-.206t} + .309$.280
6.5	DG	$2.417e^{-1.411t} + .407e^{-.185t} + .253$.240
6.5	DD	$2.985e^{-1.090t} + .285e^{-.103t} + .278$.291
6.5	BV	$2.949e^{-1.020t} + .210e^{-.038t} + .225$.369
6.5	GS	$2.595e^{-2.434t} + 1.056e^{-.339t} + .310$.291
7.0	DA	$3.228e^{-1.168t} + .132e^{-.079t} + .408$.349
7.0	RF	$2.038e^{-1.264t} + .063e^{-.160t} + .225$.223
7.0	HB	$1.921e^{-1.787t} + .727e^{-.406t} + .277$.259

TABLE 3

Individual Double Exponential Equations
for Subjects in Group H-V_{TAM}

V _{TAM}	Subject	Exponential Equation	VO ₂ rest (l/min.)
7.5	AB	$2.342e^{-1.169t} + .106e^{-.032t} + .268$.312
7.5	AO	$1.181e^{-6.690t} + 1.143e^{-.751t} + .289$.286
7.5	GT	$.609e^{-9.562t} + 1.941e^{-1.153t} + .272$.246
8.0	DH	$1.992e^{-1.393t} + .476e^{-.419t} + .273$.230
8.0	JL	$2.713e^{-1.160t} + .165e^{-.068t} + .217$.230
8.5	DM	$1.515e^{-4.083t} + .953e^{-.709t} + .210$.211
8.5	DW	$3.189e^{-1.236t} + .137e^{-.023t} + .225$.301
10.5	SP	$2.052e^{-1.820t} + .186t^{-.225t} + .302$.318
11.0	JH	$1.729e^{-2.917t} + .611e^{-.559t} + .278$.271

of the data points at 1.25 and 1.50 minutes allowed for proper curve fitting. Therefore, his results are calculated without these two data points.

Each equation was integrated over thirty minutes to determine alactic, lactic, and total oxygen debts (Appendix A). The ratio of lactic to alactic debt (debt ratio) was then calculated for each subject. These results appear in Table 4 and Table 5. Graphs of these results versus individual anaerobic threshold speeds (V_{TAM}) are represented in Figures 1 to 4.

It was not possible in this study to determine exact recovery time. The fluctuation of the fifteen second values for oxygen uptake (Appendix C) prevented the attainment of four consecutive equivalent values of resting oxygen uptake. The asymptotic value calculated by the computer program P:3R never exactly matched the resting value determined, therefore comparison of these two values would not be valid. Thus, hypotheses 5, 10, and 15 were not tested.

TABLE 4

Individual Alactic, Lactic, Total and Ratio
Oxygen Debts for Group L- V_{TAM}

V_{TAM}	Subject	Alactic Debt (liters)	Lactic Debt (liters)	Ratio Debt (Lactic/ Alactic)	Total Debt (liters)
4.0	CN	3.04	7.22	2.38	10.26
5.0	ML	2.56	1.76	0.69	4.33
6.0	RR	2.97	1.80	0.61	4.77
6.5	TB	1.62	1.89	1.17	3.52
6.5	DG	1.71	2.19	1.28	3.90
6.5	DD	2.74	2.64	0.97	5.38
6.5	BV	2.89	3.76	1.30	6.65
6.5	GS	1.07	3.12	3.92	4.19
7.0	DA	2.76	1.51	0.55	4.28
7.0	RF	2.19	0.39	0.24	2.00
7.0	HB	1.07	1.79	1.66	2.87

TABLE 5

Individual Alactic, Lactic, Total and Ratio
Oxygen Debts for Group H- V_{TAM}

V_{TAM}	Subject	Alactic Debt (liters)	Lactic Debt (liters)	Ratio Debt (Lactic/ Alactic)	Total Debt (liters)
7.5	AB	2.00	2.04	1.02	4.05
7.5	AO	0.18	1.52	8.44	1.70
7.5	GT	0.06	1.68	27.18	1.75
8.0	DH	1.43	1.14	0.79	2.57
8.0	JL	2.34	2.11	0.90	4.45
8.5	DM	0.37	1.34	3.81	1.72
8.5	DW	2.58	2.97	1.15	5.55
10.5	SP	1.13	0.83	0.73	1.95
11.0	JH	0.59	1.09	1.85	1.69

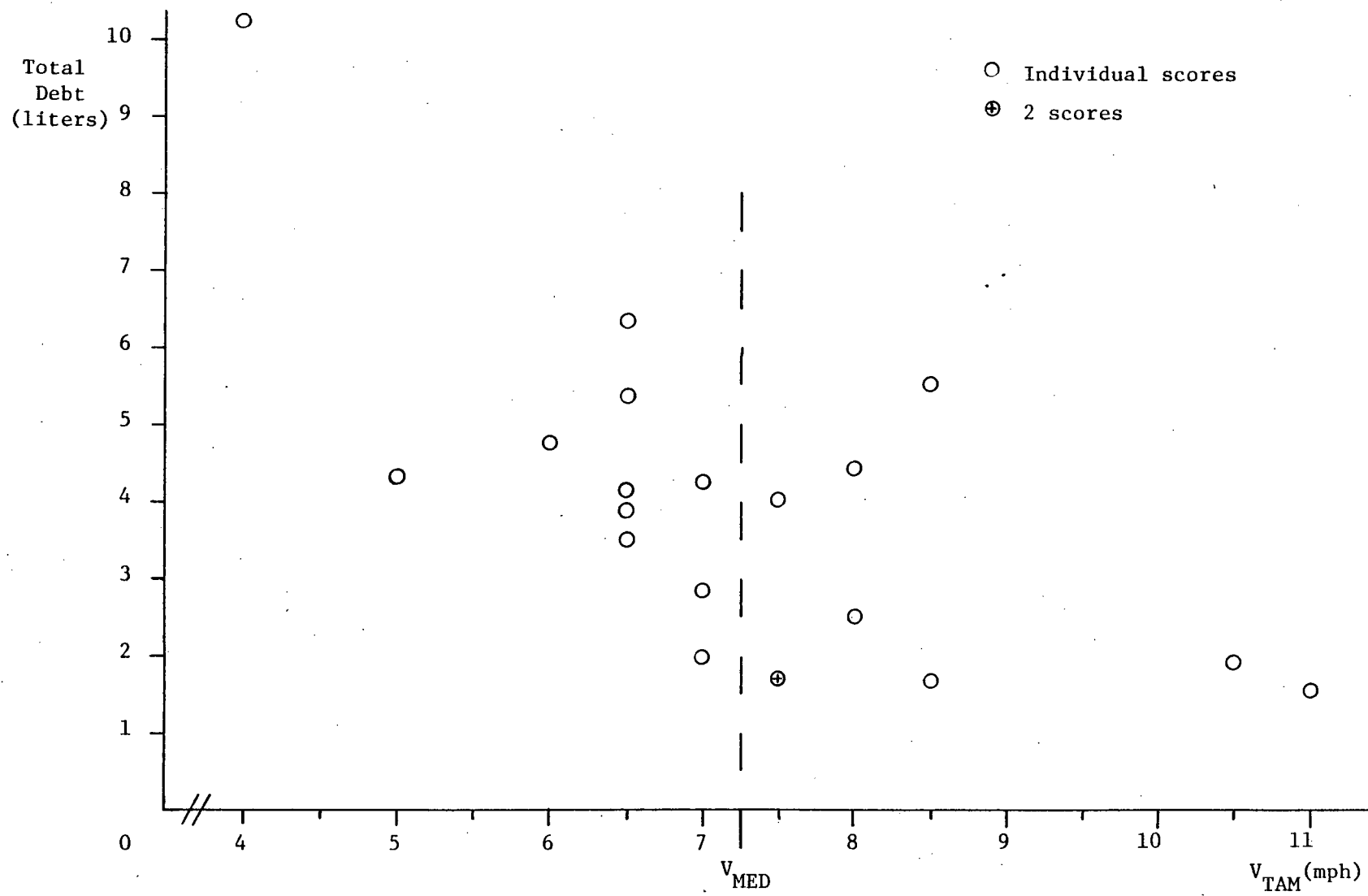


Figure 1. Graph of Total Debt and Individual V_{TAM} .

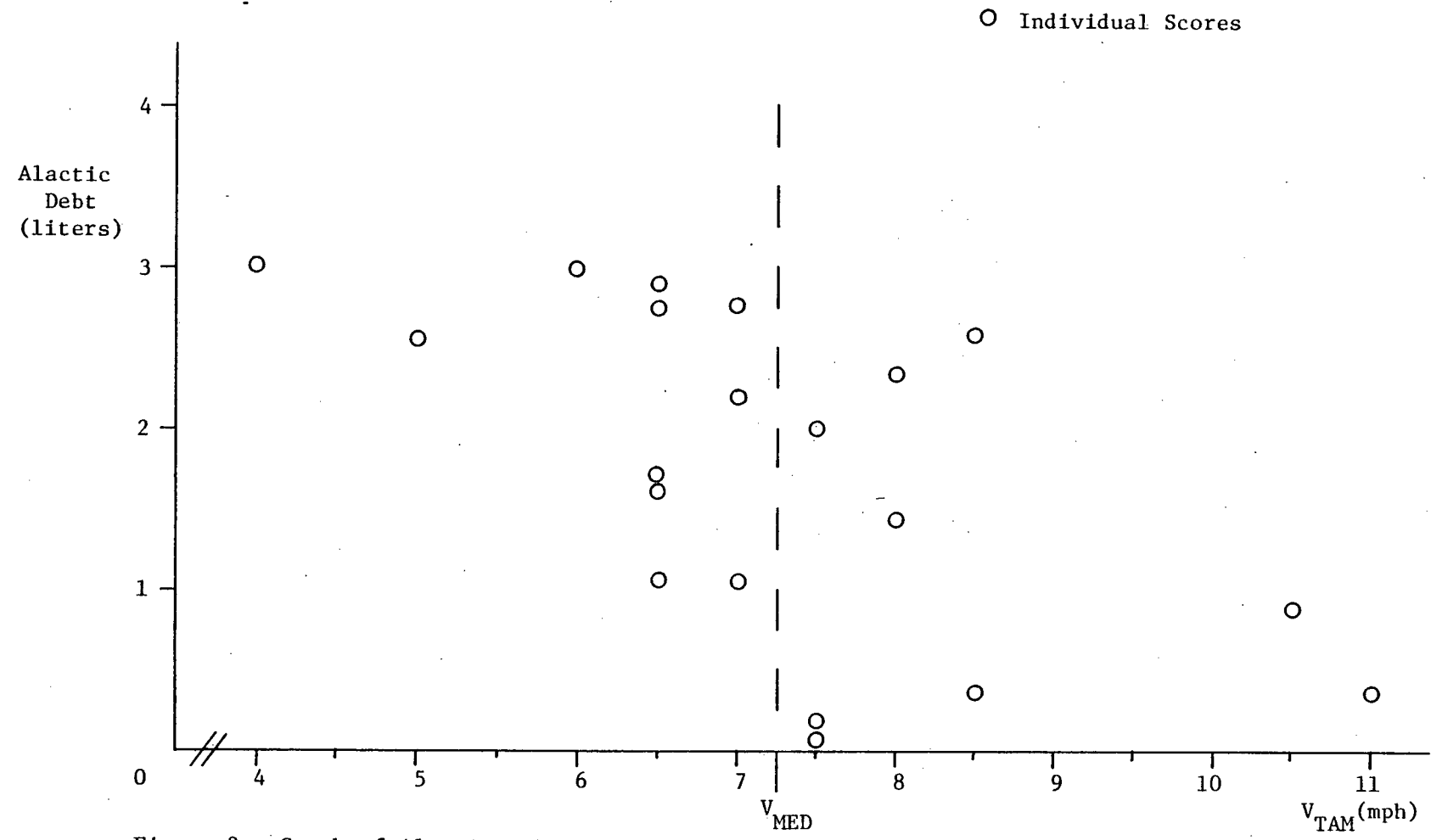


Figure 2. Graph of Alactic Debt and Individual V_{TAM}

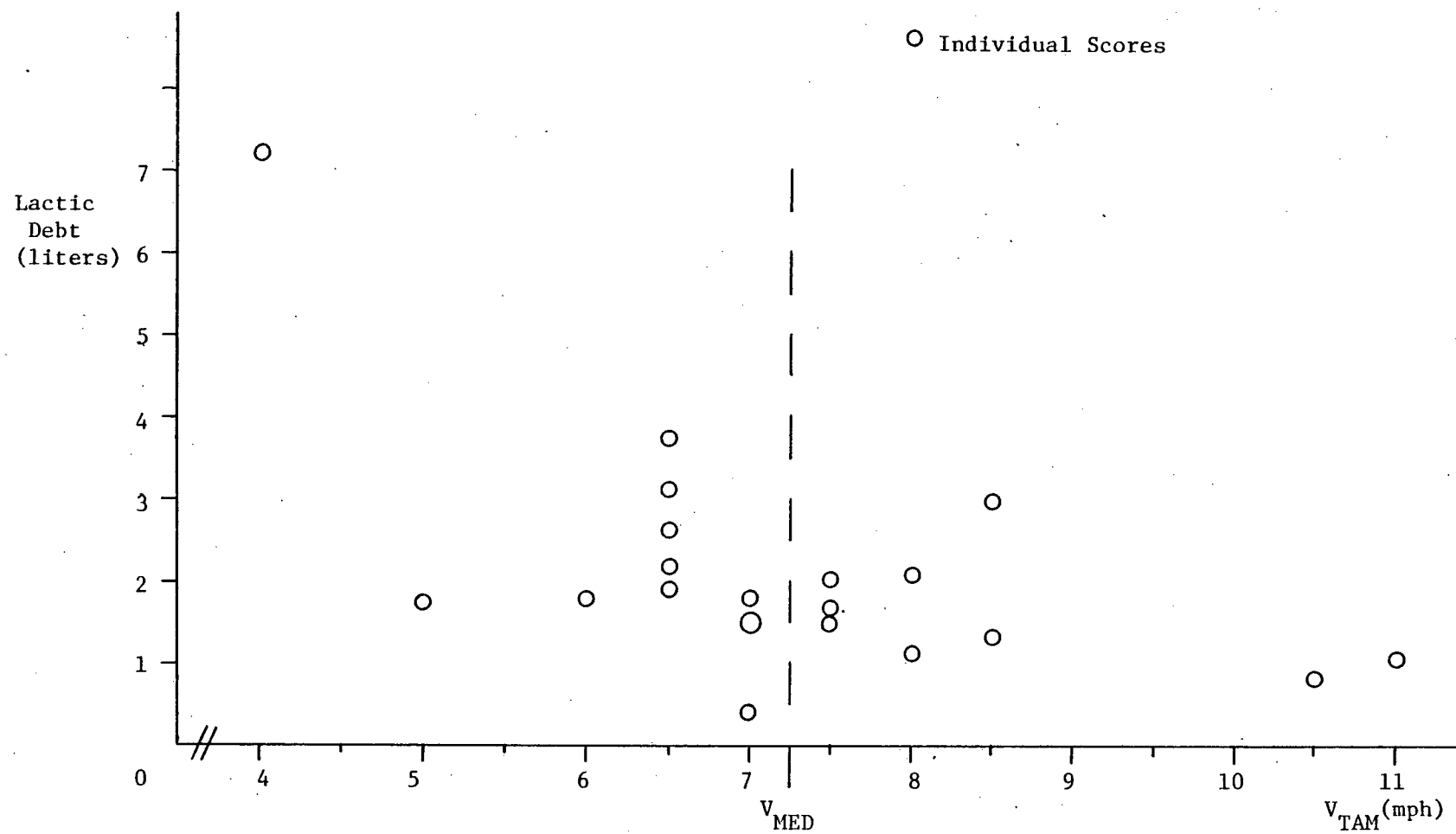


Figure 3. Graph of Lactic Debt and Individual V_{TAM}

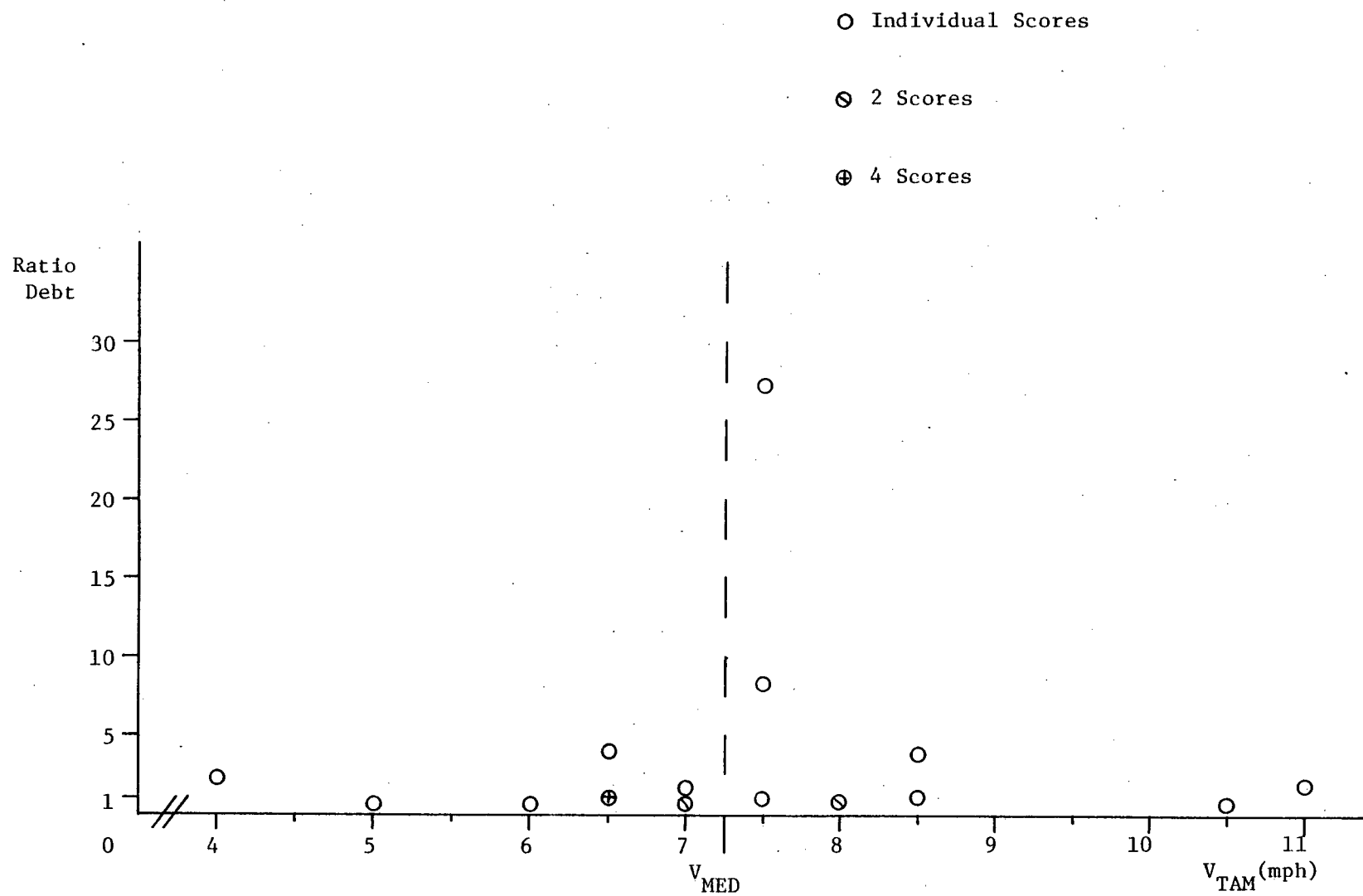


Figure 4. Graph of Individual Ratio Debt and V_{TAM}

Individual oxygen debt values were averaged for those subjects in group L-V_{TAM} and in group H-V_{TAM}. These values appear in Table 6.

TABLE 6
Means of Total, Alactic, Lactic and Ratio
Debts for Groups L-V_{TAM} and H-V_{TAM}

Group	Total Debt		Ratio Debt		Alactic Debt		Lactic Debt	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
L-V _{TAM}	4.74	2.33	1.34	1.04	2.19	0.77	2.55	1.78
H-V _{TAM}	2.83	1.43	5.10	8.65	1.19	0.95	1.64	0.65

The difference between means for total debt and ratio debt for the two groups show a significant T^2 statistic ($p < .05$). Univariate t values subsequently calculated for each dependent variable show this significance to be attributable to the difference between total debt means only ($p < .01$). Despite the large difference in means for ratio debt, significance was not obtained due to the large variance in group H-V_{TAM} (brought about by subjects AO and GT). These results are summarized in Table 7.

These results indicate rejection of the null hypothesis and therefore acceptance of hypothesis 1; that subjects in group H-V_{TAM} accumulate a lower oxygen debt compared to those subjects in group L-V_{TAM}. However, the statistical calculation does not indicate acceptance of hypothesis 4; that there is a difference between subjects in groups H-V_{TAM} and L-V_{TAM} in the calculation of a debt ratio (lactic debt/alactic debt).

TABLE 7

Multivariate Analysis of Dependent Variables
Total Debt and Ratio Debt

Dependent Variable	T^2	$p <$	t	$p <$
Total Debt	4.84	.022	9.78	.006
Ratio Debt			1.43	.248

The difference between means for alactic and lactic debt for the two groups also exhibit a significant Hotelling T^2 ($p < .01$). Univariate t values calculated for each dependent variable show this significance to be attributable to both the difference between alactic debt means ($p < .01$) and lactic debt means ($p < .05$). These results are summarized in Table 8.

These results indicate acceptance of hypotheses 2 and 3; subjects in group $H-V_{TAM}$ accumulate lower lactic and alactic debts than subjects in group $L-V_{TAM}$.

TABLE 8

Multivariate Analysis of Dependent Variables
Alactic and Lactic Debts

Dependent Variable	T^2	$p <$	t	$p <$
Alactic Debt	6.02	.01	12.1	.003
Lactic Debt			4.6	.047

Pearson correlation coefficients calculated for groups $H-V_{TAM}$ and

$L-V_{TAM}$ for V_{TAM} with total, ratio, alactic, and lactic debts appear in Tables 9 and 10.

TABLE 9

Correlation Coefficients for Subjects in Group $L-V_{TAM}$

	Total Debt	Ratio Debt	Alactic Debt	Lactic Debt
V_{TAM}	-.767*	-.210	-.506	-.729*

*Significantly different from zero at $p < .05$.

The results in Table 9 indicate acceptance of hypotheses 6 and 8; subjects in group $L-V_{TAM}$ have a negative linear relationship between total and lactic debts and V_{TAM} . These results do not indicate acceptance of hypotheses 7 and 9, therefore a decreasing function between ratio and alactic debts and V_{TAM} is not evident. It is apparent from observation of Figures 2 and 4, that both alactic and ratio debts are unrelated to V_{TAM} .

TABLE 10

Correlation Coefficients for Subjects in Group $H-V_{TAM}$

	Total Debt	Ratio Debt	Alactic Debt	Lactic Debt
V_{TAM}	-.279	-.375	-.478	-.098

The probability that the correlation coefficients for group $H-V_{TAM}$ (Table 10) are different from zero are as follows. (Rohlf & Sokal, 1969):

1. Total debt ($r = -.279$) $.4 < p < .5$
2. Ratio debt ($r = -.375$) $.2 < p < .4$

3. Alactic debt ($r = -.478$) $.1 < p < .2$

4. Lactic debt ($r = -.098$) $.5 < p < .9$

These probabilities suggest that there is not a linear relationship between total, ratio, and lactic debts and V_{TAM} . The correlation coefficient for alactic debt and V_{TAM} is very close to the confidence limit ($p < .05$) to reject the null hypothesis, suggesting a linear relationship. However, as the correlation coefficient only accounts for 23% of the variance of the sample, there is strong evidence to suggest that there is also no linear relationship between alactic debt and V_{TAM} . As a result, the null hypothesis is not rejected and remains as stated in hypotheses 12, 13, and 14; there is no decreasing function between total, alactic, and lactic debts and V_{TAM} in group H- V_{TAM} .

Discussion

The computation of double exponential equations to describe recovery oxygen consumption is consistent with the theory of decreasing payment (exponential decay) of alactic and lactic debts. The equations calculated (Tables 2 and 3) are similar to those reported by Henry and DeMoor (1950). The non-linear (α_i) and linear (a_i) parameters are of the same order of magnitude, except for those subjects in this study with small fast recovery component (subjects A0, GT and DM). Total debt, for all individuals, falls within limits of previous studies (Hill et al., 1924; Margaria et al., 1933; Henry & DeMoor, 1950; Knuttgen, 1962).

Comparison of computer calculated asymptotic $\dot{V}O_2$ rest values and determined $\dot{V}O_2$ rest values reveal similar results. However, subjects CN, BV, and DW exhibit differences greater than sixty milliliters of

oxygen per minute. Subject CN experienced difficulty in completing the 10-minute run, so that an elevated asymptotic $\dot{V}O_2$ rest is normal (Hill et al., 1924). The large variation in the resting data of subjects BV and CN may be attributed to the fact that initial resting oxygen consumption values are not indicative of their true resting state, and secondly, the lower limit of 0.225 liters of oxygen per minute set for the computer may be low.

The resulting integration of the equations to determine contributions to the total debt in this investigation, indicates that the processes involved are not as simple as originally proposed by Margaria et al. (1933). Calculation of debt ratios from the data reported by Margaria et al. (1933), Henry and DeMoor (1950), and Knuttgen (1970), show a range from 0 (mildest exercise) to about 2. These ratios do not compare with the ratios of 8.44, 27.18, and 3.81 of subjects A0, GT and DM respectively. Subjects in H- V_{TAM} who were working at a mild intensity of exercise did not exhibit ratios of 0.

At these low intensities of exercise, Berg (1947) argues that the recovery processes are better explained by a single exponential equation, thus supporting Hill et al. (1924) and Margaria et al. (1933). If this is true, single exponential equations should better describe the recovery curves in group H- V_{TAM} . Post hoc analysis of recovery curves in group H- V_{TAM} show a mean residual sum of squares of .79 (SD=.56) for a single exponential curve fit (computer program P:3R [Dixon & Brown, 1979]). The double exponential curves calculate a mean residual sum of squares of .68 (SD=.37). A correlated t-test of significance show these means to be significantly different at a level of .05. This suggests that the double exponential equation better describes recovery oxygen data points in group

H- V_{TAM} .

This result supports evidence by Knuttgen (1970), that this slow recovery phase does exist. This phase may be responsible for a return of ". . . interrelated metabolic, thermal, electrolytic and hormonal changes which the body undergoes during exercise . . ." to non-exercise levels. Piiper and Spiller (1970) also observed this slow phase in intact dog gastrocnemius muscle stimulated to do exercise that did not produce blood lactate in excess of resting values. They came to a similar conclusion in their investigation.

During oxygen deficit formation, both anaerobic glycolysis and stored high energy phosphates could be the energy sources. During the repayment phase (oxygen debt), these energy sources would be replenished at a rate dependent on the physiological make-up and the training level of the individual. This might account for some of the variability in debt ratio scores.

Results of this study indicate that recovery oxygen consumption is adequately described by a double exponential equation. However, to refer to these two phases as traditional lactic and alactic components may be questionable. The remaining discussion will use alactic and lactic terms to refer to the fast and slow recovery phases respectively.

The relationship between anaerobic threshold speed (V_{TAM}) and recovery oxygen consumption is demonstrated in the results of the hypotheses tested. The significant difference for total debt means for the two groups is consistent with observations of Wasserman et al. (1965) and Wasserman et al. (1967). The non-significant linear relationship ($r = -.279$) calculated for group H- V_{TAM} suggests that running speeds below V_{TAM} result in a relatively stable oxygen debt. The significant

negative linear relationship ($r = -.767$) calculated for group L- V_{TAM} indicates that running at increasing speeds above V_{TAM} results in increasing total debt.

The compartmentalization of total debt, into alactic and lactic debts, also produces a significant difference between the two debt components of each group (Tables 7 and 8). The only significant negative correlation calculated was for lactic debt and V_{TAM} for group L- V_{TAM} ($r = -.729$). These results suggest that the increasing total debt for group L- V_{TAM} is a result of increasing lactic debt. This may be attributable to increased blood lactate levels as suggested by Margaria et al. (1933), Henry and DeMoor (1950), Wasserman et al. (1967), and Knuttgen (1970).

Linear relationships were not observed between alactic debt and V_{TAM} in group L- V_{TAM} or between alactic and lactic debts and V_{TAM} in group H- V_{TAM} . Therefore, the relatively stable alactic component in each group, and the relatively stable lactic component in group H- V_{TAM} , suggests the significant difference between debts is a result of running above or below V_{TAM} . This implies that V_{TAM} is also critical in determining alactic and lactic debt components for a constant running speed.

Oxygen debt may be used as a criterion of fatigue and therefore used as a measure of work intensity (Simonson, 1971). The relationship between oxygen debt and V_{TAM} in this study shows that V_{TAM} is useful in determining primarily aerobic work intensity. Its relationship to increased anaerobic activity and resultant metabolic acidosis through increased utilization (recruitment) of fast twitch muscle fibers and/or increased anaerobic activity in slow twitch muscle fibers, may be very important in determining running speed for a given distance.

This relationship has recently been investigated by Weiser et al. (1978), Farrell et al. (1979), and Sucec (1979). These investigators illustrated very significant correlations between onset of anaerobic metabolism and race time. This gives strong evidence to the role of V_{TAM} in determining efficient running speed for a given distance.

In conclusion, the anaerobic threshold speed (V_{TAM}) appears to be the critical speed in determining total oxygen debt in a running exercise above V_{TAM} . V_{TAM} also appears to be critical in determining the size of the debt components, alactic and lactic. Further investigation is needed to clarify the mechanisms involved in determining the total debt and its components. Once this is accomplished, the effect of training techniques on V_{TAM} and the onset of anaerobic metabolism will be elucidated.

CHAPTER 5

SUMMARY AND CONCLUSIONS

Work intensity variation has been shown to change oxygen debt after exercise (Hill et al., 1924; Wasserman et al., 1965). Knuttgen (1962) noticed a "critical level" of work intensity which seemed to drastically affect oxygen debt size. This level of work intensity is associated with the onset of metabolic acidosis termed the Anaerobic Threshold (Wasserman et al., 1964). This study attempted to clarify the relationship between anaerobic threshold and oxygen debt after a set treadmill run.

Twenty male university students were chosen to achieve a continuum of anaerobic threshold levels. Week 1 testing sessions determined each subject's anaerobic threshold running speed (V_{TAM}) using a continuous treadmill protocol. V_{TAM} was identified by analyzing expired $\dot{V}O_2$ and $\dot{V}CO_2$ (calculated to excess CO_2 values) every fifteen seconds (Volkov et al., 1975). V_{TAM} scores ranged from 4.0 miles per hour to 11.0 miles per hour.

During the second testing session, each subject ran at the predetermined median speed of 7.25 miles per hour for ten minutes, which separated the subjects into two groups:

1. Group H- V_{TAM} --those subjects running below their V_{TAM} , and
2. Group L- V_{TAM} --those subjects running above their V_{TAM} .

Each subject's expired gases during the exercise and recovery stages were analyzed every fifteen seconds by a Beckman Metabolic Measurement Cart

interfaced into a Hewlett Packard 3052A data acquisition system.

Each individual's recovery oxygen consumption data were described with a double exponential equation by a computer. The resulting equation was integrated to determine total oxygen debt and its alactic and lactic components. The ratio of lactic/alactic debts was also calculated.

Analysis for a significance between means (Hotelling T^2) for groups $H-V_{TAM}$ and $L-V_{TAM}$ for the debt variables revealed significant ($p < .05$) differences for total, alactic, and lactic oxygen debts. No difference was evident for the ratio of lactic/alactic debt. The subjects in group $L-V_{TAM}$ had larger oxygen debts and debt components compared with those subjects in group $H-V_{TAM}$. These results suggest that V_{TAM} is critical in determining the oxygen debt after a run.

Analysis for a linear relationship between V_{TAM} and oxygen debt for each group showed total oxygen debt ($r = -.767$), and lactic debt ($r = -.729$) to be significantly ($p < .05$) linearly related to V_{TAM} in group $L-V_{TAM}$ only. This suggests that the faster the running speed is above an individual's V_{TAM} , the larger the total debt. This increasing debt is a result of the increasing lactic debt. This was not evident in group $H-V_{TAM}$.

The accumulation of an oxygen debt is indicative of fatigue (Simonson, 1971). The fact that V_{TAM} is a critical factor in determining oxygen debt suggests that work above this point results in the onset of metabolic acidosis, which limits the optimal running speed for a given distance. These findings support the evidence by Farrell et al. (1979) and Weiser et al. (1978) that the onset of metabolic acidosis is related to average race running speed. Further investigation should determine the mechanisms by which V_{TAM} is determined and changed.

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APPENDIX A

Sample Calculation of Oxygen Recovery Debts

Integration of the double exponential recovery curve allows calculation of alactic, lactic, and total oxygen debts. Integration of an exponential function is determined by the formula:

$$\int_0^t a e^{-\alpha t} dt = \left(\frac{a}{-\alpha} e^{-\alpha t} - \frac{a}{-\alpha} \right).$$

Integration of subject DH's equation

$$1.992e^{-1.393t} + 0.476e^{-0.419t}$$

over thirty minutes would be:

$$\begin{aligned} \text{Alactic debt} &= \int_0^{30} 1.992e^{-1.393t} dt = \left(\frac{1.992}{-1.393} e^{-1.393(30)} - \frac{1.992}{-1.393} \right) \\ &= 1.43 \end{aligned}$$

$$\begin{aligned} \text{Lactic debt} &= \int_0^{30} 0.476e^{-0.419t} dt = \left(\frac{0.476}{-0.419} e^{-0.419(30)} - \frac{0.476}{-0.419} \right) \\ &= 1.14 \end{aligned}$$

$$\begin{aligned} \text{Total debt} &= \text{Alactic debt} + \text{Lactic debt} \\ &= 1.43 + 1.14 \\ &= 2.57 \end{aligned}$$

$$\begin{aligned} \text{Debt ratio} &= \text{Lactic debt} \div \text{Alactic debt} \\ &= 1.14 \div 1.43 \\ &= 0.79 \end{aligned}$$

APPENDIX B

Anaerobic Threshold Curves to Determine Individual V_{TAM}

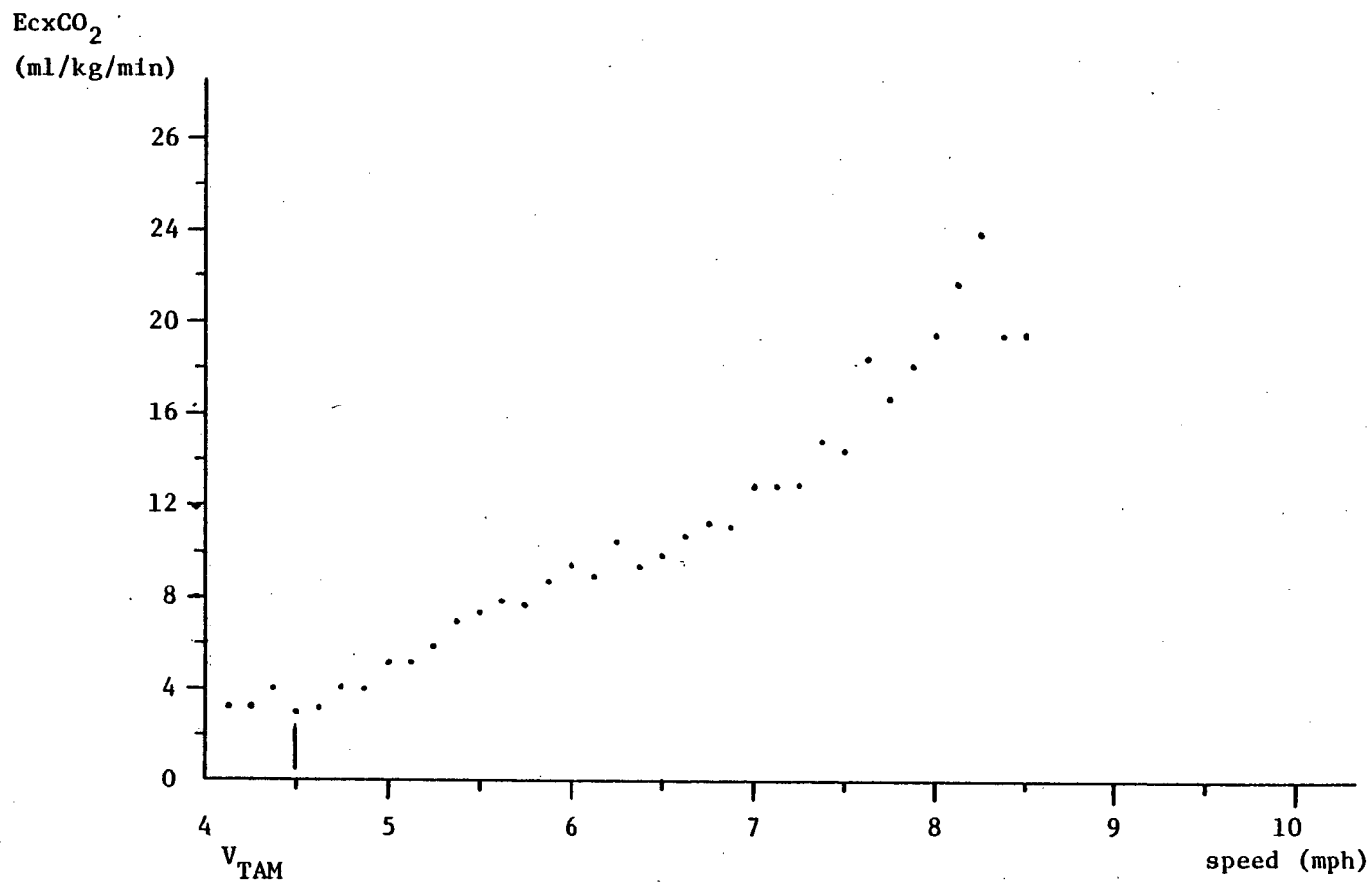


Figure 5. AT curve subject CN

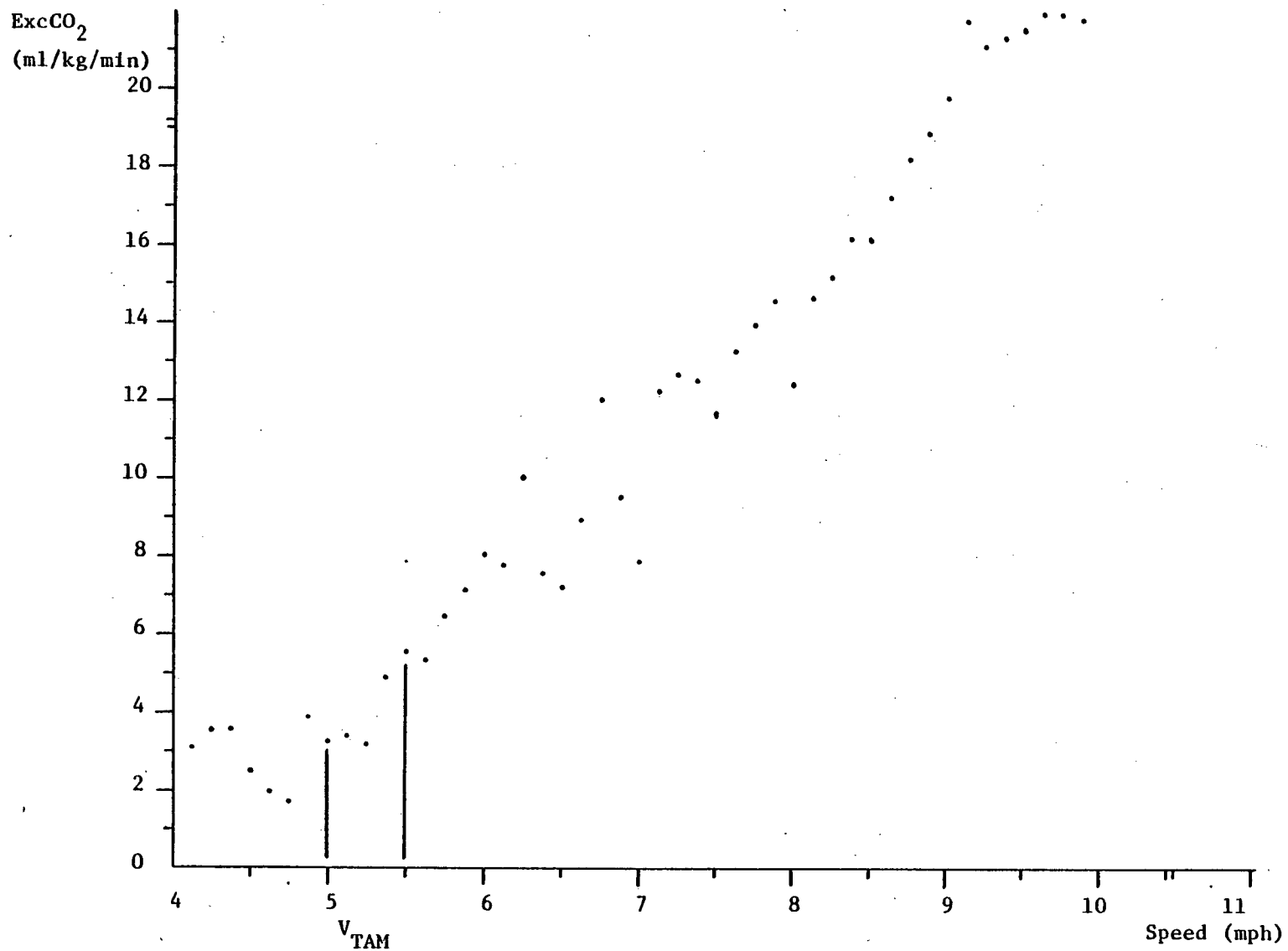


Figure 6. AT curve subject ML

ExcCO₂
(ml/kg/min)

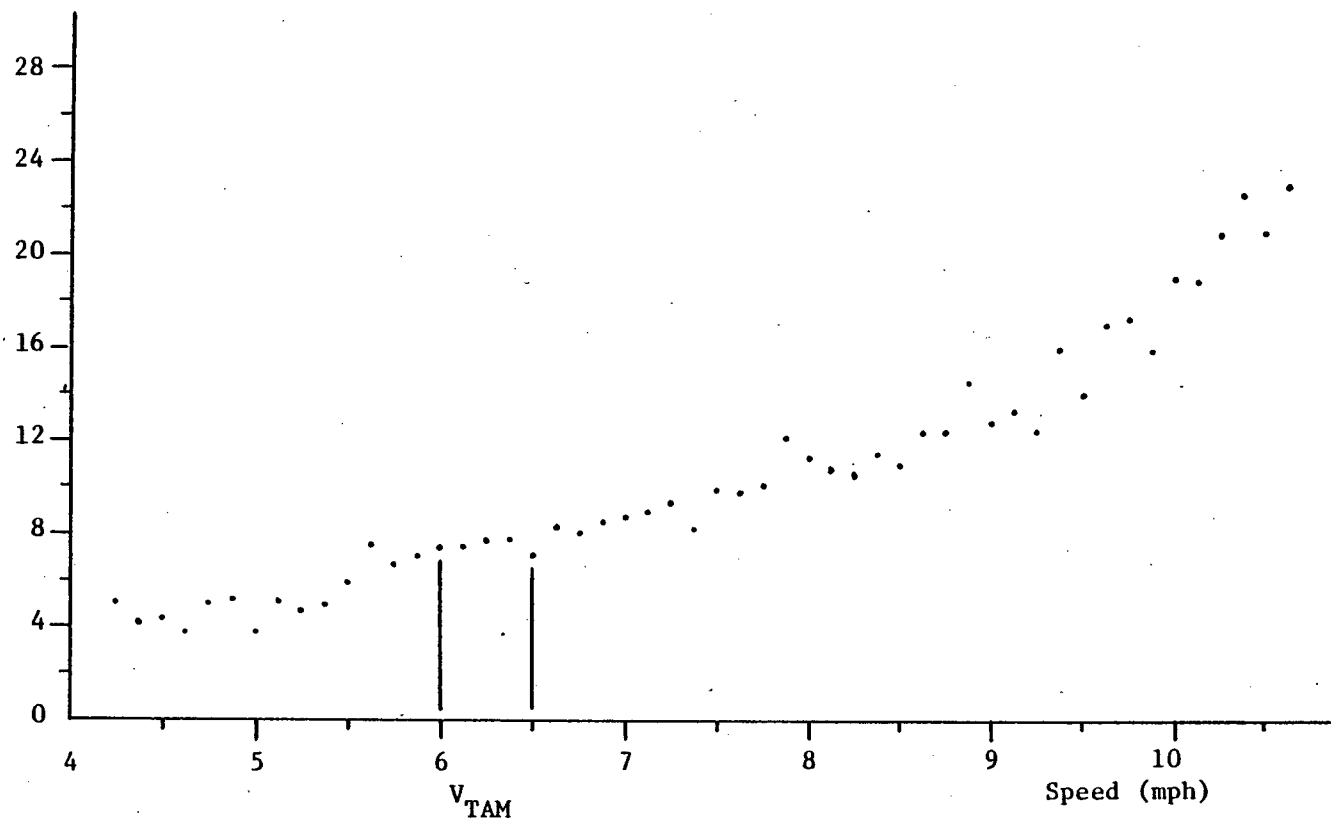


Figure 7. AT curve subject RR

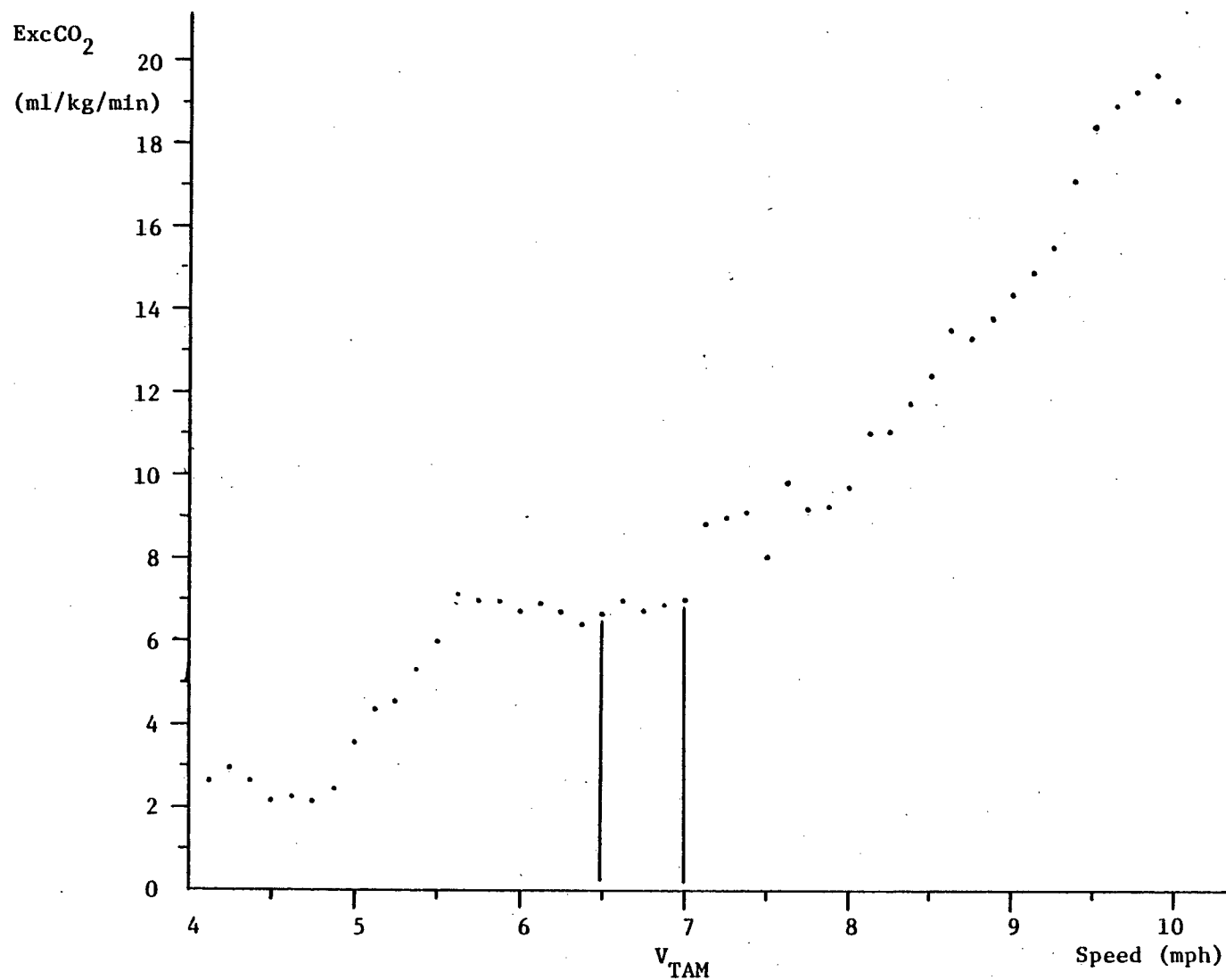


Figure 8. AT curve subject TB

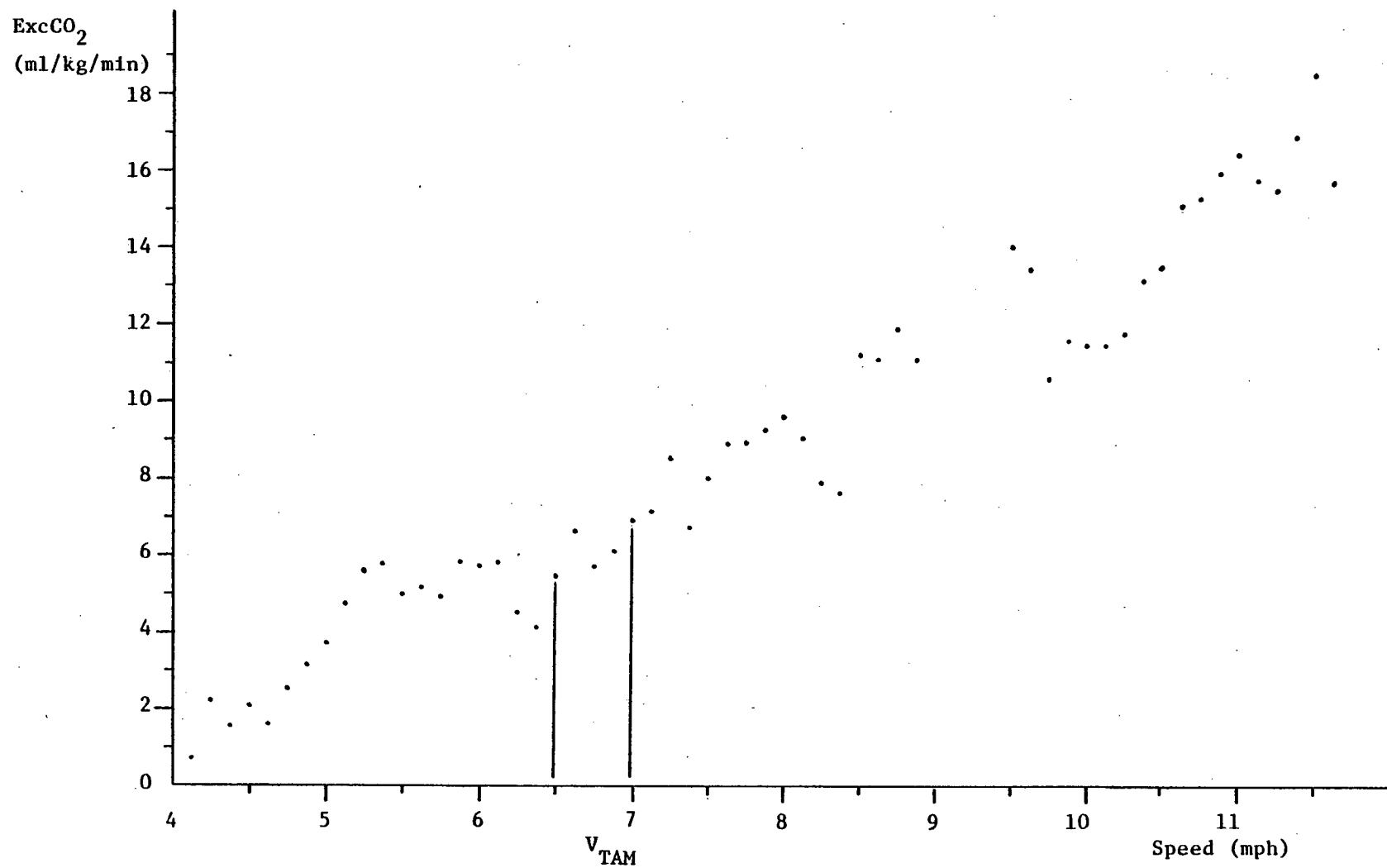


Figure 9. AT curve subject DG

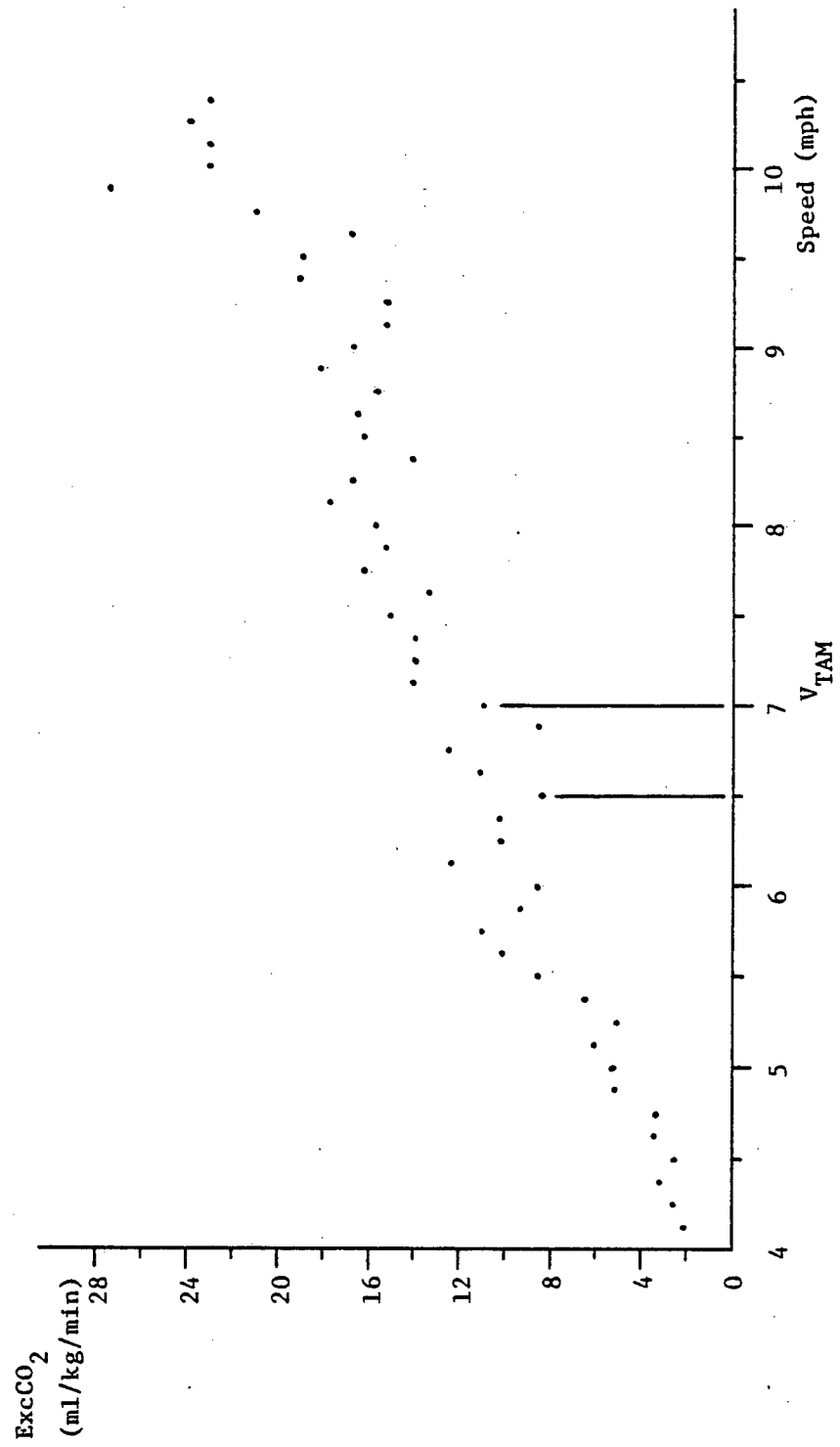


Figure 10. AT curve subject DD

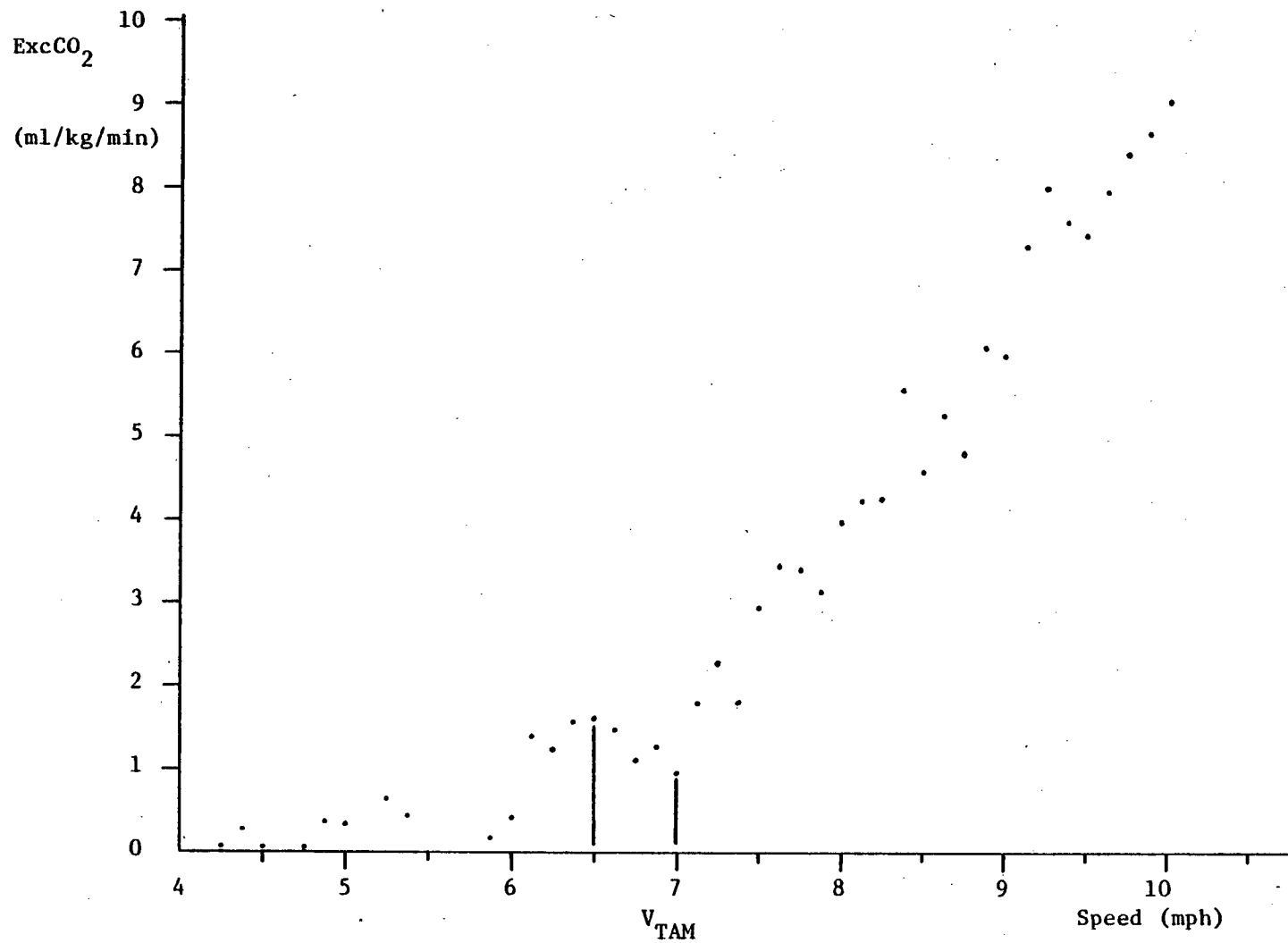


Figure 11. AT curve subject BV

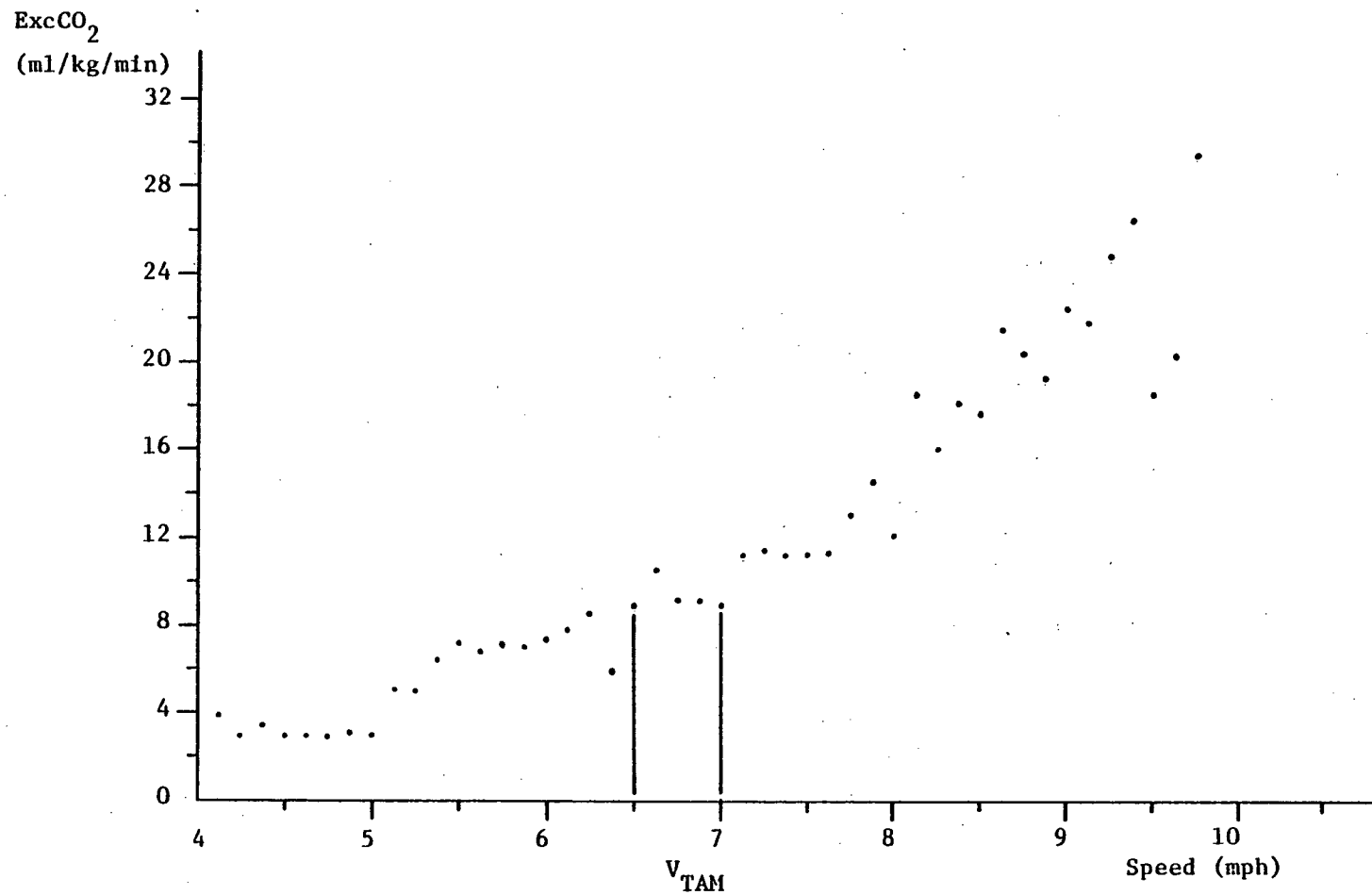


Figure 12. AT curve subject GS

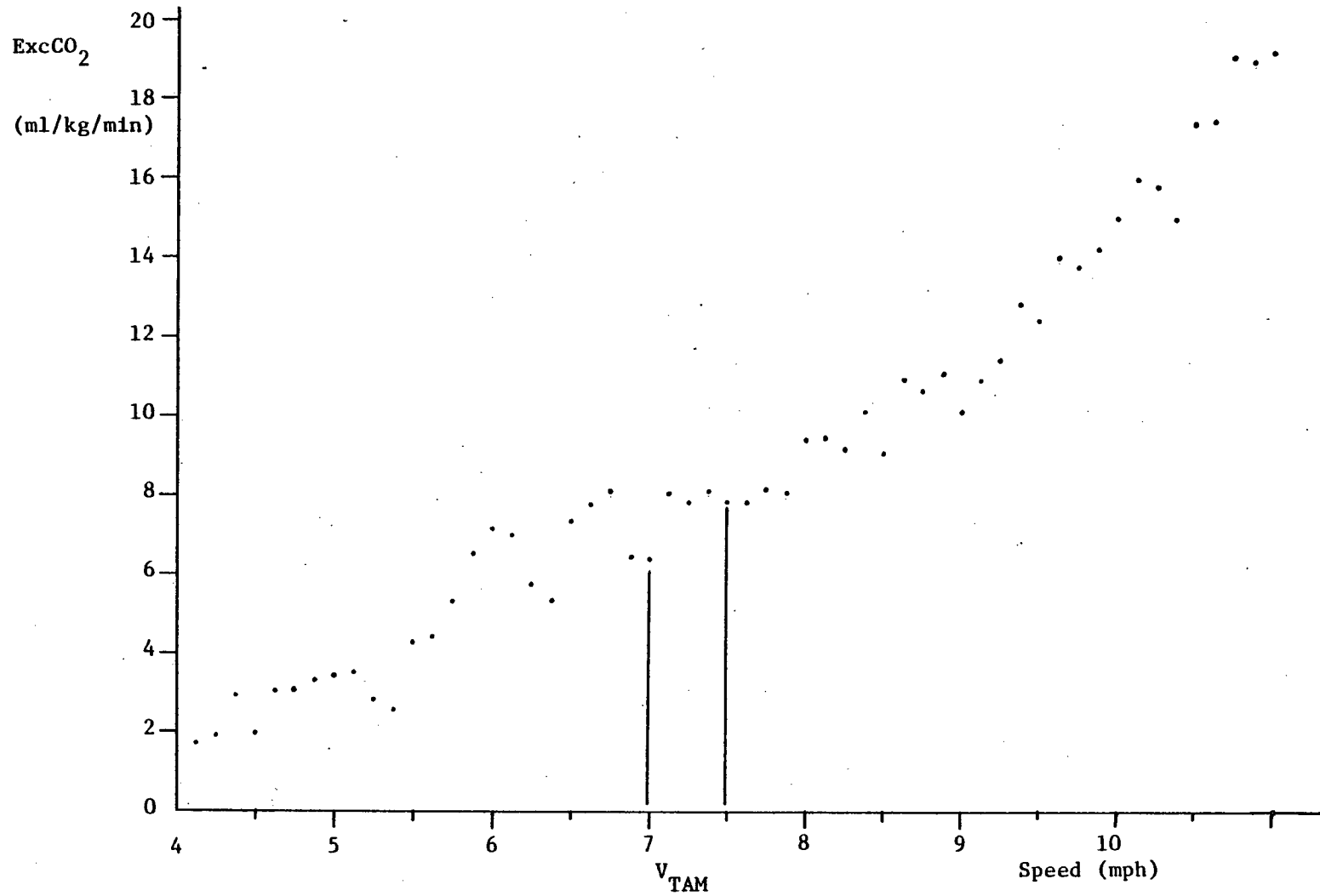


Figure 13. AT curve subject DA

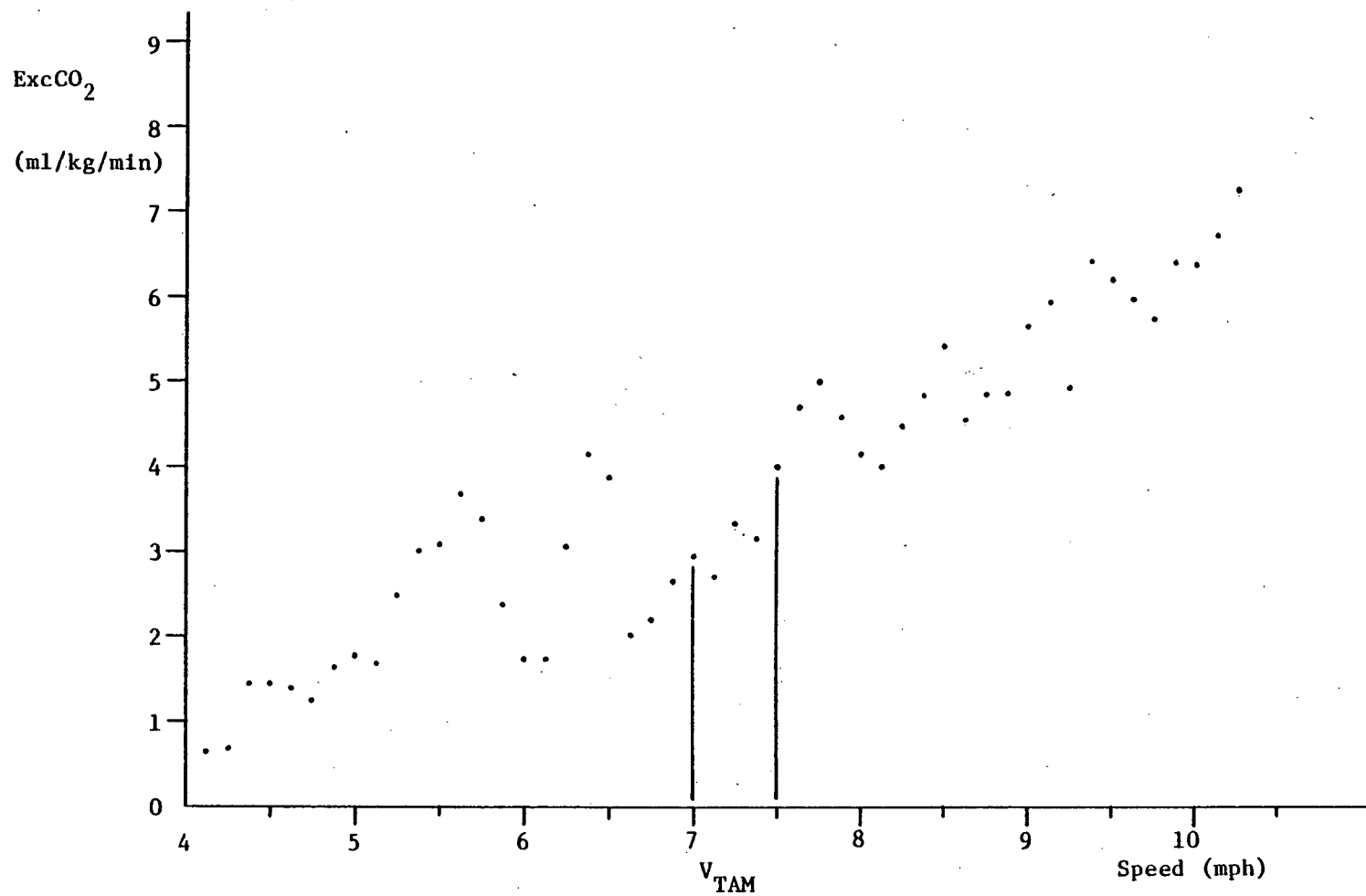


Figure 14. AT curve subject RF

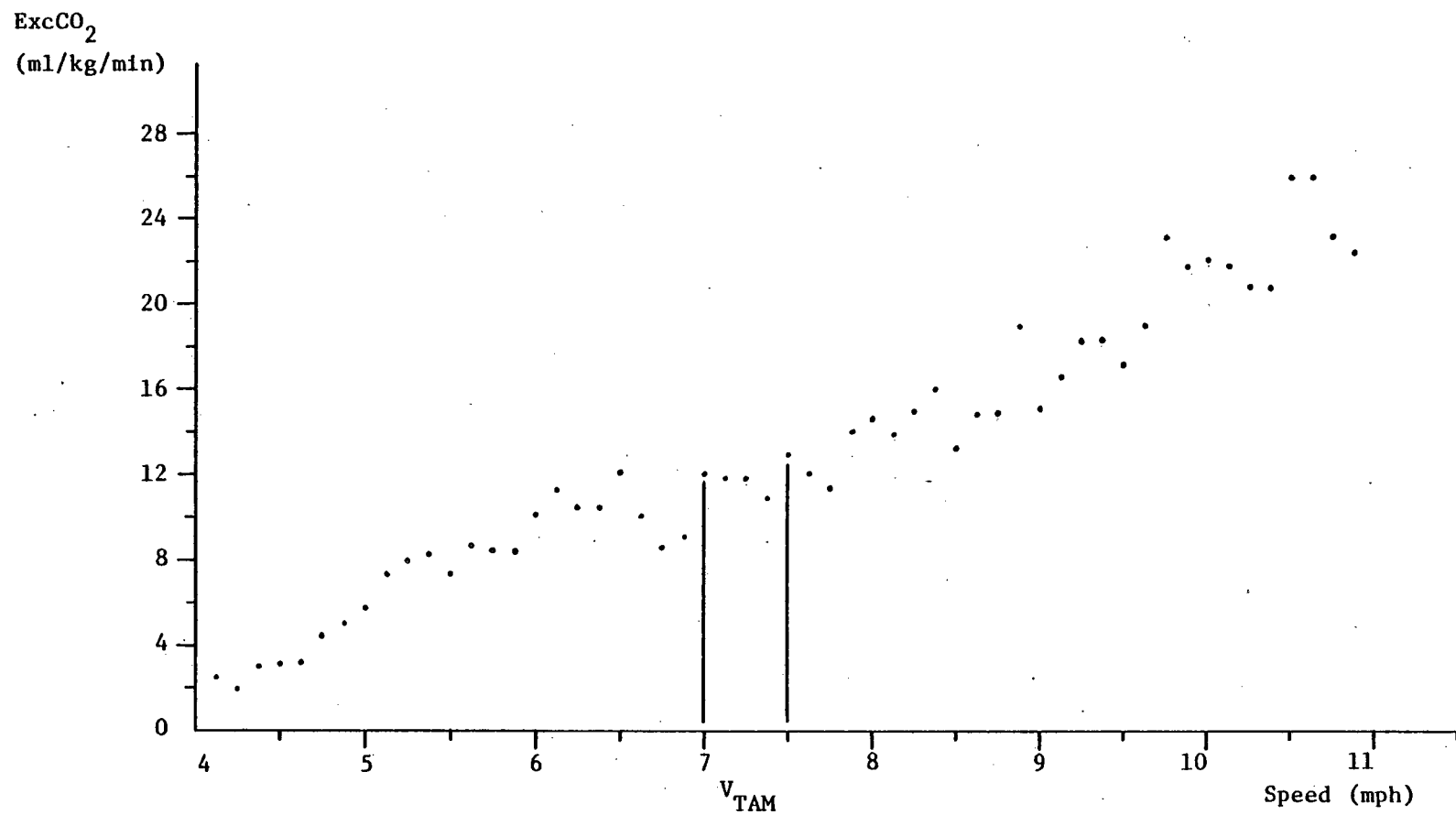


Figure 15. AT curve subject HB

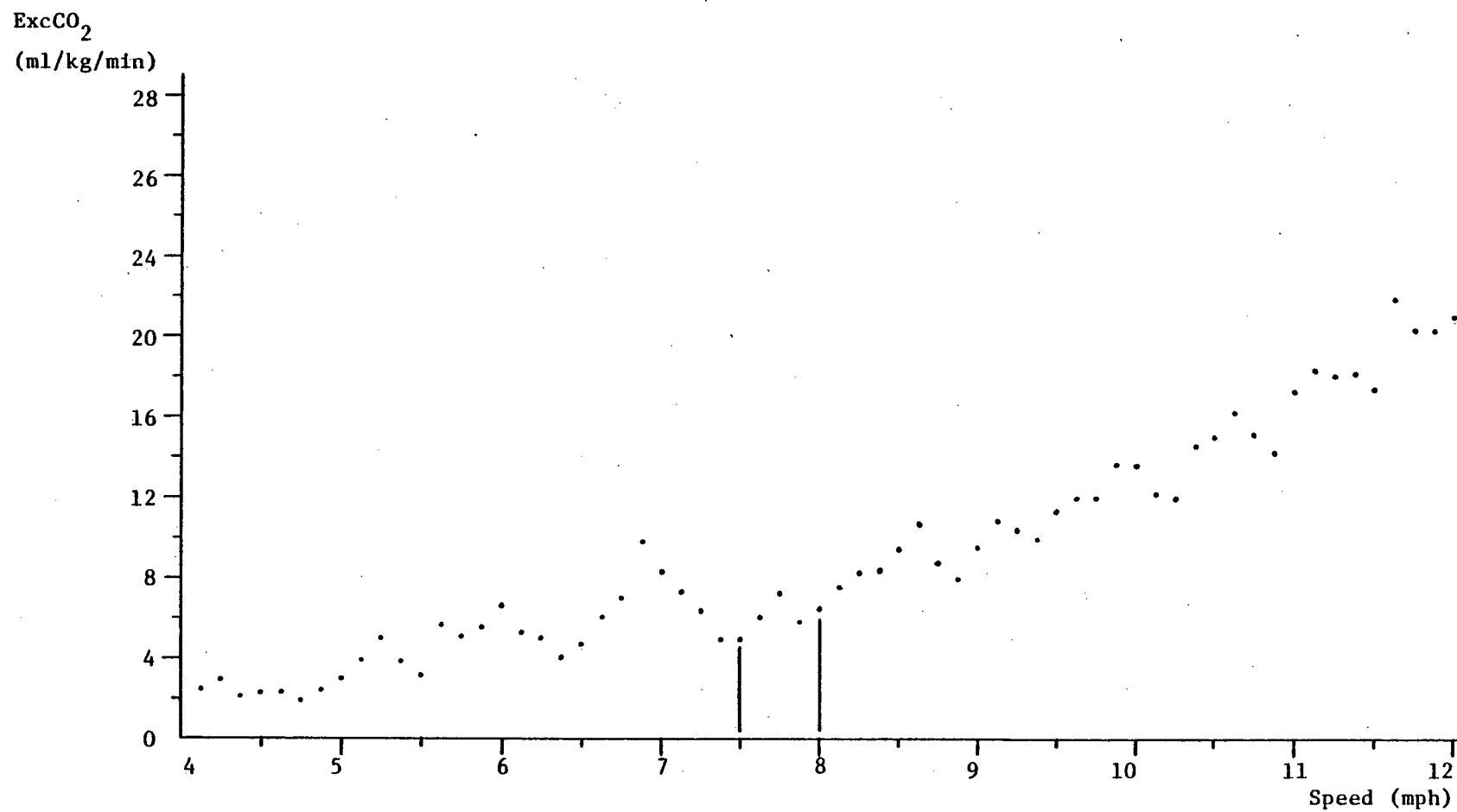


Figure 16. AT curve subject AB

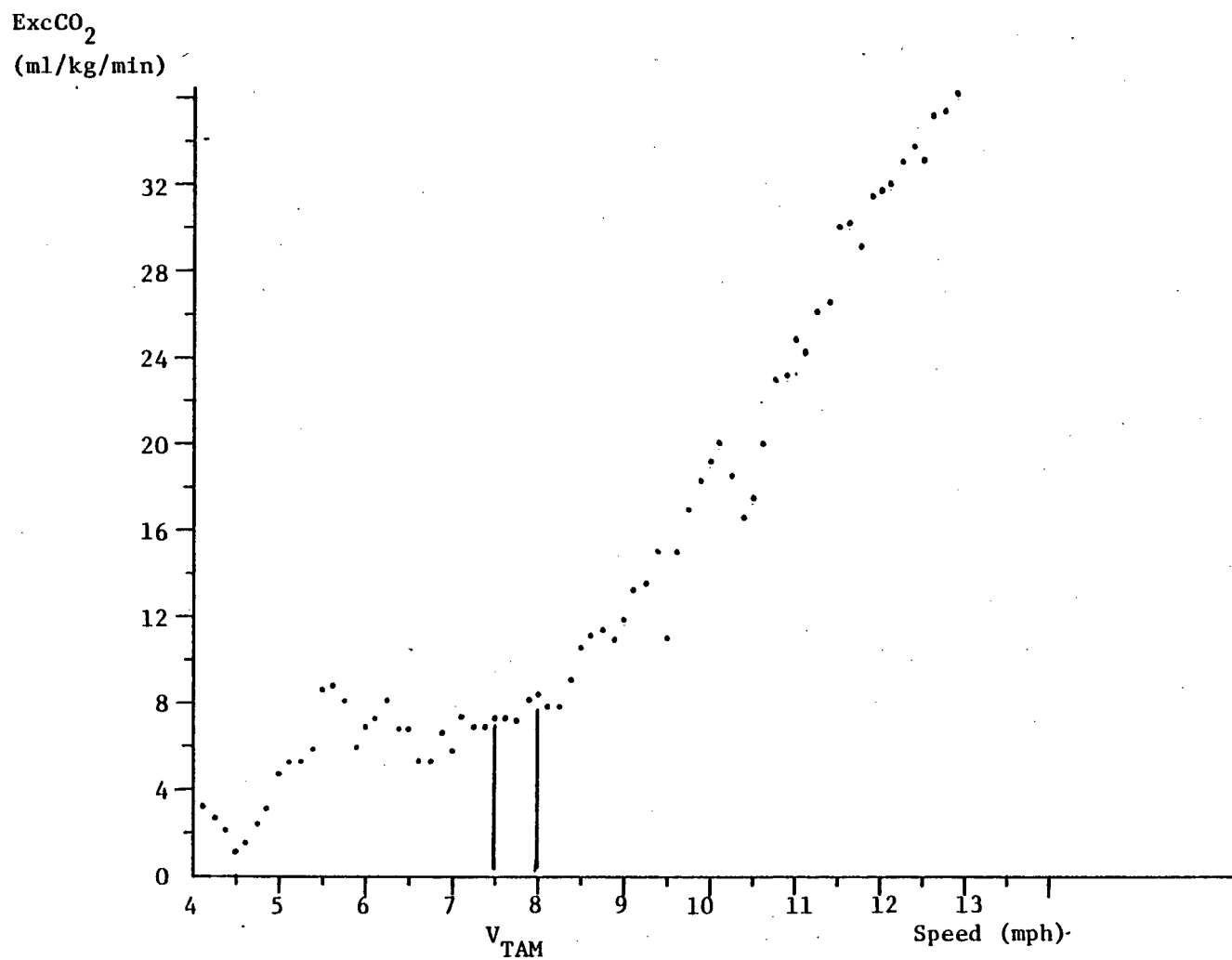


Figure 17. AT curve subject A0

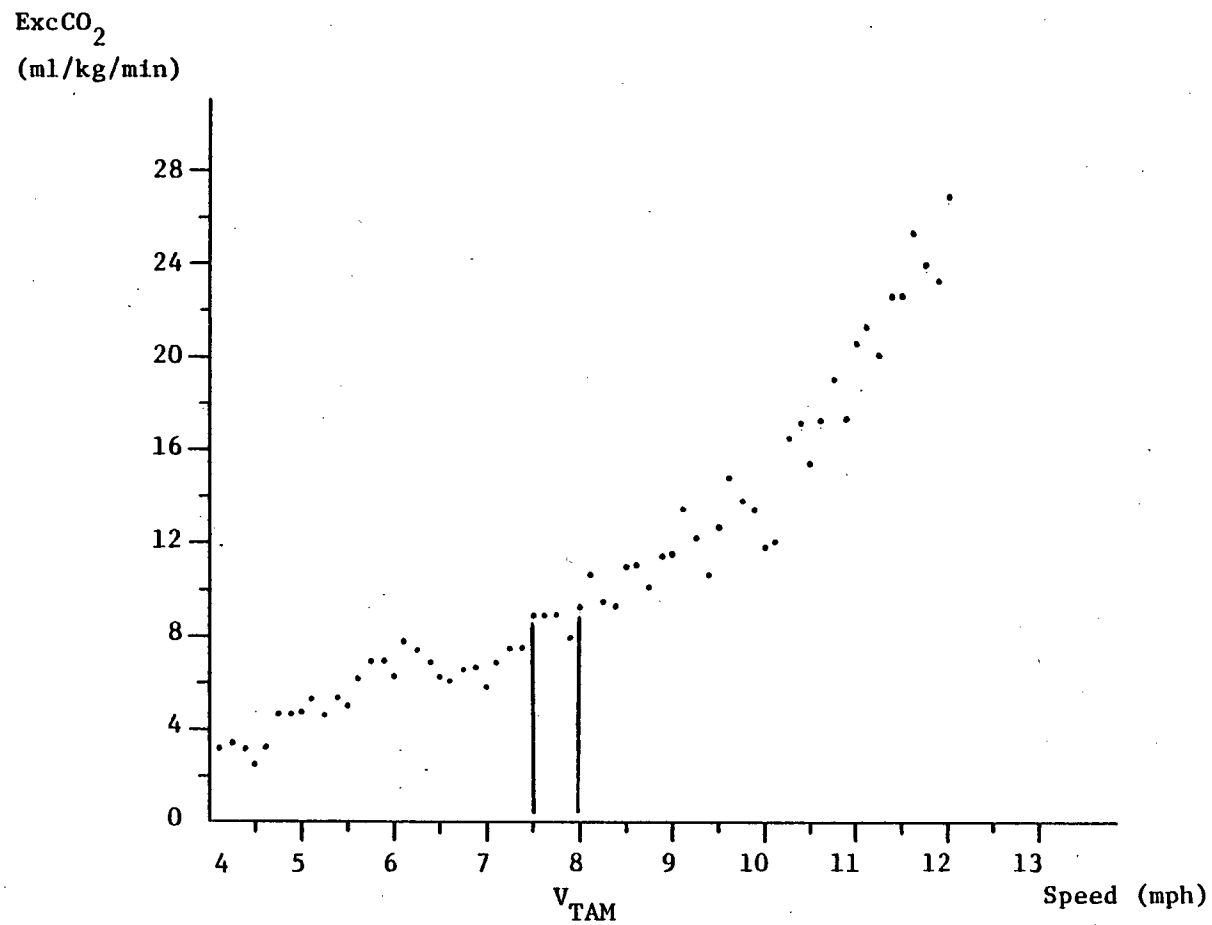


Figure 18. AT curve subject GT

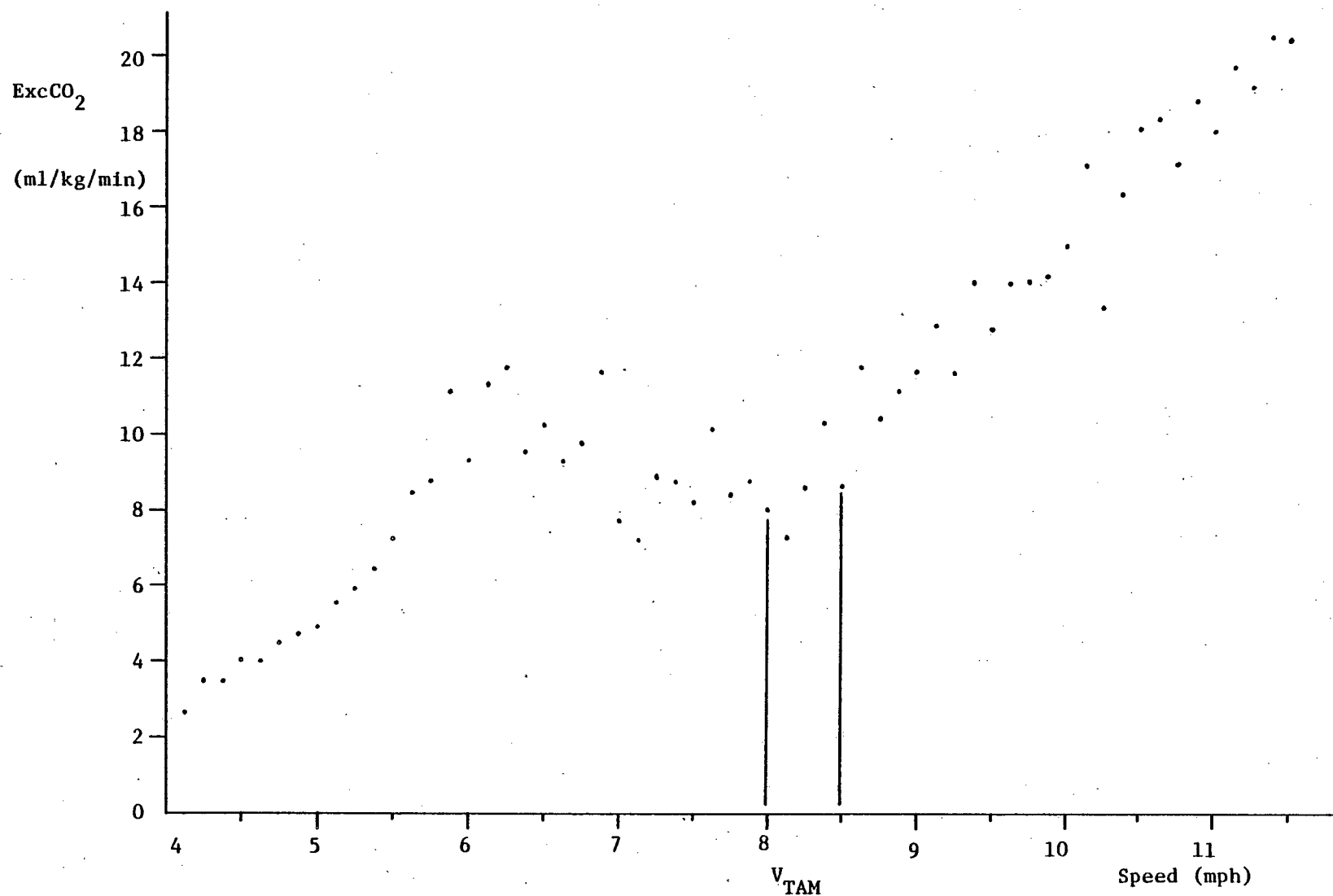


Figure 19. AT curve subject DH

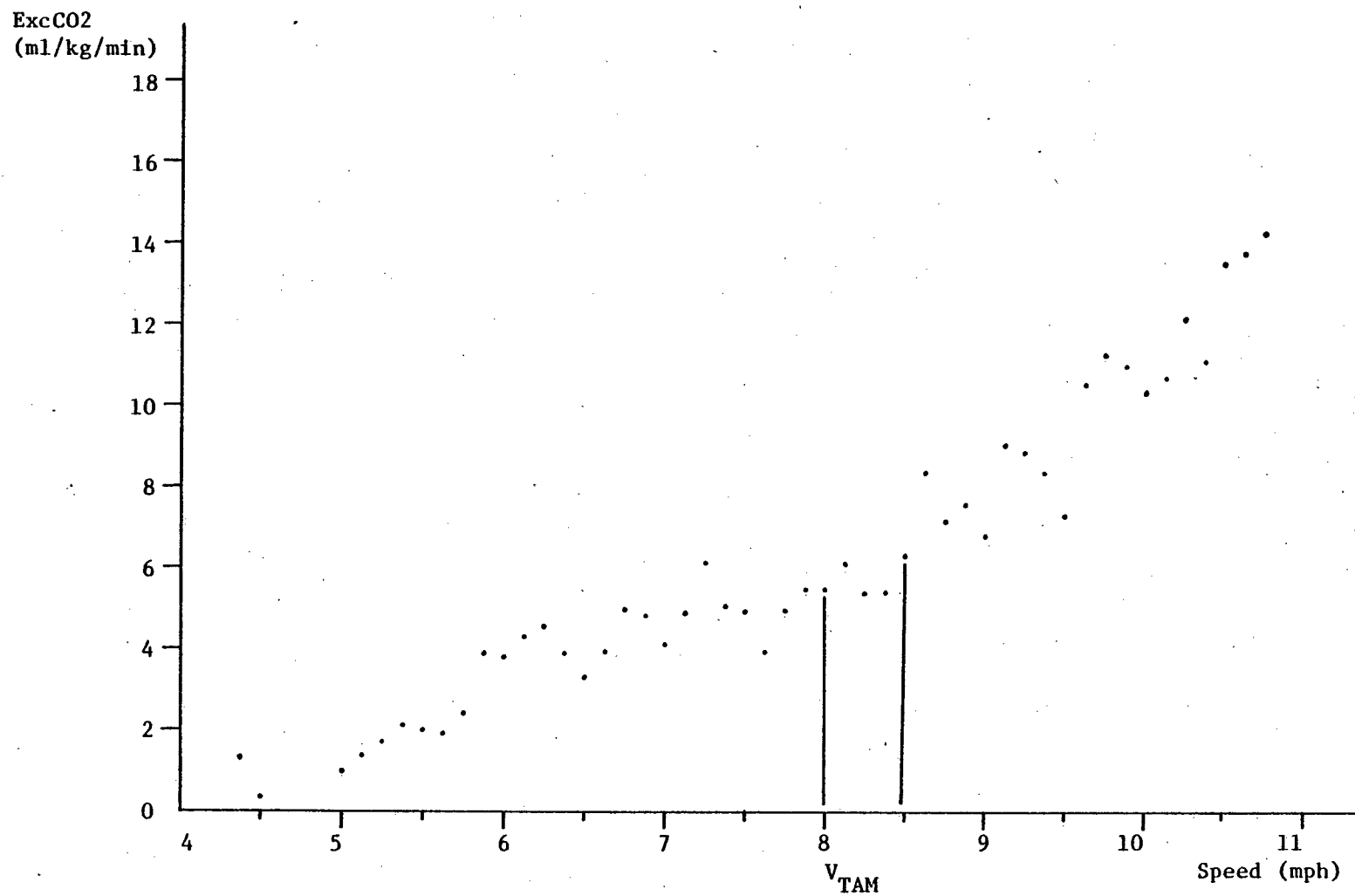


Figure 20. AT curve subject JL

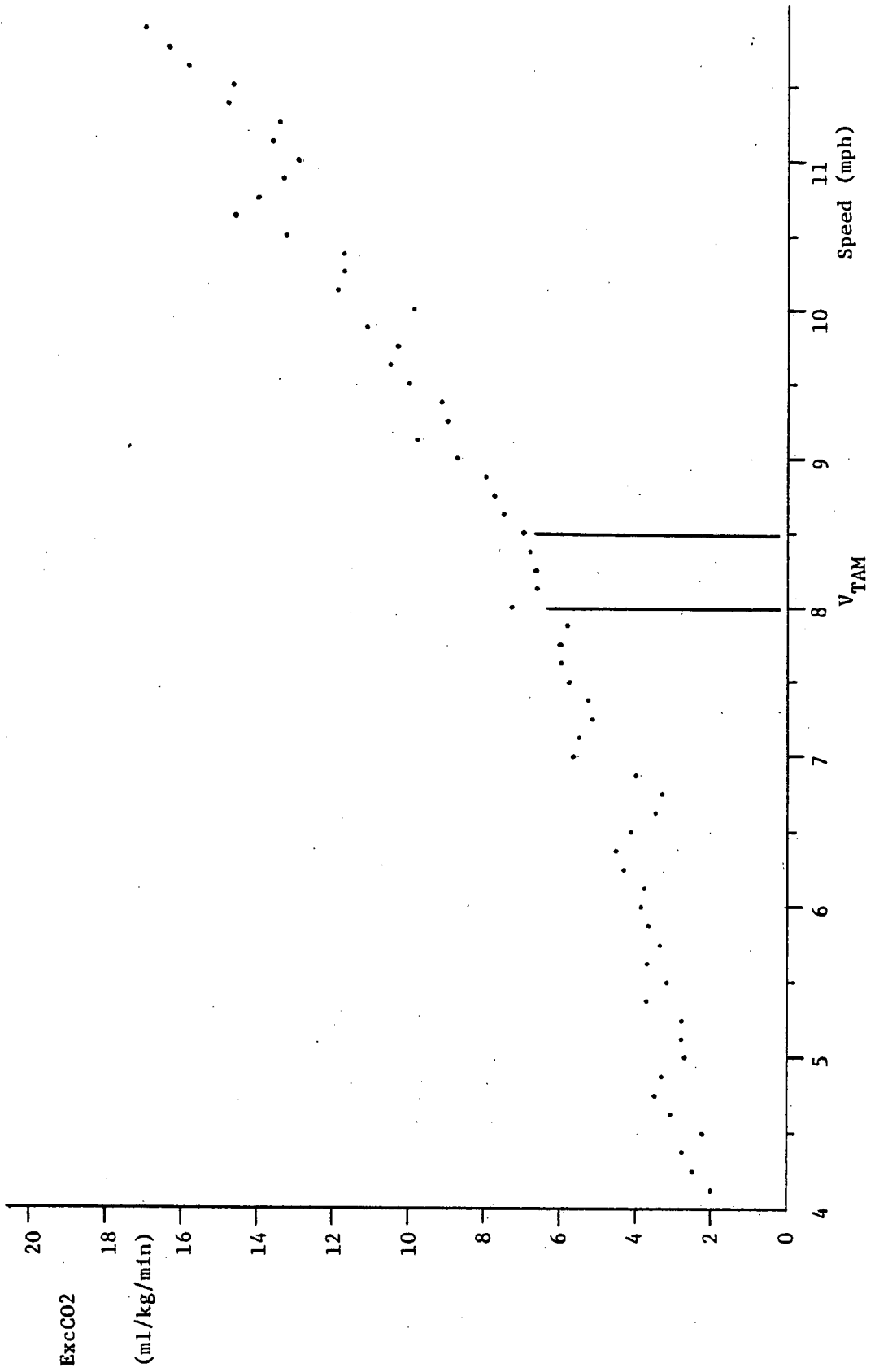


Figure 21. AT curve subject J0

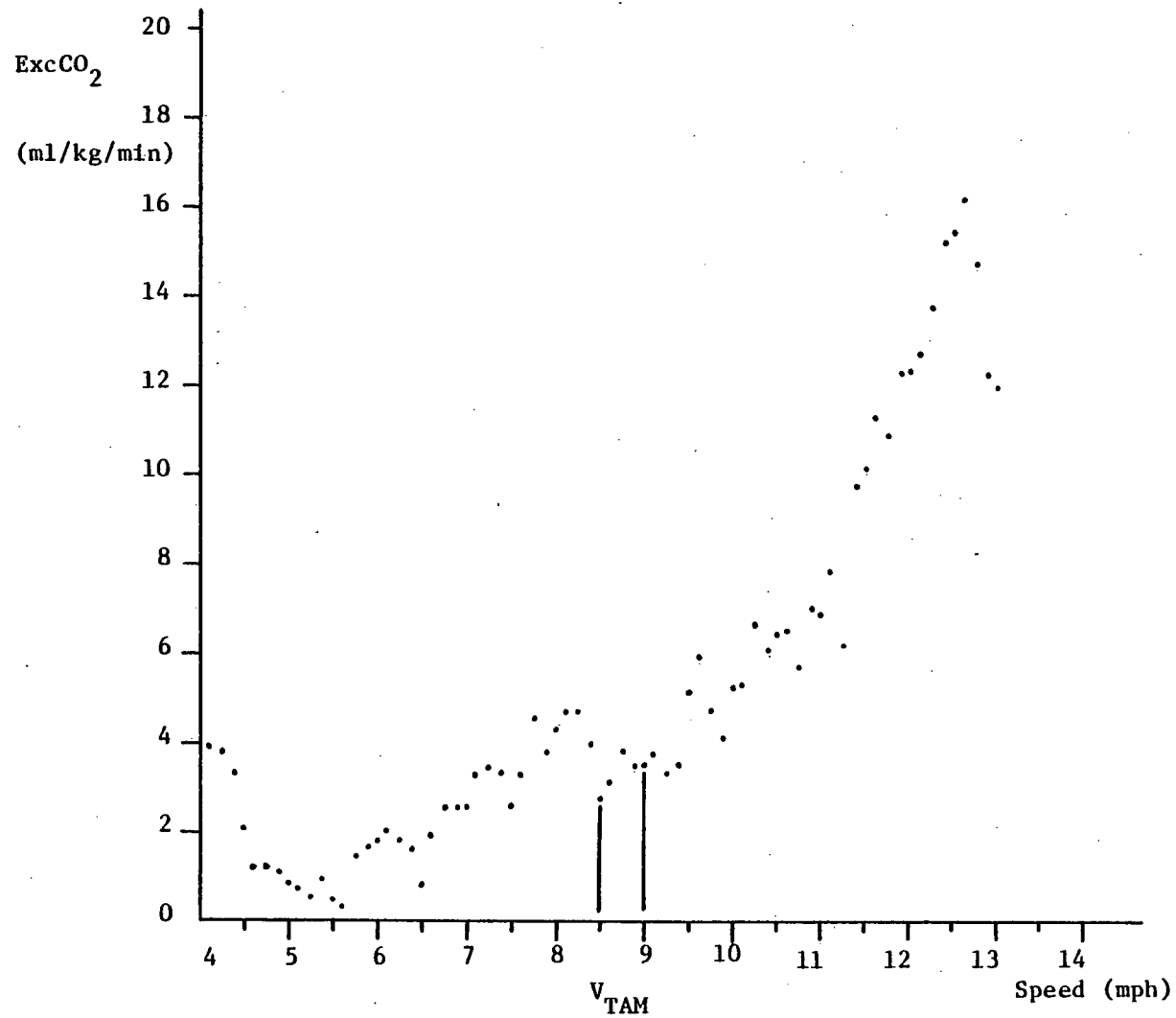


Figure 22. AT curve subject DM

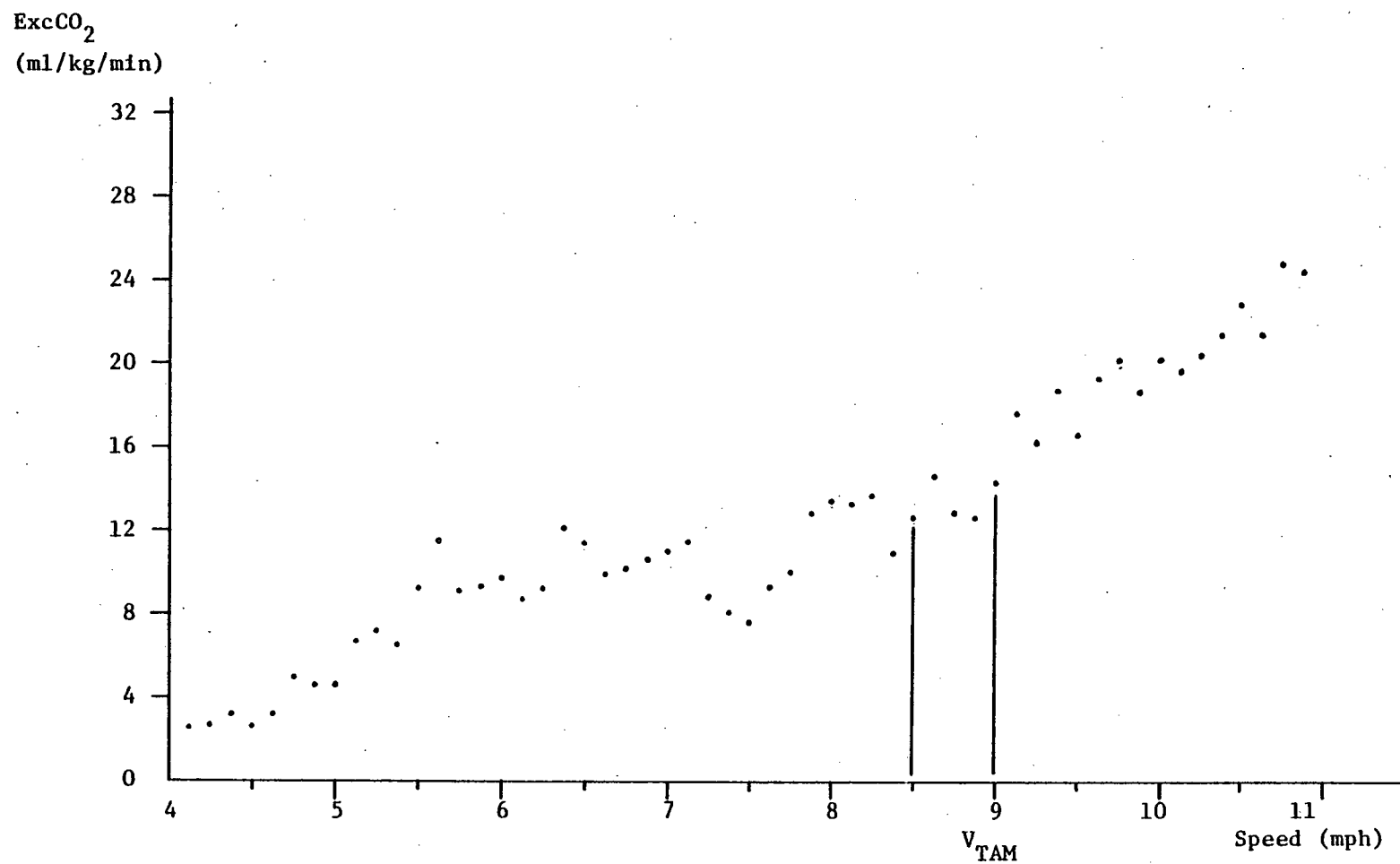


Figure 23. AT curve subject DW

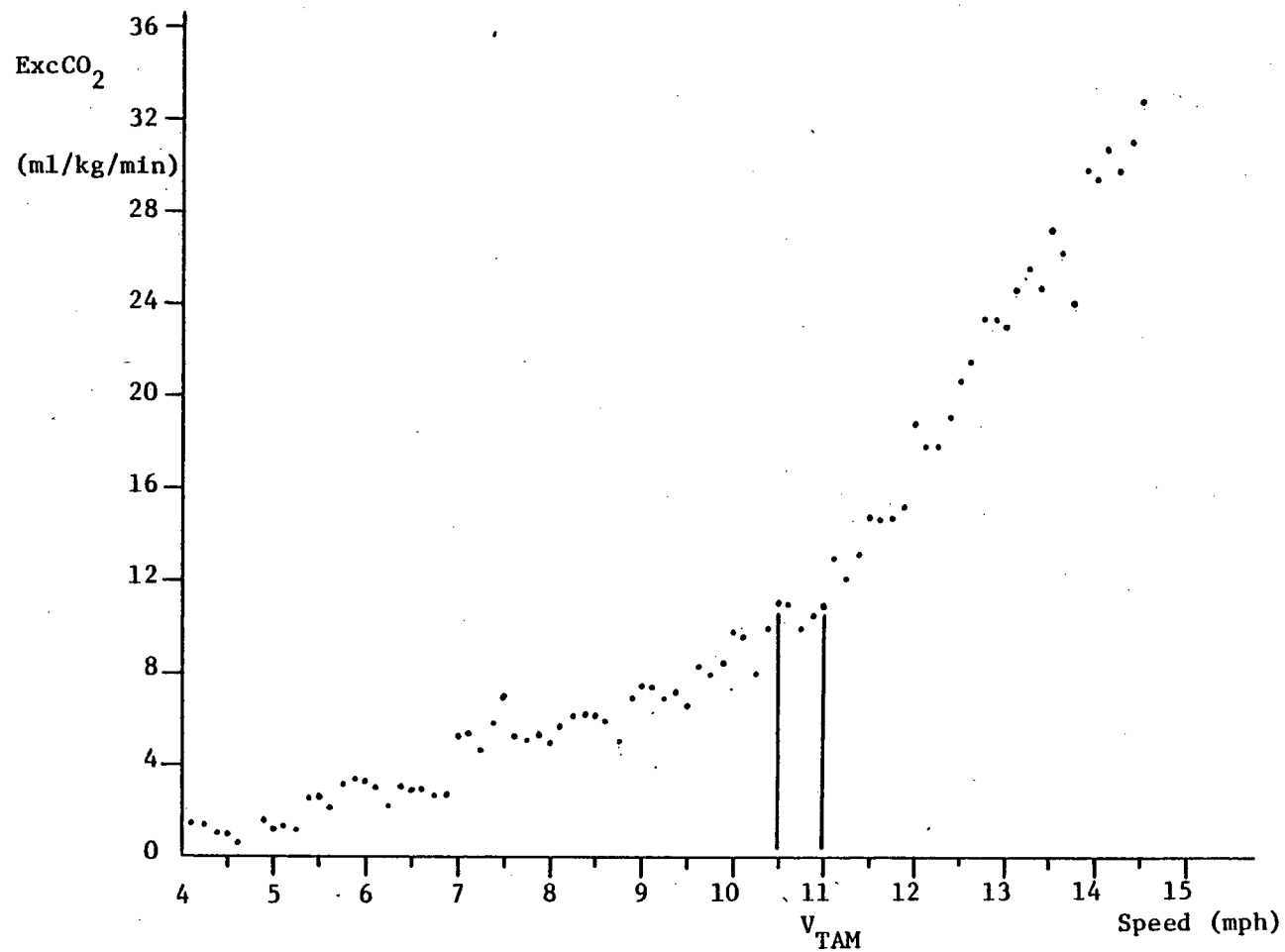


Figure 24. AT curve subject SP

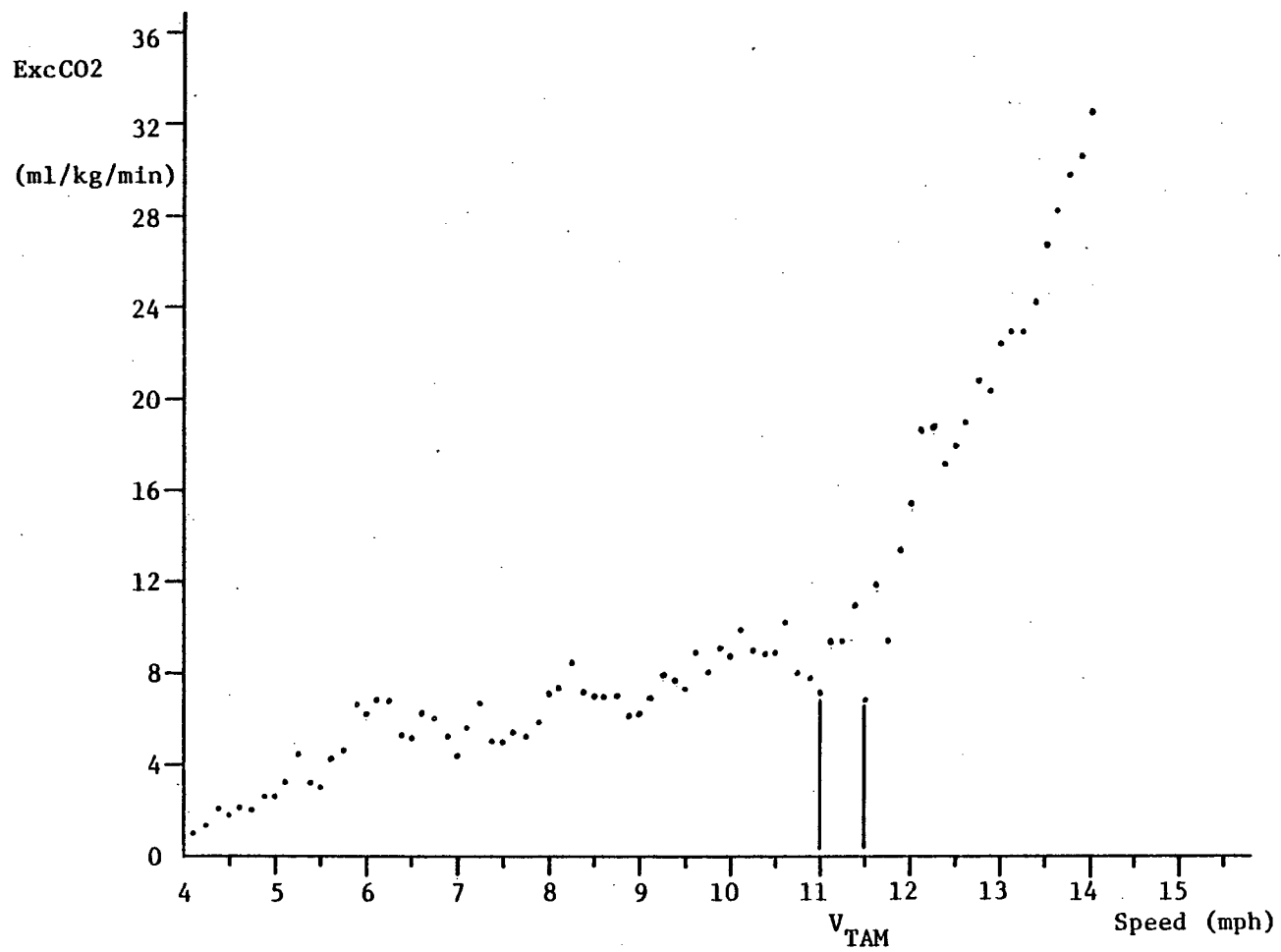


Figure 25. AT curve subject DH

APPENDIX C

Individual Oxygen Recovery Curves

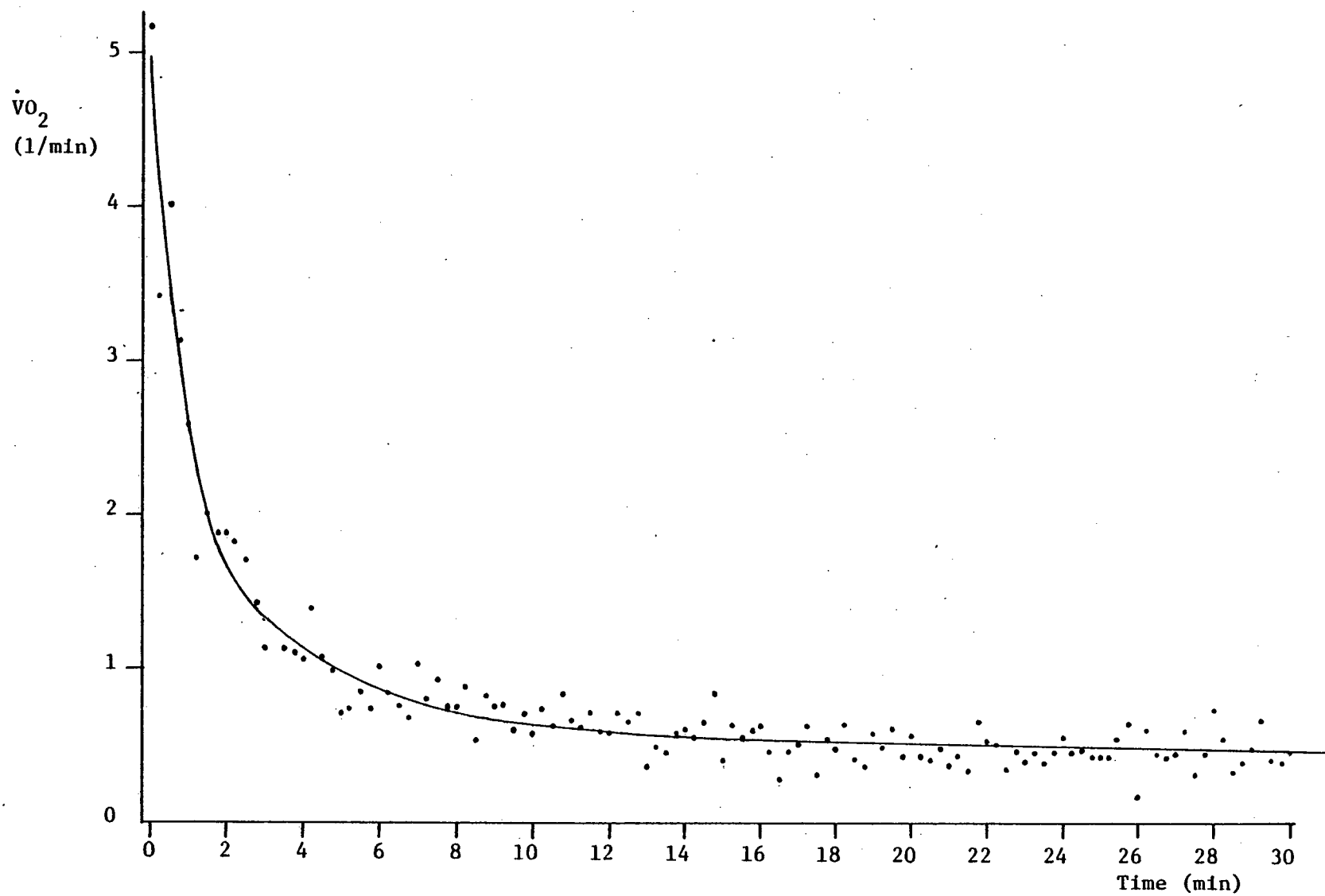


Figure 26. Recovery curve subject CN

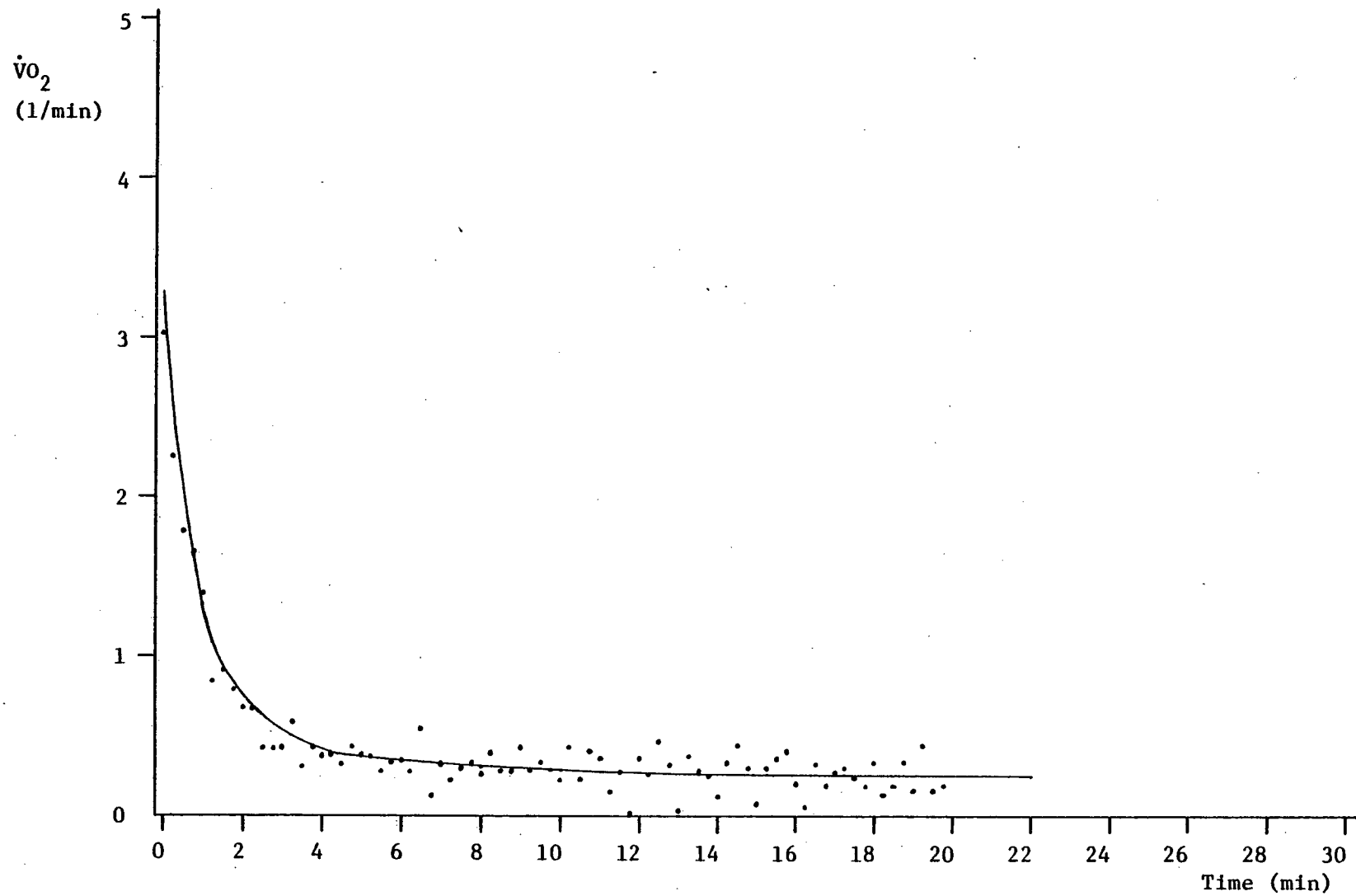


Figure 27. Recovery curve subject ML

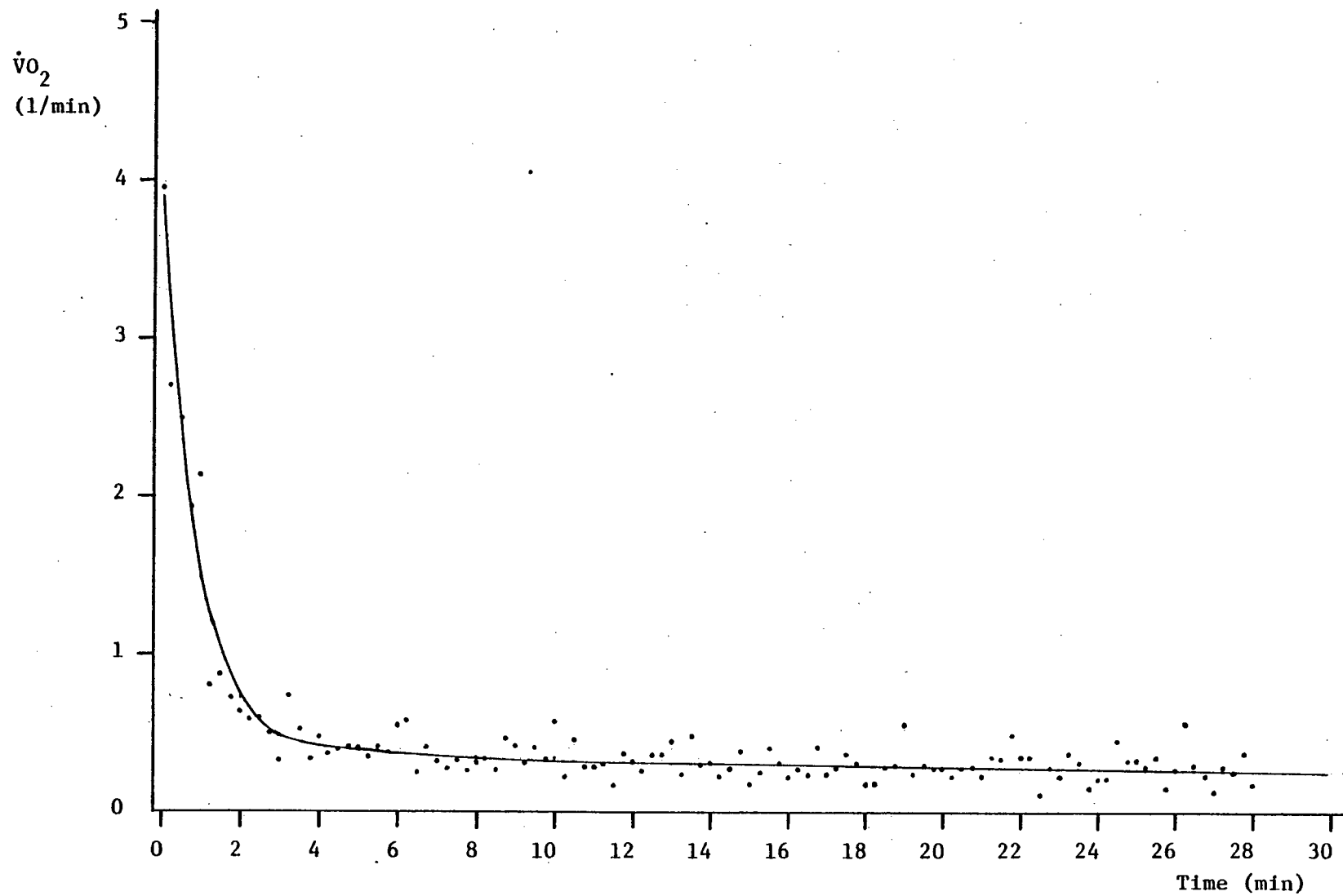


Figure 28. Recovery curve subject RR

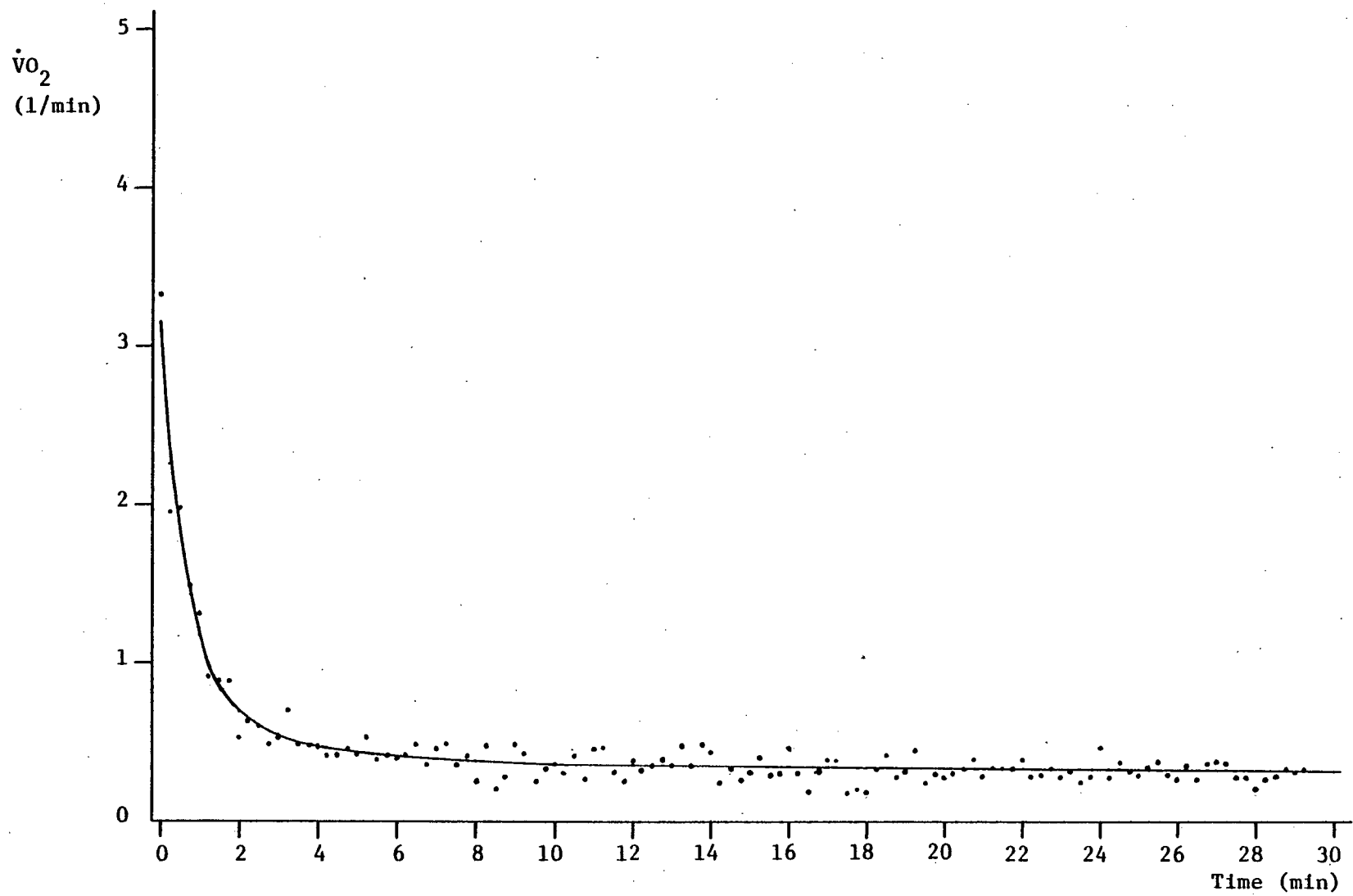


Figure 29. Recovery curve subject TB

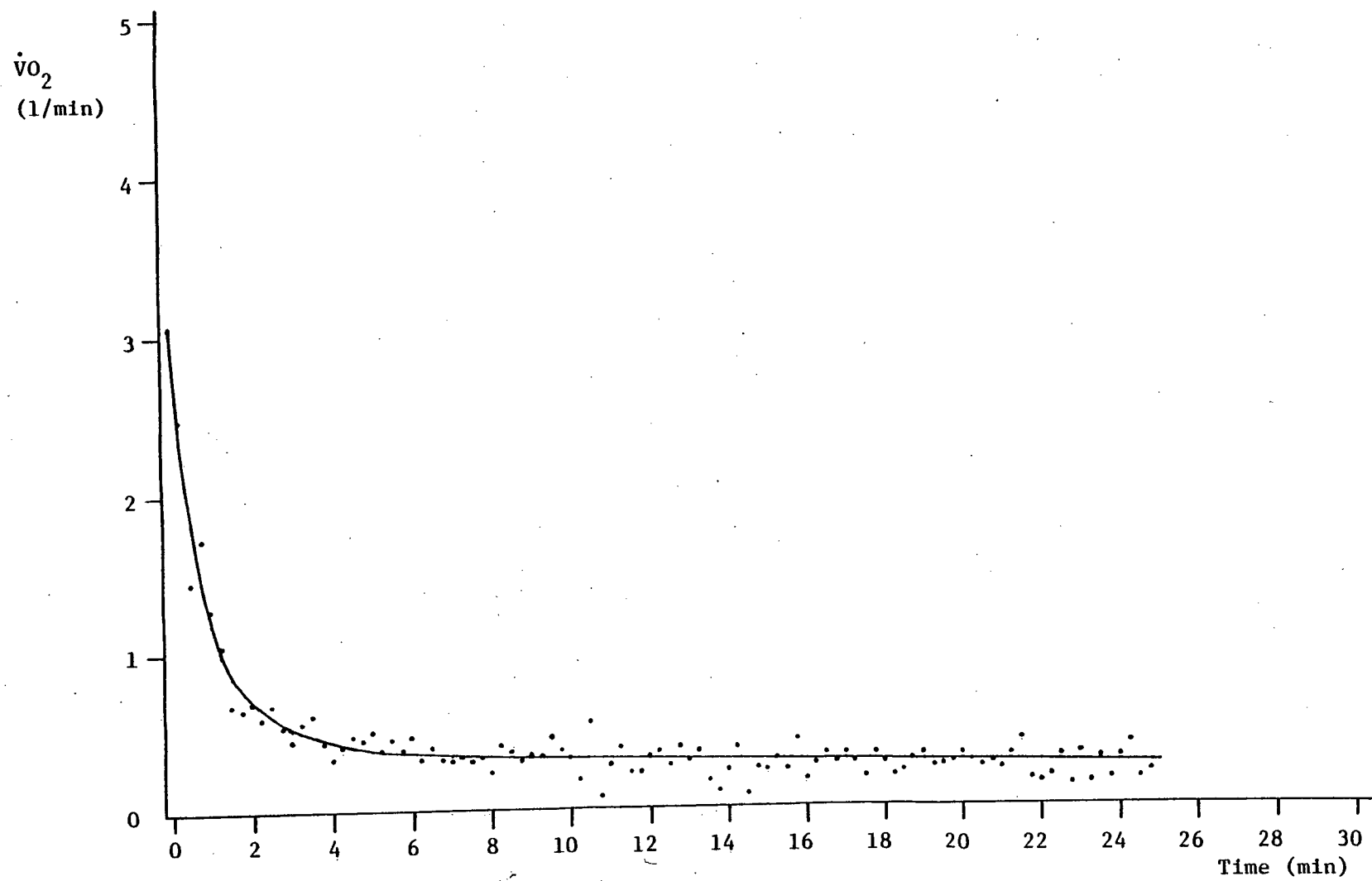


Figure 30. Recovery curve subject DG

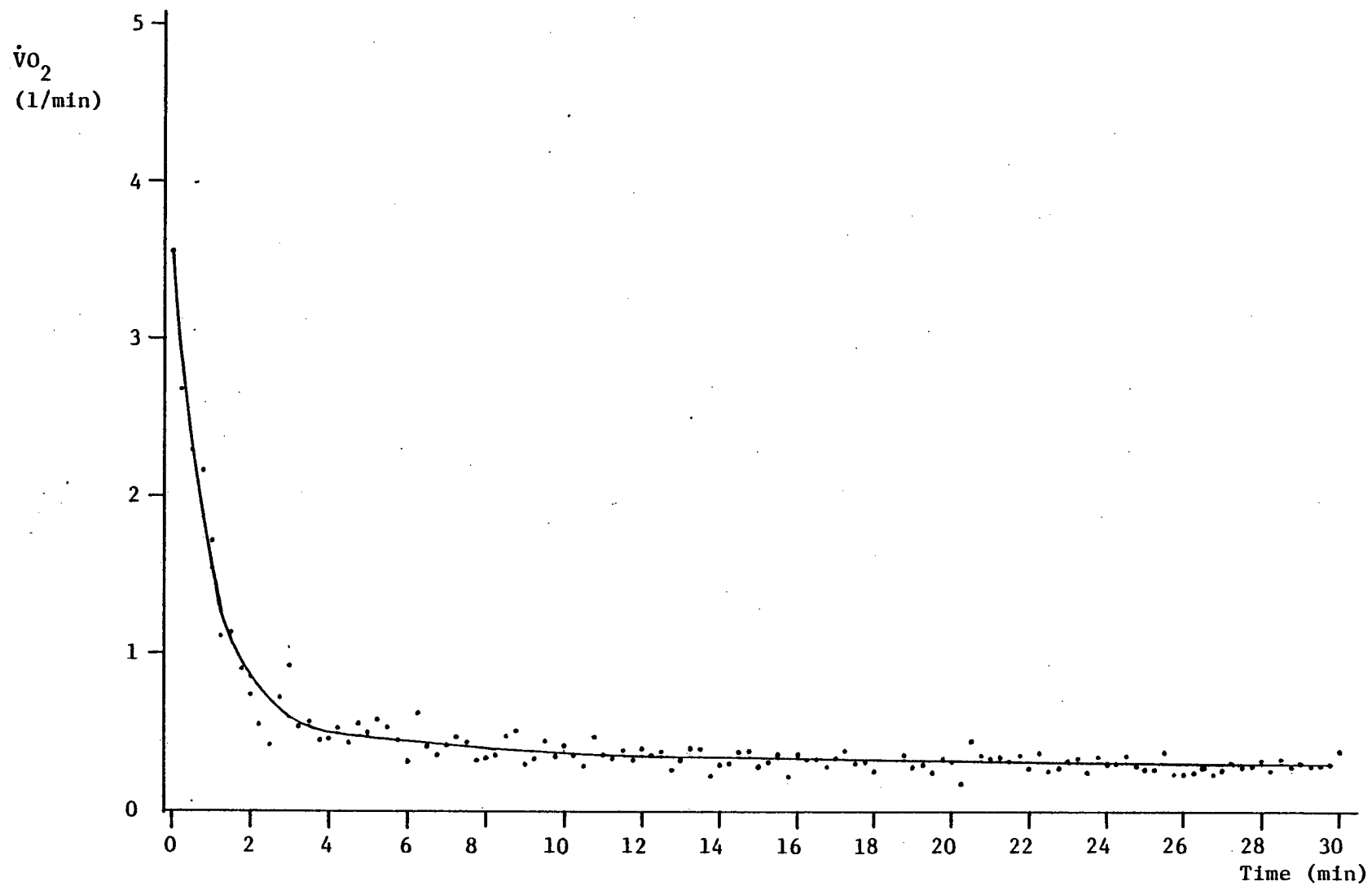


Figure 31. Recovery curve subject DD

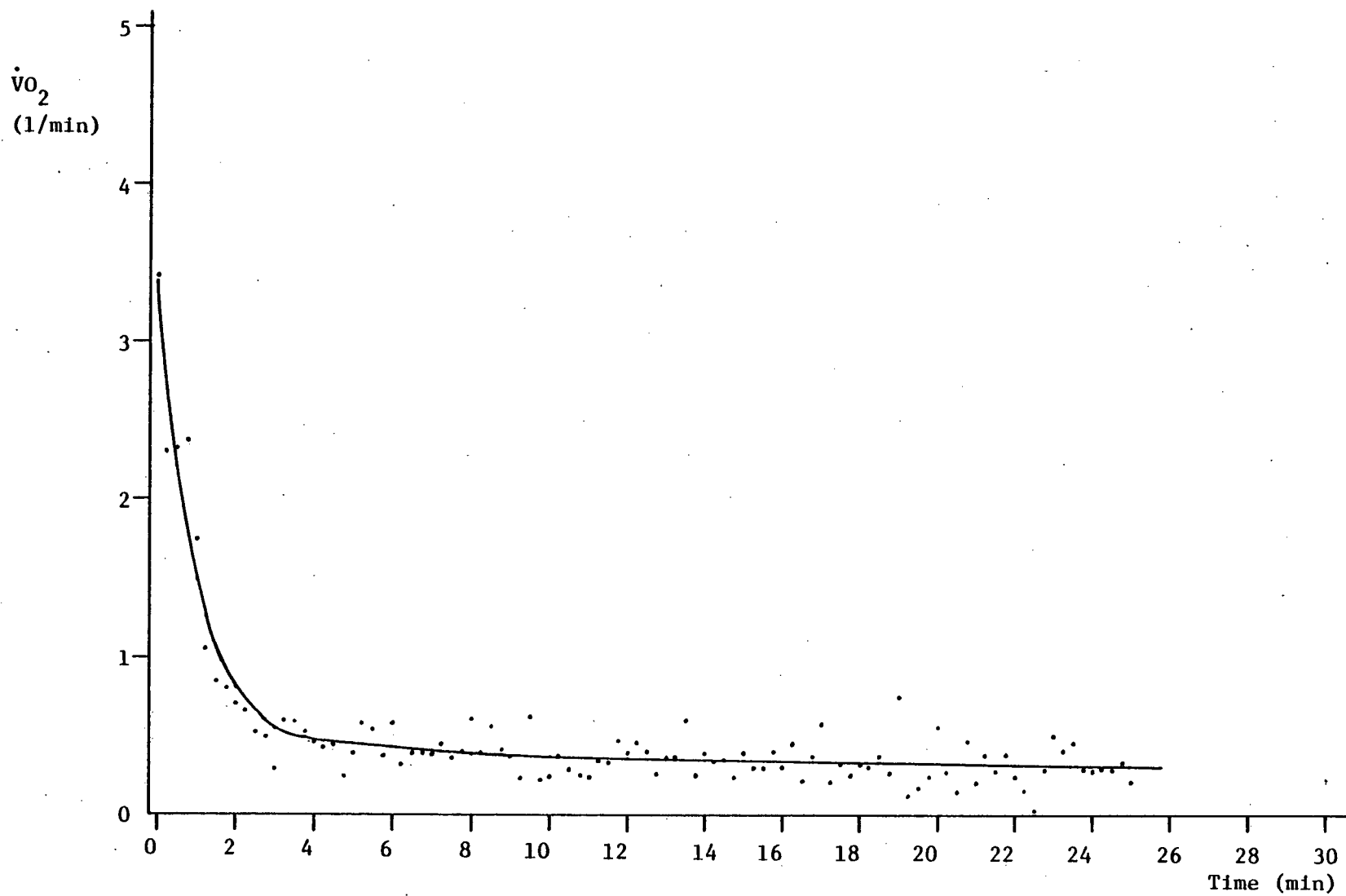


Figure 32. Recovery curve subject BV

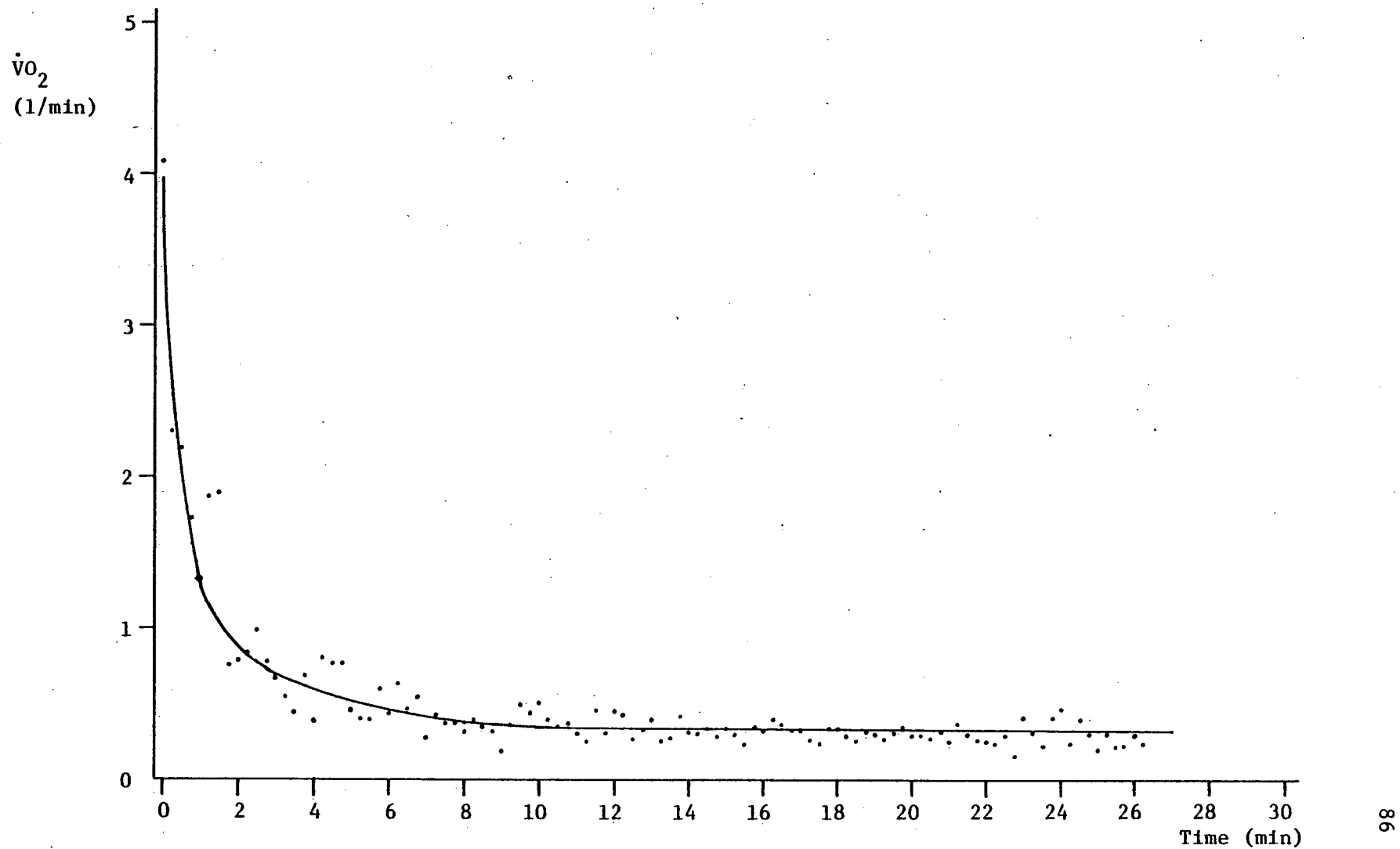


Figure 33. Recovery curve subject GS

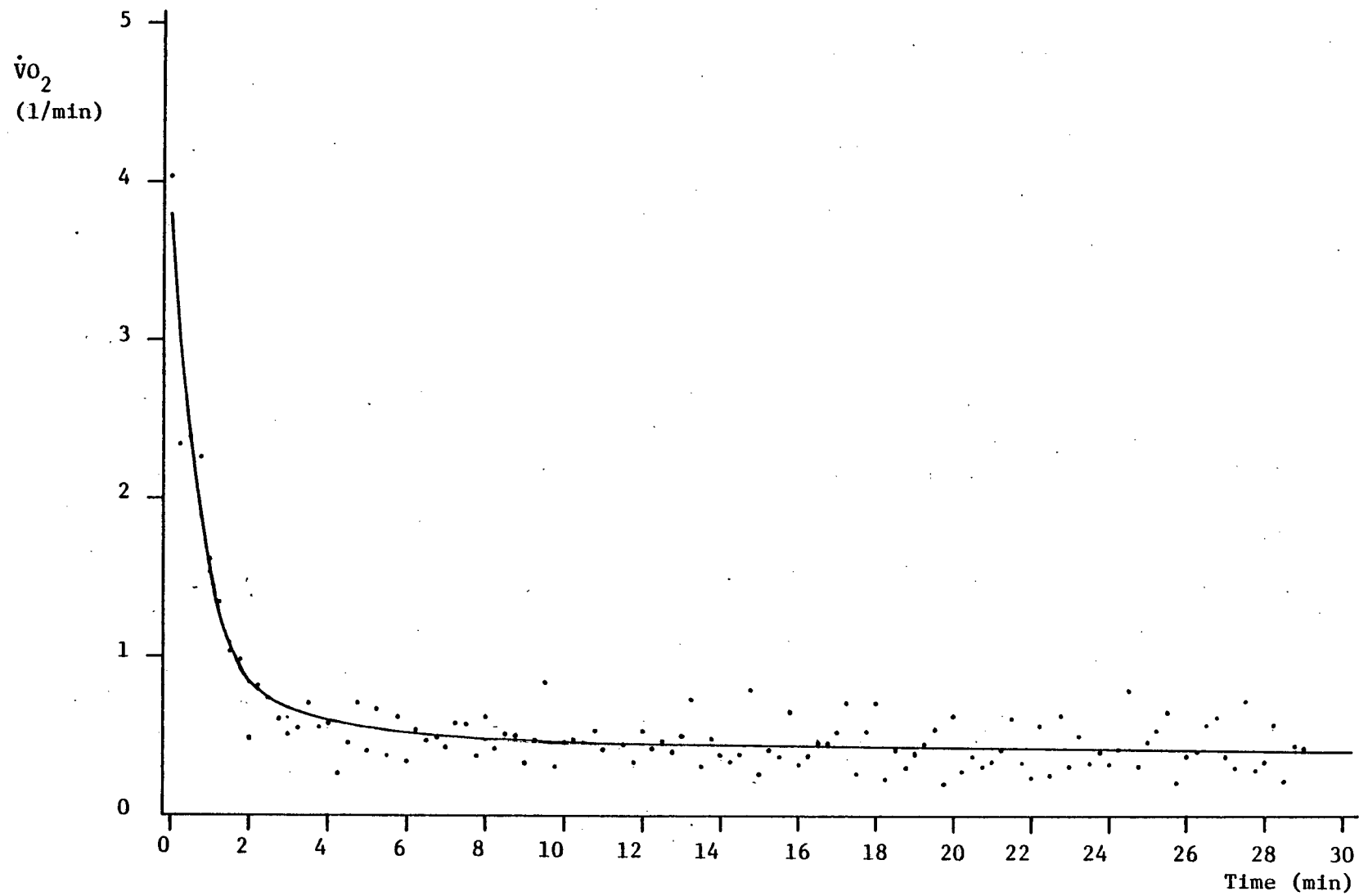


Figure 34. Recovery curve subject DA

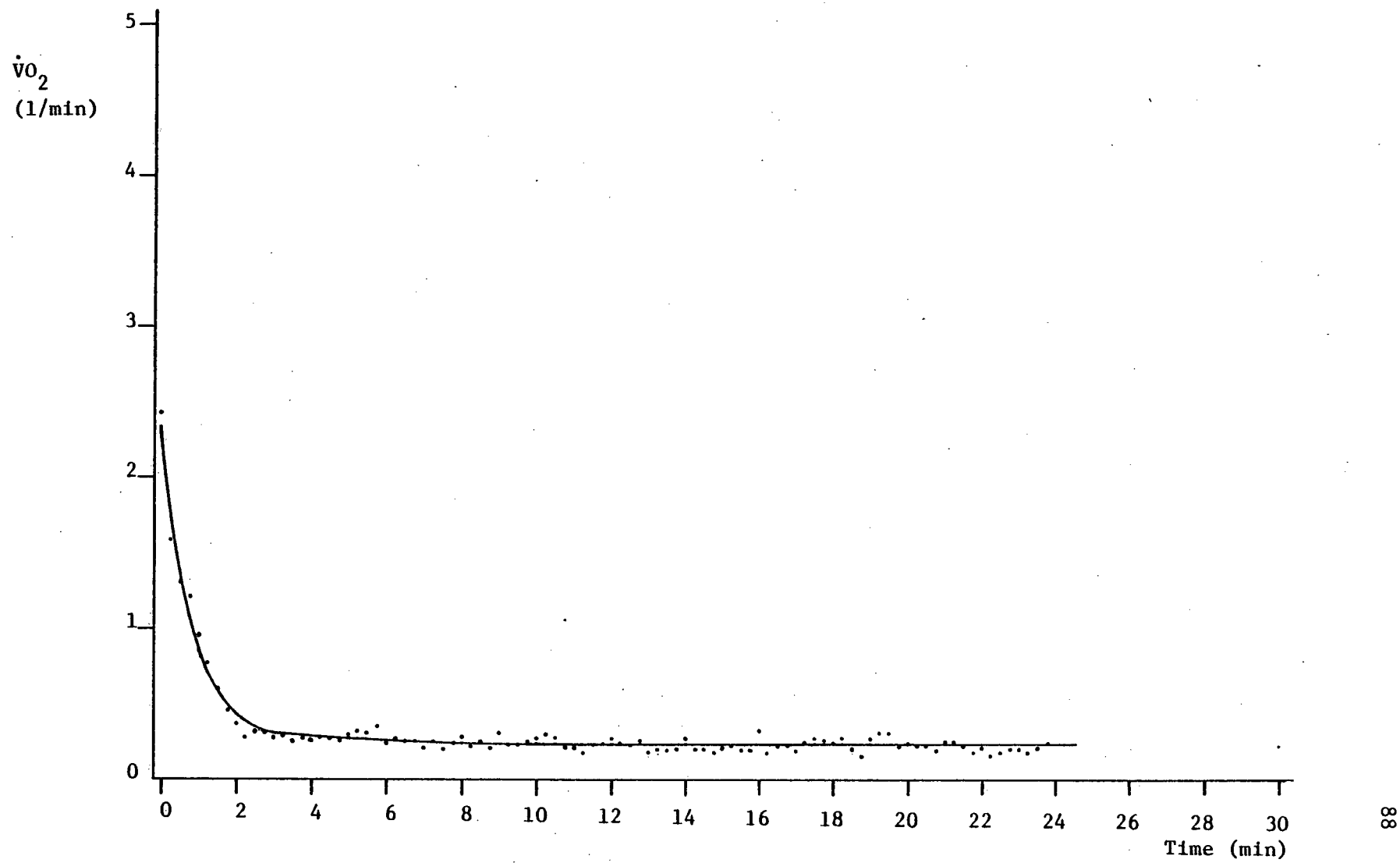


Figure 35. Recovery curve subject RF.

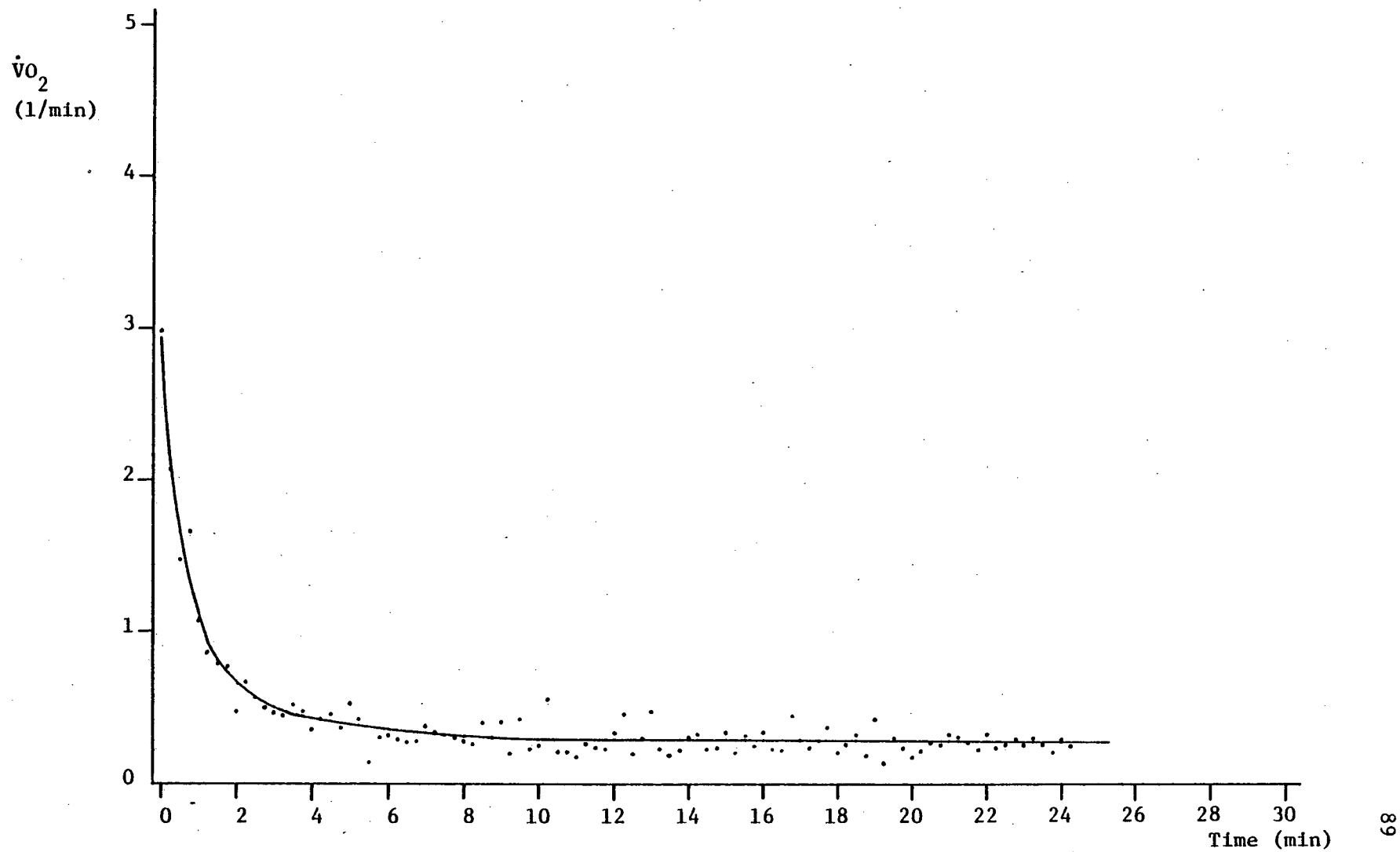


Figure 36. Recovery curve subject HB

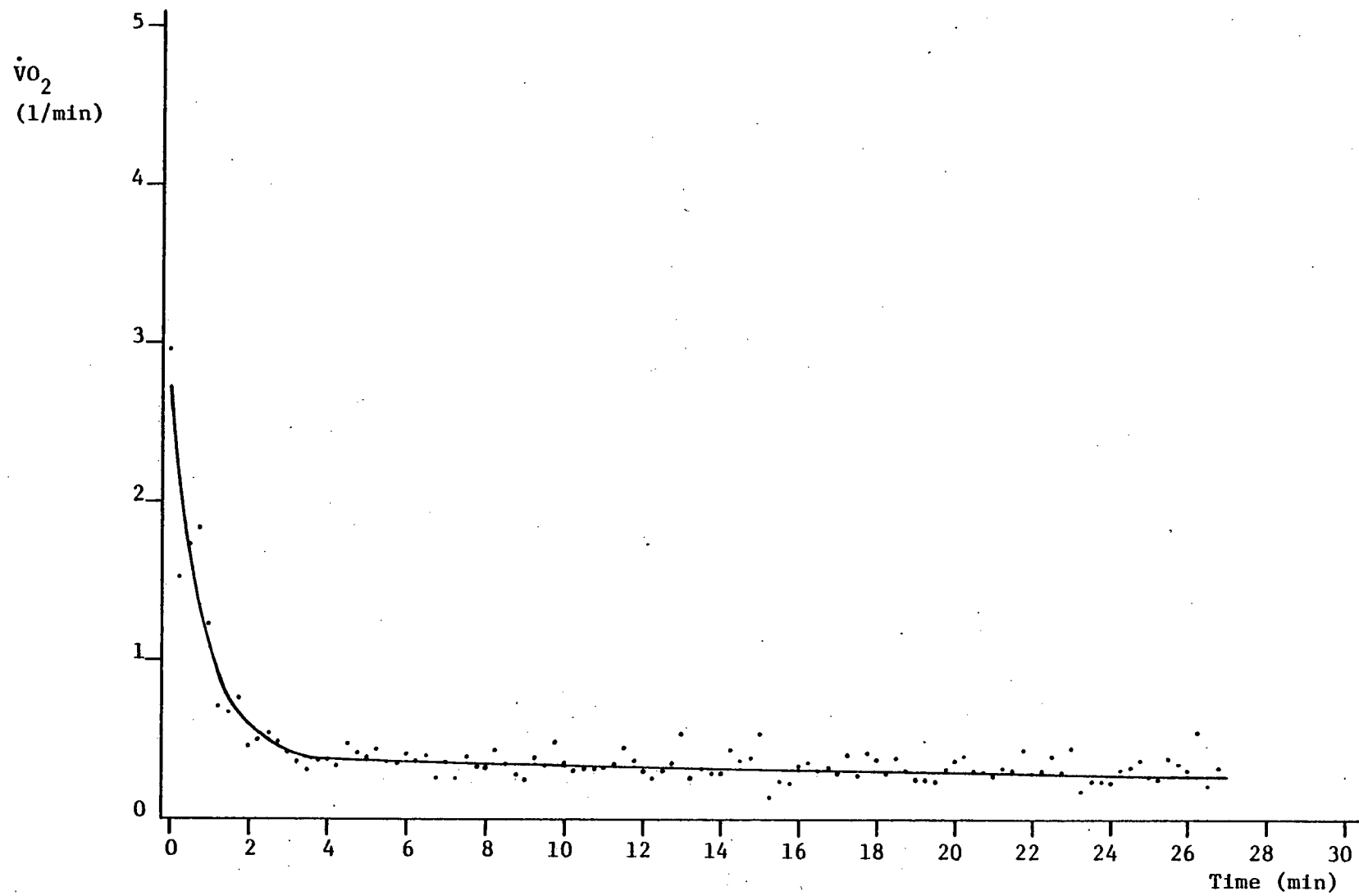


Figure 37. Recovery curve subject AB

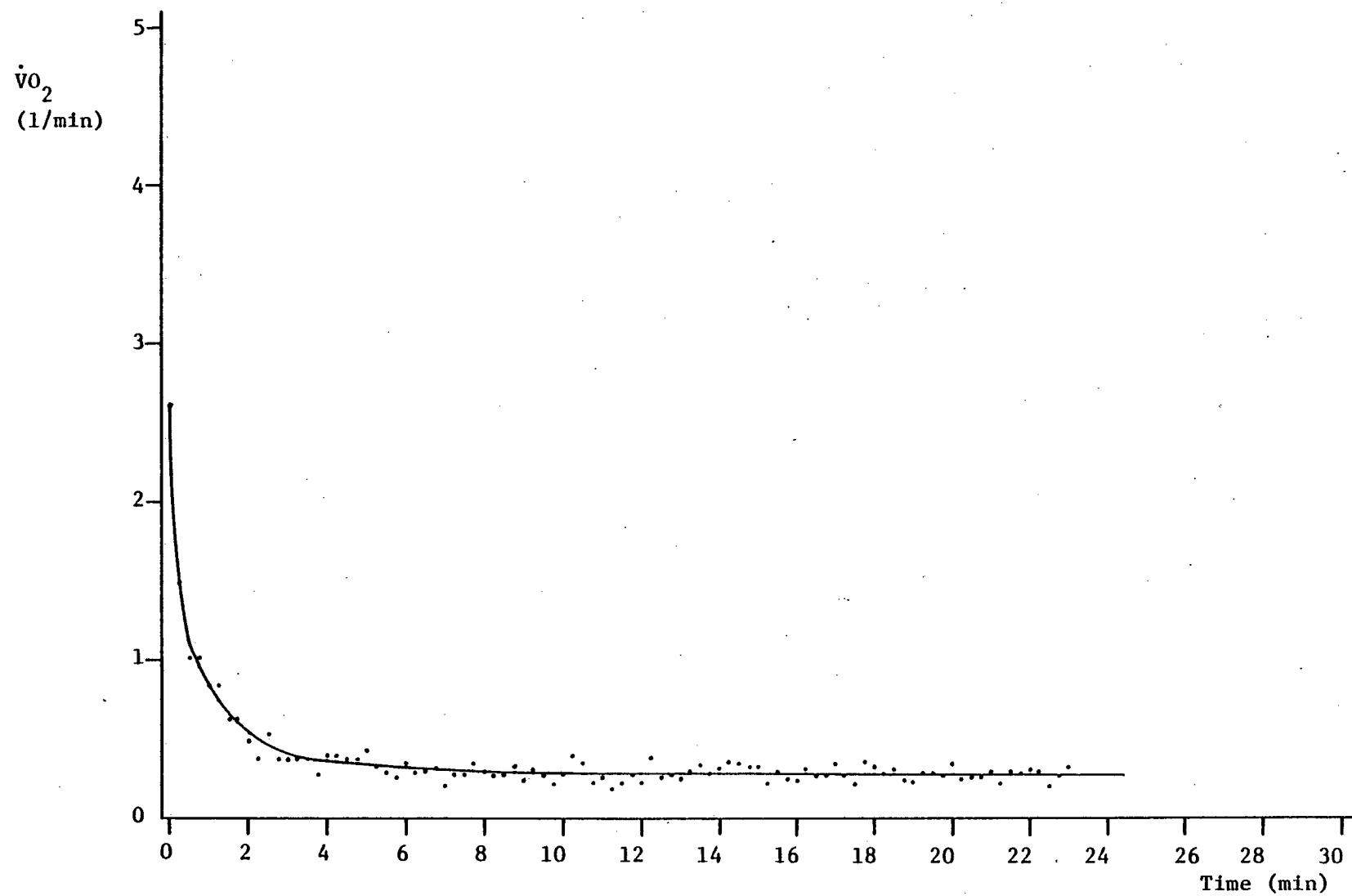


Figure 38. Recovery curve subject A0

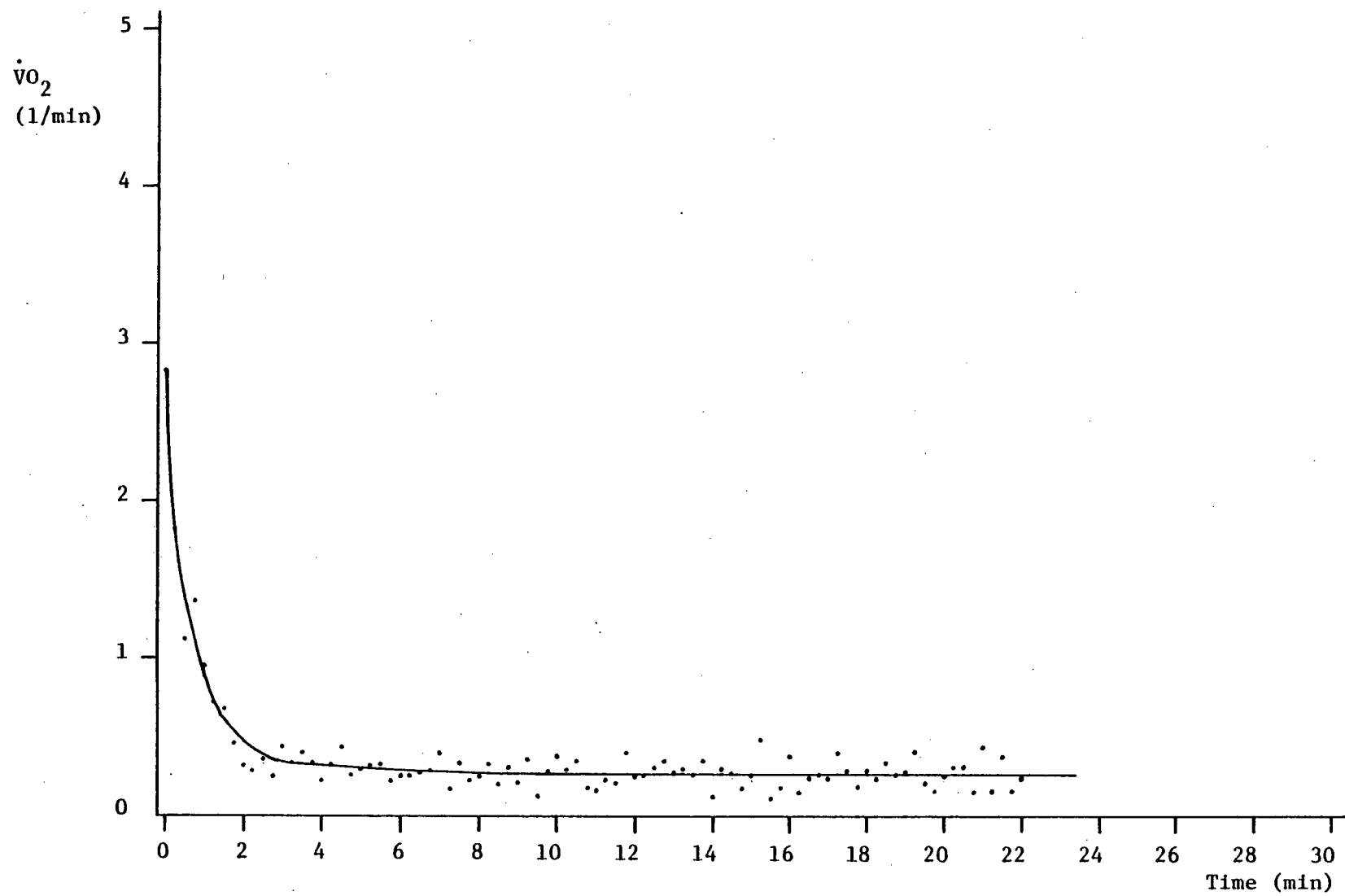


Figure 39. Recovery curve subject GT

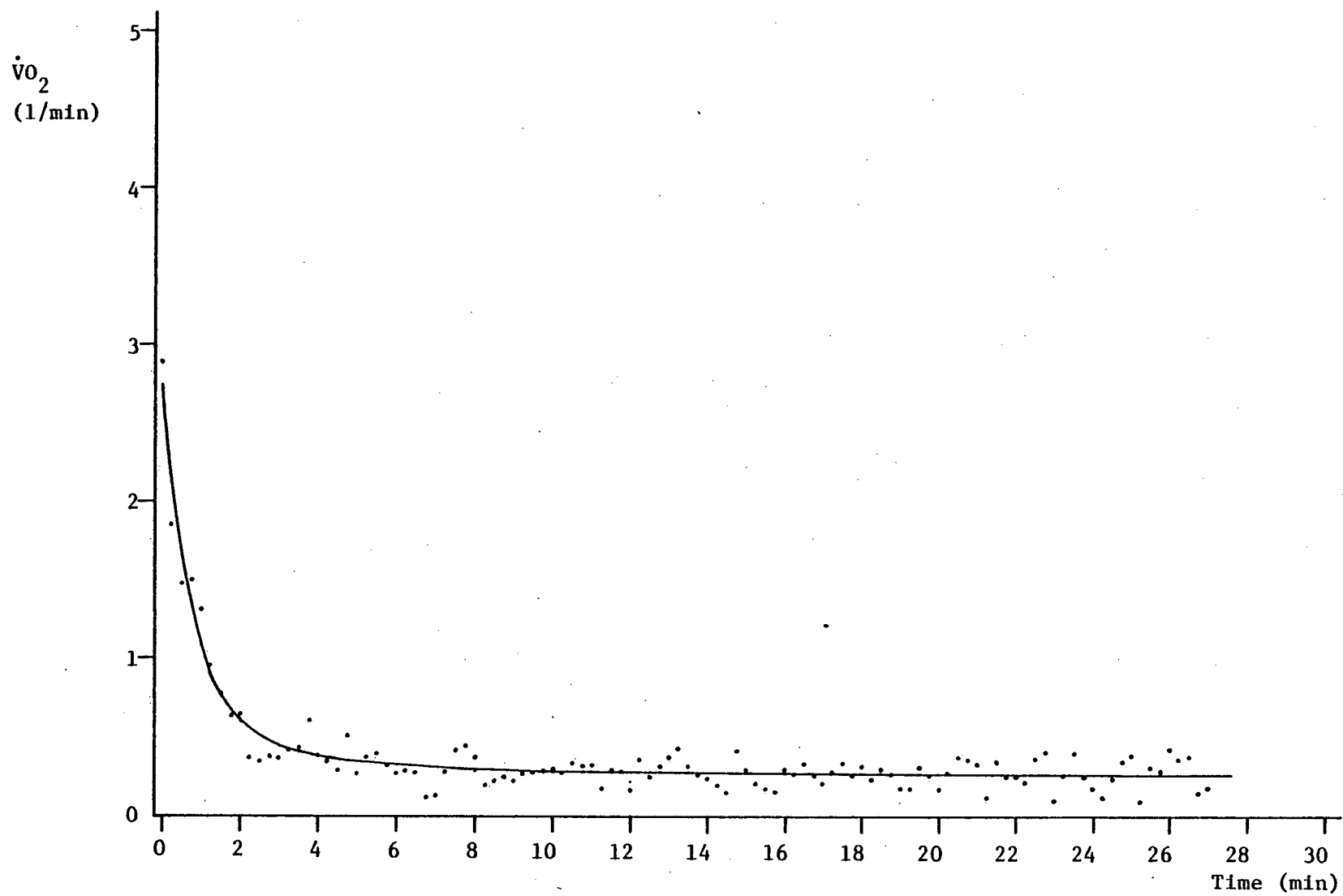


Figure 40. Recovery curve subject DH

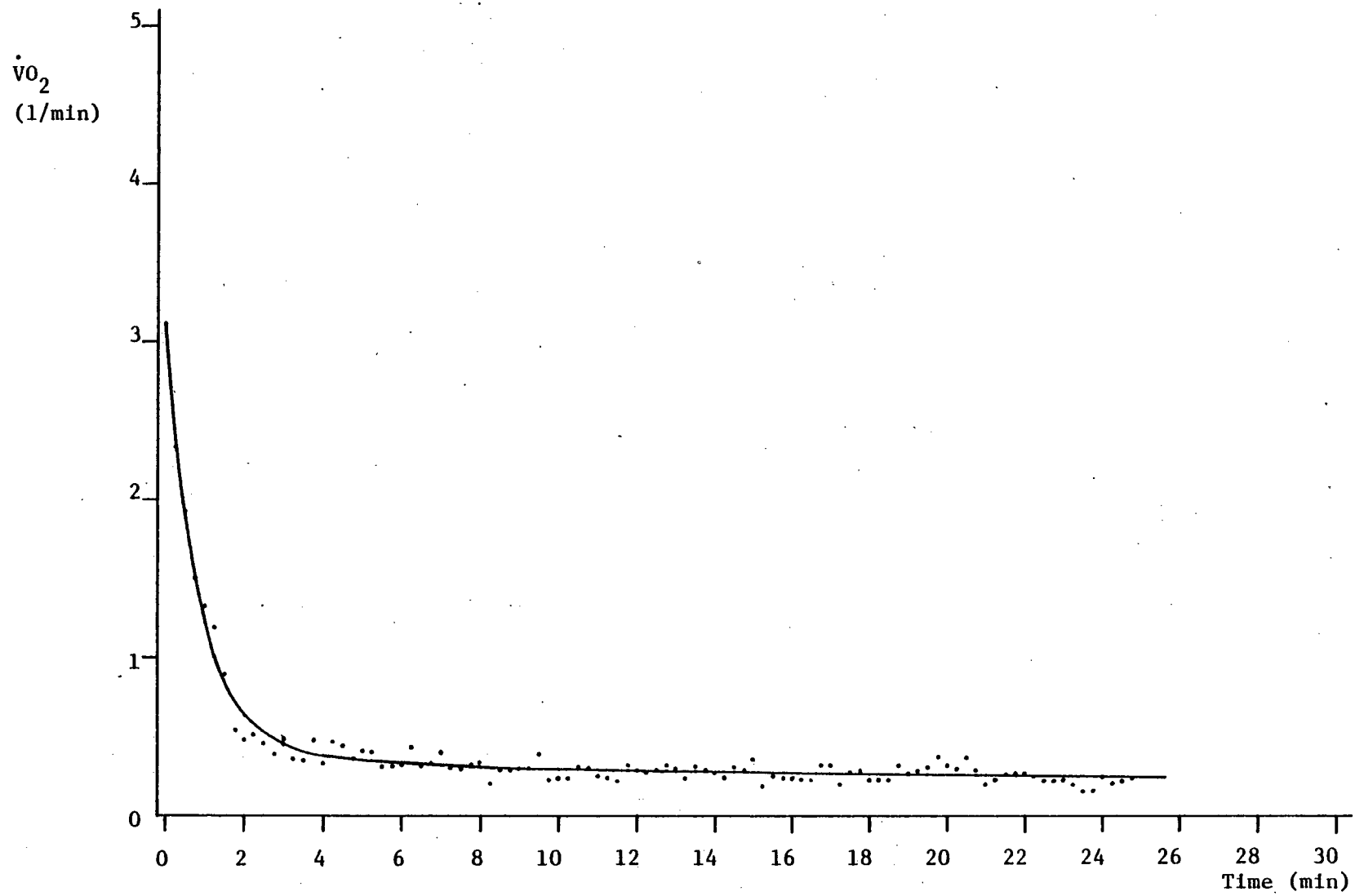


Figure 41. Recovery curve subject JL

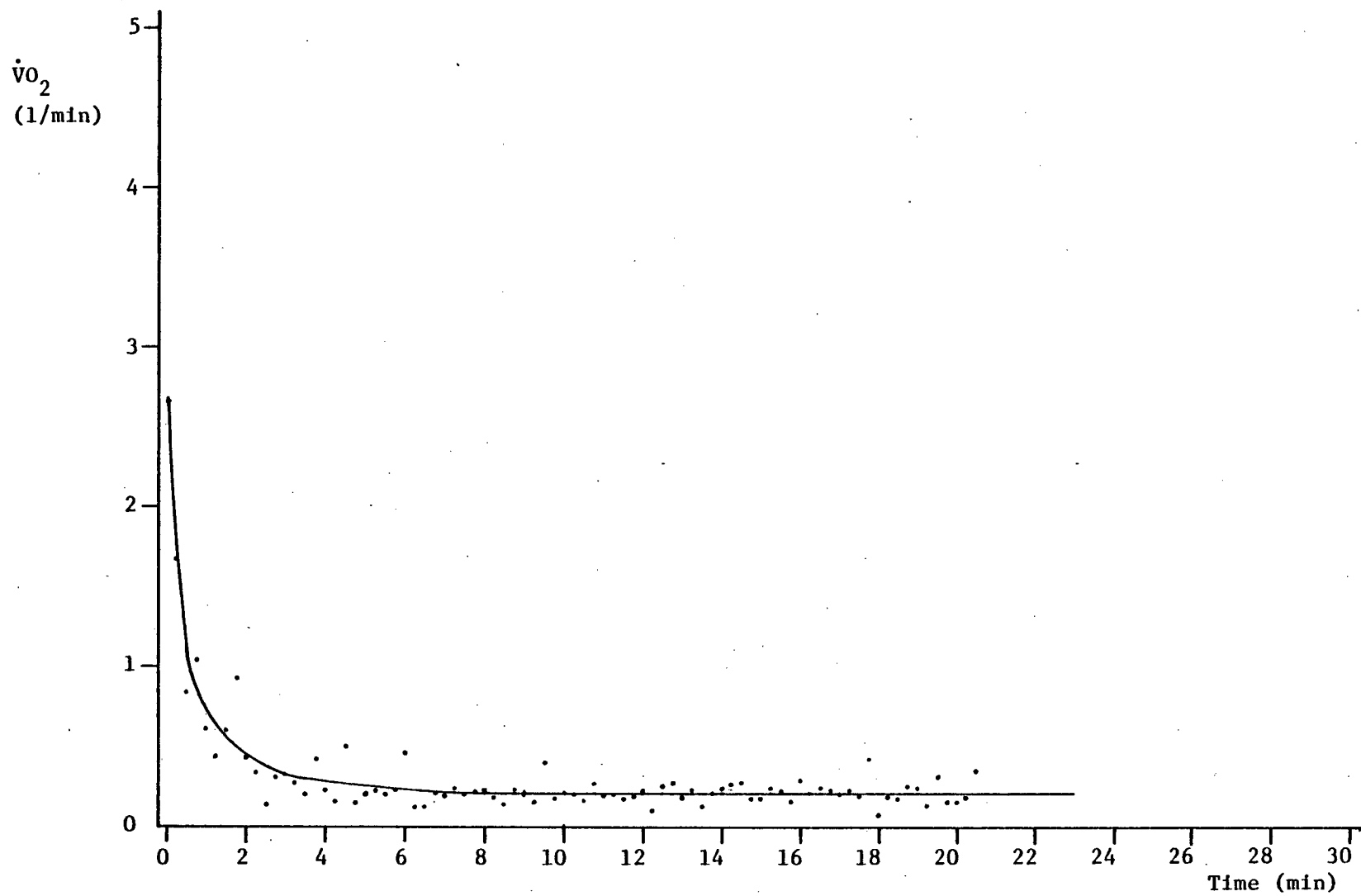


Figure 42. Recovery curve subject DM

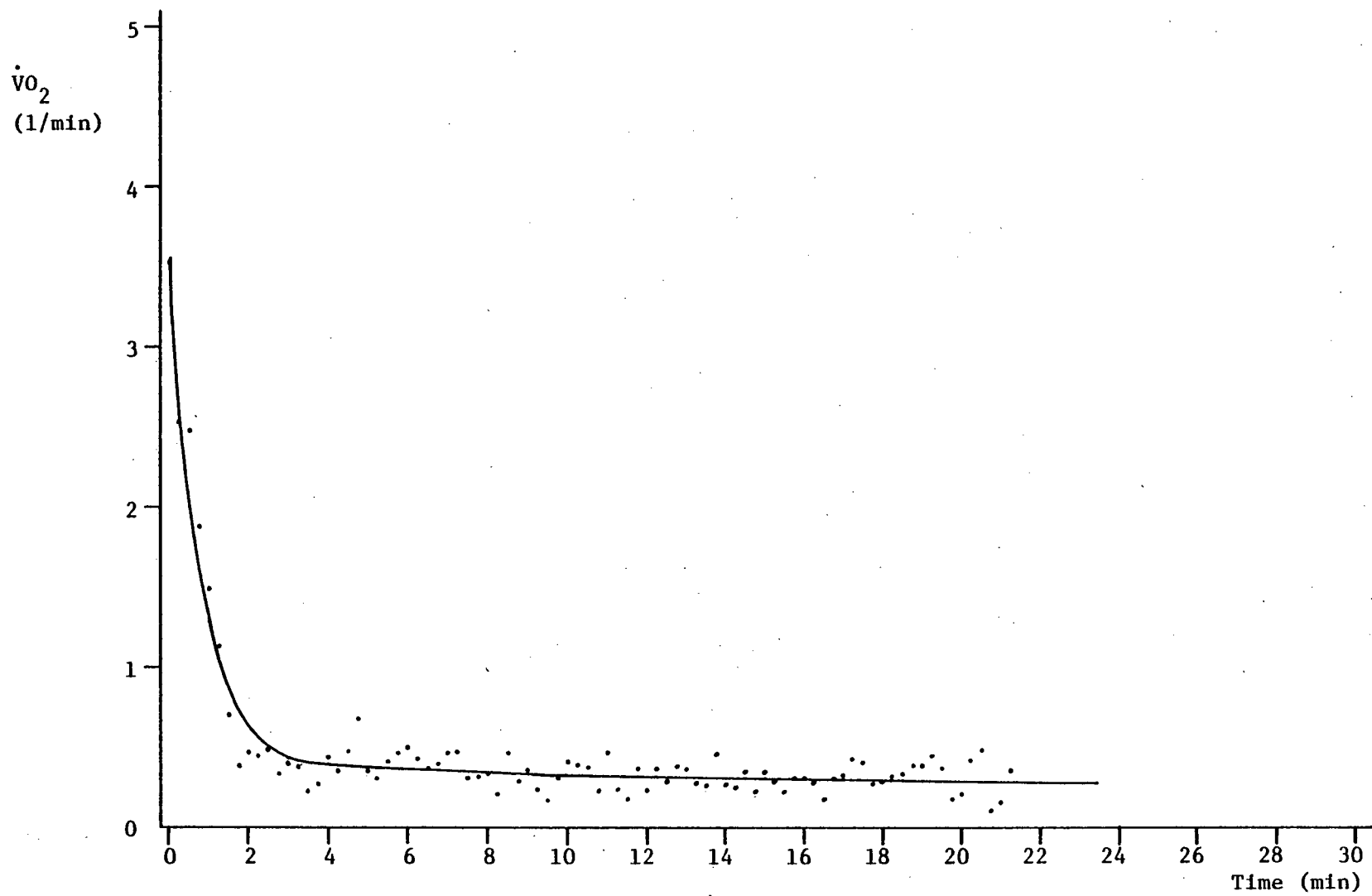


Figure 43. Recovery curve subject DW

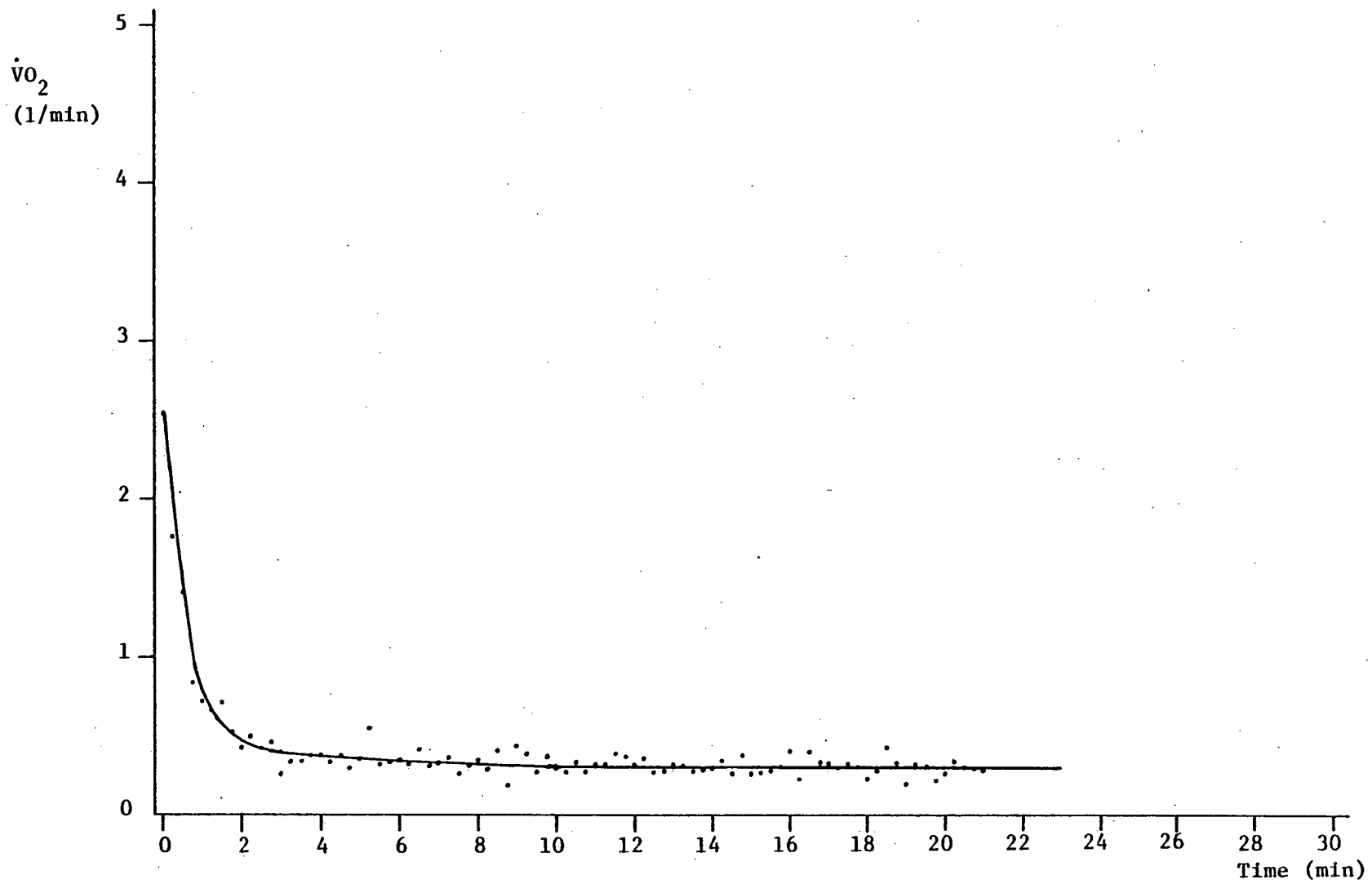


Figure 44. Recovery curve subject SP

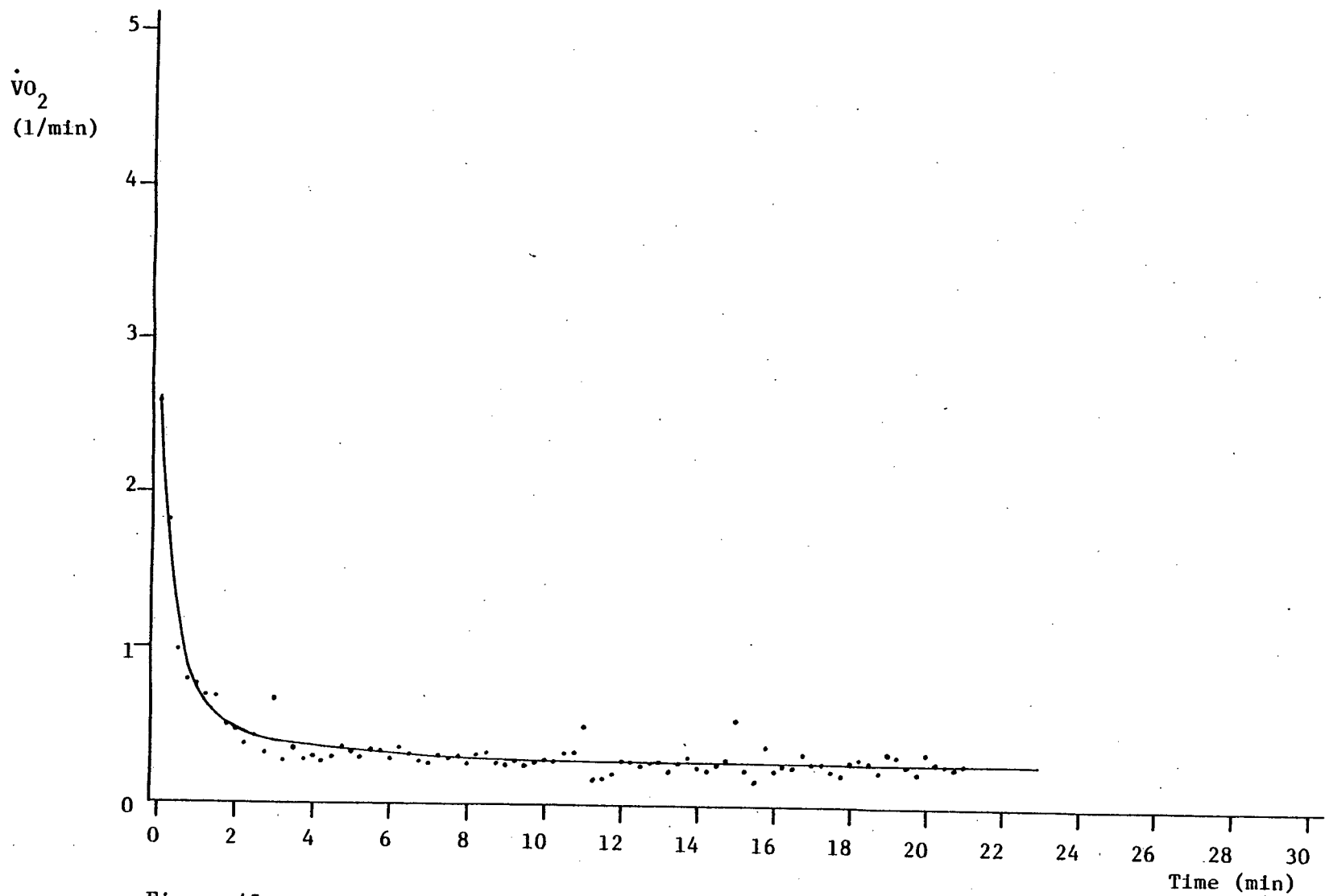


Figure 45. Recovery curve subject JH