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A SYSTEMS MODEL OF  
PHYSICAL TRAINING AND ATHLETIC PERFORMANCE

by

J.T. Corlett

B.Sc. (Biological Sciences), Brock University, 1973

A THESIS SUBMITTED IN PARTIAL PULFILLMENT OF  
THE REQUIREMENTS FOR THE DEGREE OF  
MASTER OF SCIENCE (KINESIOLOGY)

In The Department Of Kinesiology

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John T. Corlett

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## Abstract

A 25 year old male subject underwent a 24 week regimen of cycle ergometer training during which basic physiological parameters and performance in cycling and running were monitored. The amount of training was quantified in two ways, first, as cardiovascular stress as determined from ECG monitoring and, second, as the amount of physical work done as determined from ergometer speed and load settings. Standard tests for aerobic capacity, strength, and running performance were conducted weekly.

In response to the training, maximal oxygen uptake increased from a pretraining level of 52.5 ml/kg/min to a maximum value of 64.0 ml/kg/min at the end of the second phase of training. Similarly, leg strength increased from 900 pounds to 1080 pounds, cycle endurance at work rates used during the maximal oxygen uptake determination increased from 6.33 minutes to 7.70 minutes, and two mile run time decreased from 14.34 minutes to 11.67 minutes.

Using a systems theory approach, maximal oxygen uptake was successfully related to the cube root of quantified training with a simple first order differential equation where training

is expressed in units of cardiovascular stress. The time constant for this function as determined from measured data was 23 days. Leg strength was also modelled with a first order differential equation driven by training expressed in units of work done, the time constant being estimated as 60 days.

A multicomponent system was required to model actual performance data. The system is grossly described by the equation

$$\text{PERFORMANCE} = \text{FITNESS} - (\text{CHRONIC FATIGUE} + \text{ACUTE FATIGUE})$$

The dependence of these system components on training was described. Both fitness and chronic fatigue were related to the cube root of training by first order differential equations, the former with a time constant of 50 days and the latter with a time constant of 15 days. Acute fatigue was modelled as a function responding to changes in training intensity rather than to training intensity itself. The time constant for this function was 7 days. The performance model adequately simulated the characteristic features of the performance profile taken from actual measured data, thus confirming the hypothesis that the training/performance system can be described using first order differential equations.

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The folks back home

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## TABLE OF CONTENTS

Section 1: Introduction	1
Section 2: Literature Review	
a) Energy Metabolism	4
b) Substrate Utilization Capacity	6
c) Substrate Availability	9
d) Oxygen Availability	13
e) Substrate Utilization	18
f) Neuromuscular Functions	24
g) Skill	27
h) Psychological Factors	29
i) Training Programme Design	31
Section 3: Experimental Design	34
Section 4: Materials and Methods	38
Section 5: Method of Analysis	52
Section 6: Results	64
Section 7: Discussion	101
Section 8: Bibliography	120
Section 9: Appendices	
a) Appendix 1 Somatotype and Anthropometric Data	128
b) Appendix 2 Blood Chemistry Data	131
c) Appendix 3 Simulation Programmes	133
d) Appendix 4 Regression Programme	137

## LIST OF FIGURES

FIGURE	PAGE
1. A Systems Model Approach To Performance	36
2. Cardiovascular Training Profile	40
3. Work Done Training Profile	42
4. Diurnal Rhythm Of Two Physiological Parameters	44
5. Theoretical Response Of A First Order System	54
6. Calculation Of Time Constants	59
7. Response Of Maximal Oxygen Uptake To Training	69
8. Response Of Rogers Strength Index To Training	71
9. Response Of Leg Strength To Training	73
10. Response Of Blood Lactate To Heavy Exercise	75
11. Response Of Two Mile Run To Training	77
12. Response Of Cycle Endurance To Training	79
13. Simulation Of Maximal Oxygen Uptake	81
14. Simulation Of Leg Strength	83
15. Simulation Of Fitness	85
16. Simulation Of Chronic Fatigue	87
17. Simulation Of Running Performance	89
18. Simulation Of Acute Fatigue	91
19. Simulation Of Total Fatigue	93
20. Modified Simulation Of Running Performance	95
21. Modified Simulation Of Cycle Endurance	97
22. Simulation Of Performance: Weighting Constants	99
23. Systems Model Of Performance	102
24. Multicomponent Systems Model Of Performance	104
25. Hypothetical Training/Performance Example	116

## Introduction

In recent years, the effects of physical training on physiological systems have received increasingly more attention. One important aspect which has for some reason escaped scrutiny is the quantitative nature of the relationship between physical training and physical performance. The problem is of both fundamental and practical interest. Without adequate knowledge of the quantitative effects of training, the physiologist possesses a poor understanding of the determinants of human physical performance. Such knowledge could also be of considerable value to professionals in a variety of clinical contexts, among them, rehabilitation therapists and athletic coaches who are concerned with optimally increasing an individual's physical performance through training.

One approach which has been used to investigate the quantitative nature of physiological systems depends on systems modelling. In general, modelling a living system lends itself to two types of procedure. The first is what is referred to as the "black box" strategy in which the inputs and outputs are considered without reference to the individual components of the system. This strategy has the advantage of requiring only general input data but gives output only via a general transfer function which provides no insight into how the system works. The second is what might be called an "analytic" strategy in

which the system is broken down into its constituent components and each component is individually described. Such a strategy provides a detailed analysis of the system's operation but requires an accurate knowledge of each component.

In practice, a combination of the two strategies is usually used. The generalities of the black box strategy are enhanced by available knowledge of and intuitive judgements about the nature of the system's components. Two systems which have been modelled extensively in this way are the respiratory system (Milhorn and Brown, 1971) and the thermoregulation system (Stolwijk and Hardy, 1966).

The first attempt to apply such analysis to physical performance was the formulation of a theory of competitive running in which the technique by which optimum physiological efficiency over a given distance might be obtained was modelled (Keller, 1973). Two previous attempts at relating training to performance using a systems model approach have been made (Calvert, Banister, Savage, and Bach, 1976; Banister, Calvert, Savage, and Bach, 1975). The strategy employed was essentially a black box because input in the two cases examined was available only in very general terms (e.g. distance swum per month). The use of a multicomponent model was only speculative

since the natures of the intermediate variables which determine performance were not known. The present study was proposed to examine the quantitative effects of physical training on physical performance and on the physiological components contributing to physical performance.

## 2. Review of Literature

### The Determinants of Human Performance

An individual's athletic capability is dependent upon several factors. While the actual performance may assume many forms, important common components are energy metabolism, neuromuscular functions (most importantly skill or technique, and strength), and the psychological factors of motivation and competitive tactics (Astrand, 1970). These factors, in total, produce the functional characteristics of speed and endurance which together determine individual performances such as running, cycling, rowing, or swimming.

#### 2a. Energy Metabolism

Work done in an athletic performance is the ultimate result of a complex series of chemical reactions occurring at the subcellular level, governed by the laws of thermodynamics. As Lehninger (1961) points out,

"(the cell) must therefore obtain energy and use it at a fairly constant and low temperature, in a dilute aqueous environment and within a narrow range of concentration of hydrogen ions. To secure its primary energy the cell has during the eons of organic evolution perfected extraordinary molecular

mechanisms that work with great efficiency under these mild conditions."

Energy metabolism in all cells is mediated through the adenosine triphosphate (ATP) generating system in the mitochondria. For work to continue beyond the capacity of available stores of ATP (or its phosphate "storage" molecule, creatine phosphate) it is necessary to replenish the energy supply in some way. In mammalian cells this is accomplished by a complex series of reactions predicated upon the breakdown of glucose. This process is called glycolysis.

While the glycolytic pathway is accessible only to carbohydrates, the ATP generating system of the electron transport chain is accessible to fat and protein substrates as well. The production of Krebs cycle substrate from fat is accomplished by the process of beta oxidation, a progressive two-carbon release process. Since between sixty and seventy-five percent of all energy extracted from foods is supplied by triglycerides (glycerol bonded to three fatty acids), either by direct ingestion or by conversion of excess carbohydrates to fat for storage, the mechanism of fat oxidation is a significant contributor to energy metabolism.

The relationship between fat and carbohydrate metabolism is important; since the capacity to store carbohydrate is quite limited (only about 200 grams in the whole body) the conversion

of excess carbohydrate to fat is a sound physiological investment particularly since only about fifteen percent of the energy stored in a glucose molecule is lost when it is converted to triglyceride.

Under certain conditions, the building blocks of proteins, the amino acids, may be used as energy producing substrates. The process by which this occurs is called deamination. Via deamination, amino acids are converted to either carbohydrate or fat for storage and later use in energy producing pathways.

The participation in performance of energy producing pathways during physical exertion has been the focus of considerable attention and has inspired a voluminous literature. The effects of physical training on these pathways have also been widely investigated. The factors most commonly deemed important in influencing the ability of cells, particularly active muscle cells, to generate ATP are the availability of substrate, the availability of oxygen, and the activities of the enzymes involved in the energy producing pathways.

## 2b. Substrate Utilization Capacity

The ability of cells to metabolize substrate is located in the organelles, particularly the mitochondria, and the enzymes of the involved biochemical pathways. These have already been



discussed in general terms; however, the state in which they exist has been shown to be highly dependent on the activity level of the individual.

The changes in skeletal muscle mitochondria following physical training are well documented. Increases in the size (Gollnick and King, 1966; Morgan, Cobb, Short, Ross, and Gunn, 1971), the number (Gollnick and King, 1966; Kiessling, Piehl, and Lundquist, 1971), and the number of inner membrane cristae (Holloszy, Oscai, Mole, and Don, 1971), have been reported. Since the mitochondria are the sites of those enzymes involved in oxidative energy metabolism, such ultrastructural changes might be expected to reflect enzymatic adaptations and this is the case. Holloszy et al (1970) have demonstrated in the hind limb muscle of rats that training induced twofold increases in NADH dehydrogenase, succinate dehydrogenase, citrate synthetase, aconitase, and cytochrome c oxidase, all of which are involved in the Krebs cycle and electron transport. Other related enzymes have been shown to increase in different proportions after training; for example, alpha ketoglutarate dehydrogenase and malate dehydrogenase increase about fifty percent while other Krebs cycle enzymes increase only about 35 percent. This has led Holloszy to conclude that "the mitochondrial citrate cycle and citrate cycle related enzymes do not increase in parallel during adaptation of skeletal muscle to exercise" and that "as a result, there is a change in mitochondrial composition" (Holloszy et al, 1971). These

results have also been observed in studies with humans. Morgan et al (1971) demonstrated increased levels of NADH dehydrogenase, succinate dehydrogenase, and cytochrome oxidase in the leg muscles of subjects exercising two hours per day for one month on a stationary bicycle. Gollnick, Armstrong, Saubert, Piehl, and Saltin (1972) have also shown significant differences in the succinate dehydrogenase activity between trained and untrained individuals.

In contrast to the observations on oxidative enzymes, studies have shown few changes in anaerobic enzymes after training (Gollnick and Hermansen, 1973). However, the authors suggested that this "may have resulted from the use of training programs that did not stress the anaerobic system". Hexokinase is the most training-sensitive enzyme in the glycolytic pathway. It is one of the two major regulators of glycolysis. It has been observed to increase significantly after very few training sessions (Lamb, Peter, Jeffress, and Wallace, 1969; Morgan et al, 1971). A second glycolytic regulator, phosphofructokinase (PFK), does not appear to show such dramatic training responses. However, Gollnick and his coworkers (1972) have demonstrated a minor difference in PFK levels between trained and untrained men, and Holloszy et al (1971) also showed a marginal PFK increase in rats that were exercised by running. In sharp contrast to this, Eriksson, Gollnick, and Saltin (1973) have observed a 40% increase in PFK

activity in ten and eleven year old boys after two months of training. However, age differences in the training response of the anaerobic enzymes have not fully been investigated.

The training effect on glycolytic capacity is not uniform throughout skeletal muscle. Both Gollnick et al and Baldwin et al have noted that adaptation is muscle fibre type dependent. In rats, three fibre types have been indentified according to enzymatic activity patterns. Of these, Baldwin, Klinkerfuss, Terjung, Mole, and Holloszy (1972) found glycolytic capacity to increase after training only in the so called slow twitch oxidative fibres while in the fast twitch low oxidative fibres, glycolytic capacity actually decreased. In humans, where two types of muscle fibres have been identified, the fast twitch glycolytic fibres showed increased glycolytic capacity following training (Gollnick et al, 1972).

#### 2c. Substrate Availability

Carbohydrate, fat, and protein stores are all available intramuscularly as well as in extramuscular storage depots such as the liver (carbohydrates) and adipose tissue (fats). The extent to which these substrates are available for energy production is largely dependent upon diet and, to a lesser extent, activity level.

The intramuscular levels of phosphagens (ATP and creatine phosphate) is essentially fixed; Karlsson, Diamant, and Saltin (1971) have shown an increase in phosphagens in skeletal muscle following training but this increase is thought only to be secondary to an increased mitochondrial density and not to be significant in terms of anaerobic power .

Gollnick, Piehl, Saubert, Armstrong, and Saltin (1972) have demonstrated the effect of diet on intramuscular glycogen stores. A mixed diet produced a resting glycogen level of 87 mmol glucose per kg of muscle, a fat-protein diet produced a level of 43 mmol glucose per kg, while a carbohydrate enriched diet resulted in the highest level of 114 mmol per kg. The levels were independent of muscle fibre type; both fast twitch and slow twitch (white and red) showed the same results. This result has been duplicated to some extent (Hultman and Nilsson, 1971).

The amount of glucose available as glycogen in the liver is also diet dependent. Hultman et al (1971) showed that subjects at rest and on starvation diets emptied liver glycogen stores within 24 hours. Recovery occurred within one day when a carbohydrate supplemented diet was eaten. An intake of 2100 calories consisting of only five grams of carbohydrate failed to replenish adequately depleted liver stores. Similar results have been obtained by Bergstrom, Hermansen, Hultman, and Saltin

(1971).

In a muscle depleted of glycogen by maximal exercise, an inexplicable overshoot of resynthesis was observed during recovery (Hultman, Bergstrom, and Roch-Norlund, 1971). This recovery was dependent in magnitude upon the availability of substrate; the greatest overshoot occurred when glucose was infused into the blood during the recovery period. The replenishment of glycogen was regulated by the enzyme glycogen synthetase (Bergstrom and Hultman, 1966). Induction of this enzyme is controlled by intracellular levels of glucose 6 phosphate and ATP - an increase in either causing the activity of the enzymes to increase. Following muscle glycogen depletion by exercise, high activities of glycogen synthetase continued for two to four days (Bergstrom and Hultman, 1966).

Pernow and Saltin (1971) investigated the availability of substrates, particularly free fatty acids (FFA) during performance. Subjects were exercised at work rates which depleted muscle glycogen from 11.7 to 0.3 g/kg muscle, then maintained on a non-carbohydrate diet for one day. In the absence of dietary carbohydrate muscle glycogen levels increased to 2.9 g per kg of muscle within 24 hours. A second exercise session was conducted in two parts, the first under normal circumstances and the second in the presence of nicotinic acid which blocked the release of FFA from fat stores. In the first case, prolonged submaximal work continued despite lowered muscle glycogen. In the second case, work

capacity dropped by about fifty percent when both glycogen and FFA were in short supply. Total energy output decreased from 850 kcal (glycogen and FFA available) to 220 kcal (glycogen, FFA greatly reduced). These results indicated the impact of inadequate glycogen and FFA supply on performance capacity.

Although abundant in muscle, protein is essential to the structure and function of the muscle and is therefore not a desirable substrate for ATP generation. Despite this, Felig and co-workers (Felig and Wahren, 1971; Felig, Pozelsky, Marliss, and Cahill, 1970) have shown that during exercise alanine (comprising five to eight percent of muscle amino acid residues) is released into the blood at a rate high above other amino acids. They postulated that this was not the result of a protein dissolution but rather a transamination of excess pyruvate arising from the breakdown of carbohydrates and fats beyond the capacity of the Krebs cycle to use them. They presented evidence that twelve to eighteen percent of glucose used by heavily exercising muscle is derived from glucose resynthesized from blood lactate in the liver. Such systems serve not to increase the amount of available substrate, but rather to make more efficient use of that which is already there.

Gollnick et al (1972) also demonstrated an effect of training on substrate utilization in skeletal muscle using both

microscopic staining and histochemical techniques. They found that trained subjects had greater ability to accumulate muscle glycogen than untrained subjects. It was further noted that the accumulation was greatest in those muscles most involved in the subjects physical activity. For example, the leg muscles of cyclists and the arm muscles of swimmers and canoeists showed greatest glycogen levels. However, the effects of chronic exercise on-substrate levels, either intra- or extramuscular, are unknown.

#### 2d. Oxygen Availability

The utilization of oxygen as the ultimate electron acceptor of the catabolic pathways requires that sufficient exchange of oxygen occurs between the atmosphere and the mitochondria in the cell. Increased oxygen demands during exercise will clearly require increased cardiorespiratory function. The most common parameter used to appraise cardiorespiratory fitness is the maximal oxygen uptake (Pollock, 1973) which is in turn dependent upon several other factors such as cardiac output and the arteriovenous difference in oxygen concentration. In addition to these cardiovascular considerations, maximal pulmonary ventilation is an oft-quoted parameter of importance when assessing maximal oxygen uptake.

It is well accepted that cardiorespiratory fitness is dependent upon the activity level of the individual; a definite training effect upon it exists. The increase in maximal oxygen uptake capacity following training of previously sedentary individuals is well documented. The magnitude of the increase have been shown to be dependent upon the intensity, duration, and frequency of the training. Shepherd (1968) determined that intensity and duration were the most important training stimuli while frequency was less significant. Training intensity may be accurately determined by monitoring the heart rate since aerobic power has been shown to bear a linear relationship to heart rate (Wahlund, 1948; DeVries and Klafs, 1965). Those investigators seeking to standardize their training data have most frequently expressed training in terms of "percent maximal oxygen uptake" determined from heart rate monitoring and the above mentioned linear relationship (Pollock, 1973).

Many studies of the effect of training intensity on the improvement of maximal oxygen uptake have been made. The relationship appears to be a linear one (Kilbom, 1971; Sharkey and Holleman, 1967) although discrepancies due to age and initial fitness levels occurs. Davies and Knibbs (1970) trained healthy male subjects at 80, 50, and 30 percent of their maximal oxygen uptakes for eight weeks. Their results agreed with those of Shepherd (1968) and Faria (1970); the improvement



in maximal oxygen uptake was directly related to training intensity. Intensities of less than fifty percent produced no effect, that is, a "threshold" effect existed. The threshold was a function of the initial level of fitness of the individual (Durnin, Brockway, and Whitcher, 1960; Pollock et al, 1971); those with low initial oxygen uptakes showed improvement at training intensities as low as 120 beats per minute while those with high initial oxygen uptakes required heart rates of 150 or more.

The duration of a training session is also an important stimulus for the training response to occur. Wilmore, Royce, Girandola, Katch, and Katch (1970) reported that of two groups of middle aged men engaged in a ten week jogging programme, the group training 24 minutes per day showed significantly greater cardiovascular improvement than the group training 12 minutes per day. These results agreed with those obtained with women training on a bicycle ergometer (Yeager and Brynston, 1970). Such studies have been criticized for their inability to separate adequately the effects of duration from those of intensity. However, Sharkey (1970) trained six groups of college men in an array system of three intensities (130, 150, 170 beats per minute) and two work rates (7500 or 15000 kpm of total work) to determine the interactions which occurred. He concluded that if total work was constant, intensity was not a significant factor. In addition, he found that intensity and

duration did not interact to produce significantly different training responses. This supported the hypothesis of Cureton and Phillips (1964) and Banister and Taunton (1971) who suggested that total work done (or total energy cost) was the important factor in determining the amount of the training stimulus. However, the development of anaerobic power makes a significant contribution to demonstrating aerobic power by not limiting the aerobic power test (Banister, personal communication).

The frequency at which training sessions are held appears to have less effect on cardiovascular improvement than either intensity or duration. The only caution to be exercised in this regard is that the length of time occupied by the whole training programme be sufficient to allow less frequently trained individuals to overcome the initial advantage gained by those training more often (Hill, 1969). This supports the concepts advocated by Cureton and Phillips (1964) since the total work done is the same; one group merely takes longer to perform that work than the other. In contrast to this viewpoint, Jackson, Sharkey, and Johnston (1968) presented evidence which suggests that an optimum training frequency exists. College men training two or three times per week showed greater cardiovascular improvement than those training five days per week. This may, however, have been due to fatigue interfering in the cardiovascular testing in the five days per week group.

Although less frequently reported than maximal oxygen uptake, other previously mentioned cardiorespiratory parameters showed similar training effects with respect to intensity, duration, and frequency. These have been summarized comprehensively by Pollock (1973). Cardiac output, stroke volume, heart volume, maximal ventilatory volume, systolic pressure, and oxygen pulse have been shown to be subject to training effects dependent upon initial fitness level and the nature of the training programmes.

The cardiorespiratory system serves to deliver oxygen from the atmosphere to the capillaries surrounding the working cells. Training has been observed to enhance this process by increasing the capacity to take oxygen into the lungs, to pump blood from the heart to the lungs and then to the working tissues, and to invest the tissues with increased capillarization (Astrand, 1970). At the cellular level, training was also shown to increase the capacity to transfer oxygen from the capillaries to the interior of the mitochondria. Holloszy, Oscai, Don, and Mole (1971) suggested that since rat leg muscle myoglobin increased by the same magnitude as the increase in the mitochondrial inner cristae, myoglobin might be active in facilitating transport of oxygen from the cytoplasm to the mitochondrial enzymes. This hypothesis has been supported by the work of Pattengale and Holloszy (1967) and Scholander (1960).

## 2e. Substrate Utilization

The adaptations which occur in response to training have been shown to result in changes in the way in which energy production occurs. These changes are reflected in the relative rates at which various substrates are used and in the rates at which lactate, a product of anaerobic metabolism, accumulates. In both the trained and the untrained individual, utilization of substrates and the buildup of lactate has been observed to be dependent upon the type of muscle fibre predominantly used. Kugelberg and Edstrom (1971) determined that when rat muscle was subjected to repeated supramaximal shocks, the white glycolytic fibres were depleted of glycogen first at frequencies of only five per second, fast twitch oxidative fibres were depleted at frequencies of about ten per second for ten minutes, and C fibres were not depleted at all.

These differences in glycolytic energy production were observed in the induction of the enzyme hexokinase (Staneloni and Pires, 1969). White glycolytic fibres had the strongest initial activity dropping off quickly while slow twitch oxidative fibres were initially weak in hexokinase activity but increased greatly during prolonged contraction. Fast twitch oxidative fibres were intermediate between the two. Using the needle biopsy technique, Gollnick, Armstrong, Sembrowich, Shepherd, and Saltin (1973) showed glycogen depletion to be

fibre type dependent in humans as well. During short, high intensity work (150% of maximal oxygen uptake) lasting one minute, fast twitch, or white, fibres showed the highest glycolytic activity and were most rapidly depleted of glycogen. This was in contrast to moderate work rates which caused the slow twitch, or red, fibres to be depleted first. The glycogen depletion in these experiments was also shown to be diet dependent. At 74% of maximal oxygen uptake less glycogen was broken down in subjects after a fat-protein diet indicating greater use of other substrates.

1 Several investigators have suggested that glycogen depletion is a major cause of fatigue (Ahlborg, Bergstrom, Ekelund, and Hultman, 1967; Bergstrom and Hultman, 1966). Glycogen depletion accompanies fatigue during high intensity work. However, Saltin and Karlsson (1971a) found that glycogen was not significantly reduced at fatigue following prolonged work at less than 90% of maximal oxygen uptake. This was attributed to the predominant use of red oxidative fibres rather than white glycolytic ones. Even at work rates of greater than 90% maximal oxygen uptake, it appeared that ample muscle glycogen was available at the fatigue point indicating that fatigue was not simply a function of glycogen depletion. It was also noted that muscle glycogen depletion at a fixed work load was less in trained individuals than in untrained ones. This was supported by Paul and Issekutz (1971) and Paul

(1968) who suggested that during exercise FFA mobilization had a sparing effect on glycogen stores. In this regard, Froberg et al (1971) have examined local lipid stores and concluded that muscle triglycerides served as substrates for FFA production during exercise and that as little as 25% of FFA metabolized was extramuscular in origin. These conclusions were supported by Issekutz, Miller, Paul, and Rodahl (1964).

The nature of the interactions between fat and carbohydrate utilization prompted Havel to postulate that while glycogen was the preferred substrate for high intensity work, FFA were preferred to glycogen for moderate and light work rates (Havel, 1971). There is experimental evidence to support this view. Rowell (1971) and Bergstrom and Hultman (1967) showed that liver glycogen was recruited as a significant source of energy substrate in the the form of glucose during heavy exercise, sometimes exceeding 50% of the total metabolic demand. This accelerated release which occurred despite reduced hepatic blood flow coincided with reduced muscle glycogen.

Concomitantly, Havel, Carlson, Ekelund, and Holmgren (1964) demonstrated that at heavy work rates, as little as 25% of available metabolic substrate for prolonged work could be accounted for by the oxidation of FFA. Wahren, Ahlborg, Felig, and Jorfeldt (1971) and Jorfeldt and Wahren (1970) also

indicated that blood glucose uptake in exercising muscle measured by the arteriovenous glucose difference was dependent both on increasing duration and intensity of the exercise. This was in contrast to the use of fats as the major energy source for prolonged lower intensity work determined by the measurement of RQ values (Paul and Issekutz, 1967).

The increased usage of glucose as a substrate during heavy work causes an increased accumulation of lactate. Fatigue has been attributed to this by several authors (Hermansen, 1971; Karlsson, 1971a). Karlsson, Diamant, and Saltin (1971) found that the level of lactate in the muscles at the end of maximal work of varying duration was the same for work times up to seven minutes. These short intensity work periods demand high glycogen utilization. However, muscle lactate concentrations observed when exhaustion was induced by a work rates requiring twenty minutes before exhaustion occurred were considerably less. This was attributed to the greater use of FFA. Hermansen (1971) found that the subjective feeling of fatigue or complete exhaustion in thirteen male subjects was accompanied by the same approximate level of muscle lactate. Such results supported the idea that high muscle lactate concentration may be the limiting factor for work of short duration. The action of lactate as an inducer of fatigue may be mediated through a pH effect. Hermansen and Osnes (1972) showed decreases in muscle pH to levels as low as 6.80 (normal = 7.4) following

heavy lactate-producing exercise. This was thought to be a possible limiting factor in work of short duration since it has been demonstrated that decreases in pH inhibit glycolysis.

Physical training is known to reduce the concentration of muscle lactate produced during exercise. The explanations for this phenomenon are numerous. Saltin and Karlsson (1971b) have postulated one mechanism. According to these authors, training might result in a neuromuscular change which activates more red fibres than were activated in the untrained state. This would reduce the stress placed on the white fibres and, therefore, reduce glycolysis and lactate production. At a biochemical level, Saltin and Karlsson (1971b) demonstrated that eight weeks of training produced changes in the relative amounts of fat and carbohydrate used at a given submaximal oxygen uptake, providing another suggestion for how lactate buildup may be avoided. Increased mitochondrial function would counteract lactate accumulation due to increased capacity to oxidize pyruvate and incorporate extramitochondrial NADH produced by increased fatty acid production and oxidation. Holloszy et al (1971) stated in this regard that the steady state oxygen uptake in trained muscles with more mitochondria would occur at lower levels of ADP and inorganic phosphate; since glycolysis was also triggered by ADP and inorganic phosphate, it would also be induced at lower levels. The slowdown in glycolysis



(accompanied by the simultaneous increase in fatty acid oxidation) would result in less lactate production. Another way in which lactate accumulation may be avoided is to metabolize the lactate which is produced. During exercise, such a function is not accomplished in the liver since Hultman has shown that splanchnic blood flow is greatly reduced during exercise (Hultman, 1967). Jorfeldt and Wahren (1971) and Saltin and Karlsson (1971a) suggested that training might increase the ability of muscles to utilize lactate as a substrate for oxidation. This is known to occur during the recovery phase following exercise; whether or not it is significant during exercise is not known.

The once held concept that lactate is simply the result of muscle hypoxia has been demonstrated to be an oversimplification since even well perfused muscles have been shown to produce lactate (Doll, Keul, and Maiwald, 1968). The actual regulatory mechanisms controlling aerobic/anaerobic energy production are not well understood. Karlsson (1971b) summarized the functional aspects by stating that lactate accumulation begins at work rates above 50% maximal oxygen uptake and that it represents not a detrimental aspect of energy metabolism but rather a means by which work continues beyond true aerobic capacity. However, it may limit the expression of true aerobic capacity by forcing termination before aerobic capacity is reached.

## 2f. Neuromuscular Functions

Muscle strength, local muscular endurance and speed, and skill are the neuromuscular factors which determine an individual's capacity to transform the energy produced at the subcellular level into useful work.

### a) Strength; Local Muscular Endurance and Speed

The amount of pulling force a muscle can generate is its strength. It has been shown to be directly proportional to the cross sectional area of the muscle (Hettinger and Muller, 1953); the more numerous and/or the larger the muscle fibres, the stronger the muscle. Just as muscle atrophy and strength loss have been shown to occur following limb immobilization or denervation (Eichelberger, Roma, and Moulder, 1963), muscle hypertrophy and strength gain have been seen to be common phenomena of training programmes in which overloading of the muscle occurred. In addition, as atrophy was the result of decreased fibre size and not of decreased fibre number, hypertrophy was shown to be the result of increased fibre size and not of increased fibre number although some of the change in cross-sectional area was the result of increased capillarization (Rohter, Rochelle, and Hyman, 1963). While the

cross-sectional area of the muscle has been a good monitor of increased strength, the strength available to do useful work has been observed to be dependent on other factors, specifically which joint was involved and the angle at which the joint worked due to the force-length characteristics of muscles (Ramsey and Street, 1940). its effect depending on the joint and the joint angle (Clarke, Elkins, Martin, and Wakim, 1950). Strength gain showed specificity to the angle at which the training stimulus was applied (Hodgkins, 1961). Strength tested at angles differing by as little as 20 degrees from the one at which isometric training occurred showed gains as low as 50% of that measured at the exercised angle. Isotonic training showed no such specificity when performed through the full range of movement (DeVries, 1974).

The nature of the training stimulus for strength is not known. A number of physiological explanations for strength gain have been advanced but none have been experimentally proven. Among these hypothesized trophic stimuli have been hypoxia, temperature change, stretching, and cerebation (Royce, 1958). It has also been suggested that the training stimulus affects the central nervous system and that training effects are brought on by the reduction of the normal inhibitory effects of the extrapyramidal system on the motor neuron (Royce, 1958). While such a stimulus may exist, it cannot cause hypertrophy and the strength gains induced by electrical stimulation independent of the motor pathways indicated some form of intramuscular effect (Start and Graham, 1964).

Several factors other than overload training are known to affect strength. The natural hypertrophy caused by child growth and the atrophy of aging have both been shown to cause quantitative changes in strength. In both cases, the changes are attributable to gains or losses in the amount of muscle tissue. Qualitative changes in the muscle play only a minor role (Rodahl, 1961). Differences in strength between males and females have been attributed to differences in the size of the muscles (Rodahl, 1961) although qualitative differences between male and female muscle tissue have been reported (Morris, 1948). Both diurnal and annual variations in strength have been observed; in addition, the capacity for strength gain may vary with the time of year (Hettinger and Muller, 1954). Environmental effects on strength have not been widely investigated. However, alterations of arm and grip strength have been demonstrated using exposures to heat (strength increase) and cold (strength decrease) (Grose, 1958).

Local muscular endurance is largely a function of strength. Gains in local endurance have been observed to be simultaneous with increases in strength resulting from overload training (Shaver, 1970). In addition to the factors which contribute to strength, local blood flow has been demonstrated to be vital to local endurance. Isometric training has been shown to decrease the ratio of blood flow debt per unit of

exercise effort (Vanderhoof, Imig, and Hines, 1961). This may have been the result of improved peripheral circulation which accompanied muscle training.

Hill demonstrated that local muscle speed is an intrinsic property of each individual muscle. His experiments showed that the speed of contraction varied depending on both fibre type and muscle length (Hill, 1922). Hill also observed that temperature played a part in determining contraction speed, an increase of 2°C producing as much as a 20% increase in contraction speed. Neuromuscular patterns played only a small role in determining the contraction speed of a muscle and, as a result, this aspect of physical performance appears to be the least amenable to training (DeVries, 1974).

## 2g. Skill

Krech, Crutchfield, and Livson (1969) stated,

"A skill is an organized sequence of integrated patterned responses executed with precision and efficiency.

Superficially, a skill consists of many different perceptual and motor responses. When performed in a properly organized manner, both temporally and spatially, skilled behaviour results. When a sequence of acquired responses becomes a skilled action, it seems likely that some genuine reorganization at the neurophysiological level has occurred."

Motor control has been shown to involve many levels of neural organization. The relationships between them have been extensively reviewed (Evarts, 1973). Several physiological mechanisms for learning at the level of the neuron have been proposed, among them, structural changes at the synapses of facilitated pathways (Altman and Das, 1965), increases in acetylcholine levels (Rosenzweig, Krech, and Bennett, 1962) and in RNA levels (Hyden and Egyhazi, 1965). The acquisition of skilled motor behaviour is characterized by three phases: the acquisition of discrete responses, the consolidation of the discrete responses, and the conversion of the discrete responses into a "functional unit". The typical learning curve, in which skill acquisition is plotted versus training, shows an initial rapid rise due to the learning of discrete responses, a plateau due to the consolidation of discrete responses, and finally a further rise as the discrete responses are converted into a unit skill. The capacity for improvement in the third phase appears to be limited only by physiological aging and/or loss of motivation since performance continues to improve over innumerable cycles of practice (Crossman, 1959). The retention of motor skills is very long: Bilodeau (1966) has pointed out that most studies of motor skills find so little forgetting that it is impossible to determine the variables that produce it although interference by more recently acquired skills is certainly a factor.

## 2h. Psychological Factors

Kroll (1967) hypothesized that a specific set of personality traits existed which caused some people to select and participate in sports. Evidence for this view has been presented by Ogilvie and Tutko (1969) and Cooper (1969) who have demonstrated that successful athletes possessed certain traits that typify athletic achievement. These traits included high social adjustment and self-confidence, high masculinity/low femininity, and high emotional stability. Athletic activities have been classified into five categories based on their psychological demands. The five categories are 1) activities involving hand-eye coordination, 2) activities involving total body coordination, 3) activities requiring total mobilization of body energy, 4) activities in which death or injury is imminent, and 5) activities involving the anticipation of movements of other people (Vanek and Stransky, 1964). Despite the subjectivity of the system, it has proved useful in assessing the psychological demands of different sports. Psychological "types" have been identified for rugby players (Kane, 1966), auto race drivers (Ogilvie, 1968), and American football players and wrestlers (Kroll, 1968). Distance runners, cyclists, rowers, and swimmers were included in the third category and appeared to possess a distinctive psychological profile. Specifically, such athletes tended toward introversion, and possessed the emotional stability and

self-control necessary in activities in which a high level of determination, the ability to ignore pain, persistence, and durability are important to success (Vanek and Cratty, 1970). The motives of such athletes have only recently been studied.

According to Alderman (1970), in the context of human athletic performance, "motivated behaviour is the sum total of instincts and needs, motives and drives, conscious and unconscious forces, and a function of what one expects to gain from participation in sport". Vanek (1964) has constructed a multifactorial model of athletic motivation. Primary motives central to the model were the need for physical activity and the need for achievement. These basic needs were postulated to interact with secondary motives such as fear, aggression, and, in the case of individual competitors, the need for stability and predictability. This is in contrast to the desire for novelty and complexity apparent in team sport competitors. Interacting with both the primary and secondary motives are social motives such as identification (the emulation of another), suggestibility (the effect of social contacts), and intuition (realizing the emotional states of other competitors).

The interplay between the various psychological needs of an individual athlete to a certain extent has shaped individual performances. An athlete's psychological preparation,



particularly when high levels of physiological preparedness are achieved, has been thought to be the most important limiting factor in performance (Alderman, 1970). A more direct link between performance and psychological factors has been summarized by deVries (1974). Forearm flexor strength has been shown to significantly increase by 7% following a pistol shot two to ten seconds before strength effort, 12% by having the subject shouting during the application of force, and 26% by hypnosis that suggests greater strength. Such factors are not well understood and may hold great promise as training techniques in the future.

## 2i. Training Programme Design

Physical training produces both fitness and fatigue. The ultimate goal of a training programme is to maximize performance by maximizing fitness and minimizing fatigue. The type of training undertaken by an athlete is primarily determined by the type of event to be performed. Competitors in events of short duration and high intensity will train differently from competitors in events of longer duration and lower intensity. In events such as distance running and swimming, cycling, and rowing, two common approaches exist: 1) continuous training, and 2) interval training (Gordon, 1963).

Continuous training refers to covering long distances at relatively slow speeds, and is based on the principle of a physiological steady state being attained in which oxygen consumption equals energy demands following an initial lag period. The training distances have been shown to be most effective at three to five times the length of the racing event, and the time to cover the distance is such that the steady state heart rate is approximately 150 beats per minute. The philosophy of steady state training was summarized by the New Zealand coach, Lydiard (1972), who stated: "I am trying to develop my runners until they are in a tireless state. (Stamina) is the key to the whole thing..... It is merely a process of long, gradual conditioning".

A more recently-accepted method of training, developed in pre-war Germany, is interval training, which is based on repeated bursts of activity in which the heart rate reaches about 180 beats per minute alternated with short rest periods during which the heart rate is allowed to return to about 120 beats per minute. A popular variation of interval training is the Swedish "speed-play" technique in which long distances are covered with untimed pace variations (Doherty, 1963). Whatever system is used, the ability to adjust the training programme to individual differences is the underlying fundamental.

Individual athletes respond to training stimuli differently, some showing large initial increases in performance, others showing a more gradual increase. In addition, a "sports performance rhythm" (Gordon, 1963) is evident, characterized by a seasonal cycle of rising and falling performance. This is consistent with the concepts of Selye (1956) in which an adaptive reaction of the pituitary-adrenal system to stress occurs in three phases. Thus, in the context of athletics, the three phases are: 1) adaptation in which the athlete becomes accustomed to training and improves slowly, 2) completed adaptation in which the athlete is in his or her best form, and 3) readaptation in which adaptation is lost and performance decline sets in. In the preparation for competition, an optimal training programme seeks to deliver an athlete to his or her peak performance capacity at the time of important contests. To accomplish this, two banks of information are required. First, an understanding of the particular demands of the sport in which the athlete is involved is imperative. For example, the energy sources utilized by a gymnast are predominantly anaerobic and the training should reflect these needs. In contrast, a distance runner draws primarily on aerobic sources and should concentrate on developing aerobic capacity. Second, an adequate knowledge of the individual's stress response characteristics allows the training to be "fine tuned" to his or her own requirements.

### 3. Experimental Design

To examine the quantitative effects of training on physical performance, the following steps were deemed necessary:

1. Determine realistic and measurable criteria for training and performance
2. Identify as many intermediate components of the training/performance system as possible and assess their interactions
3. Decide which of the model's intermediate components would be most profitably measured and choose established experimental techniques to examine them
4. Establish mathematical techniques to test the model in the light of experimentally obtained data

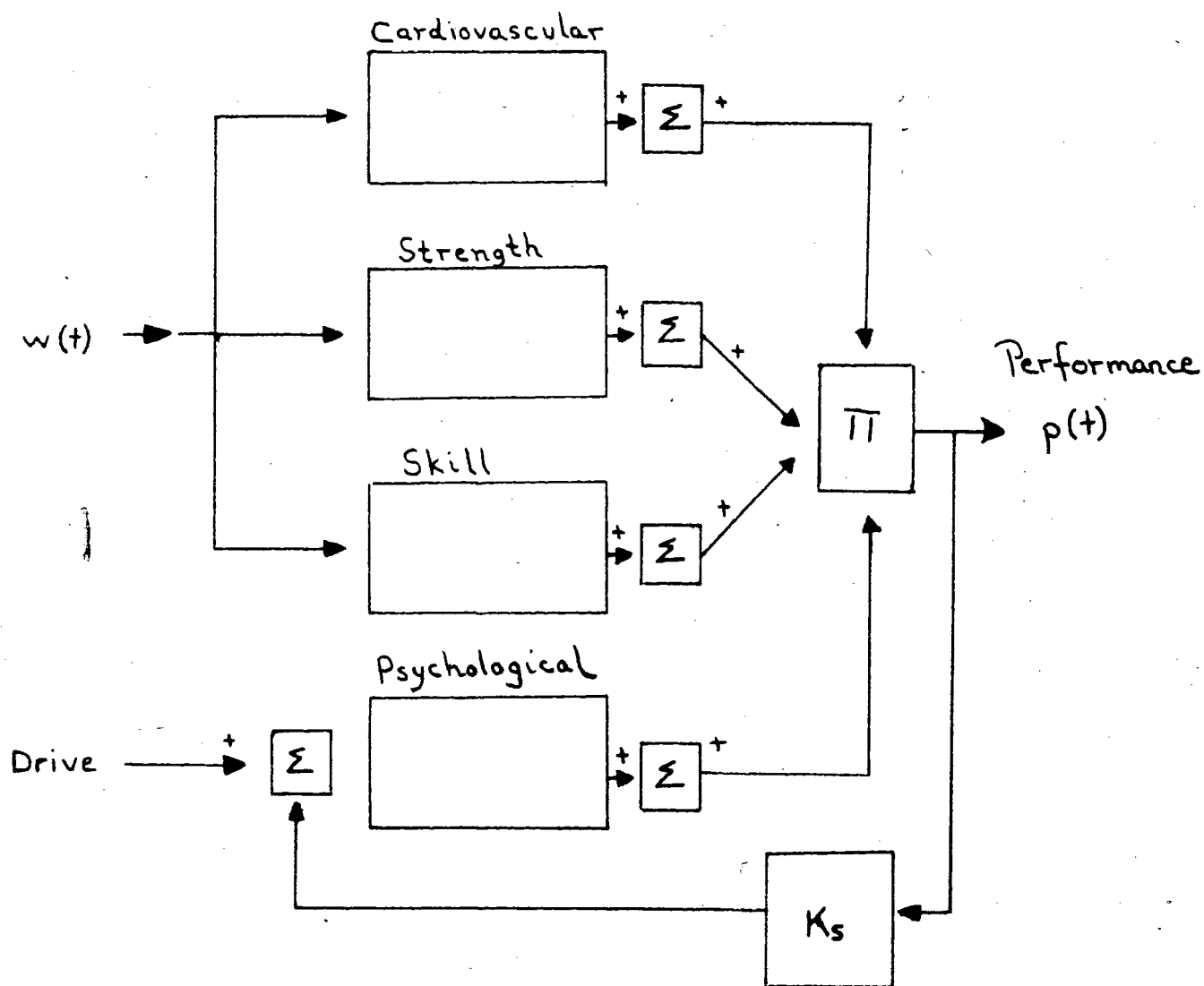
Two methods of quantifying training are available. The first was by taking some measure of physical work done (e.g. total distance run); the second, of particular importance in aerobic training, assesses the work done specifically by the cardiorespiratory system. Neither technique alone was thought to be sufficient: therefore, techniques for measuring both

simultaneously were established. To facilitate these measurements, it was decided to conduct training sessions on a cycle ergometer since work done could be easily determined in terms of force and distance (a calculation not as readily available from treadmill data) and cardiovascular stress could be simultaneously assessed via an electrocardiograph with display screen to provide the subject with continuous visual feedback of heart rate during training. To avoid possible difficulties in interpreting training data, it was decided to train exclusively aerobically. Therefore, a primarily aerobic performance criterion was established, specifically the time required to run two miles. A run performance was chosen in order to minimize the difficulties of skill and equipment associated with a cycling athletic performance.

From the literature, the large number of factors contributing to performance was evident. A general multifactorial model was constructed which described the interactions of the various factors (Figure 1) (Calvert et al, 1976). From this model, it was decided to evaluate regularly aerobic capacity and strength and ignore skill and psychological factors due to the relative unimportance of the former and the elusive quantification of the latter. It was also thought advisable to gather blood chemistry data for future reference since the study was not repeatable.

Figure 1: A Systems Model Approach To Performance

(reprinted from Calvert et al, 1976)



#### 4. Methodology

A subject underwent a four month regimen of strictly controlled physical training and physiological and athletic performance testing. The results were analyzed graphically and time constants of rise and decay of the measured parameters were determined. Time constants for additional useful parameters were taken from the available literature. The time constants were used to construct mathematical simulations of fitness and fatigue resulting from training. Fitness and fatigue were combined mathematically to obtain a simulation of performance based on the training profile. These results were tested for validity by referring to the measured values of performance.

##### A. Subject

The subject of the study was a twenty-five year old, physically active, male university student. Detailed anthropometric data was taken before the onset of training and Heath-Carter somatotype calculations are shown in Appendix 1.

##### B. Training Procedure

The subject trained each Monday, Tuesday, and Wednesday of active weeks. Physiological and performance testing was



conducted each Thursday and Friday; Saturdays and Sundays were days of physical inactivity. The 24 week training profile is shown in Figures 2 and 3. All training and performance sessions were held in the ~~late~~ morning or early afternoon. This was consistent with the peak of physiological diurnal rhythm which was measured over 24 hours by three tests: right hand grip strength, resting heart rate, and working heart rate at a work load of 990 kgm per minute. These results are shown in Figure 4. Training was done on a Monark bicycle ergometer. Pedalling speed was maintained at 60 rpm; resistance was adjusted each day to provide a steady state heart rate of 160 beats per minute. Heart rate was monitored constantly during exercise by a Physio-Control electrocardiograph.

Adjustment to steady state conditions was accomplished by a five minute period of cycling prior to the beginning of the recording of "training time".

#### C. Quantification of Training

Training was defined both in terms of total work done and in terms of cardiovascular stress. Training, in terms of work done, was defined as the product of the distance pedalled (in meters) and the cycle load (in kilograms). Training, in terms of cardiovascular stress, was defined as the amount of time of exercise at the steady state heart rate of 160 beats per minute.

Figure 2: Training, Defined as Cardiovascular Stress,  
Expressed In Training Units, Versus Time  
In Days

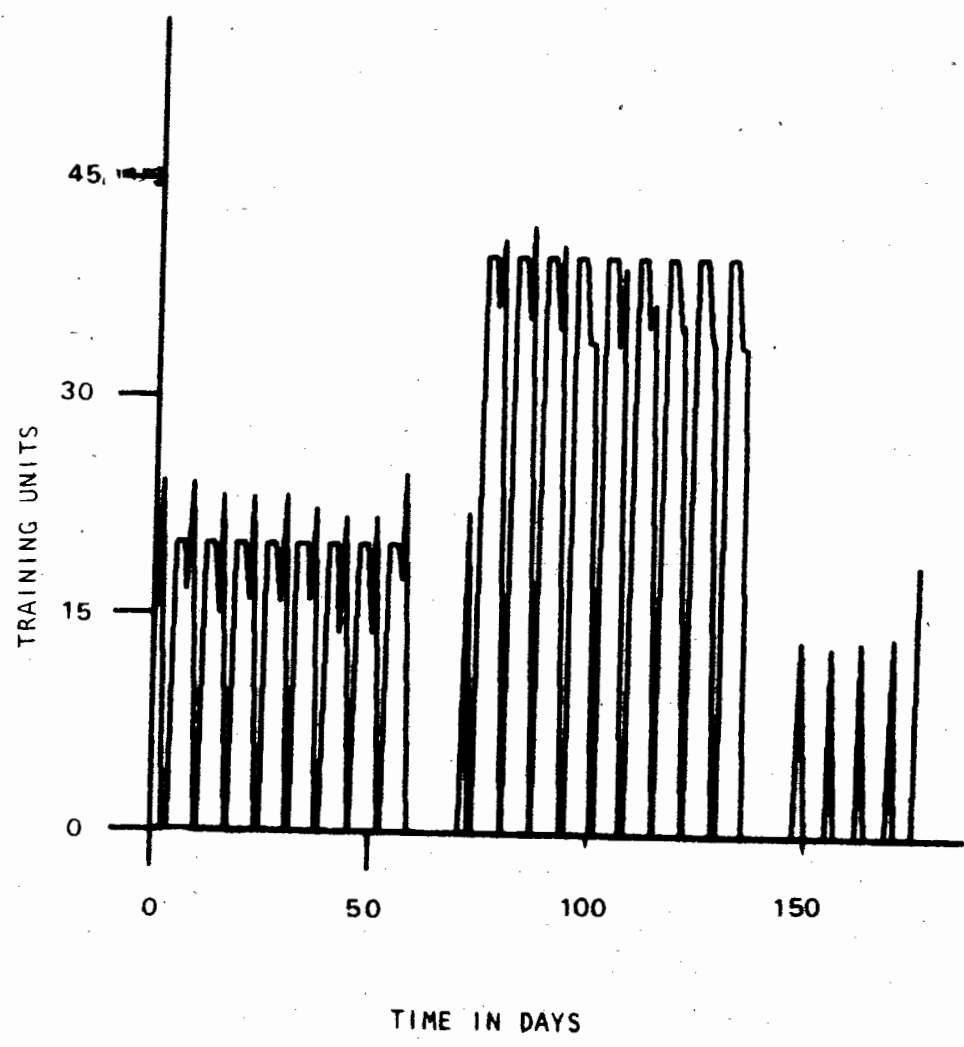


Figure 3: Training, Defined As Work Done,  
Expressed In Training Units,  
Plotted Versus Time In Days

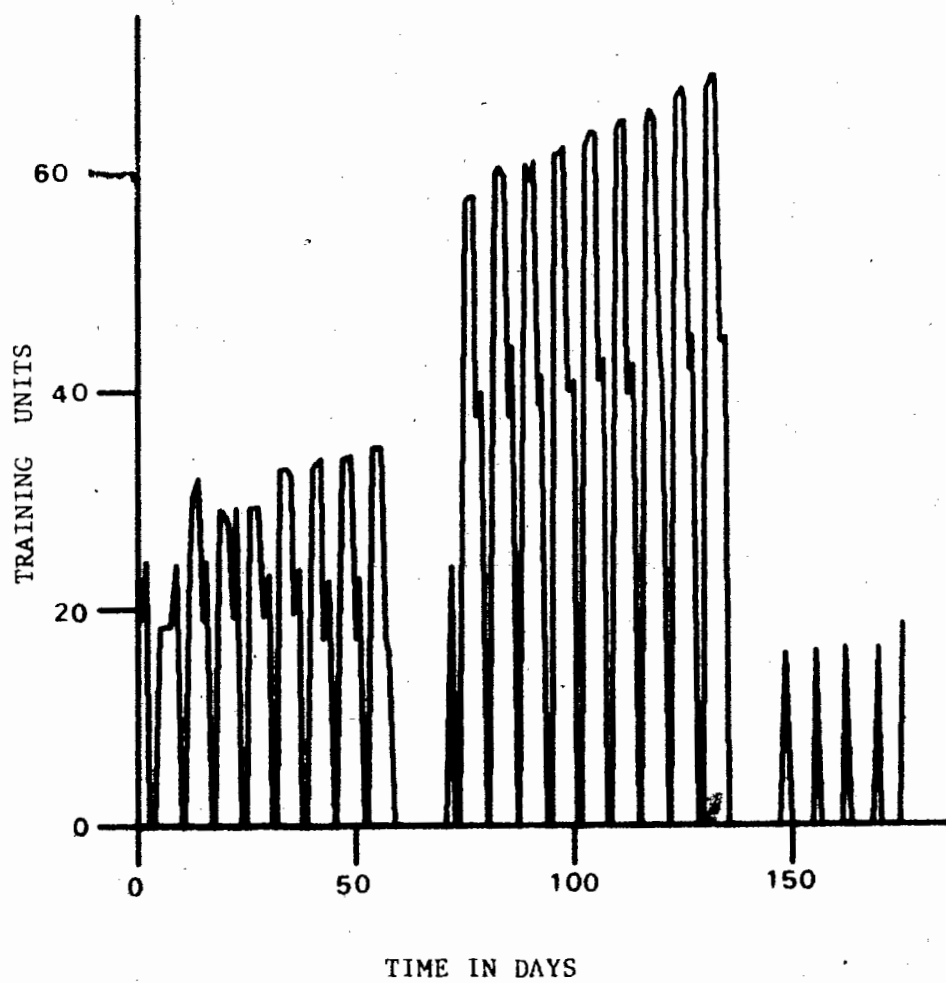
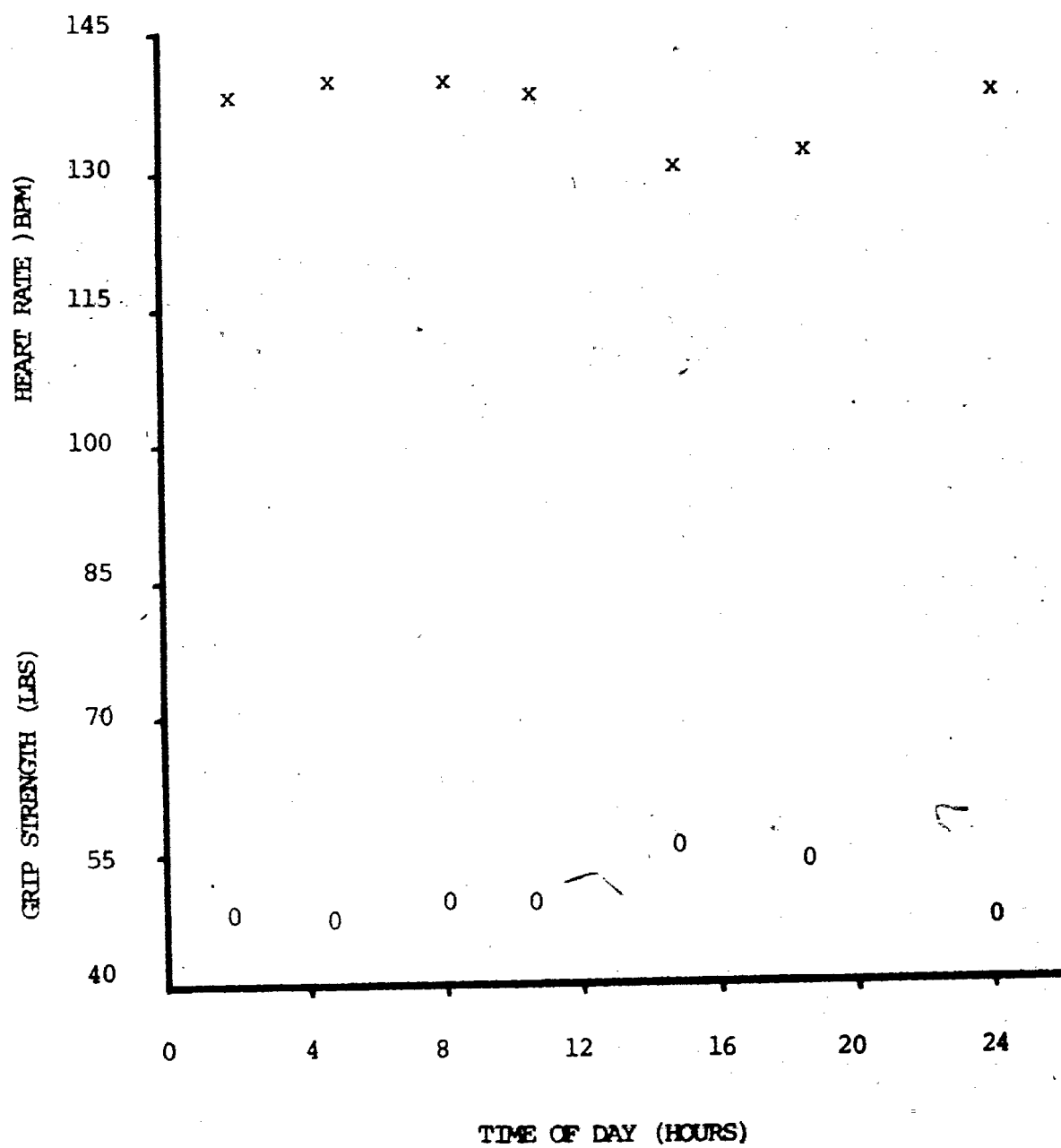


Figure 4: The Diurnal Rhythm Of Two Physiological  
Parameters

Right hand grip strength, measured in kilograms,  
and heart rate measured after ten minutes of  
cycling at a work rate of 990 kgm/min, were  
tested at three hour intervals over a period of  
twenty-four hours



Facility in data processing was enhanced by expressing training values in terms of "training units" (TU) in the following manner:

$$1 \text{ TU (work done)} = (1 \text{ km} \times 1 \text{ kg}) \div 1000$$

$$1 \text{ TU (heart stress)} = 1 \text{ minute exercising at HR} = 160 \text{ bpm}$$

The training value of both the physiological and performance testing sessions was also evaluated. The warmup prior to the maximal endurance test was conducted in three four-minute cycling phases with an increase in cycle load at the beginning of each phase. Training units for both work done and heart stress were calculated for each phase. The number of training unit equivalents (work done) was calculated as previously described. The number of training unit equivalents (heart stress) was calculated in the following manner:

$$\text{No. of TU Equivalents} = \text{cycling time} \times (\text{end phase heart rate}) \div 160$$

Values for the three phases were summed to obtain the total training value.



The maximal endurance test was conducted in phases of two minutes each with an increase in cycle load at the beginning of each phase. Training values for each phase were calculated by the method described for the warmup period and summed to obtain the total training value.

A ten minute warmup period on the cycle ergometer preceded each two mile run performance. Training values were calculated by the method described for the endurance performance warmup. The heart stress training value of the two mile run performance was calculated in the following manner:

$$\text{No. of TU Equivalents} = \text{run time} \times (\text{end run heart rate}) \div 160$$

The training value in terms of work done of the two mile run performance could not be directly calculated so was estimated in the following manner from data obtained that same day:

$$\text{No. of TU Equivalents} = \text{Warmup TU Equivalents (work done)} \times (\text{heart stress TU Equivalents of run} \div \text{heart stress TU Equivalents of warmup})$$

The training value of the 50 yard run and strength testing procedures was considered negligible and not included in the training profile.

#### D. Physiological Testing Procedures

##### i) Maximal Endurance Test

The maximal endurance test was preceded by a three phase cycle warmup, each phase lasting four minutes. The work load was increased at the beginning of each phase by increasing the belt tension on the cycle; pedalling speed was maintained constant at 60 rpm. Cycle loads were: Phase 1, 360 kgm per minute, Phase 2, 720 kgm per minute, and Phase 3, 1080 kgm per minute. Heart rate was recorded during the last ten seconds of each phase using a Fukuda recording electrocardiograph. Heart rate was plotted against work load and the physical working capacity at heart rate 170 (PWC170) was extrapolated.

The maximal endurance test was conducted on the cycle ergometer using the guidelines described by Anderson, Shephard, Denolin, Varnauskas, and Masironi (1971). The test progressed in phases of two minutes each, the work load being increased at the beginning of each phase. The cycle loads were: Phase 1, 960 kgm per minute, Phase 2, 1440 kgm per minute, Phase 3, 1800 kgm per minute, and Phase 4, 2160 kgm per minute. Heart rate was recorded during the last ten seconds of each minute using a Fukuda recording electrocardiograph. The test was concluded when the pedalling speed (initially 60 rpm) dropped below 60 rpm for five seconds.

During exercise, the subject breathed through a Collins Triple J valve from which expired gas passed through 1.5 inch diameter non-kinkable hose to a Parkinson-Cowan high precision, low resistance ventilation meter. Expired gas volume and temperature was measured each minute during exercise. During the sixth minute of exercise, a sample of expired gas was collected in a Douglas bag via a two-way valve. Oxygen and carbon dioxide composition of the sample was analyzed using the micro-Scholander technique (Scholander, 1948).

#### ii) Strength Testing

Six strength parameters testing the major muscle groups were measured weekly in accordance with the procedures outlined by Rogers (1975). Vital capacity was evaluated using a Collins 13.5 litre wet respirometer. Grip strength was measured for both right and left hands using a Stoelting rectangular hand dynamometer. Back and leg strength was tested with the Department of Kinesiology back and leg dynamometer. Arm strength was determined from the number of pushups and pullups as defined by Rogers (1975). The Rogers Strength Index was obtained in the following manner:

$$SI = VC + LG + RG + BS + LS + AS$$

where VC is the vital capacity in cubic inches, LG and RG are the left and right grip strengths in pounds, BS and LS are the back and leg strengths in pounds, and AS is the arm strength index, calculated as follows:

$$AS = (\text{pushups} + \text{pullups}) \times ((\text{body weight} \div 10) + \text{height} - 60)$$

Weight is given in pounds, height is given in inches.

### iii) Blood Chemistry Analysis

Immediately before and ten minutes after each two mile run, two blood samples were taken by medical personnel in the University Health Services Centre. Samples were taken in Vacutainer syringes. One was sent to the Coady Biomedical Laboratory, New Westminster, B.C. for SMA12 analysis as described in Appendix 2. Five mls. of the second sample was immediately transferred to a tube containing 5 mls. of 10% trichloroacetic acid in 0.5 N HCl. The mixture was agitated and centrifuged at 3000 rpm for ten minutes. The resulting supernatant was transferred to another tube and refrigerated in a sealed tube to await analysis. Lactate concentration was determined using the lactate dehydrogenase method of Bergmeyer (1962).

The effect of an impulse of training on blood lactate accumulation and clearance was also examined. Exhaustive exercise in the form of a cycle ergometer performance as described for the maximal endurance test was followed by a ten-minute post exercise blood sample and serial samples over a 24 hour period. Lactate analysis for these samples was identical to those taken following the two mile runs.

#### iv) Additional Physiological Parameters

Resting blood pressure and heart rate were measured in the University Health Services Centre prior to each two mile run performance.

#### E) Athletic Performance Testing

The maximal endurance test was conducted on the cycle ergometer as previously described.

The two mile run performance was done on the Simon Fraser University quarter-mile track. The time required to run two miles was recorded as the performance time.

The time required to sprint 50 yards has been shown to be a valid measure of anaerobic power (Margaria, 1966). Therefore, anaerobic power was measured weekly as the time to sprint 50 yards on the Simon Fraser University track.

## 5. Method of Analysis.

### a. Theoretical Overview

There are empirical reasons to expect that physiological determinants of physical performance can be described by a summation of components  $p_i(t)$  where each component is related to a profile of training by equations of the form:

$$A \frac{d^2 p(t)}{dt^2} + B \frac{dp(t)}{dt} + C p(t) = f(w(t)) \quad (1)$$

where  $f(.)$  is a non-linear function of training intensity  $w(t)$

The total performance is then given by


$$p(t) = \sum_{i=0}^N p_i(t) \quad (2)$$

The simple case where there is one component and the differential equation is of the first order is shown in Equation 3.

$$K \frac{dp(t)}{dt} + p(t) = f(w(t)) \quad (3)$$

A step change in  $w(t)$  will cause a step change in  $f(w(t))$  independent of the form of  $f(\cdot)$ . Therefore, for a step increase in  $w(t)$  followed sometime later by a step decrease, the response is that described by equations 4 and 5 and depicted in Figure 5.

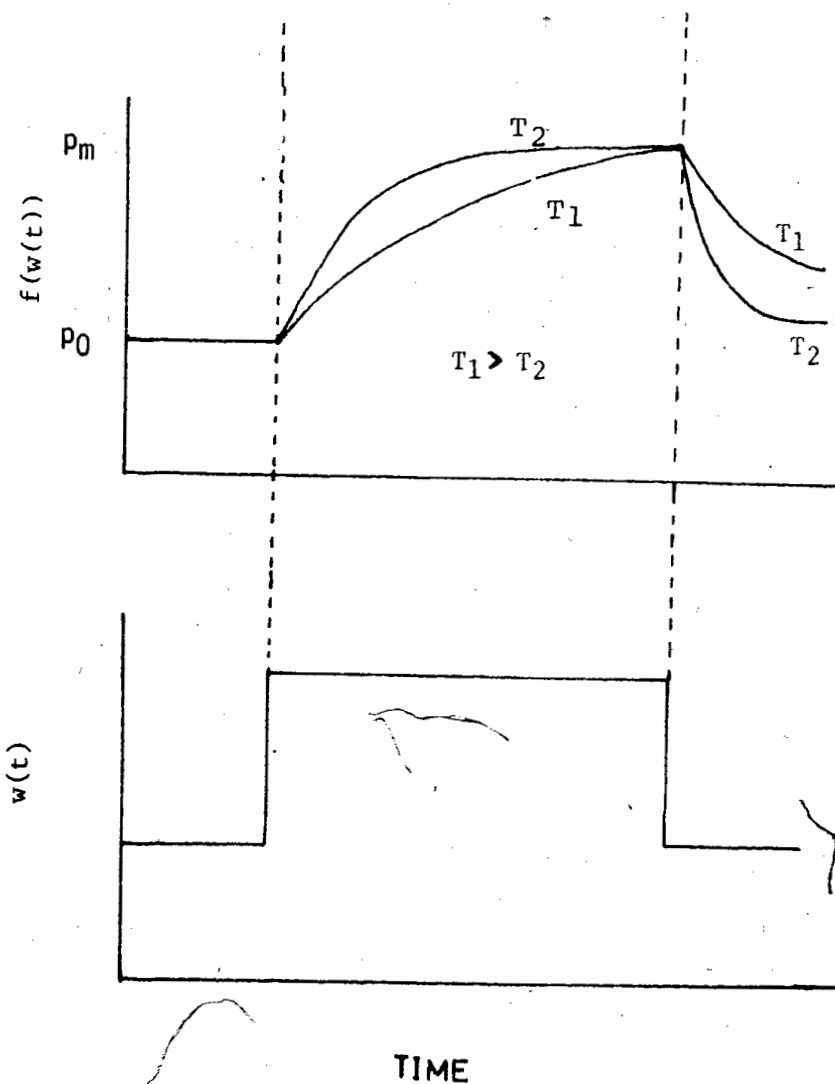
$$p(t) = (p_m - p_0) (1 - e^{-t/T}) + p_0 \quad (4)$$

$$p(t) = (p_m - p_0) e^{-t/T} + p_0 \quad (5)$$


where  $T$  is the time constant of the exponential change ( $K$  in equation 3). Very few physiological processes are completely described by a single component in the response: however, frequently one component dominates the others and it is possible to estimate the dominant time constant by observing the drop in the parameter after training ceases.

Figure 5: The Theoretical Response Of A First Order  
System to A Step Input





The purpose of the analysis is to identify the time constants and the non-linear function  $f(\cdot)$ . This can be done by choosing the constants and the non-linear function so that the variance between actual data and the model response is minimized. Formally, if data points  $p(t_1), p(t_2), \dots, p(t_n)$  represent measured values at times  $t_1, t_2, \dots, t_n$ , and  $q(t_1), q(t_2), \dots, q(t_n)$  represent the model response values at those times, then the mean squared error (variance) is

$$E^2 = \frac{1}{N} \sum_{i=0}^N (p(t_i) - q(t_i))^2$$

While some analytic methods are available to find constants to minimize the mean squared error, it will generally be necessary to use an iterative approach to find time constants and the form of the function involved.

In this method, profiles of real data are approximated by a series of components. In general, it is possible to obtain a good approximation to any profile of measured data if a sufficiently large number of components are used in the model. Thus, the best model is judged to be that in which a good approximation is obtained with a minimum of components which have been chosen with sound foundations in physiological reality.

### b. Estimation of Time Constants

The onset of training is known to induce increases in the magnitudes of those physiological parameters which contribute to fitness (e.g. maximal oxygen uptake, strength) and fatigue (e.g. lactate concentration, glycogen depletion, tissue trauma). The cessation of training results in decreases in the magnitudes of those parameters. Each of these rises and decays is characterized by a time constant,  $T$ , which is the time required for the parameter to rise to about 0.638 of its maximum value (or fall to 0.362 of its maximum value). The impulse response of a linear physiological parameter following a training session may be expressed in the following way:

$$p(t) = K e^{-t/T}$$

where  $p(t)$  is the magnitude of the parameter and  $T$  is the dominant time constant of decay of the parameter.

Because of the short duration of a training session (<1 hour) relative to the length of the time constants (>5 days) each training session may be considered an impulse.

From this relationship, it can be seen that as  $T$  decreases, the rate at which  $p(t)$  changes is increased, i.e. the smaller the time constant, the greater the rate of change

of the parameter involved. The time constants for the relevant parameters measured in this study were estimated graphically as depicted in the example in Figure 6. The measured values were plotted against time and the "smooth curve" intuitively determined to be the best fit to the points was drawn.

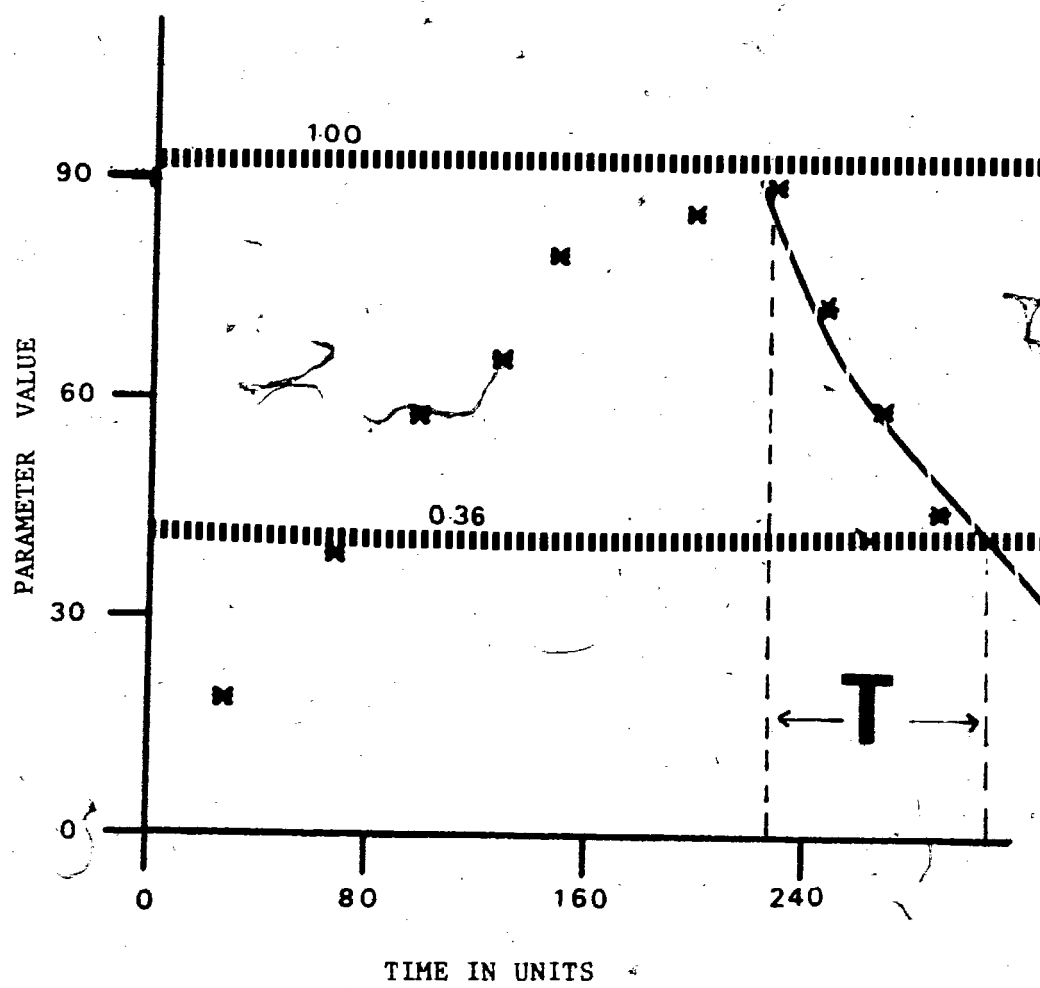
The dominant time constant of decay for each parameter was determined from the descending portion of the curve, using the time at which training ceased as the "time = 0 point" and then determining the time required for the curve to descend to about 0.362 of its maximum value. Since time constants were being estimated and not calculated exactly, it was not considered necessary to use a logarithmic plot for time constant determination.

### c. Simulation Techniques

The knowledge of the rates at which the physiological parameters changed with the onset and cessation of training provided the basis upon which the simulations were performed. The simulations were done using the computer programme ANALOG, written in APL and performed on the Simon Fraser University IBM 360 computer, listed in Appendix 3.

The simulation programme calculated simulated output by incorporating the experimentally determined time constants of the physiological parameters into a set of differential equations of the following form:

Figure 6: The Calculation Of Time Constants  
Of Physiological and Performance  
Parameters



$$B \frac{dy}{dt} + C y = D \frac{dx}{dt} + E x \quad (2)$$

where  $y$  represents the response function and  $x$  is the training function (forcing function).

Equation 2 can be rewritten as

$$\frac{B}{C} \frac{dy}{dt} + y = \frac{D}{C} \frac{dx}{dt} + \frac{E}{C} x \quad (3)$$

or

$$T \frac{dy}{dt} + y = K_1 \frac{dx}{dt} + K_2 x \quad (4)$$

where  $T=B/C$  is the time constant and  $K_1$  and  $K_2$  are the gain constants for a direct forcing function  $x$  and a forcing function dependent on the rate of change of  $x$  respectively.

From these equations, the changes in various parameters were simulated using training as input. For example, the data shown in Figure 7 clearly indicates that the response of maximal oxygen uptake is dominated by a single first order system described by the following equation:

$$B \frac{dV_{O_2}(t)}{dt} + V_{O_2}(t) = f(w(t)) \quad (5)$$

The time constant was estimated to be 23 days and the non-linear function used was

$$f(w(t)) = K(w(t))^n + V_{O_2}(0) \quad (6)$$

where  $n=0.3$  and  $V_{O_2}(0)$  is the maximal oxygen uptake in the untrained subject. The exponent  $n$  is used to describe the relatively greater effect of training at the beginning of the regimen than at the end.



#### d. Simulation Of Performance Capacity

The simulation principles applied to physiological parameters were also used to simulate functions representing "Fitness" and two types of "Fatigue", chronic and acute. Time constants for these hypothetical functions were taken from the combined information provided by the literature, experimentally obtained data, and subjective feelings of the subject during the study. The fitness and fatigue functions were combined to generate a "Performance Capacity" curve which was regressed on measured performance data as a means of testing the validity of the model described by the equation

$$PERFORMANCE = FITNESS - (TOTAL FATIGUE)$$

## 6. Results

Maximal oxygen uptake increased from a pre-training value of 52.5 ml/kg/min to a value of 60.5 ml/kg/min at the end of the first phase of training. During the second phase of training, the values increased from 57.0 ml/kg/min to a maximum of 64 ml/kg/min. The time constant calculated from the period following the final cessation of training was 23 days (Figure 7).

The Rogers Strength Index increased from a pre-training value of 2123 to a value of 2209 at the end of the first phase of training. During the second phase of training, the value increased from 2206 pounds to 2335 pounds. The time constant of decay calculated following the final cessation of training was 60 days (Figure 8).

Leg strength increased from a pre-training value of 900 pounds to a value of 1000 pounds at the end of the first phase of training. During the second phase of training, the value increased from 990 pounds to a maximum of 1080 pounds. The time constant of decay calculated from the period following the final cessation of training was 60 days (Figure 9).

Blood lactate concentration increased from a resting level of 10 mg/100 ml to a post-exercise maximum of 104 mg/100ml. The return to a normal resting level was complete within about three hours, the time constant being 60 minutes (Figure 10).

Two mile run time decreased during the training period from an initial time of 14.34 minutes to a time of 12.75 minutes at the end of the first phase of training. Following the final cessation of training, at which a best time of 11.76 minutes had been achieved, the time constant of decay was calculated to be 50 days (Figure 11).

Cycle endurance time increased during the training period from an initial time of 6.33 minutes to a time of 7.50 minutes at the end of the first phase of training. Following the final cessation of training, at which a best time of 7.81 minutes had been achieved, cycle endurance performance decayed with a time constant of 50 days (Figure 12).

Simulations of the two physiological factors hypothesized to contribute to the selected performance criteria are shown in Figure 13 (maximal oxygen uptake) and Figure 14 (leg strength). The simulated values were obtained using the calculated time constants of the actual measured values and training as the forcing function. For the oxygen uptake simulation, training

expressed as heart stress was used while for the leg strength simulation, training expressed as work done was used. Simulated values were regressed on actual values and plotted with the actual measured values.

A simulation of "fitness" was obtained by using a time constant of 50 days (the same as that calculated for the performance data) and training expressed as work done as the forcing function. The simulated values were regressed on actual values of two mile run performance and plotted simultaneously with actual two mile run times in Figure 15.

A simulation of chronic fatigue was obtained by using a time constant of 15 days (Banister et al, 1975) and training expressed as work done as the forcing function. The simulation of the chronic fatigue profile, expressed in arbitrary units is shown in Figure 16.

A second simulation of two mile run performance was generated by subtracting the chronic fatigue profile from the fitness profile. The simulated values were regressed on actual measured values and plotted simultaneously with actual values in Figure 17.

A simulation of acute fatigue was generated using a time constant of 7 days (DeVries, 1974), a delay of 3 days (based on the subject's feelings during the study), and training expressed as work done as the forcing function. Simulated values of acute fatigue, expressed in arbitrary units, are plotted versus time in Figure 18.

A simulation of total fatigue was generated by combining chronic and acute fatigue equally weighted. The simulated values were regressed on actual weekly measured values of serum bilirubin (hypothesized to be a biochemical monitor of fatigue) and plotted with actual measured values in Figure 19.

Figures 20 and 21 display respectively the simulations of two mile run and cycle endurance performance obtained by subtracting the total fatigue curve from the fitness curve. Simulated values were regressed on actual values and are plotted with the measured values.

Figure 22 shows a simulation of two mile run performance obtained by subtracting total fatigue from fitness, but weighting acute fatigue at 1.75, chronic fatigue at 1.00 and fitness at 1.00. the weighting constants were obtained via regression analysis (Appendix 4) and refined iteratively.

Anaerobic power as measured by the time required to run 50 meters did not show an improvement with training.

Blood chemistry data, taken each week following the two mile run performance, is shown in Appendix 2.

Figure 7: The Response Of Maximal Oxygen Uptake  
To Training

Maximal oxygen uptake was evaluated weekly during the training regimen. These values in ml/kg/min are plotted versus time in days.

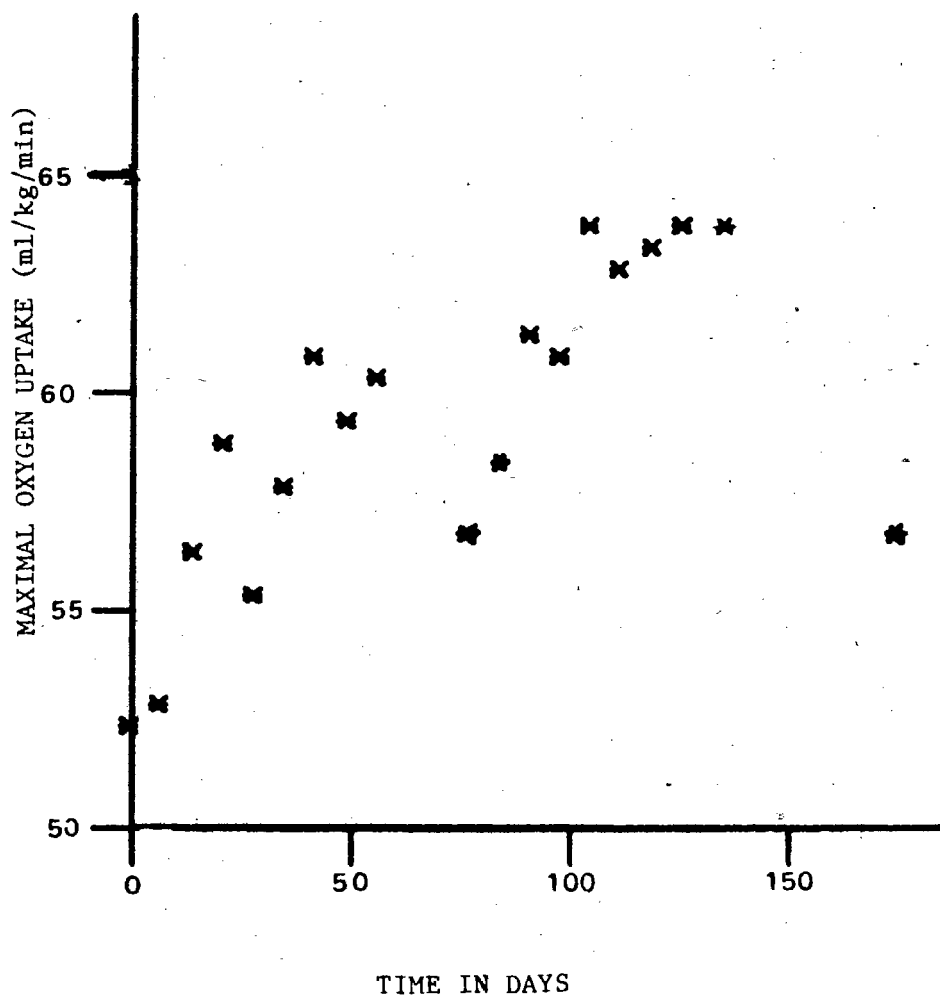




Figure 8: The Response Of The Rogers Strength Index  
To Training.

The Rogers Strength Index was evaluated weekly during the training regimen. These values, measured in pounds, are plotted versus time in days.

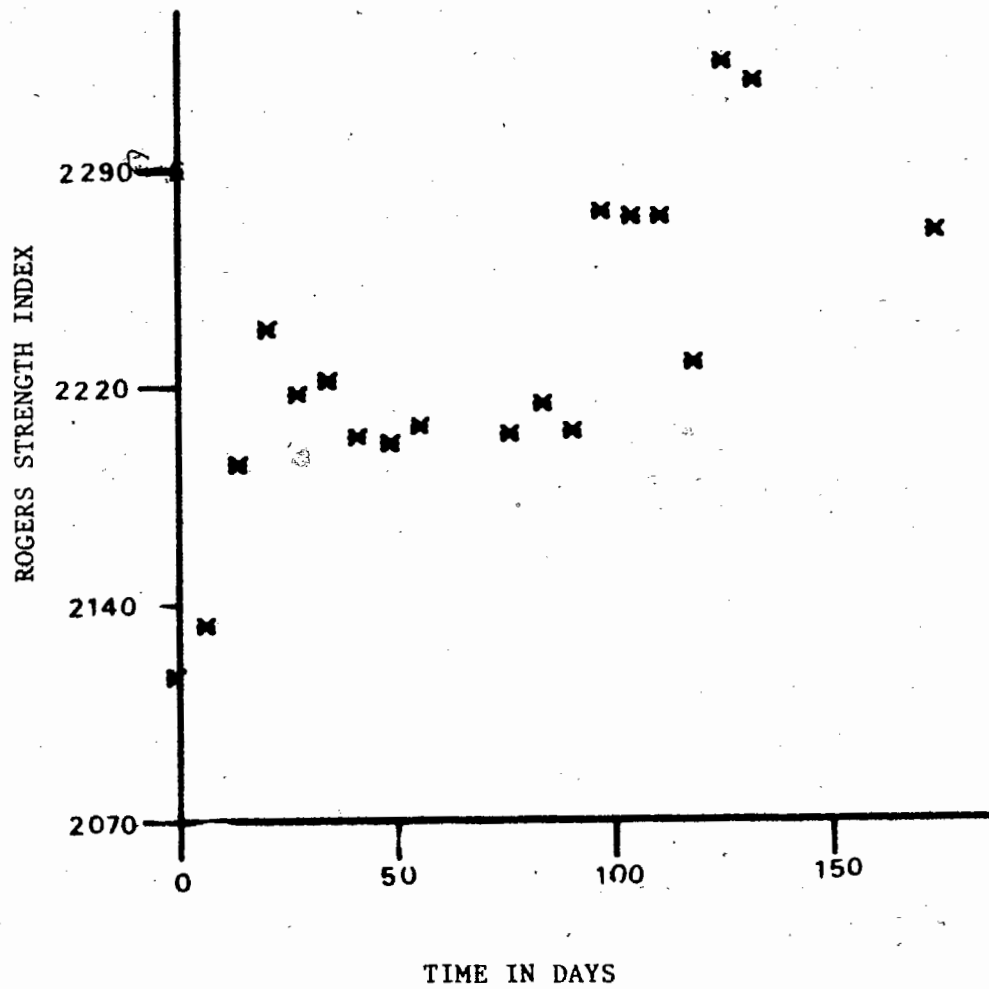


Figure 9: The Response of Leg Strength  
To Training

Leg Strength was evaluated weekly during the training regimen. These values, measured in pounds, are plotted versus time in days.

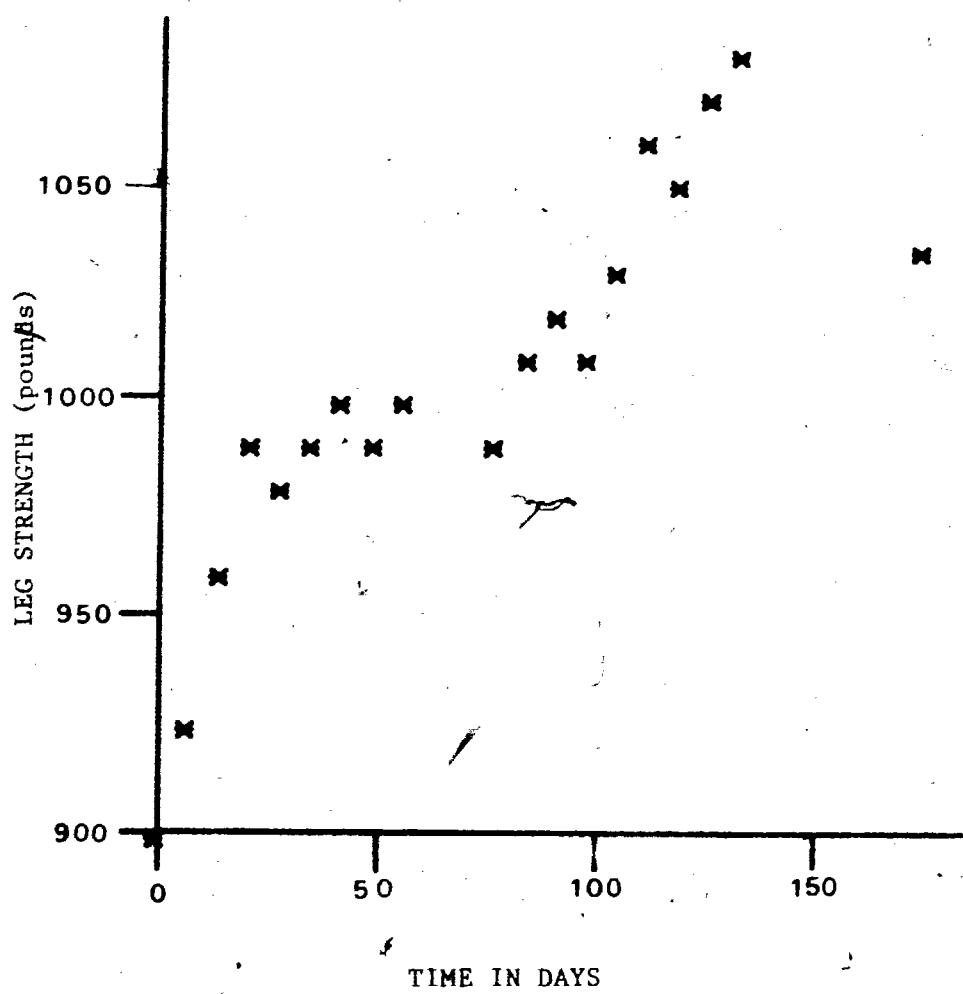


Figure 10: The Response of Blood Lactate  
Concentration To Heavy Exercise

Blood lactate concentration was determined at regular intervals for two hours following a bout of exhaustive exercise on the cycle ergometer. These values, measured in mg lactate/100 mls blood, and the pre-exercise concentration, are plotted versus time in minutes.

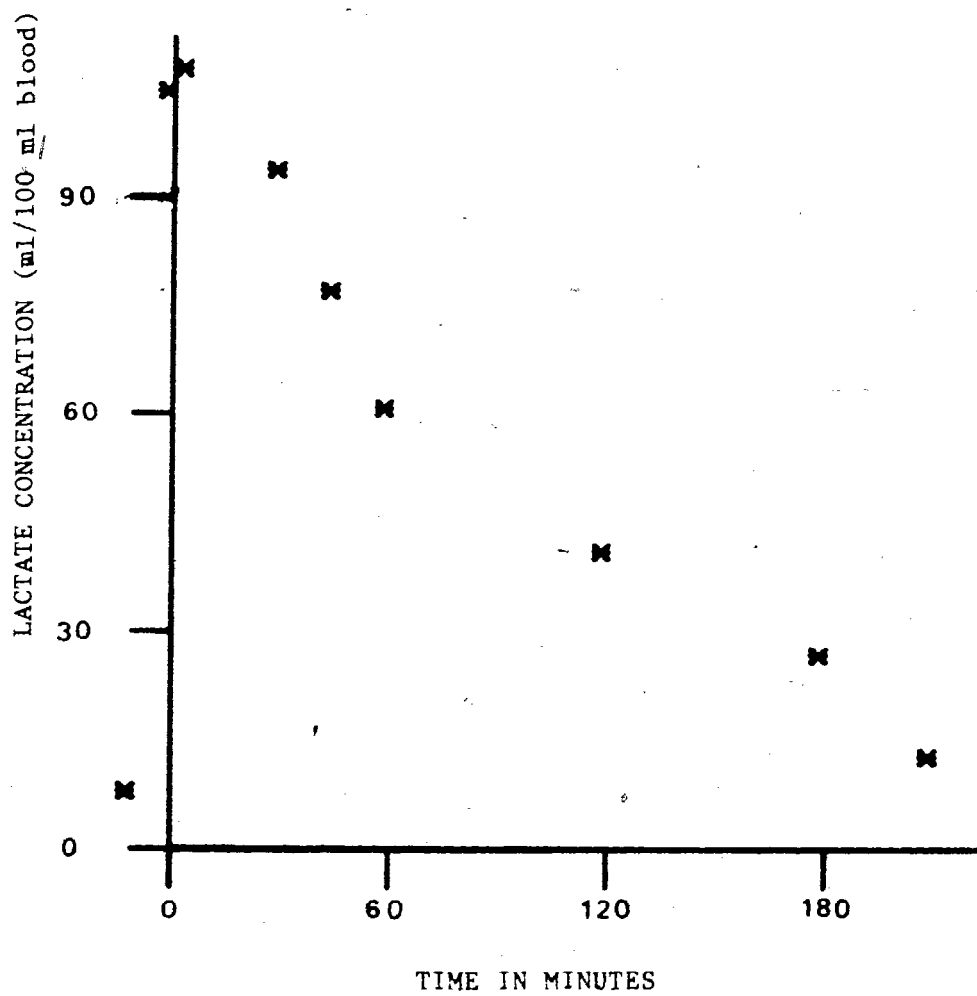


Figure 11: The Response of Two Mile Run  
Performance To Training

The time taken to run two miles  
was evaluated weekly. These values,  
measured in minutes, are plotted  
versus time in days.

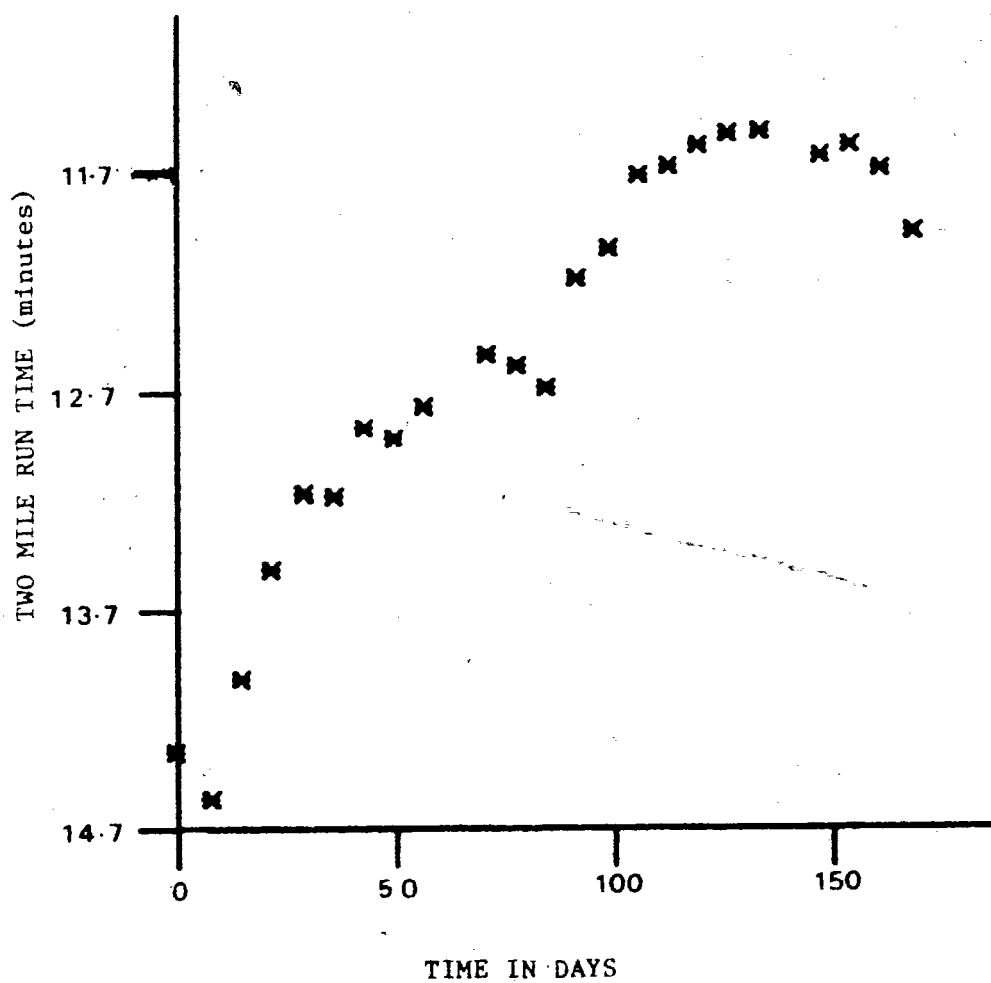
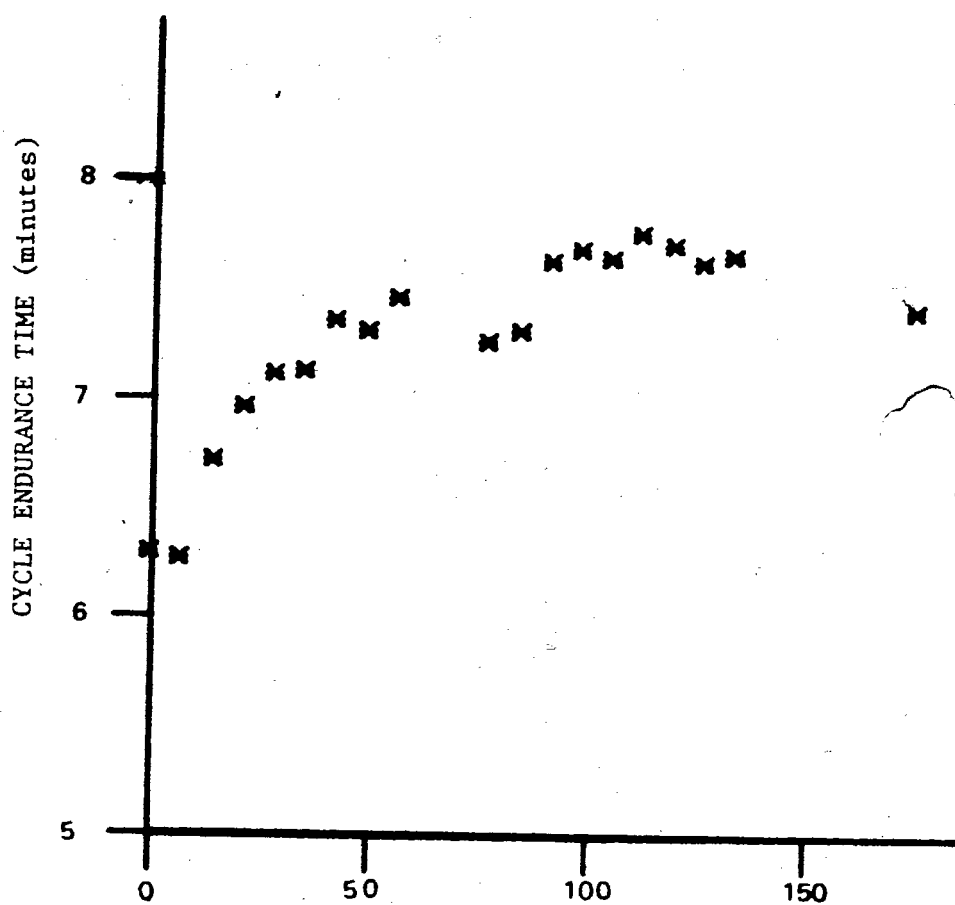




Figure 12: The Response of Cycle Endurance  
Performance To Training

Endurance performance on the cycle  
ergometer was evaluated weekly.

these values, measured in minutes,  
are plotted versus time in days.



TIME IN DAYS

Figure 13: The Simulation of Maximal Oxygen  
Uptake

The simulated profile of maximal oxygen uptake, performed using a time constant of 23 days and the cube root of training (Figure 1) as the forcing function, is plotted versus time in days. Actual maximal oxygen values (Figure 5) are simultaneously plotted.

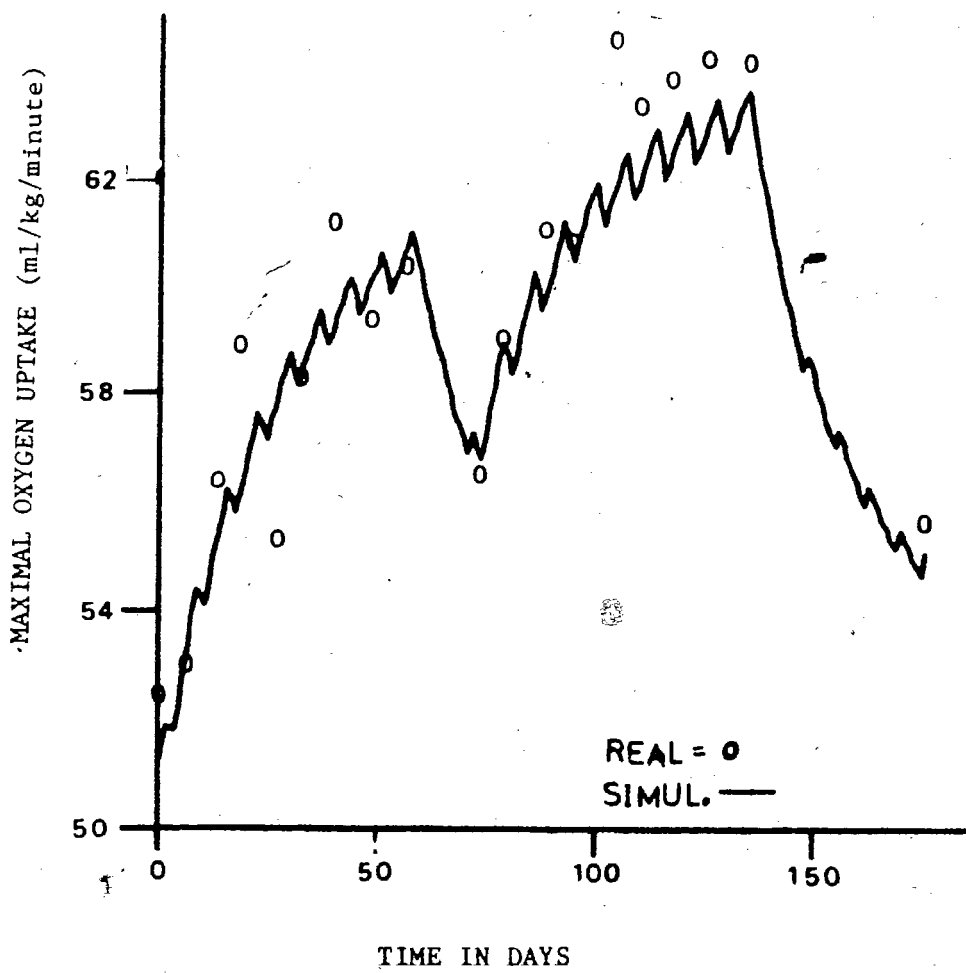


Figure 14: The Simulation of Leg Strength

The simulated profile of leg strength, generated using a time constant of 60 days and the cube root of training as the forcing function (Figure 2) is plotted versus time in days. Actual values of leg strength (Figure 9) are simultaneously plotted.

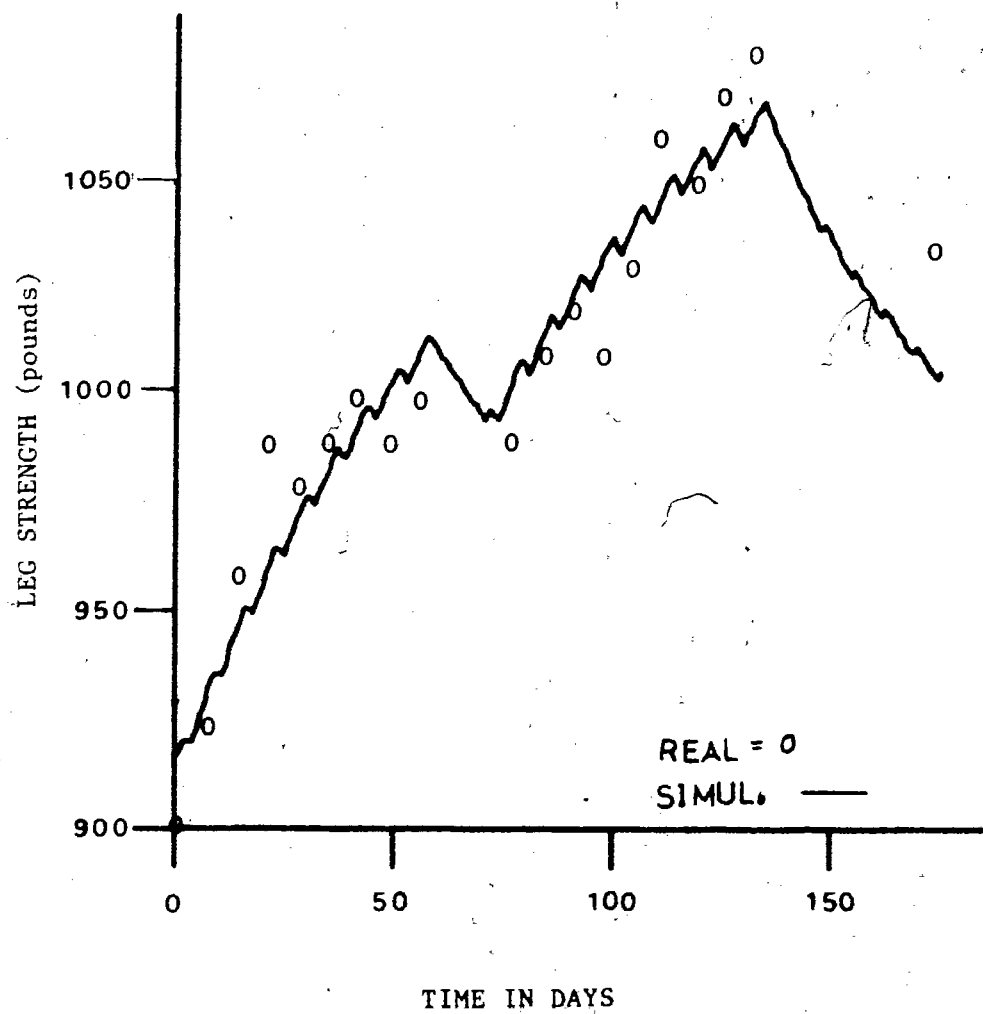


Figure 15: The Simulation of Fitness

The simulated profile of fitness, generated using a time constant of 50 days (the same as that of performance) and the forcing function seen in Figure 2, is plotted versus time in days. The actual values of two mile run performance are simultaneously displayed.

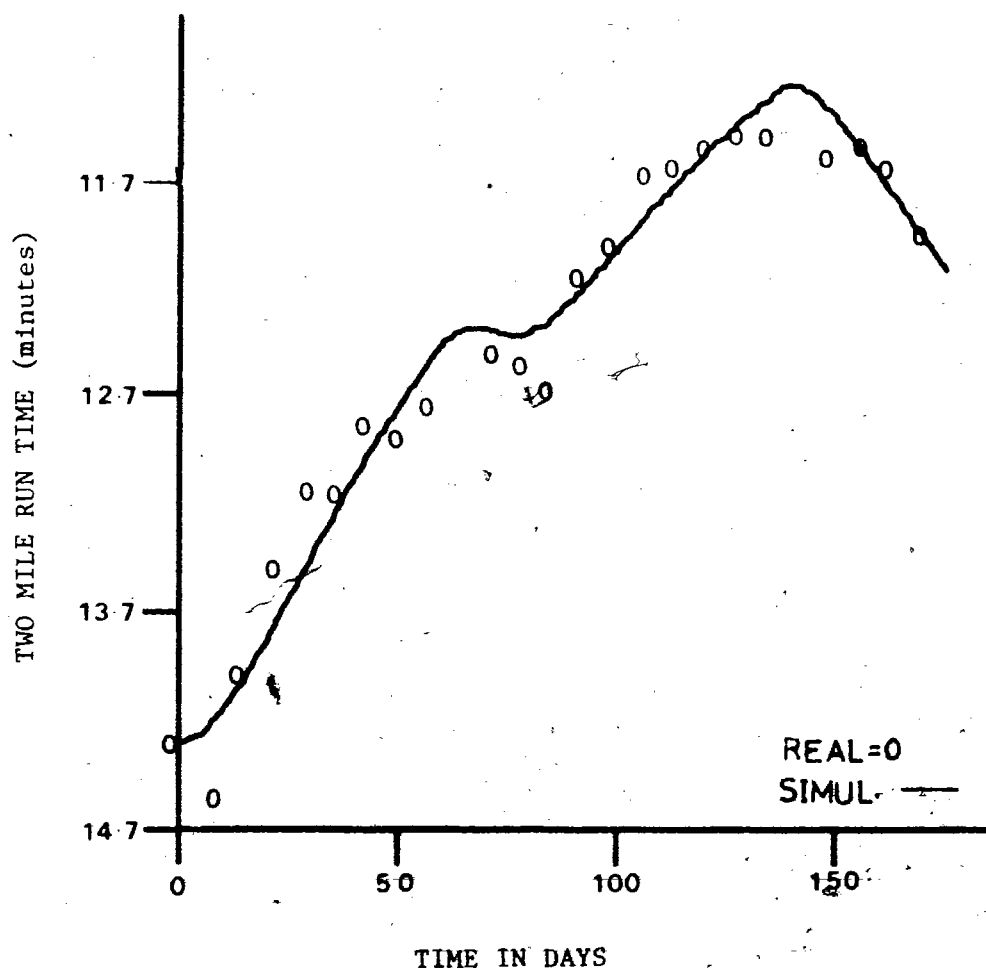




Figure 16: The Simulation of Chronic Fatigue

The hypothesized profile of chronic fatigue, generated using a time constant of 15 days (Calvert et al, 1975) and the forcing function seen in Figure 2, is plotted versus time in days.

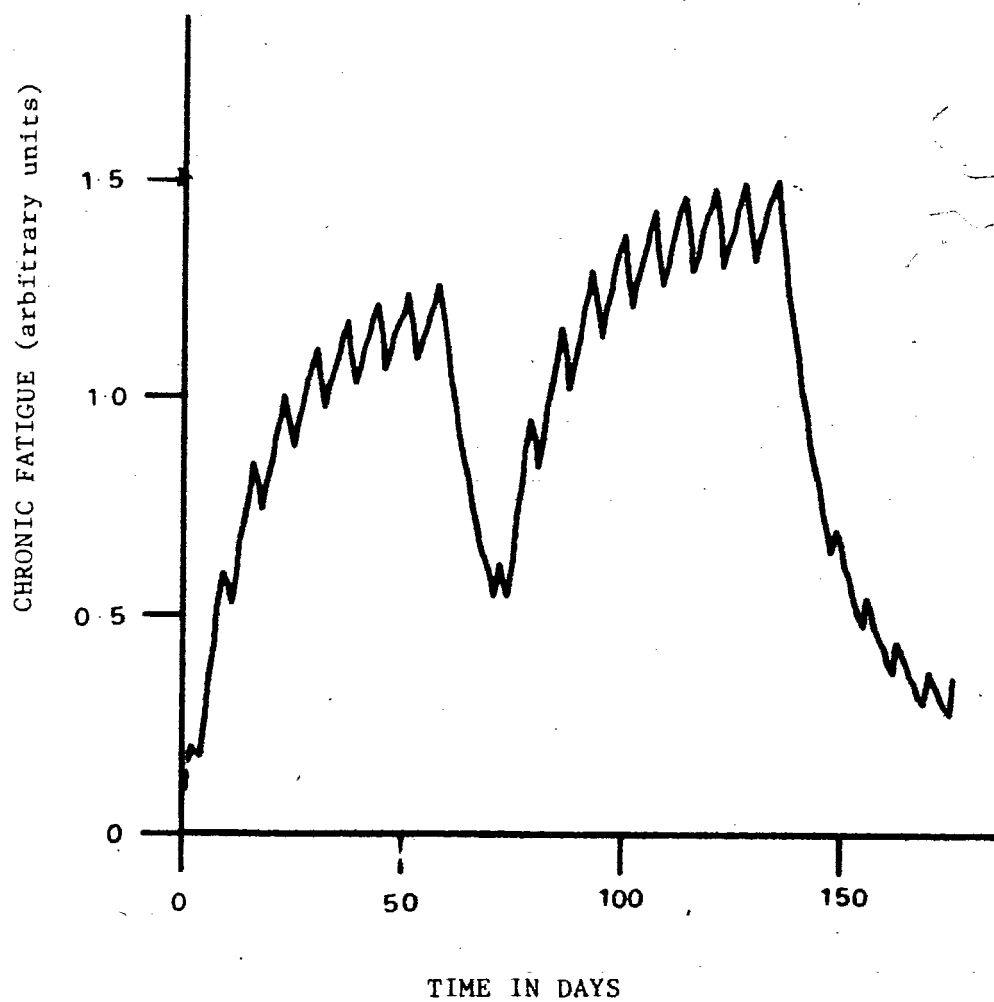


Figure 17: The Simulation of Performance

The simulated profile of performance generated by subtracting the fatigue curve in Figure 15 from the fitness curve in Figure 15, is plotted versus time in days. The actual values of two mile run performance in minutes are simultaneously displayed.

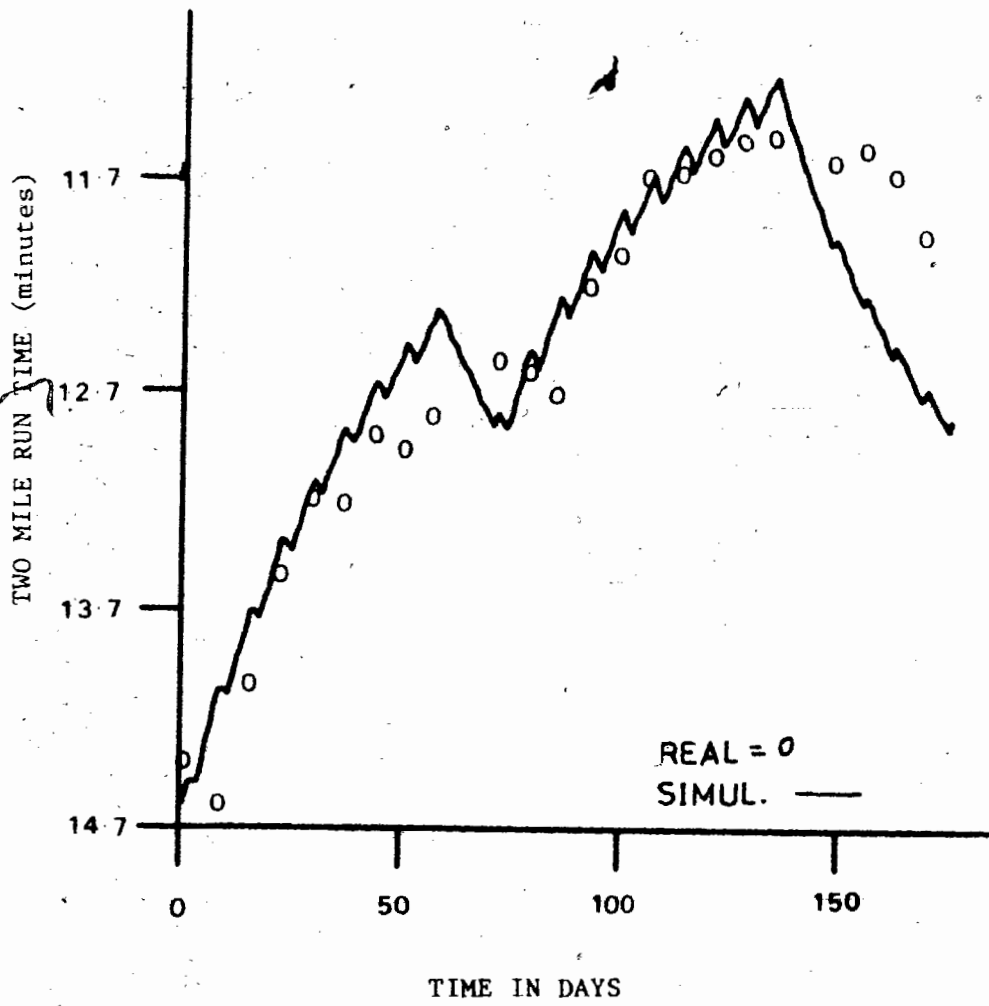


Figure 18: The simulation of Acute Fatigue

The simulation of acute fatigue, a component which responds to changes in training intensity, generated using a time constant of 7 days, a delay of three days following the onset of training, and the forcing function seen in Figure 2, is plotted versus time in days.

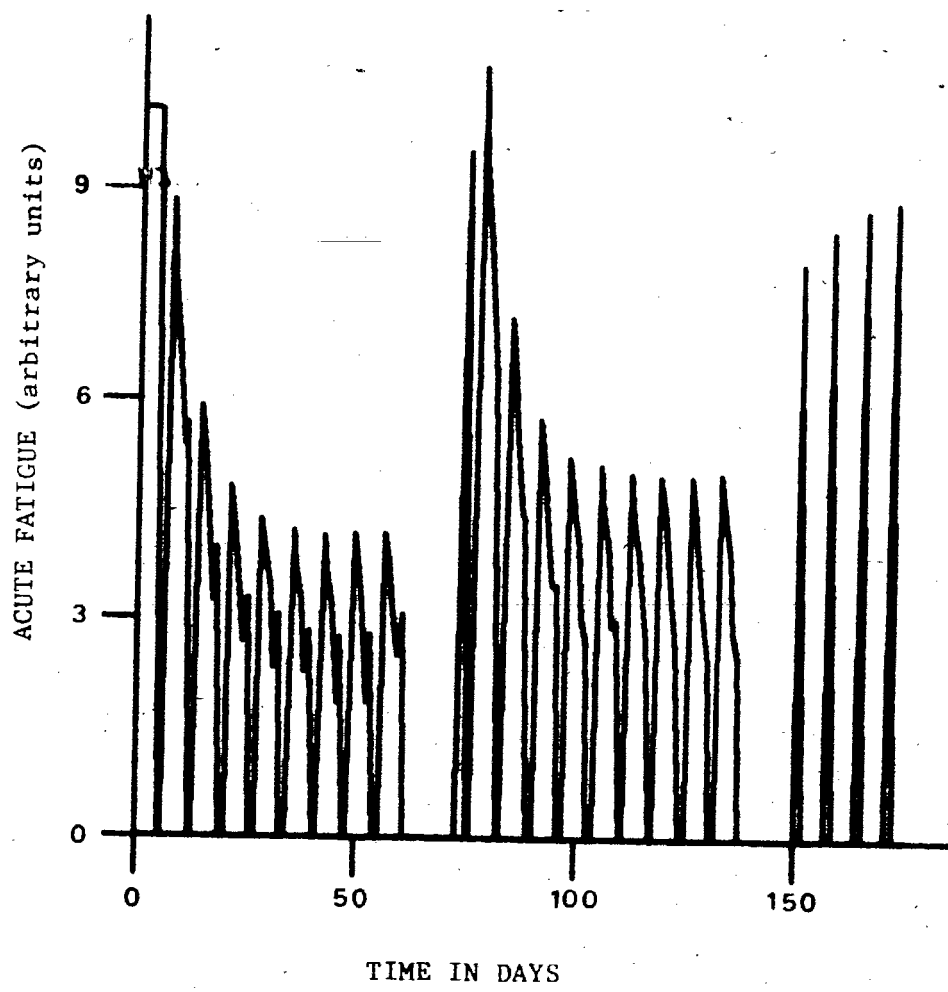


Figure 19: The Simulation of Total Fatigue

The simulation of total fatigue, generated by adding the curves of chronic and acute fatigue (Figures 16 and 17), is plotted versus time in days. The actual values of serum bilirubin, measured in mg/dl blood, are simultaneously displayed.

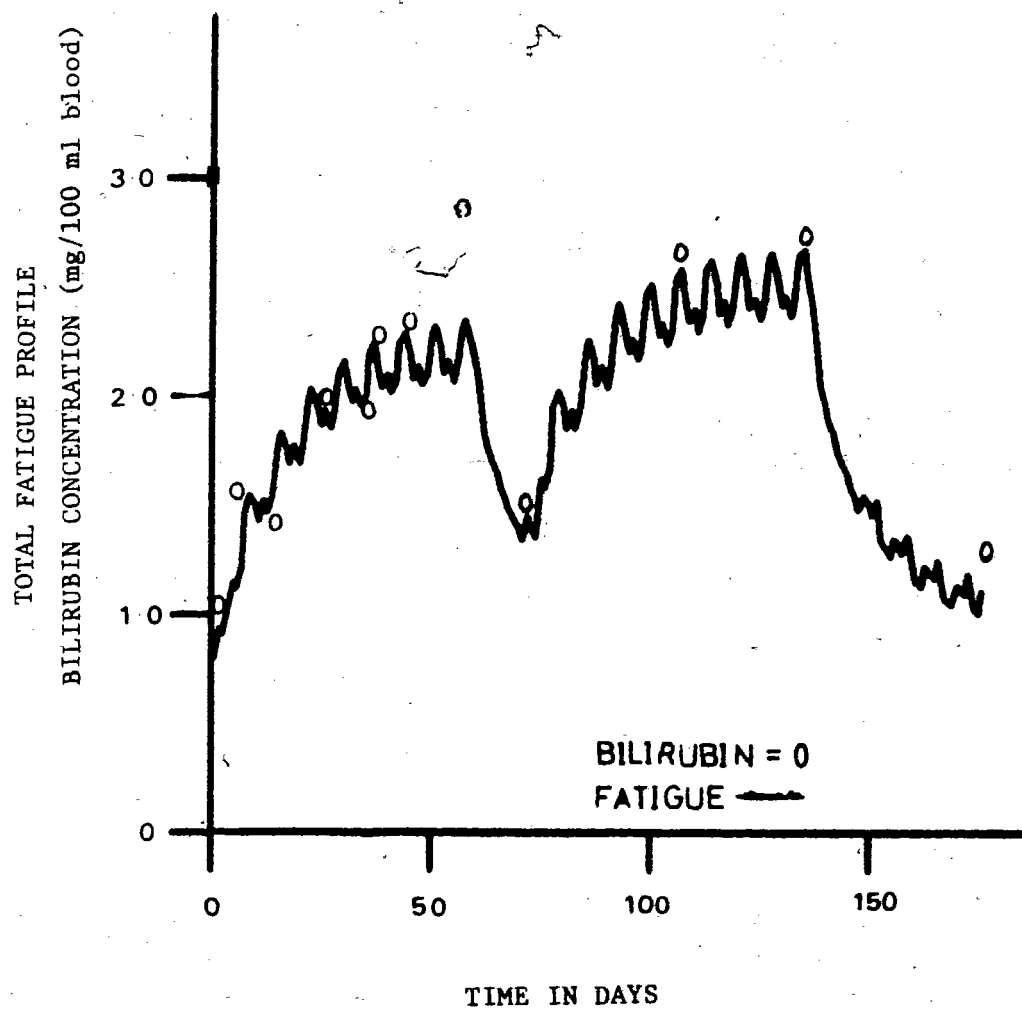




Figure 20: The Simulation of Performance,  
Modified To Account For The  
Inclusion of Acute Fatigue

The simulation of performance,  
generated by subtracting the  
total fatigue simulation (Figure  
17) from the fitness curve  
(Figure 15), is plotted versus  
time in days. Actual two mile run  
performance is simultaneously plotted.

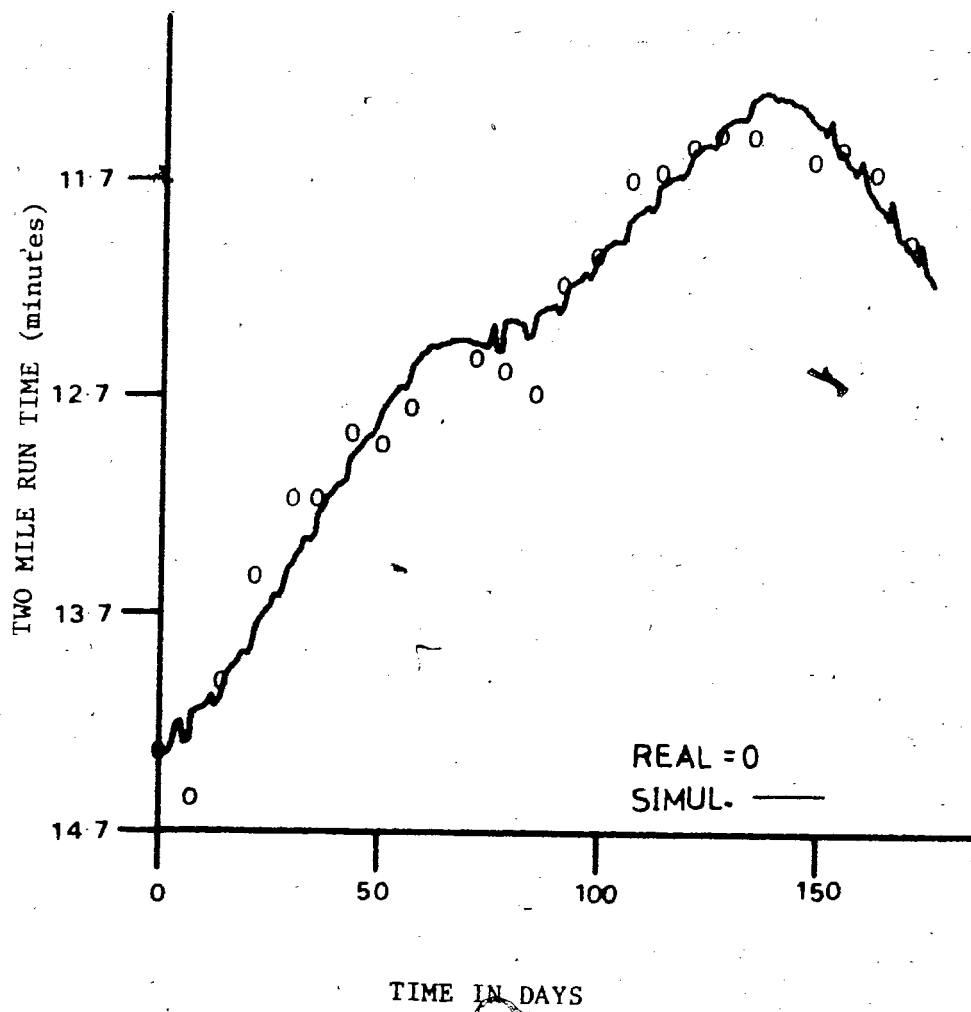


Figure 21: The Simulation of Performance  
Accounting For The Inclusion  
of Acute Fatigue.

The simulation of performance,  
generated by subtracting the  
total fatigue curve (Figure 17)  
from the fitness curve (Figure 15),  
is plotted versus time in days.  
The actual values of cycle endurance  
are simultaneously displayed.

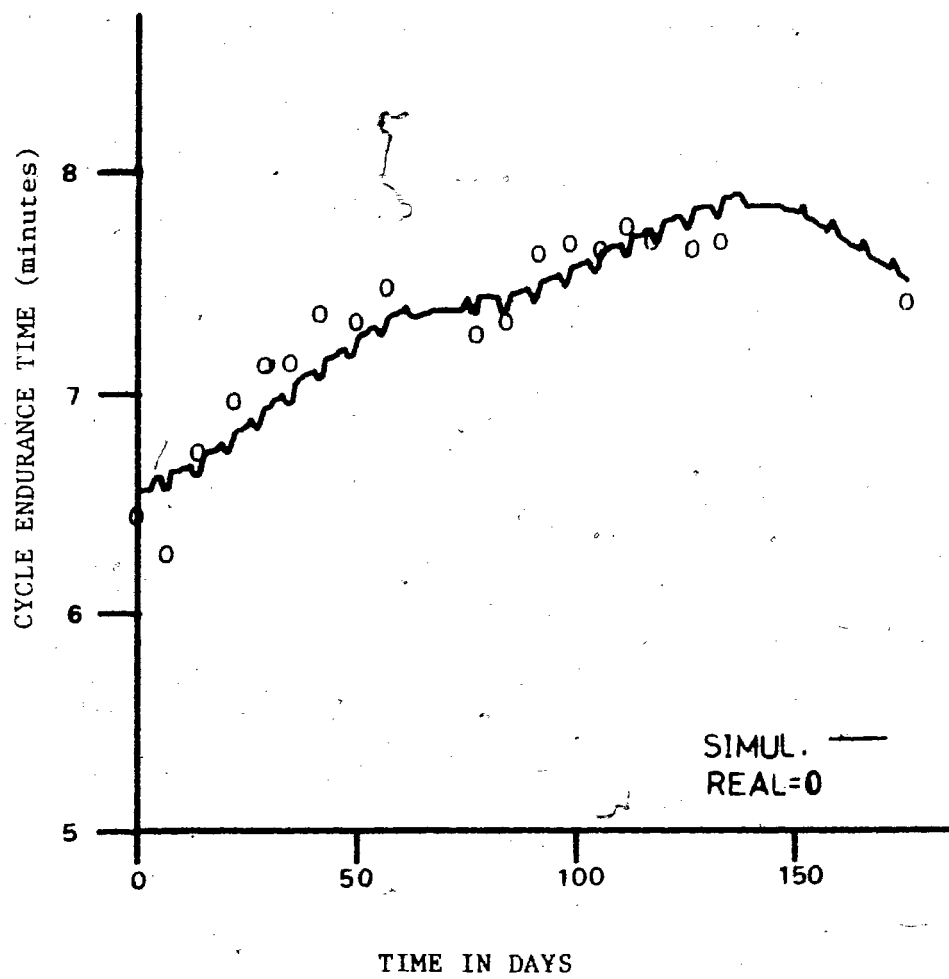
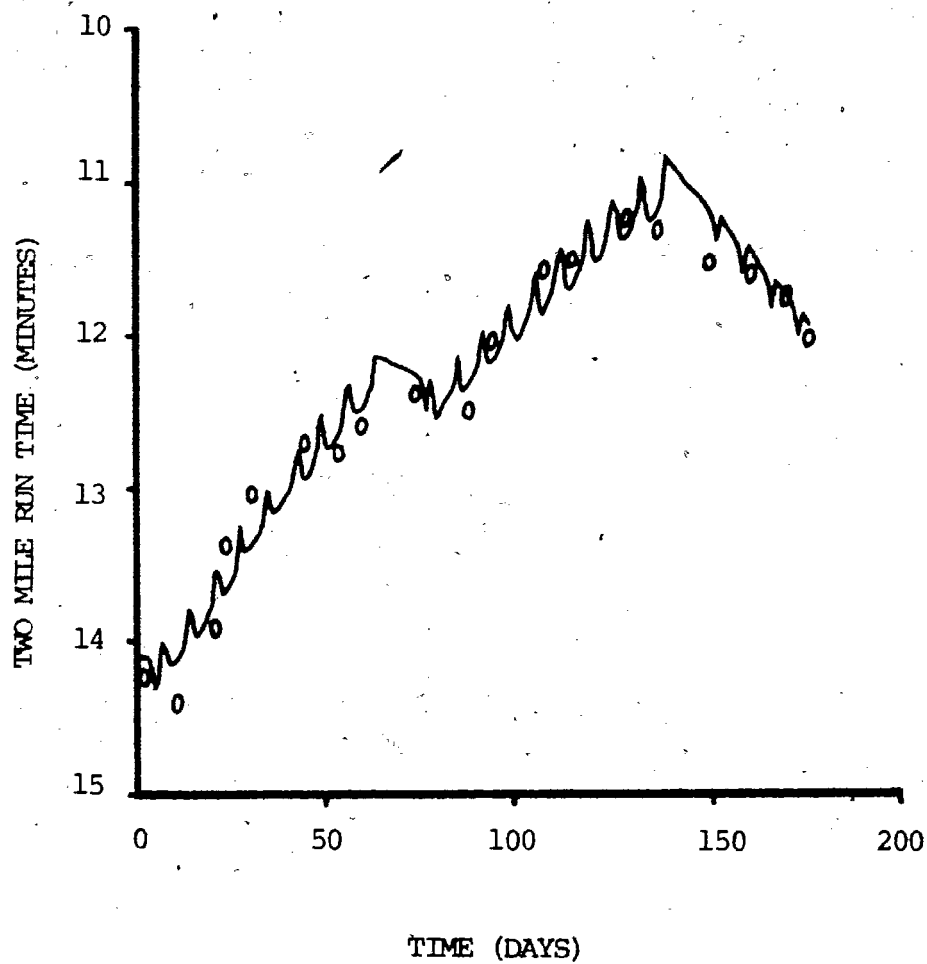


Figure 22: The Modified Simulation Of  
Performance Using Weighted  
Values For Fitness, Chronic  
Fatigue, and Acute Fatigue

The simulation of performance  
was calculated as in Figures  
20 and 21 except that the  
weighting constants  $A=1.0$ ,  $B=1.0$ ,  
and  $C=1.75$  obtained using the  
regression programme in Appendix  
4 were used iteratively to modify  
the relative values of Fitness,  
Chronic Fatigue, and Acute Fatigue.

$$PERF = A \times FIT - (B \times CHR.FAT + C \times AC.FAT)$$



## 7. Discussion

Physical (athletic) performance is a complex phenomenon. However, Banister et al (1975) and Calvert et al (1976) suggested that a system grossly describing the relationship between input (training) and output (performance) existed as shown in Figure 23. The system was based on the concept that each training session could be regarded as an "impulse" because of its relatively short duration when compared with the time constants of the physiological responses to training. Each training session was thought to produce an impulse of fitness and an impulse of fatigue, each characterized by a decay time constant. In the case of the model of the swimmer, the time constants of decay were iteratively determined to be 30 to 50 days for fitness and 15 days for fatigue. By summing the individual impulse responses, a profile of both fitness and fatigue was generated. Performance was simulated by subtracting the fatigue curve from the fitness curve, i.e.,

$$\text{PERFORMANCE} = \text{FITNESS} - K \times \text{FATIGUE}$$

The present study was designed to elaborate upon the gross system previously described. A different system was hypothesized and is shown in Figure 24.

Figure 23: A Systems Model Of Performance



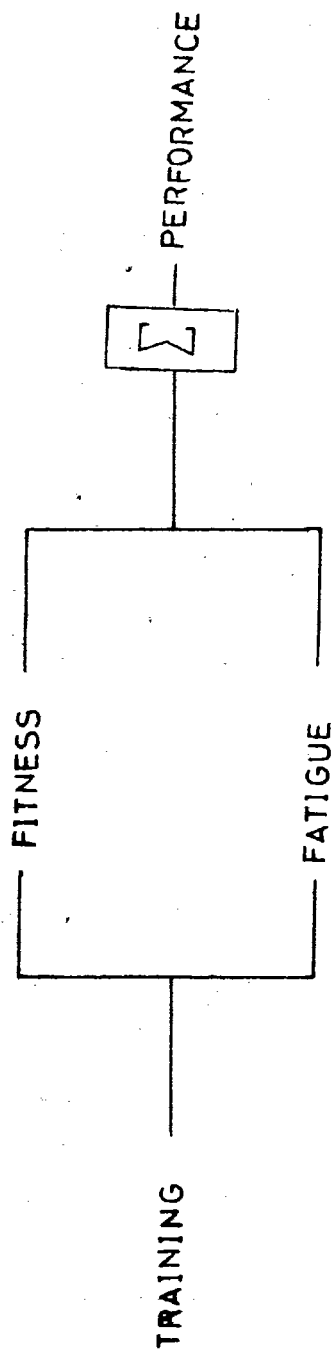
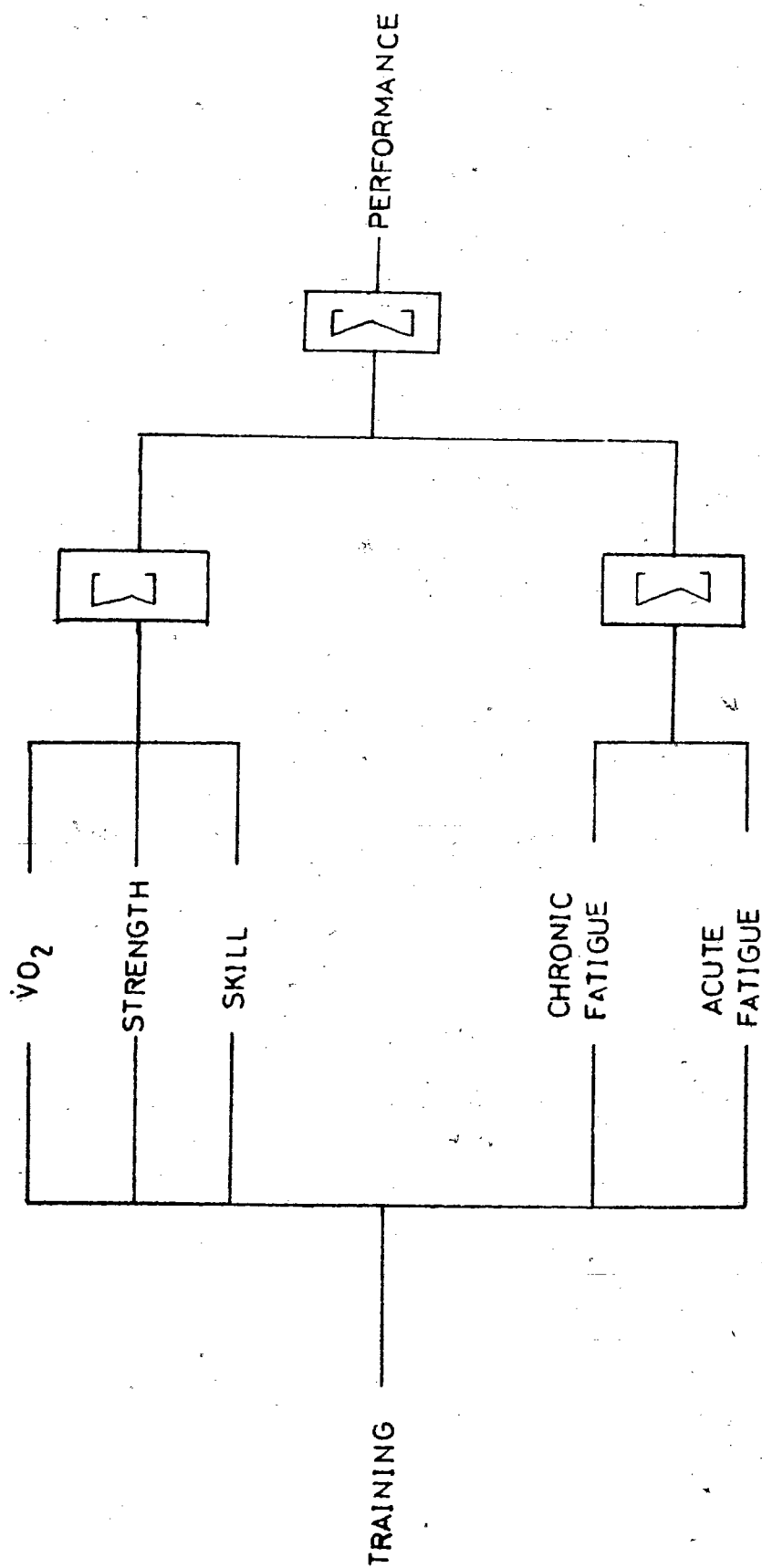


Figure 24: A Multicomponent Systems Model Of Performance



Fitness was hypothesized to be a function of three primary factors under the performance conditions of the study. These factors were skill, aerobic capacity, and strength. Because of the fundamental nature of the primary performance criterion (a two mile run), and because of an adequate background in competitive athletics possessed by the subject, skill was not considered to represent a significant factor in improved performance due to training. However, in cases in which the performance is of a more complex nature and/or the subject is not experienced in the skills required, skill might constitute a major input to improved performance and should not be overlooked. However, in this study, aerobic capacity and strength were considered to be the most important contributing factors to increased fitness.

Aerobic capacity (as measured by maximal oxygen uptake) is shown in Figure 7. The decay time constant for the maximal oxygen uptake curve as estimated by the method described in Chapter 5 was found to be 23 days. Using this information, maximal oxygen uptake was simulated using the daily training (shown in Figure 2) as the forcing function. The results of this simulation, displayed simultaneously with the actual values in Figure 13, indicated that aerobic capacity was related by a first order differential equation to the cube root of training.

The qualitative knowledge that the function relating aerobic capacity to training is non-linear is a useful concept. It is important to recognize that doubling the amount of training only causes a 23% increase in aerobic capacity, not a 100% increase as predicted from a linear model (Davies and Knibbs, 1967). In effect, a "law of diminishing returns" is at work and increases in training should be approached from a carefully examined cost-benefit point of view.

The description of the quantitative response of maximal oxygen uptake to training provides a means by which aerobic capacity can be predicted for any time during a training programme. Therefore, the amount of training required to maintain a desired level of cardiorespiratory fitness can be calculated. Such a practical application could be of use to clinicians working in physical rehabilitation programmes and individuals involved in their own exercise regimens.

From an athletic viewpoint, it is interesting to note that aerobic capacity has decreased significantly when the maximum of the modelled performance curve occurs. This is somewhat counter-intuitive and clearly suggests that a factor or factors other than maximal oxygen uptake are implicated in the determination of a performance of a predominantly aerobic nature. Such may not be the case for top class athletes who have been training at high levels for long periods of time.

These individuals may reach a plateau of aerobic capacity and, while they must train to maintain this level, it is probable that increasing training will not significantly increase their maximal oxygen uptake.

The Rogers Strength Index shown over the course of the training programme in Figure 8 possessed features similar to those of maximal oxygen uptake; however, the data was very "noisy". This was probably the result of week to week variations in those factors such as arm strength which were not affected by the training and which were not measured by techniques of high accuracy. The parameter of the Strength Index which was most affected by the bicycle training was leg strength. This is shown in Figure 9. It can be seen that leg strength, like maximal oxygen uptake behaved as an exponential rising response to a step input (Figure 5). The time constant of decay for leg strength was graphically estimated to be 60 days. Using this information and using the weekly training from Figure 3 as the forcing function, leg strength was simulated. The results of this simulation are shown in Figure 14. Like maximal oxygen uptake, leg strength was fairly successfully modelled as an exponential function of the cube root of training. The actual values of leg strength rose slightly more quickly and fell off slightly less quickly than the modelled values. This was possibly due to a learning effect involving a neural reorganization which occurred very rapidly as the

training programme began and then remained long after training ceased much as the capability for riding a bicycle lingers long after one has stopped riding a bicycle. The relative contributions of increased aerobic capacity and increased strength to improved fitness, and therefore, to improved performance were not immediately obvious. From the performance data shown in Figure 10, a decay time constant was estimated to be 50 days. The time constant for Fitness was accordingly considered to be the same. Maximal oxygen uptake and strength were combined in varying proportions and the decay time constant for each weighting was estimated. The ratio of maximal oxygen uptake to strength which yielded a time constant of 50 days was found to be 0.50. This points to the importance of musculoskeletal development even in a performance of a predominantly aerobic nature. Exercise programmes to improve individual's cardiovascular fitness should be designed so that a lack of musculoskeletal capability is not the limiting factor in the physical activity.

Although performance behaved generally in the same way as the strength and oxygen uptake parameters identified with fitness, Figure 15 shows that there are sufficient dissimilarities to consider at least one additional factor. This finding is in agreement with Calvert et al (1976) who found that swimming performance did not parallel simulated fitness during the training programme. This second factor was

fatigue. In Figure 16, the simulation of chronic fatigue is shown. It resembles closely the fitness profile with the exceptions that it rises more quickly upon the onset of training and falls off much more rapidly upon the cessation of training. Such a result is reasonable in the context of training for athletic competition. If fatigue and fitness possessed time constants of equal magnitudes, no significant training effect would be possible since the influence of each fitness impulse would be negated by an equally long lived fatigue impulse.

The simulation of performance, calculated as

$$\text{PERF} = \text{FITNESS} - \text{CHRONIC FATIGUE}$$

is shown in Figure 17. This concept of performance is clearly preferable to the simple FITNESS model of Figure 14 in that it quite accurately models the decline in performance which occurs after training has stopped for periods of more than a week or two. However, it does not adequately model the initial drop in performance which occurs when training begins and it does not accurately account for the rise in performance which is seen one to two weeks after training stops.

Therefore, a second fatigue factor, acute fatigue, was hypothesized. This factor was thought to respond to CHANGES in



training intensity rather than to training intensity itself.

Acute fatigue was simulated using a time constant of 7 days and a time delay of three days at the onset of training (Figure 18). These values were derived from an analysis of the subject's feelings of secondary muscle soreness as described by DeVries (1976). At the beginning of the training programme, the soreness appeared in leg muscles during the third day necessitating the three day delay function. The secondary soreness gradually diminished as the musculoskeletal system adapted to the stress from training; the time constant for this diminution was estimated to be seven days. In Figure 17, it can be seen that during the breaks in training of two weeks duration, the acute fatigue factor decays to zero, a result corroborated by the subject's feelings during the training programme.

A simulation of total fatigue was calculated by adding the profiles of chronic and acute fatigue with equal weighting constants. This is shown in Figure 19. The curve rises very quickly as training begins, levels off quite quickly relative to both fitness and chronic fatigue alone, and drops off very quickly when training first stops and more slowly as the period of no training continues. Serum bilirubin (a breakdown product of hemoglobin) is simultaneously plotted with the total fatigue curve. Blood hemoglobin levels have been used by athletic coaches to assess the fatigue levels of athletes in danger of suffering from overtraining (J. Bloomfield, personal

communication). When a sudden drop in blood hemoglobin is seen, this is taken as being indicative of high fatigue and training is tapered appropriately. In this study, blood hemoglobin did not vary greatly from week to week (Appendix 2) and certainly did not vary in a way resembling the hypothesized fatigue profile. However, the subject was not in a situation where overtraining was probable and it is possible that blood hemoglobin was not a sufficiently sensitive indicator of fatigue. Conversely, serum bilirubin maintains a striking fidelity to the total fatigue curve and it is suggested that it may be a more sensitive monitor of fatigue than hemoglobin. As such it may be useful as a fatigue indicator when training is not highly intense (such as when preparing for athletic competition). No other blood parameter measured in this study demonstrated the same potential usefulness in monitoring fatigue. Figures 20 and 21 are simulations of running and cycling performance calculated as

$$\text{PERF} = \text{FITNESS} - \text{TOTAL FATIGUE}$$

These show a significant improvement over the model shown in Figure 17. The general rising trend of performance with training is accurately modelled as is the general falling trend as training is suspended for more than a few weeks. However, unlike the model which accounted only for chronic fatigue, the model represented by Figures 20 and 21 accurately describes the

rise of performance one to two weeks after training ceases. This can be accounted for by the much more rapid decay of total fatigue relative to fitness. When training stops, fitness levels remain quite high, decaying with a time constant of about 50 days. Since the nature of a time constant is such that a parameter decays back to its "resting" level after about three time constants, the fitness resulting from a training programme of the type in this study will require about five months to disappear completely. However, the total fatigue curve decays much more quickly: within one month, it has virtually disappeared. The even more rapid disappearance of the acute fatigue (evident as symptoms of muscle soreness) is in large part responsible for the increase in performance seen shortly after training stops.

The phenomenon of the initial decrease in performance at the onset of training is not fully explained by the model used to construct Figures 20 and 21 although the correct trend is visible. In Figure 22, a complete model which accurately handles all of the major features of performance as seen in this study is presented. It was calculated as:

$$\text{PERF} = A \times \text{FIT} - (B \times \text{CHRONIC FAT} + C \times \text{ACUTE FAT})$$

The weighting constants A, B, and C were calculated using the regression programme listed in Appendix 4 and refined

iteratively. For the conditions in this study, the values which provided the best model of performance were  $A = 1$ ,  $B = 1$ , and  $C = 1.75$ . This indicates that while fitness and chronic fatigue were equally weighted, acute fatigue is a more significantly weighted factor. This underscores the importance of starting exercise programmes slowly, particularly with naive subjects and, further, points to the importance of developing the musculoskeletal system simultaneously with the aerobic systems.

The model presented in this study, when provided with sufficient information, becomes predictive in the sense that it is able to model performance under hypothetical training situations. Such capabilities, if used under the constraints resulting from individual differences and factors not accounted for by this study (e.g. psychological input), could be of value to athletic coaches, rehabilitation therapists, and workers in extreme environments who require the capacity to prescribe individually designed programmes of training for athletic competition, for recovery from medical trauma (e.g. myocardial infarction), or for predicting limits to performance in unusual environmental conditions.

An example of the "predictiveness" of the model is shown in Figure 25. One hypothetical performance date, day 125, representing a major meet during a track or swimming season of 150 days' duration was arbitrarily selected. Performance was then simulated as a function of training utilizing the

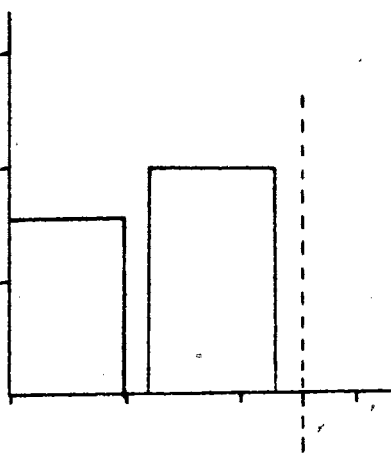
techniques previously described. Time constants for aerobic power, strength, and fatigue functions were as reported in Chapter 6. By iterative modification of input (training, Figure 25a), the best possible output (performance, Figure 25b) was generated, thereby giving a good indication of the optimum training profile for the meet (Day 125) in consideration). In this case, training profile four was the best of the given examples.

The case described by Figure 25 is a simple one. Few competitive seasons have only one meet and it is rare that consecutive meets will be as far apart as those chosen in the given example. The problem, in a situation where many important competitions exist quite close together, is one of optimal control theory and is much more complex than the scope of the present study. However, such problems are soluble with available mathematical techniques and the physiological concepts presented here.

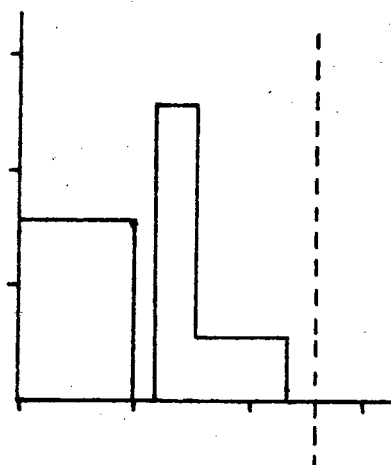
Although this study was undertaken on only one subject, the concepts described are of applicability to other individuals. There is no reason to believe that the subject in this study encountered any abnormalities in his response to training or that his responses are qualitatively different from those described in the literature. In applying the model to other individuals, quantitative differences in the training

Figure 25: A Hypothetical Training/Performance Example

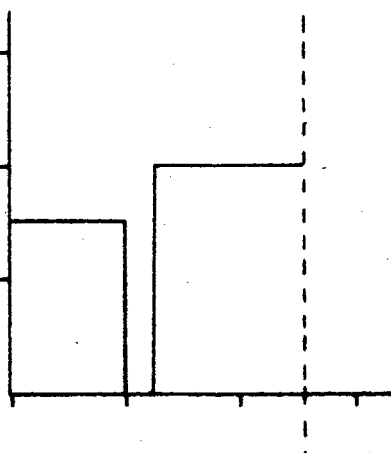
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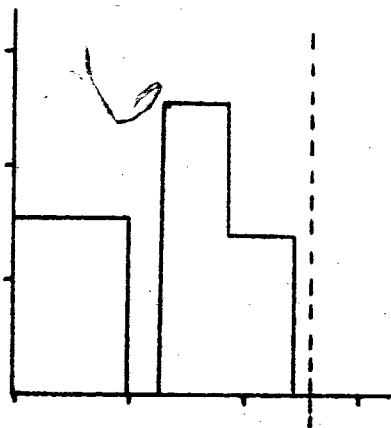
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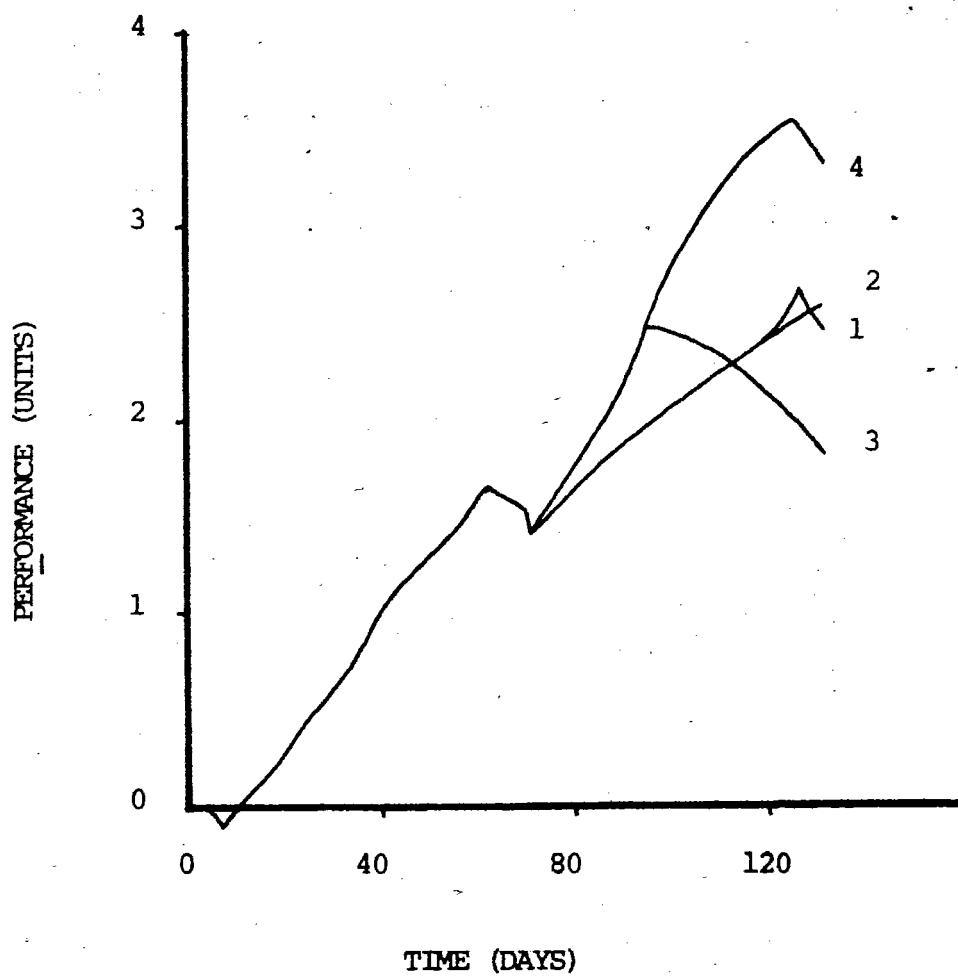


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4







response are to be expected due to genetic variability. Indeed, for a given individual, time constants should be expected to change over a long training period as more permanent functional changes are affected. However, although the numbers involved may change, the concepts should be valid, and the model may have wide-ranging usefulness.

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Appendix 1: Somatotype and Anthropometric Data

ANTHROPOMETRIC DATA

1. Name:	<u>CORLETT</u>			
2. Student Number:	<u></u>			
3. Date of Birth:	<u>22</u> day	<u>JUNE</u> month	<u>1950</u> year	
4. Height (cm.)	<u>174.7</u>			
5. Weight (kg.)	<u>62.1</u>			
6. Triceps skinfold (mm)	<u></u> (1)	<u></u> (2)	<u></u> (3)	<u>4.3</u>
7. Subscapular skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	<u>7.9</u>
8. Suprailiac skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	<u>4.2</u>
9. Calf skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	<u>3.9</u>
10. Front thigh skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	
11. Rear thigh skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	
12. Abdomen skinfold	<u></u> (1)	<u></u> (2)	<u></u> (3)	
13. Right humerus diameter (cm)				<u>6.82</u>
14. Left humerus diameter (cm)				<u>9.55</u>
15. Right femur diameter (cm)				
16. Left femur diameter (cm.)				
17. Right flexed biceps girth (cm.)				<u>29.0</u>
18. Left flexed biceps girth (cm.)				
19. Right calf girth (cm.)				<u>34.2</u>
20. Left calf girth (cm.)				

HEATH-CARTER SOMATOTYPE RATING FORM																												
NAME <b>CORLETT</b>		SEX: M F		AGE		NO:																						
OCCUPATION		ETHNIC GROUP		DATE																								
PROJECT:		MEASURED BY:																										
Skinfolds (mm):		TOTAL SKINFOLDS (mm)																										
Triceps = 4.3		Upper Limit	10.9	14.9	18.9	22.9	26.9	31.2	35.8	40.7	46.2	52.2	58.7	65.7	73.2	81.2	89.7	98.9	108.9	119.7	131.2	143.7	157.2	171.9	187.9	204.0		
Subscapular = 7.9		Mid-point	9.0	13.0	17.0	21.0	25.0	29.0	33.5	38.0	43.5	49.0	55.5	62.0	69.5	77.0	85.5	94.0	104.0	114.0	125.5	137.0	150.5	164.0	180.0	196.0		
Suprailiac = 4.2		Lower Limit	7.0	11.0	15.0	19.0	23.0	27.0	31.3	35.9	40.8	46.3	52.3	58.8	65.8	73.3	81.3	89.8	99.0	109.0	119.8	131.3	143.8	157.3	172.0	188.0		
TOTAL SKINFOLDS = 16.4																												
Calf = 3.9																												
		FIRST COMPONENT	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9	9½	10	10½	11	11½	12			
Height (m.)	= 174.7	55.0	56.5	58.0	59.5	61.0	62.5	64.0	65.5	67.0	68.5	70.0	71.5	73.0	74.5	76.0	77.5	79.0	80.5	82.0	83.5	85.0	86.5	88.0	89.5			
Bone: Humerus (cm)	= 6.82	5.19	5.34	5.49	5.64	5.78	5.93	6.07	6.22	6.37	6.51	6.65	6.80	6.95	7.09	7.24	7.38	7.53	7.67	7.82	7.97	8.11	8.25	8.40	8.55			
Muscle: Biceps (cm)	= 9.55	7.41	7.62	7.83	8.04	8.24	8.45	8.66	8.87	9.08	9.28	9.49	9.70	9.91	10.12	10.33	10.53	10.74	10.95	11.16	11.37	11.58	11.79	12.00	12.21			
-(triceps skinfold)	= 27.8	23.7	24.4	25.0	25.7	26.3	27.0	27.7	28.3	29.0	29.7	30.3	31.0	31.6	32.2	33.0	33.6	34.3	35.0	35.6	36.3	37.1	37.8	38.5	39.3			
-(calf skinfold)	= 33.8	27.7	28.5	29.3	30.1	30.8	31.6	32.4	33.2	33.9	34.7	35.5	36.3	37.1	37.8	38.6	39.4	40.2	41.0	41.8	42.6	43.4	44.2	45.0	45.8			
		SECOND COMPONENT	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9								
Weight lb.	= 62.1	Upper limit	11.99	12.32	12.53	12.74	12.95	13.15	13.36	13.56	13.77	13.98	14.19	14.39	14.59	14.80	15.01	15.22	15.42	15.63								
Mt. $\sqrt{W}$	= 3.9	Mid-point	and 12.16	12.43	12.54	12.85	13.05	13.26	13.46	13.67	13.88	14.01	14.29	14.50	14.70	14.91	15.12	15.33	15.53									
		Lower limit	below 12.00	12.33	12.54	12.75	12.96	13.16	13.37	13.56	13.78	13.99	14.20	14.40	14.60	14.81	15.02	15.23	15.43									
		THIRD COMPONENT	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9								

## Appendix 2: Blood Chemistry Profile

	2	9	16	23	30	37	44	51	58	65	72	79	86	93	100	107	114	121	128	135	142	149	156	163	170	176
Ca	9.7	9.9	9.4	9.7	10	9.7	10.2	9.5																		
P	4.8	4.7	4.1	4.8	4.5	4.2	4.5	4.5																		
Gluc	9.1	10	9.1	10.1	10.7	12.1	9.0	10																		
BUN	15	17	15	23	20	15	18	13																		
Uric Acid	5.3	5.9	5.7	6.3	6.0	5.7	6.0	5.5																		
Prot	7.2	7.3	7.2	7.7	7.3	7.2	7.3	7.0																		
Chol	175	154	155	160	170	146	157	147																		
Album	4.5	4.4	4.5	4.6	4.5	4.7	4.5	4.4																		
Alk Phos	55	44	48	53	49	46	42	40																		
LDH	153	137	153	168	162	155	140	126																		
SGOT	40	30	36	41	35	34	35	35																		
Bili	1.1	1.5	1.4	2.0	1.9	2.2	2.3	2.9	1.4					2.5				2.6								1.3
Hb	15.5	15.1	15.2	15.4	15.3	15.2	15.1	15.4		15.4	15.1	15.4	15.1	14.9	15.6	15.3	15.3	15.1								15.2

### Appendix 3: Simulation Programmes

```

V INIT
[1]  PP+4
[2]  I+1
[3]  II+0
[4]  A+PAA+XXYY+DELX+0
[5]  'HOW MANY FIRST ORDER DIFFERENTIAL EQUATIONS OR TRANSFER FUNCTIONS?'
[6]  NT+
[7]  +(NT=0)/L19
[8]  A+(NT,7)P0
[9]  II+1
[10] 'THE EQUATION IS OF THE FORM:-'
[11] '          A2*ΔV/ΔT + A1*V = B1*U'
[12] ''
[13] 'SPECIFY THE COEFFICIENTS A1,A2,B1 IN TURN.'
[14] 'USE THE FORMAT  A1 A2 B1'
[15] 'FOR EACH DIFFERENTIAL EQUATION'
[16] ''
[17] L1:'EQUATION'
[18] II
[19] AT+
[20] A[II;]+AT[1 2],0,AT[3], 0 0 0
[21] 'NOW SPECIFY THE INITIAL CONDITION FOR V I.E. V(0)'
[22] Y0+
[23] A[II;6]+Y0
[24] +(NT≥II+II+1)/L1
[25] L19:+'HOW MANY SECOND ORDER DIFFERENTIAL EQUATIONS OR '
[26] 'TRANSFER FUNCTIONS ARE REQUIRED?'
[27] ND+
[28] +(ND=0)/L17
[29] +'THE EQUATION IS OF THE FORM:-'
[30] ''
[31] '          2      2'
[32] '          A3*Δ V/ΔT  + A2*ΔV/ΔT + A1*V = B2*ΔX/ΔT + B1*X'
[33] ''
[34] 'ENTER THE COEFFICIENTS AS A VECTOR WITH THE FORMAT'
[35] 'A1 A2 A3 B1 B2'
[36] A+((NT+ND),7)P((,A),((ND*7)P0))
[37] IID+1
[38] II+NT+1
[39] L1A:'EQUATION'
[40] IID
[41] AT+
[42] A[II;]+AT, 0 0
[43] 'SPECIFY INITIAL CONDITIONS ON V, ΔV/ΔT, AND U'
[44] 'V(0)'
[45] Y0+
[46] 'ΔV(0)/ΔT'
[47] YD0+
[48] 'U(0)'
[49] X0+
[50] A[II;6]+Y0-(A[II;5]*X0+A[II;2])
[51] A[II;7]+YD0
[52] IID+IID+1
[53] II+II+1
[54] +(ND≥IID)/L1A
[55] L17:+'HOW MANY DIFFERENTIATORS ARE REQUIRED ?'

```



```

[56] NE←□
[57] →(NE=0)/L16
[58] DIFF←NEρ0
[59] L16:□←'HOW MANY PIECE-WISE LINEAR (PWL) UNITS ARE REQUIRED ?'
[60] NP←□
[61] →(NP=0)/L22
[62] II+1
[63] XXYY←(NP,2,15)ρ0
[64] L20:□←'HOW MANY POINTS IN PWL ?'
[65] NXX←□
[66] XXYY[II;1;15]←NXX
[67] JJ+1
[68] 'TYPE THE POINTS IN PAIRS - X THEN Y'
[69] L21:XXYY[II;1 2 ;JJ]←□
[70] →(NXX≥JJ+JJ+1)/L21
[71] →(NP≥II+II+1)/L20
[72] L22:□←'WHAT IS TIME STEPSIZE ?'
[73] ΔT←□
[74] □←'HOW MANY DELAYS ARE REQUIRED ?'
[75] NDEL←□
[76] →(NDEL=0)/L24
[77] DELX←(NDEL,51)ρ0
[78] SDEL←NDELρ0
[79] II+1
[80] 'SPECIFY DELAYS IN TURN'
[81] L23:□←II
[82] DELX[II;1]←□
[83] SDEL[II]←DELX[II;1]
[84] DELX[II;1]←[(DELX[II;1]+ΔT)
[85] →(DELX[II;1]<51)/L25
[86] 'DELAY TOO LONG - REINITIALIZE'
[87] →0
[88] L25:→(NDEL≥II+II+1)/L23
[89] L24:□←'ASSIGN INPUT TO X'
[90] X←□
[91] N←ρX
[92] Y←Nρ0
[93] SA←A
[94] SPA←PAA
[95] SDIF←NEρ0
[96] SXY←XXYY
[97] XT←X

```

## ▽ REINIT

```

[1]  A+SA
[2]  I+1
[3]  PAA+SPA
[4]  DIFF+SDIF
[5]  XXYY+SXY
[6]  DELX+((ρSDEL),51)ρ0
[7]  DELX[;1]+SDEL
[8]  X+XT
[9]  N+ρXT
[10]  □+'DO YOU WISH TO CRANGE X ?'
[11]  YN+□
[12]  +(YN='N')/L80
[13]  □+'SPECIFY NEW X'
[14]  X+□
[15]  N+ρX
[16]  XT+X
[17]  Y+Nρ0
[18]  L80:□+'SPECIFY NEW TIME STEP-SIZE'
[19]  ΔT+□
[20]  +(NDEL=0)/0
[21]  DELX[;1]+SDEL+ΔT
[22]  DELX[;1]+[DELX[;1]
[23]  XT+X
[24]  Y+Nρ0
[25]  SSΔT+ΔT

```

▽

## ▽ RUN

```

[1]  I+1
[2]  VO2+Nρ0
[3]  LL:VO2[I]+1 DE1 X[I]
[4]  I+I+1
[5]  +(N≥I)/LL
[6]  'DONE'

```

▽

#### Appendix 4: Regression Programme

▽ REGRESS

[1] 'PREDICTION FROM MODEL'  
 [2] PRED+□  
 [3] 'ACTUAL PERFORMANCE'  
 [4] AA+□  
 [5] 'DAYS ON WHICH PERFORMANCE OCCURRED'  
 [6] DD+□  
 [7] PP+PRED[DD]  
 [8] NN+ρAA  
 [9] Z+(NN,2)ρ0  
 [10] Z[;1]+NNρ1  
 [11] Z[;2]+PP  
 [12] CC+AA⊗Z  
 [13] 'RESULT STORED IN PERF'  
 [14] PERF+CC[1]+(CC[2]×PRED)

▽