PHYSICAL TRAINING IN MIDDLE-AGED MEN

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BY

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DECLARATION

The initiative and design of this study was entirely that of the author. Two experimental aspects of this study were carried out in collaboration with other investigators.

Haemodynamic function was assessed by the technique of radionuclide ventriculography using the equipment and personnel of the Cardiac Department, Western Infirmary, Glasgow. The technique was performed by the cardiac physicians, Drs. I. Findlay and H. Dargie and the collection and analysis of the radionuclide data carried out by a medical physicist, Mr. J. Wilson.

Plasma lactate concentration was assessed by Mr. A. Rumley of the Biochemistry Department, Western Infirmary, Glasgow.

The author was entirely responsible for the interpretation of the results obtained from these two methods. All other exprimental assessments in this study were performed by the author with the assistance of one technician, Mr. J. Wilson, Institute of Physiology.

Some of the results of this study have been published:

1. Taylor R.S (1986) The physiological effects of a 30 week program of marathon training in sedentary middle-aged men. Eds. McGregor, J.A & Moncur, J.A. 'Sport & Medicine'. Proceedings of the VIII Commonwealth and International Conference on Sport, Physical Education and Health, Glasgow, July 18-23, 1986.

2. Findlay I.N, Taylor R.S., Grant S., Rumley A., Pettigrew A., Manzie A., Dargie H., Clelland, J. and Durnin, J.V.G.A. (1987) Cardiovascular effects of marathon running in unfit middle-aged men. British Medical Journal 295 (6597): 521-4.

3. Rumley A.G, Taylor R.S., Grant S., Pettigrew A. and Findlay I.N. (1988) Effect of marathon training on the plasma lactate reponse to submaximal exercise in middle-aged men. British Journal of Sports Medicine 22: 31-34.

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SUMMARY

Previous aerobic endurance studies have suggested that middle-aged groups have a reduced magnitude of physiological training response in comparison to their young adult counterparts. However, this observation is based on only a few studies involving middle-aged groups, often with a less demanding program of training compared to their younger counterparts. The purpose of this study was to examine the physiological effects of a comparatively strenuous program of aerobic endurance training in a group of middle-aged men.

Physiological assessments were performed prior to (T1), after 15 weeks (T2) and 30 weeks (T3) training. VO_2max was assessed directly by means of a continuous graded bicycle ergometer test to exhaustion. This test also permitted the measurement of hemodynamic function by radionuclide ventriculography. The plasma lactate response to bicycle ergometer exercise was also assessed . In order to assess any training specifity effect a submaximal treadmill exercise test was also performed. Body composition was assessed by total body weight, limb circumferences and body fat content. Body fat was determined using the skinfold and densitometric methods. Four skinfold sites were assessed: biceps, triceps, subscapular and supra-ilac. The training program consisted of a graded 30 week

program of running training leading to participtaion in the 1984 City of Glasgow Marathon. Training prescription was based on exercise session frequency an duration. The intensity of training, although not prescribed, was sampled in some subjects during the study. The energy cost of training was predicted from subject training diaries using the speed and duration of their running during each training session.

216 telephone responses were obtained to a newspaper article advertising the study. After selection and medical/physical activity screening, 53 sedentary males aged 35 to 50 yrs. (40.0 ± 4.3* yrs.) were considered acceptable. Over the 30 weeks of training, 14 (26%) of the subjects dropped out. The main reason for drop-out was lack of time and loss of interest. The results of the present study are based on the 39 subjects who completed the 30 weeks training.

VO_2max increased significantly ($P < 0.001$) over the first 15 weeks training from 33.9 ± 4.0 ml/kg/min (T1) to 39.0 ± 5.6 ml/kg/min (T2). No significant change in VO_2max was observed between T2 and T3 (38.8 ± 5.2 ml/kg/min). This increase in VO_2max was the result of an increase in maximal cardiac output (T1-T3: 18.4 ± 4.0 to 22.0 ± 3.1 l/min) as the value of maximal arteriovenous oxygen difference did not significantly change (T1-T3: 14.4 ± 2.4 to 14.0 ± 1.9 ml/100ml). Maximal stroke volume increased with training while maximal heart rate fell (T1-T3: 103 ± 25 to 127 ± 16

* mean ± standard deviation

ml; 179 ± 12 to 173 ± 11 beats/min). Submaximal treadmill and bicyle ergometer heart rate, oxygen uptake and pulmonary ventilation were all significantly reduced during the period of the present training study. A training specifity effect was suggested in this study as the magnitude of reduction in these submaximal parameters during treadmill exercise exceeded that of bicycle ergometer exercise. Submaximal bicycle ergometer plasma lactate concentration was reduced during the initial 15 weeks training, although this pattern of change was reversed from T2 to T3 with an increase in submaximal lactate concentration increased from T2 to T3 (OBLA: 126 ± 30 to 152 ± 28 to 132 ± 34 Watts). The factors potentially responsible for this reversal in submaximal plasma lactate response are discussed in the text. Total body weight and the sum of skinfolds significantly fell ($P < 0.001$) during both the first and second 15 weeks of training (79.9 ± 10.1 to 77.2 ± 10.7 to 75.4 ± 11.1 kg and 56.0 ± 11.8 mm to 48.1 ± 11.1 to 48.1 ± 11.1 mm). Body density increased (1.047 to 1.053 to 1.058 $kg/m^3 \times 10^3$) over the period of training resulting in a reduction in total body fat content (22.7 ± 3.7 to 20.0 ± 4.0 to 17.4 ± 7.4 %). The energy cost of training exceeded the energy value associated with the loss in body fat suggesting a reduction in energy intake over the period of the study (T1-T3: +47751 ± 250521 kcal/min).

The relative magnitude of this increase in VO_2 max

(15%) observed during the period of the present study is no greater than reported by previous studies involving less strenuous training programs. The results of submaximal treadmill testing suggest that this failure to increase VO_2max in excess of previous studies is probably reflective of training specifity rather than aging per se. In agreement with previous studies investigating middle-aged groups, the heamodynamic results of this study suggest that the factors responsible for increasing VO_2max are central rather than peripheral. The reductions in total body weight and body fat content observed over the period of the present study are considerably greater than those typically reported by previous studies involving both middle-aged and young adult groups.

In conclusion, the middle-aged subjects in the present study achieved a similar magnitude of cardiorespiratory adaption with training to that reported in previous young adult studies. However, in support of the hypothesis that training adaptability is reduced with aging, the present training program is more physically demanding than previous studies involving both young and older subjects.

CHAPTER 1

INTRODUCTION

1.1 GENERAL

'Physical training' can be defined as a program of repeated exercise bouts. Over a period of time, training can result in physiological adaptation. This training induced adaptation can take the form of a change in body structure, a change in body function or changes in both body structure and function.

Different types of training will produce different patterns of physiological adaptation. Training which induces brief muscle contractions at maximal or near maximal effort will increase skeletal muscle mass and strength. Exercise of a more prolonged nature at a lower intensity will generally elicit an improvement in aerobic endurance. Variations of these adaptations can be observed with intermediate forms of these two types of training.

The process of physiological adaptation which results from training can enable the body to respond more easily to subsequent exercise. An increase in strength can be defined as an improved ability to exert muscular force for a brief period of time. An improvement in aerobic endurance is defined as an enhanced ability to persist in physical activities that rely heavily upon an input of oxygen for the metabolism

of energy.

The efficiency with which the body deals with exercise is referred to as 'physical fitness'. Training is generally considered to be the primary means of improving physical fitness and although physical fitness consists of a number of different components it has been traditionally simplifyed to aerobic endurance.

The evaluation of those training adaptations which could be associated with aerobic endurance exercise was initially performed by comparing athletic groups with their sedentary colleagues. The role of training, per se, in accounting for the differences in fitness observed in these cross-sectional studies is difficult to interpret because of the effects of genetic factors. To overcome these diffculties cross-sectional studies have been replaced by longitudinal aerobic endurance studies.

However, the range of such longitudinal aerobic endurance training studies remains limited in spite of the number of such studies which have been performed. Although these studies have attempted to examine the effect of variations in exercise mode and effort in terms of duration, intensity and frequency on the magnitude of training adaptation, the extent of these training variations has been small. The majority of studies have examined the training load of 3 sessions of 20 to 30 minutes duration per week, at 60 to 70 %VO_2max for a 10 to 20 week period. Few groups have

reported on the effects of training programs which require a much greater training effort in terms of increased program duration and session frequency and duration. The probable explanation for the limited number of investigations which have involved more demanding training programs is the expectation of a high subject drop-out rate. Never-the-less the disadvantage of poor training compliance may be offset by an increased magnitude of physiological benefit.

Few studies have examined the effect of aerobic endurance training on older individuals, the majority of studies using young adults of 16 to 25 years. This trend presumably reflects the greater ease in recruiting such groups, from instance for a University student population. Interestingly those studies that have investigated the training response of middle-aged individuals report that increasing age appears to reduce the magnitude of physiological adaptation to aerobic endurance training. However, as the number of studies involving older individuals remains relatively limited it is difficult to confirm or refute this hypothesis. This particularly important in view of the number of all age groups that can now be seen participating in aerobic endurance exercise.

The 26.2 mile marathon foot race is an event of virtually unequalled aerobic endurance demand and presumably for this reason has traditionally been restricted to small numbers of highly trained athletes.

Never-the-less in recent years the number of individuals taking part in marathons has vastly increased. These participants vary considerably in both age and previous physical activity background. The training effort involved in adequately preparing a previously inactive individual for a marathon is considerable. However, the motivation required to complete the such a training program is provided by the considerable prestige associated with participation in this event. The training program for marathon was selected as the vechicle of aerobic endurance training in the present study because of these features ie. demanding training effort, participation by older indivduals and its motivating nature. Finally, despite its popular of the modern day marathon, the physiological effect a novice preparing for a marathon remains relatively undetermined.

1.2 PURPOSE OF THIS STUDY

To examine the physiological effects of a comparatively strenuous program of aerobic endurance training in a group of previously inactive males aged 35 to 50 years. For this purpose a 30 week training program leading to participation in a marathon was chosen. The physiological training adaptations observed in this study will be compared with those observed in previous traditionally less-demanding aerobic training studies involving young adult subjects. This comparison

is intended to enable assessment of the effect of age and training program effort on aerobic endurance trainability.

1.3 REVIEW OF RELATED LITERATURE

Aerobic endurance is generally agreed to be a complex measure made up of a number of physiological variables.

In this chapter these physiological variables will be examined and their relative importance assessed in order to establish the limiting physiological components of aerobic endurance. The methods whereby these components can be experimentally evaluated will be described and their relative advantages and disadvantages considered. Finally, to provide a perspective from which the present training program can be evaluated, the methods and findings of previous aerobic endurance training studies will be discussed. The detailed physiological findings of these earlier studies will be outlined in Chapter 4 in the relevant sections.

1.3.1 Components of aerobic endurance

Aerobic endurance can be defined as the ability of the cardio-respiratory systems to deliver oxygen to the working muscles during physical exercise which involves the rhythmic contraction and relaxation of large muscle groups for periods of 5 minutes or more (Lamb, 1978).

Within the oxygen transport system oxygen from the atmosphere enters the lung during inspiration and passes down the airways to the pulmonary alveoli. Oxygen then diffuses across the alveolar membrane and into the blood where it is mostly carried in chemical combination with haemoglobin. At the same time carbon dioxide diffuses out of the blood to be expelled during expiration. The oxygenated blood leaving the pulmonary system then enters the left side of the heart from which it is distributed to the general circulation. Oxygen is extracted from arterial blood by exercising skeletal muscle and is utilized in the production of adenosine tri-phosphate which is in turn utilized in muscle contraction when carbon dioxide is produced. The resulting de-oxygenated blood then returns to the right side of the heart from which it re-enters the pulmonary system and another circulatory cycle commences. Thus the physiological mechanisms which may play a role in limiting aerobic endurance are the respiratory and cardiovascular factors involved in transporting oxygen to active muscles as well as factors which affect the capacity of the muscles to utilize oxygen.

It is widely agreed that, other than in cases of lung disease or exercise at high altitude, the pulmonary factors of perfusion, ventilation and diffusion capacity, are not limiting in endurance activities (Astrand & Rodahl, 1977; Margaria & Cerretelli, 1968). This belief has arisen from the

following sources of experimental evidence. At an extremely heavy work load which can be tolerated for only a few minutes at the most, pulmonary ventilation is greater than at a somewhat lower but still maxmimal load, which can be tolerated for about 6 min. The oxygen uptake is nevertheless the same in both cases (Astrand & Saltin, 1961). At maximal workload it is possible to voluntarily increase ventilation thus showing that the ability of the respiratory muscles to ventilate the lungs is not exhausted during spontaneous breathing (West, 1979). Finally, at peak exercise, arterial blood drawn from a brachial artery has been shown to have a partial pressure that is unchanged from that observed at rest. The partial pressure tends to fall during high intensity workload due to increasing concentrations of lactate and falling pH. Both these latter observations point not only to adequate transport of oxygen to, and removal of carbon dioxide from, the alveolar membrane surface by the ventilation function but also to adequate perfusion of the alveolar capillaries (Asmussen & Neilsen, 1946).

Since, under normal conditions, pulmonary factors do not limit aerobic endurance, then the limiting factor must be either the ability to deliver oxygen to the working muscles or the ability of these working muscles to utilize this oxygen. The body of experimental evidence for each of these possibilities will be given in the Section 4.2.

1.3.2 Methods of aerobic endurance assessment

(a) Aerobic Power

The gold standard for quantification of aerobic endurance has for a number of years been maximal oxygen uptake (VO_2max) or aerobic power (Mitchell et al, 1958). VO_2max is defined as the maximal rate of oxygen consumption that can be achieved during exercise. An increasing VO_2max is considered to indicate improved aerobic endurance (Astrand & Rodahl, 1977). Homozygous and heterozygous twin studies have demonstrated that up to 93.4% of variability in VO_2max is heritably dependant (Klissouras, 1971). Although often dismissed on these genetic grounds, more recent study has suggested that this estimate of hereditability has been considerably over-estimated (Bouchard et al, 1986).

A very high reliability of VO_2max measurement can be expected, regardless of the mode of exerise, the population being tested, the protocol, or the equipment. The range of the reliability coefficient is from 0.74 to 0.98, with an average coefficient of variation of 9.1% (McArdle et al, 1973, Cunningham et al, 1977, Taylor et al, 1955). This variability was examined by Katch and his colleagues (Katch et al, 1982) and found to be largely the result of biological rather than technical factors. The Fick relationship best describes the variables which determine the maximal oxygen uptake: VO_2 = CO x AVO_2D. This

relationship states that the amount of oxygen consumed (VO_2) depends on the amount of blood pumped (cardiac output: CO) and the amount of oxygen extracted from the blood (arterial venous oxygen difference: AVO_2D). Thus maximal VO_2max is determined by the product of maximal cardiac uoput and maximal arterio-venous oxygen difference.

The direct assessment of VO_2max has a number of essential requirements. The exercise should be of a dynamic nature, involve large muscle groups and be performed to maximal physical effort (Mitchell et al, 1958). Typically used exercise modes include treadmill walking and running, upright bicycle ergometry and stepping (Astrand & Rodahl, 1977). Although the subject on a cycle ergometer remains stationary and thus allows for the taking of ancillary measurements, studies have shown that cycling consistently leads to values of direct VO_2max that are on average 7-8% lower than that of treadmill exercise (Boileau et al, 1978; Shephard et al, 1968). This is because the high proportion of total power output developed by the quadriceps muscles during cycling resricts blood flow, limits venous return and thus limits effort peripherally by pain and weakness (Shephard, 1969).

The duration of the exercise should be at least 5 minutes to allow adequate time for circulatory adaptation (Astrand & Saltin, 1967). VO_2max is usually assessed by expired air collection at peak effort

followed by gas and volume analysis (Haldane & Priestley, 1956). The protocol of a maximal test is usually graded with exercise workload increasing progressively with time (Shephard et al 1968b; Hammond & Froelicher, 1984). This progression can be either continuous or discontinous. The former has the advantage of reducing the total period of testing, useful if a large number of tests are being performed, but some investigators have found the values of VO_2max from continuous tests to be less than from discontinous (Froelicher et al, 1974; Zauner & Benson, 1981). Other investigators have found no such discrepancy (Fardy & Hellerstein, 1978).

A possible source of error in the determination of direct VO_2max is failure to achieve true maximal effort. The classical concept of VO_2max (Hill & Lipton, 1923) assumed that as power output increased a plateau of oxygen consumption was reached (Taylor et al, 1955). Never-the-less there is still some disagreemnt as to how many subjects can achieve such a plateau (Cunningham et al, 1977). Other physiological indicators which have been used in establishing true maximal effort include attainment of age predicted peak heart rate, peak respiratory exchange ratio, peak blood lactate and percieved exertion (Shephard, 1984).

(b) Heart rate

Because of the subject effort, tester time, requirement for medical coverage and problems in

establishing maximal effort, indirect methods for the evaluation of VO_2max have been developed. These methods usually involve the measurement of steady state heart rate at one or more submaximal workloads (Astrand, 1960; Martiz et al, 1961; Shephard et al, 1968b). This information on submaximal performance is then extrapolated to a predicted maximal value using the assumptions of linearity of heart rate versus oxygen consumption, a known maximal heart rate and, if oxygen consumption is not measured, a consistent mechanical efficiency of exercise (Astrand & Rodahl, 1977). Departures from linearity of oxygen consumption and heart rate have been noted at high work loads (Davies, 1968; Shephard, 1967). Age predicted maximal heart rate standard errors of 10 to 25 beats/min have been reported (Hammond et al, 1983). Mechanical efficiency shows a 4 to 5% variation in cycle ergometry, a 7% variation in stepping and a 10% variation in treadmill walking (Shephard et al, 1968b). Additionally, the prediction equations or nomograms of VO_2max (Astrand & Rhyming, 1954) were derived from healthy young subjects who were usually familiar with that mode of exercise. Applying such population specific equations to other groups will lead to error although this error was reduced in the above nomogram in terms of potential age differences by age correction factors (Astrand, 1967). The end-result is that indirect estimates of VO_2max, when compared with direct values, often show individual

errors of 10 to 20% and about 10% for mean group values (Shephard et al, 1968b). Despite this prediction error indirect VO_2max is, like its direct counterpart, thought to demonstrate trends in aerobic endurance fitness trends with training.

(c) Plasma Lactate

Studies in recent years have often shown little correlation between VO_2max and aerobic endurance performance (Jacobs 1986; Kinderman et al, 1979; Katch et al, 1982) and increases in performance with training have been documented despite little or no change in VO_2max (Daniels et al, 1978).

A number of studies have reported a reduction in submaximal lactate concentration during aerobic exercise with aerobic endurance training (Saltin et al, 1968, Ekblom et al, 1968, Hurley et al, 1984). These later studies, and others, have shown that this change in submaximal lactate response is more highly correlated with performance changes with training than with VO_2max (Daniels et al, 1978). Thus in recent years a more commonly used index of aerobic endurance is submaximal plasma lactate concentration. This strong relationship between aerobic endurance performance and lactate levels is believed to be attributable to lactate levels during exercise reflecting not only the functional capacity of the central circulatory apparatus to transport oxygen to the muscles, but also the peripheral capacity of the musculature to utilise

this oxygen (Jacobs, 1986).

For convenience, plasma rather than muscle levels have been measured when assessing lactate response to exercise. However, it has been shown that the circulating levels of lactate in venous blood accurately reflect levels within the muscle (Jacobs & Kaijser, 1982). Plasma lactate concentration at low intensities of exercise is the same as at rest but as the intensity of exercise increases there is a critical point at which lactate begins to accumulate, a point termed 'Owles's Point'. In assessing the plasma lactate response to exercise it has been traditional to assess the changes in blood lactate response over a wide range of submaximal workloads (Saltin et al, 1968; Ekblom et al, 1968). In recent years rather than compare the plasma lactate reponse over the range of submaximal effort, a single point of the submaximal blood lactate concentration has been chosen to represent the response. As Owles's point is often poorly defined (Wasserman et al, 1977) the generally used method of lactate assessment is the approach of a defined lactate concentration. This is commonly taken at $4mmol^{-1}$ (Kinderman et al, 1979) and is referred to as the OBLA: 'onset of blood lactate accumulation' (Sjodin et al, 1981 & 1982). A study comparing values of OBLA in 14 subjects, obtained during identical incremental bicycle ergometer tests, found coefficients of variation of 2.9% and 3.86% when quoting OBLA with regard to oxygen

uptake and workload respectively (Sjodin et al, 1982).

(d) Body composition

Many previous aerobic endurance training studies have included the assessment of body composition (Pollock, 1973; Wilmore, 1983; ACSM, 1978). This inclusion probably reflects the finding that body composition is an important determinant in aerobic endurance performance. Increasing body fat content, and thus total body weight, increases the submaximal oxygen cost of walking and running (Durnin & Passmore, 1967). Such an increase would not necessarily increase VO_2max and thereby increase the relative proportion of VO_2max required to perform this workload. Such an increase in standard submaximal workload oxygen cost, ie. a decrease in aerobic efficiency, has been shown to be inversely related to aerobic endurance performance (Astrand & Rodahl, 1977).

Body composition has generally been evaluated by assessment of total body weight and body fat content. Fat content is traditionally assessed by the skinfold or densiometric methods. Like the previous factors, significant alterations in body composition have been observed with aerobic endurance training (Wilmore, 1980; Pollock, 1973)

1.3.3 Methods and findings from previous aerobic endurance training studies

The last 20 years or more have witnessed an

abundance of studies examining the effects of aerobic endurance training (Pollock, 1973, ACSM, 1978, Wilmore, 1983). However, because of the lack of standardization of testing protocols and procedures, the lack of methodology in relation to training procedures and experimental design and a lack of preciseness in the documentation and reporting of the quantity and quality of training prescibed, overall interpretation of this data is sometimes difficult.

Although these studies have examined the effect of aerobic endurance training programs on a variety of physiological parameters, the majority have assessed VO_2max. Therefore it is possible to standardize and summarise their findings in terms of changes in VO_2max.

(a) Training program

Aerobic endurance training programs are generally defined in terms of duration (overall and session), frequency, intensity and exercise mode. The importance of each of these parameters will be reviewed below.

i. Intensity

With regard to training intensity, it was found that the minimum intensity of training needed to achieve a training effect was about 130 to 150 heart beats/min which is equivalent to 60% of the difference in resting and peak exercise heart ('heart rate reserve') or 50% VO_2max (Karvonen et al, 1957). However, it has been suggested that in unfit older

individuals a heart rate of 100 to 120 beats/min may be sufficient (Pollock,et al, 1978). This was confirmed in a study where 2 groups of subjects of low fitness and high fitness were subjected to 10 minutes of exercise per day, five days per week for 5 weeks (Gledhill & Lynon, 1972). Although 120 beats/min was sufficient to achieve a training effect in the high fitness group it did not do so in the low fitness group. It is generally believed that if the minumum training intensity is achieved then the magnitude of improvement in VO_2max is directly related to the intensity of training (Pollock et al, 1978).

ii. Duration

The training session duration has also been shown to be directly related to the increment of VO_2max increase with training (ACSM, 1978). A 20 week study which involved a fixed training intensity of 85-95% of heart rate reserve and a frequency of 3 days per week found increases of VO_2max of 8.5%, 16.1% and 16.8% from session durations of 15, 30 and 45 minutes respectively (Pollock et al, 1978). It appears that the intensity and duration of training are closely related and that the total amount of work done is critical to the training effect (ACSM, 1978, Pollock, 1973). High intensity training periods of 10 to 15 minutes can be just as effective as longer duration programs if the total energy cost is the same for each. One study (Pollock et al, 1978) found that 20 weeks of 40 minutes

of walking 4 days per week at 65 to 75% heart rate reserve produced a similar improvment in VO2max as a 20 week program of jogging 3 times per day for 30 minutes at 85 to 90% heart rate reserve. Attrition rates of 30% or more have been noted in programs that have utilized high intensity short duration training (ACSM, 1978).

It has been suggested that the overall aerobic endurance program duration is an important training determinant. Evidence in support of this hypothesis comes from a review by Pollock (1973) of 51 training studies involving some 450 subjects ranging in age from 11 to 75 years. The improvement in VO_2max observed with training programs with duration approximately 10 weeks was 4 to 14 % and for programs of 20 weeks or more was 14 to 93 %. However, the majority of these previous studies had a similar range of duration (17 to 20 weeks) with a few studies investigating durations in excess of this.

iii. Frequency

Several studies have placed less emphasis on training frequency as an important stimulus for training and more on intensity or duration (Pollock et al, 1984). These studies have kept their comparative programs of training frequency the same with regard to total ammount of work or total work output and therefore, rather predictably, found frequency to be unimportant. For example the change in VO_2max was found

to be the same after 13 weeks of three 30 minute sessions per week as for five 30 minute sessions for 8 weeks. However, when the studies are compared after 8 weeks, the magnitude of increase in VO_2max was greater with the program of higher frequency which suggests that training frequency is an important factor in determining the effectiveness of an aerobic training program. The importance of training frequency is confirmed by the results of a 20 week program in which training sessions were each of 30 minutes duration at 85 to 90% heart rate reserve and increases in VO_2max of 8, 13 and 17% were found for training frequencies of 1, 2 and 3 days per week respectively (Pollock et al, 1969). However, it appears that with training frequencies of 3 times per week or more, the magnitude of change in aerobic power tends to plateau (Gettman et al, 1976). In addition, those individuals who trained 5 days per week complained of chronic soreness and required considerable motivation and encouragment to complete the study. In support of the importance of training frequency . Reanalysis of the this review reveals a highly significant correlation ($r = +0.66$, $P < 0.001$) between training frequency and the change in VO_2max with training.

iv. Exercise mode

Theoretically the mode of aerobic exercise would not effect the magnitude of increase in VO_2max if the intensity, duration and frequency of the programs were

the same. A 20 week study confirmed this hypothesis when it was found that a 30 minute session at 85 to 90% heart rate intensity, 3 days per week, which compared cycling, walking and running resulted in very similar increases in VO2max (Pollock et al, 1975). The mode of testing and training in the study was the same. Other studies have reported training specificity in that the increase in VO_2max is reduced when the mode of testing is different to that training (Pecher et al, 1974; Magel et al, 1975).

Although these previous studies have examined the effects of the various training determinants on the magnitude of training effect, the range of training effort examined is relatively small. Indeed although there is evidence to suggest that increasing training duration and frequency results in an increased magnitude of training effect, this hypothesis has not been rigoursly investigated. The training load of the present training study is considerably greater than the traditional previous training studies and this effect will be assessed with regard to changes in VO2max, submaximal physiological function and body composition.

(b) Subject age

The majority of previous aerobic endurance training studies have been limited to young male adults aged 18 to 30 years with few studies investigating the training effect on older groups (Pollock, 1973).

Early studies involving middle-aged subjects

reported decreased absolute and relative magnitudes of change in increases in VO_2max in comparison to training programs involving young adult groups (Benestad, 1965). However, the intensity, duration and frequency of these training programs, suggests that this reduction in trainability may be more the result of reduced training effort than ageing. Indeed more recent studies of comparable training effort have shown relative increases in VO_2max in middle aged subjects is similar to those of younger age groups. (Saltin et al, 1969; Ekblom et al, 1968; Wilmore et al, 1970).

It has been suggested that the time span of the training program of previous studies on older groups may not have been sufficient to demonstrate an equivalent training effect to their younger counterparts (Pollock, 1973). Middle aged sedentary men may take several weeks to adapt to the rigors of training and may thus need more time to gain full benefit from a program. For example, two investigations conducted with middle-aged men who exercised either 2 or 4 days per week found that both groups improved in VO_2max. Mid-test results of the 16 and 20 week programs showed no difference between groups, while subsequent final testing found the 4 day groups to improve significantly more (Pollock et al, 1969; Pollock et al, 1971).

To provide a basis for more informed comment on the effect of aging on trainability and VO_2max more

studies involving the training of middle-aged groups are needed, particularly those which include programs more demanding of effort and time. One of central purposes of this study to provide such extra information.

CHAPTER 2

METHODS

2.1 STUDY REQUIREMENTS

The experimental design of this study did not demand that the selected group represent a statistically random sample. Statistical random sampling can involve considerable practical difficulties. It was however , recognised that certain subject selection criteria would need to be met in this study.

The subjects were all required to have a 'sedentary background' prior to participation in the study. This criteria would provide a consistent base line within the study group from which all subjects would have the maximum opportunity to respond to the training program and at the same time allow across subject comparisions to be performed. As outlined in Chapter 1, some studies have shown that individuals with a previous exercise background will demonstrate a reduced magnitude of physiological adaptation to training when compared to their sedentary colleagues (Pollock, 1973, Astrand & Rodahl, 1977). A 'sedentary background' was defined as having abstained from any other form of regular recreational or sporting activity for five years or more prior to selection. Similar criteria of inactivity have been used in other aerobic

endurance training studies (Saltin et al, 1968; Ekblom et al, 1968).

Previous exercise programs quote compliance rates which range from 30 to 70% and although these studies estimated training compliance by different means, (eg. specific percentage threshold of training sessions performed, attendance at supervised training sessions or the ability to 'complete' the training program) they demonstrated that compliance is dependant on the type and intensity of the training program, the time frame of the study and the population involved (Dishman, 1982). The time course and nature of the present training program can be considered as physically demanding in comparison with previous aerobic endurance studies and it follows from this that training compliance in the present study would only be achieved if the population selected was as highly motivated as possible. In an attempt to ensure this objective, subjects were selected only if they intended to run and applied for entry in the 1984 City of Glasgow Marathon prior to hearing of the study.

To examine the effect of age on training response an age group of between 35 and 50 years was chosen for this study. This age group can be regarded as presenting a 'middle-aged' range.

The demanding nature of the training program and the testing procedures made it necessary for all participants to be free from any disease or injury that

may put them at risk or reduce their ability to complete the study.

2.2 SUBJECT SELECTION AND SCREENING

An advertisement which publicised the study and outlined the need for suitable volunteers was placed in a national newspaper two days after the entry forms the 1984 City of Glasgow Marathon were released. This article is shown in the appendix A.

From the respondents to this article a selection of study participants was made via a number of screening levels.

2.2.1 Telephone Enquiry

Interested readers were asked to respond to the newspaper article by telephoning into the laboratory details of age, previous medical history and a short summary of physical activity background. Exclusion of all individuals with one or more inappropriate reponses was made. A telephone enquiry form is shown in the appendix B.

2.2.2 Meeting of subjects

The subjects selected by means of the above telephone enquiry were contacted and invited to attend a meeting where the background and methodological details of the study were explained.

During this meeting a detailed presentation of the testing procedures and the training program requirements was given. Emphasis was placed on the

demanding nature of the study which required considerable commit ment in both time and physical terms. It was emphasised that only those fully committed both to participation in the study and completion of the marathon should take part. At this point subjects were given the opportunity to withdraw from the study.

2.2.3 Medical Examination

A full physical examination of all the volunteers from the above meeting was carried out.

Standing and supine blood pressure was assessed and a 12-lead electrocardiogram performed. A 12 hour fasting blood sample was taken and a full blood count performed in addition to plasma lipid determination. Finally the subjects were asked to complete a detailed health questionnaire. This questionnaire was based on that designed by The World Health Organization committee to screen individuals for cardiovascular disease by obtaining details of past and present, personal and family medical history (Appendix C). This medical examination and evaluation was entirely performed by the Cardiac and Biochemistry Departments of the Western Infirmary, Glasgow.

Any individuals found unacceptable for participation in this study on the grounds of medical health were excluded.

2.2.4 Detailed Physical Activity

To ensure the selection of a suitably inactive

study group, those participants who passed the medical examination were asked to complete a detailed questionnaire on their past and present levels of occupational and recreational physical activity. This information also allowed a quantification of the previous physical activity background of the study group.

This questionnaire is a modified version of the population questionnaire designed by the University of Toronto Department of Physical Education and Recreation. A copy of this questionnaire is shown in the appendix D.

2.3 SELECTION OF TESTING PARAMETERS

The many aerobic endurance training studies programs have examined the changes effected by training on a variety of physiological parameters (Astrand & Rodahl, 1977).

It was outwith the remit of this study to examine the effect of marathon training on all these parameters. However, only a limited number of these physiological parameters have been consistently reported, in particular VO_2max, submaximal heart rate and plasma lactate and body weight and fat content. These parameters formed the central core of the present study together with a limited number of ancillary measures.

The following physiological parameters were

examined in this study:

Body Composition

- Total body weight
- Height
- Body fat content

Maximal & submaximal exercise capacity

- Oxygen uptake
- Heart rate
- Pulmonary ventilation
- Plasma lactate
- Cardiac output
- Blood pressure

Resting cardiovascular function

- Heart rate
- Blood pressure

2.4 SELECTION OF TESTING PROCEDURES

Although the physiological parameters to be examined during this program of marathon training had been selected, a number of factors still remained to be taken into account before the final test procedures could be determined.

For the assessment of cardiac output and plasma lactate the author had neither the experimental expertise or equipment. To assess these parameters assistance was sought from investigators in these methodological areas. The extent of this collaboration is discussed below.

The method of cardiac output determination in this study, Radionuclide Ventriculography, required the chest to be in a relatively stable position during the measurement. This technique cannot therefore be performed during treadmill exercise and is usually limited to bicycle ergometry where the upper body is in a stable position. Thus, to enable cardiac output determination during exercise in this study, the bicycle ergometer was selected as the testing mode.

It is suggested that maximal exercise testing, particularly if performed in previously sedentary individuals of 30 years or over, should be performed in the presence of a physician (AHA, 1967) and since a physician was also required to be in attendance to perform the above method of cardiac output determination it was decided that this bicycle ergometer test would be maximal.

As discussed in the review, the greatest magnitude of training adaptation is usually seen when the mode of exercise used in testing is the same as that of training. If therefore the only means of assessing the proposed training program was a maximal bicycle ergometer test it might be expected that the efficacy of the program would be under-estimated. To counter any such possible effect it was considered important to also introduce treadmill testing and as the subjects were already expected to perform one maximal test it was thought best that this treadmill test be

submaximal.

It was not possible to assess all the above parameters in one day because of their number and the importance of adequate rest between the performance of the two exercise testing proceedures. The tests were therefore divided into two non consecutive days and performed in two separate laboratories.

Day 1 (Western Infirmary Cardiac Dept.):

Maximal bicycle ergometer test

Day 2 (Institute of Physiology):

Body composition assessement

Pulmonary function assessment

Submaximal treadmill test

These tests were performed prior to training (T1) and immediately after training (T3) ie. after 30 weeks. In addition, to assess the time course of the change of these parameters with training, these tests were also performed midway through training (T2) ie. after 15 weeks.

2.5 STANDARDIZATION OF TESTING PROCEEDURES

The examination of changes in physiological function relies on the standardization of testing procedures and subject preparation at each stage of testing (Shephard et al, 1968a,b). The experimental procedure of each test is outlined below and was rigidly adhered to for each subject at each stage of testing.

2.5.1 Subject Preparation

The physical condition of an individual immediately prior to testing has been clearly demonstrated to be an important factor in determining their subsequent test performance (ACSM, 1980; AHA, 1979).

The recomendations of the American Heart Association on subject preparation prior to exercise testing were followed (AHA, 1972) ie. avoid physical fatigue and extremes of climate, be in a post-prandial state and abstain from cigarette smoking and alcohol.

To avoid fatigue the subjects were requested to avoid all forms of exercise and extremes of climate on the day preceding their test (McArdle et al, 1973). To ensure that the subjects were post prandial, they were instructed to avoid food in the two hour period immediately prior to testing and advised that the last meal should be light.

Blomquist and his colleagues (1970) have found that alcohol intake shorly prior to exercise testing, although having no effect on maximal performance, increases heart rate, blood lactate and oxygen uptake at a standard submaximal workload. For this reason, and in accordance with AHA recommendations, the subjects were asked to abstain from alcohol during the 24 hours prior to testing.

Smoking has been shown to reduce both VO_2max and performance capacity as well as increasing submaximal

heart rate (Ekblom & Hout, 1972). This effect was observed if testing was performed 10-45 minutes after smoking (Juurup & Muido, 1946). In an attempt to avoid these acute physiological effects of smoking the subjects were asked to refrain from smoking in the 2 hours immediately preceding the test.

Health of the subject is critical for testing safety and the reliability of the test results. The subjects were asked to report any illnesses prior to testing so that the test date could be rearranged.

The eating, smoking and physical activity patterns and health of the subjects were checked by questionnaires on arrival at the laboratory. In the uncommon situation where one or more of the recommendations were not met, the subject was asked to return the following day, or the soonest alternative.

A copy of the subject's testing instructions and the observer pre-test check list are shown in appendix E and F respectively.

2.5.2 Time of testing

It is widely accepted that the time of day can alter the value of various physiologial parameters. Factors that have been reported to be affected by circadian rhythms include resting and submaximal heart rate, oxygen uptake, plasma lactate concentration and VO_2max (Reilly et al, 1984).

To take account of possible circadian variations in the physiological values being assessed in this study,

the subjects were tested (within a two hour period) at the same time of day, at each stage of testing. All testing was performed between the hours of 0800 and 2000.

2.5.3 Laboratory Conditions

The environmental conditions during which a test is performed, like the initial condition of the subject, are also important to the standardization of testing procedures.

Although temperature and humidity have been shown to have little effect on maximal exercise performance (Saltin, 1964), an increase in temperature (> or = 25 °C) has been reported to result in an increased submaximal heart rate (Williams et al, 1962). Plasma lactate concentration is also reported to increase with a rise in environmental temperature (Fink et al, 1975). Rowell et al (1964) suggests an optimal temperature of 17 °.

The environmental conditions for the tests in this study were set in agreement with the recommendations of the WHO. Expert Committee (Shephard et al, 1968 a,b). All tests were performed within the 'thermal comfort' zone, defined as being within a temperature range of 18 to 22 °C and a relative humidity of 35 to 45 %. To aid cutaneous heat loss by convection, a fan was directed at the subjects during exercise.

2.6 EXPERIMENTAL DETAILS OF TESTING PROCEEDURES

2.6.1 Maximal Bicycle Ergometer Test

This test was carried out in the cardiac assessment laboratory of the Western Infirmary, Glasgow.

(a) Selection of exercise protocol

This protocol required to be designed so as to enable both submaximal metabolic parameters and maximal oxygen uptake assessment.

There is no standard protocol for assessing maximal oxygen uptake that is completely satisfactory for all subjects (Shephard et al, 1968a). However, there are a number of standard testing principles to be followed when assessing when selecting a protocol to assess maximal oxygen uptake. These priniciples refer to the type and duration of the exercise as well as the achievement of true maximal effort.

Because the increased activity of skeletal muscles accounts for most of the increased oxygen uptake during exercise, it is critical that large muscles are used if maximal oxygen uptake is to be obtained. It is reported that about 50 per cent of the total muscle mass must be engaged in exercise before maximal oxygen uptake can be achieved (Rowell, 1974). Routine testing of maximal oxygen uptake has been accomplished chiefly with three methods of exercise: treadmill, bicyle ergometer and bench stepping (Nagle, 1973). Studies have found that step and bicycle ergometer tests repeatedly

underestimate the value of maximal oxygen uptake determine by treadmill exercise by some 5-10 %. Moreover, treadmill tests are subject to the most differences in skill and efficiency between subjects (Nagle, 1973).

Despite the advantages of treadmill testing in obtaining a true value of VO_2max, bicycle ergometer exercise was selected for this study. The choice of bicycle exercise was made because of the ancillary measurements which were made. As described in the previous section, the method of cardiac output assessment in this study demanded that the subject's chest be in a relatively fixed position during exercise. This method is discussed below. Furthermore, in cycling, the relatively motionless upper body allows easy monitoring of electrocardiogram and blood pressure and withdrawl of blood for lactate determination.

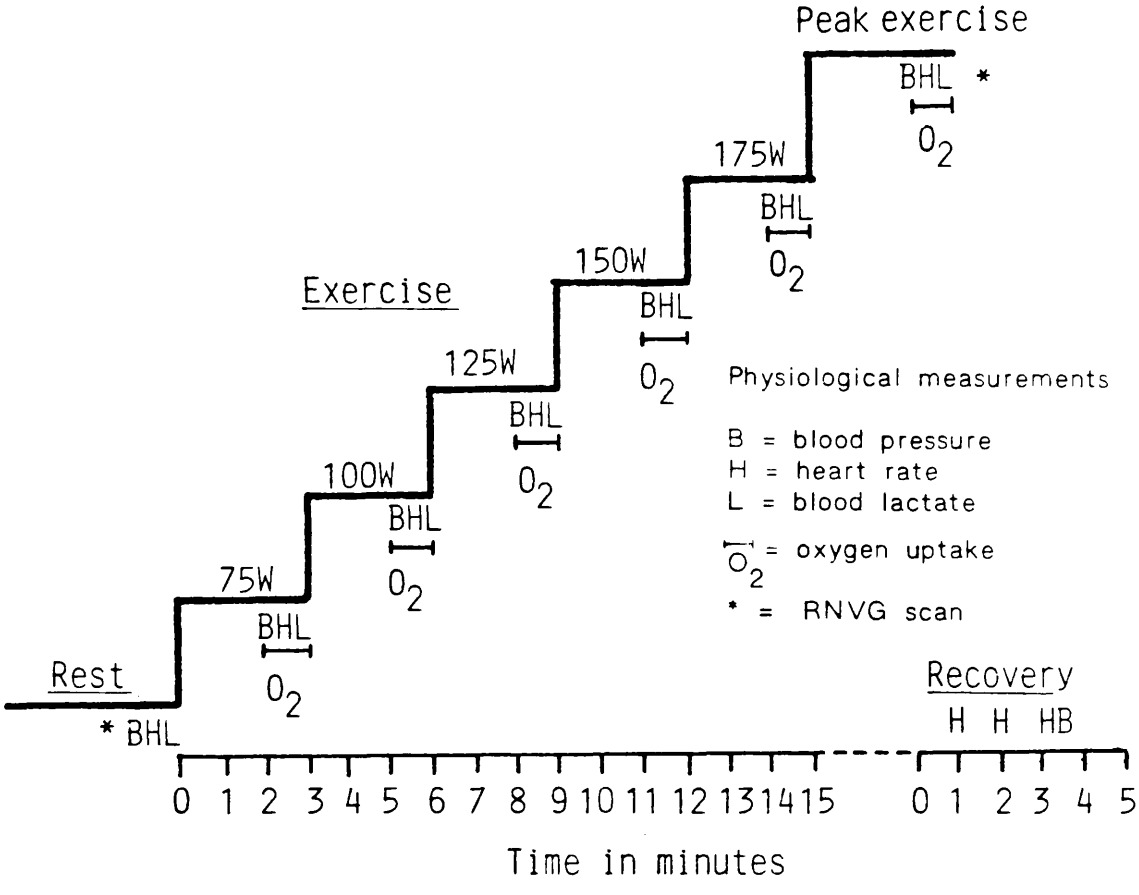
It has been shown that a discontinous exercise protocol can often give rise to a slightly higher value of maximal oxygen uptake (2.7 %) than continuous protocols (Shephard et al, 1968a). Never-the-less there a number of practical problems associated with discontinous protocols. A number of discontinous protocols require that the subject return the labortaory on one or more occassions (Taylor at al, 1955; Shephard et al, 19868a). If the time scale between these visits is prolonged the aerobic endurance

fitness of the subject may change. Many of the subjects in the present study, being tested during the working day had too little time for such a return visit. Moreover the time required to perform one discontinous test takes overall more time than a continous protocol of the same exercise time. Because of the routine clinical demands of the hospital laboratory, the time available to test the study participants was limited. Discontinous tests are often used in exercise tests where the arms are moving during exerice, such as treadmill running, is so great as to hinder removal of a blood sample or assessment of blood pressure. In the case of bicyde ergometer exercise the arms are maintained in a relatively fixed position and therefore do not hinder any measurments using the arms. In view of the practical problems associated with discontinous exercise it was decided that the maximal exercise protocol in the present study be continous.

Previous studies have observed that achievement of maximal oxygen uptake in less than 5 minutes results in its underestimation (Astrand & Saltin, 1961). This presumably reflects insufficient time for cardiovascular adjustment. Prolonged exercise tests also leads to maximal oxygen uptake underestimation, probably because blood flow to the skin increases and this may reduce blood and oxygen supply to the working muscles (Froelicher et al, 1974). Regardless of the subject population, it is advised that the exercise

FIGURE 1

Maximal bicycle ergometer exercise; timings of exercise increments and measurements



test should progress from initial exercise loads which can be accomplished without difficulty to higher loads that which lead to maximal exercise in about 10-20 minutes (Lamb, 1984).

In order to conform with recommended exercise duration, a bicyle ergometer* protocol was selected with a initial workload of 75 watts and which increased by 25 watts every 3 minutes until exhaustion. This protocol is illustrated in figure 1. The initial workload of 75 watts, has a predicted oxygen cost of 1.50 litres/min (ACSM, 1980). Based on age predicted oxygen uptake values for the present study group (Astrand, 1960), this initial workload corresponds to about 40 per cent of maximal effort. This initial workload would be expected to be performed comfortably.

To enable achievement of maximal effort within 10 to 20 minutes it is important that the increase in workload increment be sufficient. However, as with the initial workload it is important that this increase not be greater than the rate of cardiovascular adaption. The 25 watt increment of workload increase corresponds to a predicted oxygen cost of 0.3 litres/min or about 3 to 6 ml/kg/min for subjects with weight range 50 to 100kg. This increase in oxygen cost is within the recommended range of exercise workload increments recently suggested by Jopker (1981). Based on the age predicted VO2max (Shephard, 1966) for this group it was estimated that the subjects would achieve this value in

* see Appendix J

some 15 minutes, a value within the recommended exercise duration.

The metabolic measurements of oxygen uptake, heart rate and plasma lactate concentration were required to be made during submaximal exercise. It was important that sufficient time be given to allow the attainment of steady state for each of these parameters with respect to the particular workload being performed. Studies have shown that the criteria of steady state can be met within 2 to 3 minutes of exercise for oxygen uptake and heart rate , particularly if the exercise protocol is continous (Astrand & Rodahl, 1977) and presumably also if a standard magnitude workload increment is applied. The time to steady state for plasma lactate is less well documented and will be discussed in more detail in Section 4.4 of the Discussion chapter.

(b) Method and equipment

On arrival at the laboratory the subjects were asked to change into shorts and training shoes. In order to ensure the subject was a standardized resting condition they then lay supine on a couch in a 'quiet room' for 15 minutes. At the end of the rest period a physician administered an injection of lignocaine to the antecubital area of the right forearm and shortly after inserted a teflon catheter into the medial antecubital vein.

The subjects were then taken into the cardiac

testing laboratory and placed on an electrically braked upright bicycle ergometer (Siemen Elan Schonander, Sweden). Their feet were secured to the pedals by means of foot clips. The height of the saddle was adjusted so that the subject's legs were vitually fully extended at the bottom of the pedal stroke. It was checked that this position was comfortable and that it allowed an efficient pedalling action.

Familiarization, with exercise mode and equipment, is an important aspect of subject preparation. It has been shown that the reduction in anxiety and improved mechanical efficiency asociated with familariztion result in reduction in both exercise heart rate and oxygen uptake (Shephard, 1969). A 3 minute period of pedaling at zero resistance prior to the actual test was allowed to enable familiarization.

The bicycle test started at a workload of 75 watts and pedal frequency of 50 revolutions per minute. Workload increased by electrically increasing the resistance applied to the bicyle flywheel while the pedalling rate remained constant. At high workloads if the subjects found it uncomfortable to pedal at this initial pedalling frequency they were permitted to pedal at the faster rate of 65 revolutions per minute. If the subjects could not maintain a pedal frequency of at least 50 for the 3 minutes, it was taken that the subject had reached exhaustion and the test was terminated. All the subjects were encouraged to

exercise to exhaustion.

When vigorous exercise is terminated suddenly there is tendancy for blood to pool in the veins of lower extremities. This pooling may result in reduction in venous return which in turn reduces cardiac output, thereby compromising cerebral and coronary blood flow at a time when myocardial oxygen demand remains high. To prevent such venous pooling, the flywheel resistance was turned to zero immediatly after the achievment of peak effort and the subjects were directed to keep their legs turning thereby maintaining venous return by the skeletal muscle pumping action.

(c) Physiological parameters assessed

(i) Respiratory parameters

Assessment of respiratory factors was made using the standard Douglas bag method (Haldane & Priestley, 1956).

After mounting the bicycle ergometer the subjects were asked to insert a mouthpiece attached to a valve, which they breathed through for the remainder of the test. The valve was connected via a 1 metre length of 40 mm, diameter plastic hosing (Falcona Ltd.) to an evacuated Douglas bag (Collins Ltd., USA) fitted with a 3-way tap. Initially the position of this tap was turned so that the subjects breathed through the system and into the atmosphere rather than the into the Douglas bag.

A Y-shaped respiratory valve (Edwald Koegal Co.,

San Antonio, Texas, USA) was used in this study which has a number of characteristics for exercise testing purposes. These include low weight (72.5g), small size (92cm long), low resistance to air flow (1.0/2.0 cm.H20 for flow rate of 360 to 624 l/min) and finally a low dead space (64ml) (Lenox & Keogal, 1974). The valve was regularly checked during the study for leaks by passing a known volume of air intermittantly throught the valve and measuring the volumes with a Tissot spirometer (Collins Ltd, USA).

At the beginning the final minute of each 3 minute bicycle ergometer workload period the 3-way tap was turned so that the subjects breathed directly into the Douglas bag and their expired air was collected. At the end of the 3 minute period the tap was turned so that the contents of the Douglas bag became completely shut off and again the subjects breathed into the atmosphere. Generally a 60 second period of expired air collection was made although during the peak exercise load this was often reduced to 30 seconds to ensure a collection. Care was take to accurately time each sample so as to reduce possible volume error (Philips & Ross, 1967).

To prevent any gas diffusion, the contents of each Douglas bag was analysed immediatly at the end of the exercise test. Intially a volume of 0.5 litres of expired air was drawn from the Douglas bag sidearm by the intake pump of an infra-red carbon dioxide analyser

(P.K. Morgan Ltd.) which in turn pumps its exhaust into a paramagnetic oxygen analyser (Taylor Servomex Ltd.). Readings were taken from the meters when their gas concentration indicators ceased to fluctuate by more than 0.05 percent full-scale deflection. Both these analysers were calibrated on the morning of testing and then every 4 hours thereafter with oxygen and carbon dioxide mixtures drawn from cylinders (BOC Ltd.) of known composition. These cylinders were calibrated to within ± 0.03% on delivery by chemical analysis using a Lloyd-Haldane apparatus. These procedures are in accordance with metabolic measurement recommendations (Weber & Janicki, 1986).

The contents of the Douglas bag were then drawn through a dry gas meter (Parkinson-Cowan Ltd.) by a vacuum pump (Hoover Ltd.) a constant flow rate of 120 liters per minute and the volume of the bag measured to th nearest 0.1 litre. The dry gas meter was regularly calibrated using a 300 litre spirometer (Collins Ltd, USA). A thermistor probe sealed within the gas meter measured the temperature of the expired air to the nearest 0.1 °C.

All the appropriate respiratory calculations were carried out on a programmed CBM Series 3032 Professional computer. These calculations and an example print-out are shown in Appendices G and H respectively.

(ii) Heart rate

Bicycle exercise heart rate was determined in this study by electrocardiographic monitoring using a 3 channel electrocardiograph (Fenduka Denshi Cardio Auto D-38). This electrocardiograph met the frequency response specifications for exercise monitoring as outlined by the American Heart Association (AHA, 1972). Heart rate was determined by measurement of the average R-R wave interval taken over 20 successive cardiac cycles.

The electrocardiogram was also used to monitor the condition of the myocardium. The physician in attendance continously examined the electrocardiogram for any changes suggestive of ischemic or rhythm abnormalities. The selection of a 3-lead (V_4, V_1 & V_5) electrocardiogram was made to enable a comprehensive assessment of the overall electrical activity of the heart as possible.

Placement of the recording electrodes on the limbs is not practical during exercise. Therefore the 4 limb leads were placed on the extremities of the trunk, on the shoulder and upper waist, and two electrodes placed in the positions of V_1 (right sternum margin at the 4th intercostal space) and V_5 (junction of left anterior axillary line at the level of the 5th intercostal space).

In order to achieve a satisfactory electrocardiographic trace, care was taken to ensure that the skin was suitably prepared and the electrodes

properly attached. The skin was initially cleansed of oils with an alcohol-soaked gauze snd then abraded by running over the area with guaze containing containing an abrasive gel. Small self-adhesive silver-silver chloride eletrodes were attached to the skin. To reduce the risk of electrode slippage with sweating, the electrodes were further bound to the skin with the use of elastic adhesive tape.

The 3 leads were continously recorded throughout exercise at a paper speed of 5 mm/sec while during the final 30 seconds of each minute of exercise the paper speed as increased to 25 mm/sec so that sufficient R-R wavediscrimination occured to allow accurate heart rate determination.

Heart rate was also assessed by the same method during the period of supine rest and immediatly prior to exericse while the subject sat on the bicyle ergometer as well each minute during the first 3 minutes of recovery.

The electrocardiographic monitor was regula rly calibrated for paper speed and voltage, the former being critical for accurate heart rate determination. In accordance with the American Heart Association recommendations, the accuracy of the paper speed was always 99.5 per cent or better (AHA, 1967).

(iii) Blood pressure

Basis of method

Arterial blood pressure was assessed both at rest

and during exercise for both physiological and medical purposes.

When evaluating the haemodynamic response to exercise, the measurement of both arterial blood pressure and cardiac output (discussed below) allows calculation of the peripheral vascular resistance to be made (Clausen, 1977). This calculation will be discussed in more detail in section 4.2.

Resting and exercise blood pressure was used as a medical screening technique in addition to electrocardiographic montoring. In accordance with World Health Organiztion recommendations a failure to increase systolic blood pressure despite increasing worklod was indicative of an abnormal cardiovascular reponse, suggestive of ventricular failure, and the test was immediatly terminated (Andersen et al, 1971).

Method and equipment

Arterial blood pressure was assessed indirectly by ascultation using a pressure cuff around the left forerarm, attached to a mercury sphygmonometer. Care was taken to ensure that the arm during this measuement was relaxed and at the same level as the heart. All measurements were carried out by the attending physician.

Arterial blood prssure was determined after supine rest in both the supine and a standing position. Because of the noise during exercise it is not practical to assess the diastolic value by indirect

means. Systolic readings were taken during the final 30 seconds of each 3 minute bicycle ergometer workload and immediately prior to the termination of exercise. In addition systolic and diastolic readings were taken 3 minutes post exercise.

(iv) Plasma lactate concentration

Although the author withdrew some of the blood samples, the analysis of the blood to determine the lactate concentration was carried out entirely by the Biochemistry Department of the Western Infimary, Glasgow.

The blood samples for lactate determination were drawn from the indwelling catheter in the antecubital vein of the right arm.

Samples were drawn after the period of 15 minutes supine rest and again during the last 30 seconds of each 3 minute bicycle workload and finally just prior to exhaustion.

In all 7 mls., of blood were withdrawn at each sample. The initial 4 mls. of catheter dead space was discarded and the remaining 3mls. used for lactate determination. To prevent glycolysis after collection, each sample was placed in a labelled 5 ml. flouride oxalate tube and shaken well.

Following the comp letion of the exercise test the samples were spun in a centrifuge at 5000rpm. for 10 minutes, the plasma was then pipetted off and placed in 5 ml labelled plastic tubes. The plasma samples were

then frozen at - 20 °C and maintained in this condition until analysis was carried out 2 to 3 days later.

The lactate concentration of the samples was determined using a fully enzymatic UV. spectrophotometic assay (Boeringer Corp. Ltd, Mannaheim, West Germany). The test principle of this assay is described in the appendix.

The onset of blood lactate accumulation (OBLA) value was determined using the standard of method Sjodin et al (Sjodin et al, 1979). The plasma lactate concentration for each individual was plotted sep rately versus bicycle ergometer workload, oxygen uptake and percent VO_2max. OBLA was determined by extrapolation of this plot at a plasma lactate value of 4 mmol/l. This method is demonstrated (for workload) in figure 2.

(v). Cardiac Output

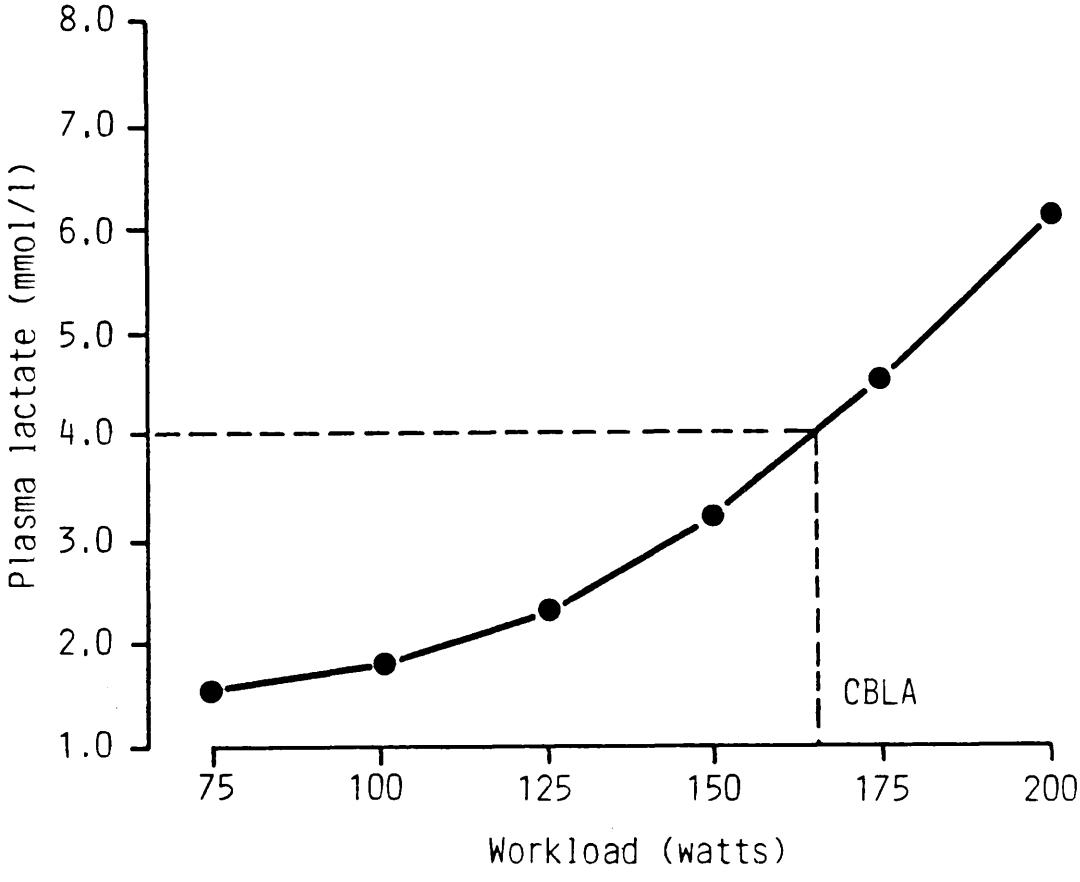
Basis of Method

Cardiac output is the volume of blood output ejected from the left ventricle of the heart each minute. Because of the technical difficulties in measurement, cardiac output has not been commonly assessed in previous training studies.

According to the Fick principle the cardiac output, when taken in conjunction with oxygen uptake, makes it possible to indirectly assess the arteriovenous oxygen difference of the overall vasculature.

FIGURE 2

Prediction of OBLA submaximal bicycle ergometer plasma lactate response



$A\text{-}V.dO_2 = VO_2 / CO$

Where $A\text{-}V.dO_2$ is the

arteriovenous oxygen difference

CO is the cardiac output

and VO_2 is the oxygen uptake

Thus by assessment of cardiac output in this study a fairly detailed examination of the oxygen transport system can be made.

Method & Equipment

The only method of directly measuring cardiac output is cannulation of the ascending aorta, thereby measuring the volume of blood ejected by the left ventricle. Such proceedure is not possible.

The direct Fick method is generally accepted as the most accurate method of cardiac output determination and is the standard method in clinical settings (Carpenter et al, 1985). Cardiac output is calculated by arithmetically dividing the oxygen uptake by the arteriovenous oxygen difference.

The accurate assessment of oxygen uptake is a relatively straight forward procedure. However, the direct assessment of the arterio-venous oxygen difference involves right heart catheterization in order to sample mixed venous blood. Catheterization of the heart is a procedure that must be carried out under surgical conditions by a trained physician and even then this technique has a risk of complications. The

direct Fick technique is therefore considered inappropriate for assessment of cardiac output in healthy individuals particularly when exercising.

The indicator-dilution method for determination of cardiac output was first introduced in 1897. It is a invasive technique which does not involve right heart catheterization or blood gas analysis. Injection of a known amount of material is made into the circulation and measurement made of the dilution of the material 'downstream' during a known period of time. Dyes such as indocyanine green and Evans Blue are generally used with the concentration of the dye being measured from small blood samples drawn continuously from an artery by an electrical photosensitive device. Excellent agreement has been reported between cardiac output values obtained during exercise when measurements were taken simultaneously with both the dye-dilution technique and the direct Fick technique (Saltin, 1973). In most, if not all, previous longitudinal studies which assessed the effects of training on cardiac function the technique adopted has been that of dye-dilution. The main limitation of this technique is that it involves the attainment of steady-state, a requirement that is difficult to achieve at high exercise intensities. Although the risks of the method are somewhat less than the direct Fick, the fact remains that it is an invasive technique and requires extensive subject cooperation.

Several non-invasive techniques of cardiac output determination have been developed with varying degrees of success. A commonly used modification of the indicator-dilution method is the thermodilution technique (Riedinger and Shedlock, 1984). Carbon dioxide rebreathing in particular, has been a method commonly used. Although the basis of the technique is simple and it requires minimal equipment, it is subject to considerable error. Error estimates as high as 20% have been reported when compared to dye dilution methods. However, the true error value is thought to lie within +10% (Cunningham & Patterson, 1976).

In this study a technique of cardiac output determination called First Pass Radionuclide Ventriculography (RNVG) was used. RNVG is a relatively recent technique (Van Dyke et al, 1972). Although RNVG is routinely applied in the assessment of cardiac function in patient populations it can also be used as a safe and accurate method of assessing cardiac output in normal populations (Reych et al, 1980). In addition RNVG, unlike previously described methods, allows not only the measurement of cardiac output but also end-diastolic and end-systolic cardiac volumes. As will be discussed in section 4.2, a method that enables determination of cardiac volumes allows determination of some of the factors determining cardiac output.

The method requires the injection of a dose of

radioactive substance into a peripheral vein. When this substance, now in the bloodstream, passes through the heart radioactivity is accurately measured. From this information it is possible to construct a time-activity curve which corresponds to the cardiac cycle. By applying a length-area formulae to the image of the left ventricle at different stages of the cardiac cycle it is possible to accurately determine the end-systolic and end-diastolic volumes. The difference between these two volumes, together with a measurement of heart rate, allows the determination of cardiac output. Although reportedly not as accurate as cardiac catheterization (Froelicher, 1983), RNVG allows the safe assessment of cardiac function during both rest and exercise.

The application of the RNVG technique and the analysis required to obtain cardiac volumes was carried out entirely by the staff of the Cardiac Department, Western Infirmary, Glasgow. A physicist operated both the gamma camera and the dedictated computer analysis system necessary for the measurements while the isotope was injected by a cardiologist.

A multicrystal gamma camera (Baird Atomic Inc, MA, USA.) equipped with a 1.0 inch collimator was used. This has the advantage of suitability for data aquisition and processing at a high count rate. When the subjects were comfortably at rest on the bicycle ergometer, the gamma camera was positioned anterior to their precordium. Proper positioning wih respect to the

heart was verified by an injection of a tracer dose of isotope.

The radiopharmaceutical, Technetium-99m-sodium pertechnate ($NaTcO_4$), produced from a commercial nuclear reactor was prepared in a 0.5 ml. bolus. With the subject sitting on the bicycle ergometer prior to exercise, this bolus was postioned in the connective tubing and flushed into the circulation as a discrete bolus by means of a second injection immediately after a first of 20 ml. of saline. With 2 studies to be performed, rest and exercise, the radiopharmaceutical was divided up into 2 doses of 300 and 500 MBq. (total 800 MBq.= 20mCi). First pass RNVG is dependant on the transit of a complete bolus through the central circulation. To prevent fragmentation of the bolus, care was taken to ensure that the injections were made as rapidly as possible at a time when the subject was not breathing heavily (only possible in the resting study). After this resting injection of isotope, 1 to 2 minutes were allowed for the counts acquiral, after which the execise protocol began.

During the last 30 seconds of peak exercise a second injection of the isotope was made in the same way as described above. To stabilize the chest and ensure adequate positioning of the heart, the subject's chest was sometimes held against the camera. The analysis of the counts data obtained from each test was analysed by the dedicated system computer.

A recent study in the present laboratory based on 17 subjects assessed at rest and during exercise was performed to assess the reproducibility and accuracy of the RNVG method. (Clelland et al, 1987). A high correlation was observed between contrast angiography RNVG ($r = \pm 0.90$, $P < 0.001$) with a standard error of estmate on EDV of 24 ml. High values of reproducibility values were observed for cardiac output during both rest and exercise ($r = \pm 0.81$ and $r = \pm 0.70$ respectively). The figures for this laboratory compare favourably with those published from laboratories in the United States (Froelicher, 1983).

2.6.2 Submaximal Treadmill Test

This test was carried out in the metabolic assessment laboratory of the Institute of Physiology, University of Glasgow. The entire experimental work in this test was performed solely by the author with the assistance of one technician.

(a) Selection of exercise protocol

To obtain the greatest amount of information from a submaximal test it is important to design a test protocol that will ellicit as wide a range of physical effort as possible yet not fatigue a subject to exhaustion (Shephard et al 1968b).

It is important that the initial level of submaximal work is sufficient to stress the subject to an extent where their physiological reponses reflect

the level of exercise rather than the effects of the testing environment and psychological factors placed on the subject (Shephard et al, 1968b). For young adults this minimum level of work is taken as that which achieves a steady state heart rate of 100 to 120 beats/min or more (ACSM, 1980). This is particularly important if accurate extrapolation of this submaximal data to peak exercise is to be performed (Maritz et al, 1961).

The selection of the appropriate submaximal protocol is dependant both on the fitness and age of an individual to be tested (ACSM, 1980). A 'fit' individual is defined as being able to perform a standard submaximal workload test at a lower heart rate than their less fit counterparts. A standard protocol may elicit a suitable range of physical effort in a fit individual but overtax an unfit one. It is well documented that maximal heart rate falls steadily with age (Astrand, 1960). Although an older person may be able to perform a given level of work for the same heart rate as a younger individual, the older person will be working at a higher proportion of their maximal heart rate. In selecting a standard treadmill protocol for this study it was therefore important to consider the age range of the group and their initial range of fitness.

Data has been previously collected in this testing laboratory on young adult males taken from a University

student and technician population. This data was taken from the test protocol of 5 minutes walking at 3 mph on the level, then 5 minutes walking at 4 mph on the level then finally 5 minutes walking at a speed of 4 mph on a 5% grade or 10% grade for fit individuals. The average heart rates for this group at these three levels of exercise were approximately 100 beats/min, 130 beats/min and 160 beats/min. (Durnin, 1986). This range of heart rates was deemed most suitable for this population who, using the data from Astrand's laboratory, were calculated as having a mean predicted maximal heart rate of 195 beats/min (Astrand, 1960).

In order to assess the adequacy of this protocol for the present population, a pilot study was performed in which 10 sedentary and 10 active males aged 35 to 50 years (mean 38 yrs) were taken from a population of University staff. The sedentary group was defined as those individuals who did not participate in aerobic endurance sport on a regular basis. The active group was defined as those individuals who had been performing aerobic endurance exercise at least 3 times per week for 1 year or more. They performed the same protocol as described above except that the active group performed all 4 levels of work. Heart rate was recorded during the final 3 minutes of each 5 minute period. Table 1 summarizes this data.

The three levels of exer cise in the sedentary group appeared to ellicit a heart rate range that

TABLE 1

Summary of pilot study evolving treadmill protocol
(means and SD's)

Treadmill work level	Heart rate (beats/min) Sedentary Groups	Active Groups
3mph 0% grade	101±6	89±7
4mph 0% grade	132±8	112±10
4mph 5% grade	158±10	128±12
4mph 10% grade	-	156±12

exceeded 100 beats/min yet did not exert the subjects to maximal. The range of predicted maximal heart rate for this group was 170 to 185 beats/min (Astrand, 1960). In the active group, these initial three levels of exercise were not sufficient to ellicit an adequately high heart rate range. The inclusion of a fourth level of exercise increased heart rate to a level similar to that in the sedentary group.

In view of these findings it was decided that prior to training (T1) the intitial three levels of treadmill work would be performed while midway (T2) through training and at the end of training (T3) the fourth level of exercise would be included (see Figures 3 & 4). It was decided that for ease of direct comparison these protocols would be strictly adhered to even if a subject failed to produce an appropriate heart rate. However, it was hoped that by performing the above pilot study the incidence of such a situation would be minimal.

Some previous studies have shown that maximal walking protocols give rise to an unreliable value of VO_2max (Shephard, 1984). In this test it was intended that the subjects performance would always be submaximal. A walking protocol has the advantage that it is safer, less liable to variability and requires less time for familiarization than treadmill running (Shephard, 1984).

(b) Method and equipment

FIGURE 3

Submaximal treadmill exercise; timings of exercise increments and measurements (T2 and T3)

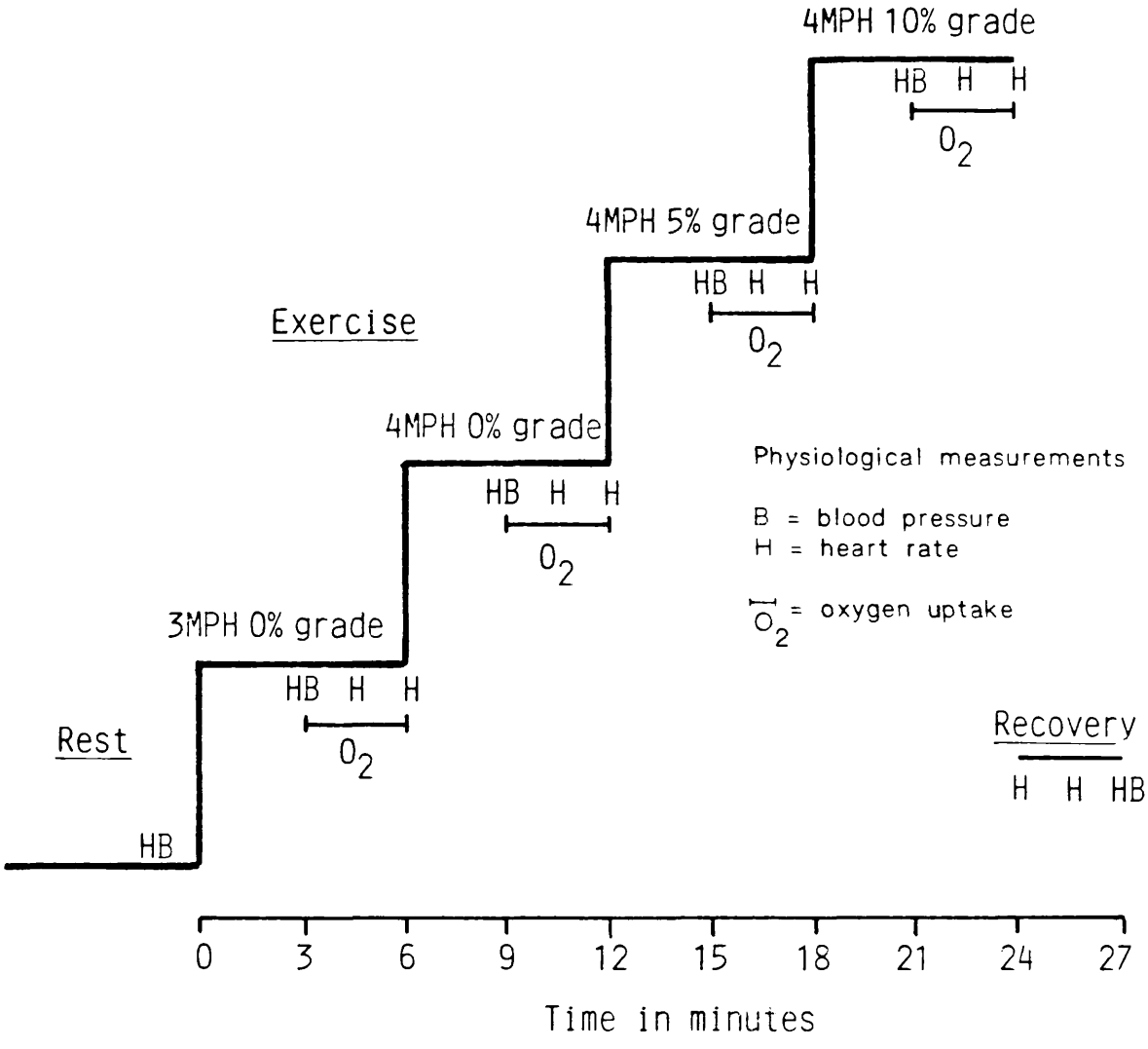
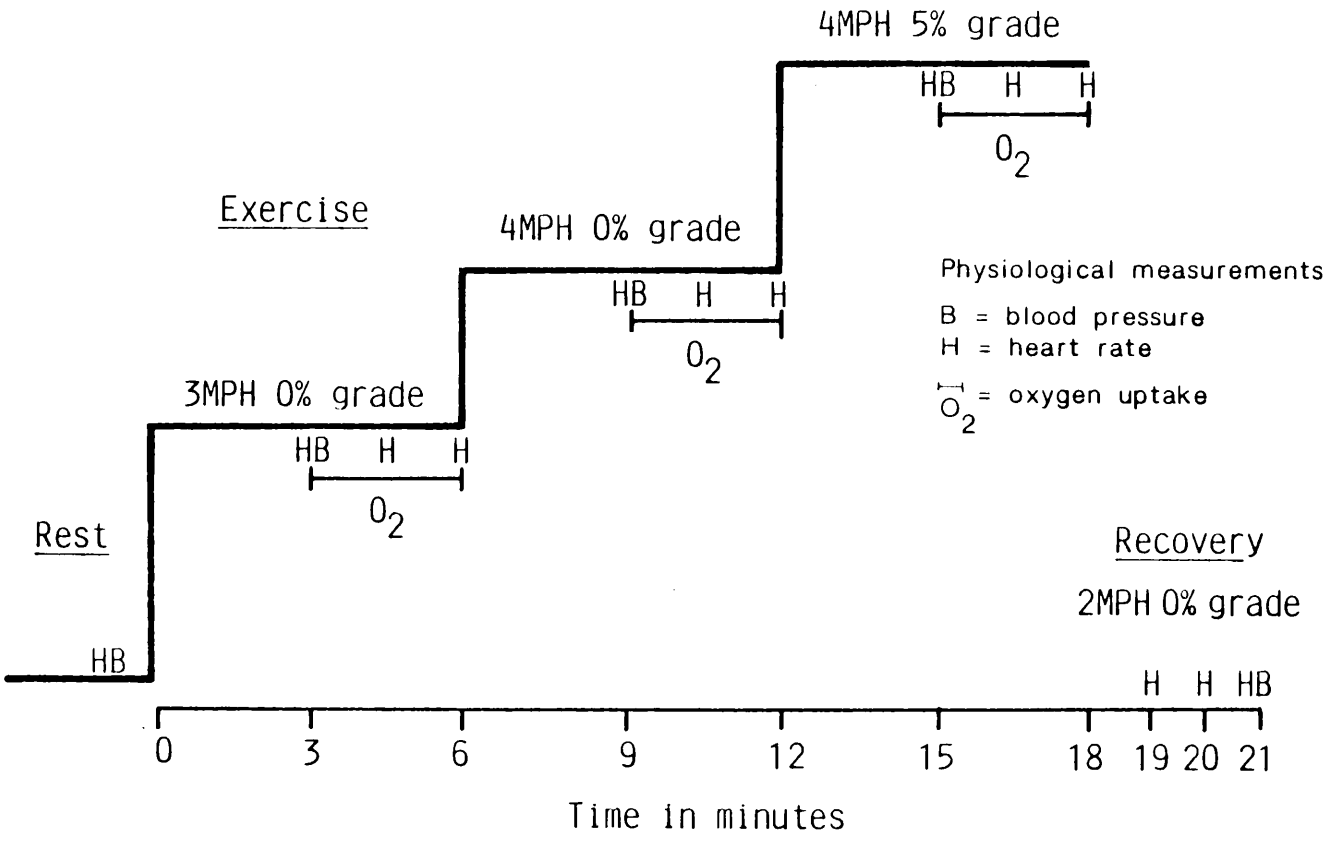


FIGURE 4

Submaximal treadmill exercise; timings of exercise increments and measurements (T1)



This test was performed at least 2 days after the maximal bicycle test to ensure adequate recovery.

The effects of learning and habituation in treadmill exercise are considerable and outweigh that of the bicycle ergometer (Anderson et al, 1981). Shephard (1970) examined the physiological reponse of a group of naïve subjects on two consecutive days. They found that there was an average fall in heart rate at a given level of treadmill exercise from day 1 to day 2. In the present study, on their first visit to the laboratory, the subjects were given a 10 minute period of slow treadmill walking to familiarize themselves with the experimental set-up. The subjects were asked to walk as naturally and as economically as possible. Care was taken to ensue that the subjects walked without holding onto the handrails, an action that is known to reduce the oxygen cost of the exercise (Hammond & Froelicher, 1984).

Both speed and gradient of the treadmill were operated by the observer with an increase in workload occuring every 5 minutes. The treadmill was regularly calibrated for both speed and gradient throughout the study. If the subjects heart rate exceeded their age predicted maximum (Astrand, 1960) or if they felt undue discomfort or fatigue the exercise was terminated.

At the end of the exercise protocol the subjects were asked to continue walking while the treadmill speed was gradually reduced to the 2 mph. level

where it remained for 3 mins. As discussed previously this active recovery was used to reduce the risk of venous pooling. The subjects were asked to keep exercising until their heart rate returned to 100 beats/min or less.

(c) Measurements

(i). Respiratory factors

These measurements were carried out using the Douglas bag technique as described above for bicycle exercise.

At the beginning of the third minute of each treadmill workload the 3-way tap was turned so that the expired air of the subject could be collected. At the end of the sixth minute of this exercise load this tap was turned so that the contents of the Douglas bag became shut off. Analysis of the expired air was carried out as above and the appropriate respiratory calculations performed on a programmed PET computer. A summary of these calculations is shown in Appendix G.

(ii) Heart rate

Heart rate was measured prior to, during and immediately after exercise by electrocardiographic monitoring using a single channel ECG recorder.

The ECG was recorded from the lead position CM_5. The indifferent electrode was attached to the manubrium of the sternum, the exploring electrode placed in the V_5 position and the neutral electrode placed on the back of the chest. This lead was chosen because its

simple electrode placement avoided the large skeletal muscle masses that would lead to interference. CM5 also usually senses a large QRS complex which makes heart rate assessment by R-R interval measurement simple (Blackburn et al, 1967).

The ECG was continuously displayed on a VDU monitor throughout the test. Heart rate was determined during the last 20 seconds of each of the last 2 treadmill workload minutes from the average R-R on a 25 mm/sec read-out. The values of treadmill exercise heart rate reported in this study are the average for the 2 minute recording period. Heart rate was also measured minute-by-minute during the 3 minutes immediately post-exercise.

(iii) Blood Pressure

Arterial blood pressure was assessed using the indirect method described above for the maximal bicycle ergometer test.

Prior to the commencement of exercise, triplicate measurements of systolic and diastolic blood pressure were taken with the subject in a seated position. The lowest of these readings was recorded. The systolic was assessed during the last 30 seconds of the 2 final minutes at each level of exercise. Systolic blood pressure was measured during last 30 seconds in the 3 minute period immediately post exercise.

2.6.3 Body Composition and energy metabolism

These evaluations were carried out in the Metabolic Assessement Labotaory of the Institute of Physiology, University of Glasgow. The entire experimental work was carried by the author except for assistance by one technician.

(a) Selection of Testing Procedures

As described in Section 1.3.3, previous studies that examined the effect of aerobic endurance training on body composition have in general restricted themselves to the analysis of the parameters of total body weight and body fat content (Wilmore, 1983).

These studies have assessed body fat principally by the technique of skinfold measurement although a limited number of studies have also determined body density by hydrostatic weighing. The skinfold sites and regression equations of body fat calculation from these sites chosen by these training studies reveals considerable variation (Oscai, 1973).

In accordance with these previous studies it was decided that the present study would base its analysis of the effect of training on body composition on the measurements of total body weight and the proportion of that weight which was fat. The skinfold sites and regression equation for body fat determination chosen was that of Durnin & Womersley (1974). These sites (biceps, triceps, subscapular and supra-iliac) are relatively easy to assess and have been shown to have a high degree of measurement reproducibility (Durnin &

Womersley, 1974). Moreover these equations, unlike other regression equations, have proved not to be population specific in that they have been found to accurately reflect body fat content in other groups (Wilmore, 1983). Additionally, since the subjects of the present study were selected, like those of Durnin and Womersley, from a Glasgow population it might be expected that this would further reduce the possible prediction error of these equations.

In addition to the measurements mentioned above, assessment of body circumferences was also made. It was intended that these later measurements in combination with body fat would give a crude estimate of changes in skeletal muscle content of different body areas (Weiner & Lowrie, 1969).

(b) Methods and Equipment

(i) Anthropometry

The anthropometric measurments were made prior to treadmill exercise using the following proceedures.

Stature.

Each subject, with his heels together and stretching upwards to his fullest extent, stood on the horizontal platform of a Harpenden wall-mounted stadiometer. His back was as straight as possible against the back of the stadiometer and his Frankfort plane was checked to be horizontal. He was asked to "take a deep breath" in order to make him stretch up after which the head-bar was brought down onto his

head. The subject's heels were always watched to ensure that they were not raised. Readings were taken to the nearest millimeter.

Body Weight.

Weighing was carried out with the subject clad only in underwear or light sportswear. This was done at the same time as underwater weighing, to the nearest 0.1 kg. on an Avery beam balance (Model 3302 ABN).

Skinfold thickness.

This method is based on the assumption that a predictable proportion of fat is situated subcutaneously and therefore if subcutaneous fat is measured indirectly from skinfolds, it will provide an estimate of the total body fat content.

These measurements were done on the right side of the body with the subject standing in a relaxed condition, although no statistical difference between measurements on either side of the body have been found (Womersley & Durnin, 1973).

The instrument used was a Harpenden caliper (Holtain Ltd., Bryberian, Crymmych, Pembrokshire). This was regularly checked for consistancy of pressure over the full range of jaw opening (ie. 10±2 g./mm^2 surface area of jaw) and accuracy of reading for various distances between the jaws. The skinfolds were picked up between the thumb and forefinger and the caliper jaws applied, approximately 1 cm. below the forefinger and thumb, at the skinfold site. The reading was taken

2 seconds after the full pressure of the caliper jaws was applied to the skinfold. Each reading was taken to the nearest 0.2 mm.

In choosing suitable skinfold sites, several basic factors must be taken into account. The fold must be relatively easily picked up and not too firmly attached to the deep fascia. The site must be accurately definable, since the difference between 2 sites of only a few cms. apart can be considerable (Garn, 1954). In addition, the few sites measured must be representative of total body fat. Based on these considerations and as recommended by the International Biological Program (Weiner & Lowrie, 1969), Durnin and Womersley choose the following 4 sites and these were selected for this program.

1. Biceps: The skinfold was picked up on the front of the relaxed arm at the midpoint of the muscle in a vertical direction.

2. Triceps: The skinfold was taken at the back of the relaxed arm, at the mid-point between the acromion process and the olecranon process. The measurement was taken at this midpoint and directly in line with the two processes (this site was marked on every subject) in a vertical direction.

3. Subscapular: The skinfold was picked up under the angle of the scapular, and just below the tip of the inferior angle of the scapular, at an angle of about 45 degrees to the vertical, with the fingers touching the

bone.

4. Supra-iliac: This measurement was taken just above the iliac crest, on the mid-axillary line with the skinfold in a vertical position.

Each of these measurements was taken in triplicate and the mean, to the nearest mm., was recorded.

Although it is estimated that about half the total body fat is subcutaneous (Keys & Brozek, 1953), the precise proportion of total fat found in the subcutaneous tissues has been shown to be dependant on sex and age (Durnin & Womersley, 1974). The sex and age dependant logarithmic equations of Durnin and Womersley (1974) have been used in this study to predict body density from the sum of the biceps, triceps, subscapular and supra-iliac skinfolds (see Appendix). Based on the 2 compartment model of the body (ie. 'Fat free mass' and body fat) each of which have a constant and known density then body density was then converted into body fat content (percent fat), using the equation of Siri (1956). These equations are shown below:

Durnin and Womersley equations

Age 30-39 yrs: $D = 1.1422 - 0.0544 \times \log SUM$

Age 40-49 yrs: $D = 1.1620 - 0.0700 \times \log SUM$

where D is the body density (kg/m^3)

SUM is the sum of 4 skinfolds

Siri Equation

$$\%F = (4.95/D - 4.50) - 100$$

where %F is the body fat content (percent)

D is body densiity (kg/m^3)

Body circumferences.

Assessment was made of body circumference in order to gain a crude estimate of the relative amounts of fat and skeletal muscle within this area or limb of the body.

The measurement was again made on the right side of the body with a flexible steel anthropometric tape (Holtain Ltd., Bryberian, Crymmych, Pembrokshire). Measurement was made to the nearest 0.1 cm.. The following sites of body circumference were assessed.

1. Upper Arm: With the subjects arm hanging relaxed and just away from his side, the horizontal circumference was taken midway between the inferior border of the acromion process and the tip of the olecranon process. This measurement overlapped the skinfold site.

2. Calf: The subject was seated on a table with his legs hanging freely and the back of his knee touching the table. By moving the tape up and down his leg the maximum horizontal circumference was located and measured.

3. Thigh: With the subject standing, feet slightly apart and weight evenly distributed on both feet, the measurement was taken with the tape placed horizontally around the thigh and with its top edge just under the gluteal fold.

4. Buttocks: The maximum horizontal circumference was measured.

(ii) Hydrostatic Weighing.

This measurement was made after submaximal treadmill exercise.

The overall density of the body is determined by use of Archimede's Principle: weighing the subject first in air and then in water, correcting the weight in water for the volume of air in the lungs. Assuming the body consists of 2 compartments (the 'Fat free mass' and body fat) of constant and known density, the relative proportions of these 2 compartments and the absolute fat content of the body can be estimated using the appropriate equations.

Each subject was weighed in air as described above. Underwater weight and the lung volume at the time of underwater weighing were assessed by the procedure described below.

The subject entered a tank of water heated to a temperature of 36 to 38 $_{\circ}$C, taking care to rub their hands all over their body (particularly scalp, axillae and pubic regions), in order to remove any entrapped air bubbles. The subject then sat on a seat in the tank attached to a load-cell which in turn was supported by a hook onto the ceiling above the tank. The load-cell gave a display of weight on a digital voltmeter to the nearest 0.01 kg. Just prior to the experiment this voltmeter was 'zeroed' for the weight of the weighing chair. The subject then fitted a nose-clip and practised expelling air from his lungs and gently

ducking himself so that he was just totally immersed. Three 3-litre rubber aneasthesia bags with 2-way taps were filled with a known volume of pure oxygen from a Benedict-Roth spirometer. The subject was now ready to begin the actual experimental procedure.

The subject sat perfectly still, expired as much as he could comfortably and then very gently (to prevent oscillations of the weighing chair and load cell) ducked his head under the water so that he was completely immersed. A maximal expiration (ie. to residual volume) was considered unnecessary as it has been previously demonstrated that the volume of expiration has little effect on the accuracy of the lung volume determination (Durnin & Satwanti, 1982). Moreover, a maximal expiration often makes the subject nervous toward the procedure. This immersed position was held for 3 to 5 seconds until a stable reading could be taken from the voltmeter. As soon as a reading was obtained, the subject raised their head out of the water on a signal and without breathing any atmospheric air, were connected to an anaesthesia bag containg pure oxygen. The experimenter then opened the 3-way tap towards the subject who took in a deep breath of oxygen, expelled it and then breathed in and out twice more.

The 3 breath procedure allows thorough mixing of the gas in the alveolar spaces with the oxygen in the bag (Rahn et al, 1949). The tap was then closed and the

oxygen and carbon dioxide contents of the bag determined using paramagnetic (Servomex Ltd.) and infra-red (P.K. Morgan Ltd.) analysers respectively. These analysers were calibrated prior to tests on each subject by using standard gas mixtures contained in cylinders which, in their turn, were calibrated using a Lloyd-Haldane apparatus. The details of this calibration are discussed in the appendix. The nitrogen concentration was determined by difference (ie. by subtraction of the carbon dioxide and oxygen content from 100%). The volume of air in the lungs was then determined by nitrogen dilution based on the nitrogen content of alveolar air being 80%. This and the appropriate calculations for determination of body density and body fat content were performed on a programmed CBM Series 3032 Professional computer.

The whole procedure was carried out in triplicate with an interval of at least 5 minutes between measurements. In the unusual event of there being poor agreement between these three sets of results, a further measurement was performed.

(iii) Calculation of the energy cost of training.

The direct measurement of the energy cost of exercise involves the performance of that particular activity inside a calorimeter. This technique is limited because it requires the aquisition of an expensive non-portable calorimetric chamber. Although portable calorimetric suits have been developed in

recent years (Webb et al, 1969), the measurement of energy expenditure is usually performed indirectly (Durnin and Passmore, 1967). Probably the most commonly used indirect technique is that of the Douglas Bag method. The amount of oxygen consumed is measured by this method and can then be converted to a given energy value taking account of the relative proportion of carbon dioxide produced (Weir, 1949). This procedure although considerably more easy to perform than direct calorimetry, is not sufficiently portable for taking field measurements. The measurement of oxygen uptake is a relatively simple procedure during treadmill running in the laboratory but is impractical during a normal outdoor training session.

It was necessary in this study to find a method for determining the energy cost of training without the restriction of cumbersome equipment for taking field measurements. A traditional method for the calculation of energy expenditure where no physiological measurements are required is the use of standard tables of physical activity energy values previously determined from population studies. Standard energy values have been determined for a variey of activities such as standing, sitting and a various speeds of walking (Durnin and Passmore, 1967).

The determination of the standard energy cost of running has been the topic of research for a number of years (Hill and Lipton, 1923). The information from

these studies has generally been limited because of small sample numbers (often 3 or less) and the selection of only elite or good distance runners.

In the 1950's the concept emerged of the existence of a linear or a very nearly linear relationship between running speed and energy expenditure during treadmill running. Early proponents of the linear relationship concept stated that it was possible to estimate energy expenditure from running speed alone and this value was independant of other factors such as body weight (Henry, 1951). However, Margaria and his co-workers (1963) found that the energy cost of running was dependant not only on the speed of running but also on body weight - increasing body weight resulting in an increased energy cost of running at a given speed. They calculated the net cost of running to be 1 kcal/kg/km and developed a nomogram for the determination of energy-cost for various running speeds and body weights.

The study of Margaria et al, was performed on relatively few young adult males with a well-conditioned background and small range of body weight. Never-the-less the values of the energy cost of running as determined from this study have been applied to individuals with a variety of training and body composition backgrounds (McArdle et al, 1981). Indeed Dill (1965) noted differences of up to 16% lower weight corrected oxygen cost of submaximal running in elite

runners when compared to their non-elite counterparts.

In the late 1960's, Shephard (1968) undertook to investigate the relationship between running speed and energy cost on a comparitively large group of men (n = 24), of varying age (20-40 yrs.), body weight and activity backgrounds, and over a variety of treadmill speeds (5-17 mph). Like Margaria and others, Shephard observed that running speed and weight corrected energy-cost were linearly related. He calculated an equation for the oxygen-cost of running based on speed and body weight:

$$VO_2 \text{ (ml/kg/min)} = 4.61V + 7.7$$

where V = running speed in km/hr

This equation can be redefined in terms of energy expenditure:

Assuming that 1 l. of O_2 = 5 kcal

$$EE \text{ (kcal/min)} = ((4.61Vx + 7.7) \times TBW) / 200$$

where EE = energy cost running

Vx = running speed in mph

TBW = total body weight in kg

In all the above studies, the energy costs were determined from treadmill running. Pugh (1970) examined the possible difference that might ex ist between standard treadmill and out-door running and noted that although over the same range of speeds treadmill energy cost vs speed was linear, a curvilinear relationship exsisted between track running and energy-cost. Pugh concluded that this extra energy demand was due to the

cost of overcoming the air resistance of still air. This extra energy increased both with increasing running speed and increased subject surface area.

Increase in VO_2 due to air

resistance = $0.00418.Ap.V^2$

where Ap is the projected body area

(ie. 0.266 x surface area)

V is running speed in m/sec

It was calculated that this energy cost of overcoming air resistance can amount to up to 7.5% of the total energy cost of running at middle distance speeds.

The energy cost for each training session in the present study was determined by using Shephard's equation. Body weight was taken as the previously determined laboratory value. Running speed of each training session was calculated from the reported training time and distance in the subject's training diary. It was assumed that the average running speed was indicative of training effort. Training speed was calculated by dividing the total time for a training run by the distance covered. The energy cost of the training session was calculated by multipying the energy cost per minute for this speed by the ssion duration. These running energy costs were corrected for outdoor running using the above equation of Pugh (1970) and an average value of surface area for the study group. It was found that for running speeds of 8 mph, a significant alteration in energy cost due to wind

resistance occured. The following corrections were applied to the Shephard figures to take this into account:

8mph: +0.5 kcal/min, 9mph: +0.75 kcal/min,
10mph +1.0 kcal/min

(iv) Calculation of changes in energy intake with training

As the subjects prior to this study were of relatively constant weight it can be assumed that they were in a relative state of energy balance prior to training ie. their energy expenditure was equal to their energy intake.

During the present training program, if this energy balance was no longer held then there would be changes in the body's energy store. Fat deposited in situations of energy intake excess and fat lost if energy expenditure was in excess. Energy intake was not directly measured in this study and it was therefore not possible to directly measure any changes in energy intake that may have taken place throughout the duration of this study.

As described above the change in energy expenditure as the result of exercise was estimated. Assuming the major change in the body's energy store with training was in fat content, it was possible to estimate this change from the measurements of body fat content of the subjects. Using these two values it was possible to assess the change in energy intake of the

subjects during this training program.

Change in EI = Change in EE - Change in ES

Where, EE = energy cost of training

ES = energy store as assessed by body fat content

EI = energy intake

The validity of the assumptions behind these calculations will be considered in the Section 4.5.3.

2.6.4 Training Program.

(a). Principles of Training.

The success of any exercise program in obtaining a physiological training effect is dependant on the so-called 'overload principle' ie. the body's energy systems only adapt in situations where they have conditions imposed on them that exceed their normal range of function (Astrand & Rodahl, 1977).

As was discussed previously in section 1.3.3, it has been suggested that in order to obtain such a training effect a minumum of 3 exercise sessions per week of 20 to 30 minutes at an intensity of 60% VO_2max or 70% heart rate reserve needs to be performed. This minimum threshold may be slightly less in older individuals or subjects of poor initial condition. This exercise should be 'aerobic' in nature ie. involving continuous rhythmic contractions of large muscle groups against little resistance.

Although overload can lead to a training effect,

too great an overload may result in injury (Pollock et al, 1977). Previous studies have shown that even modest degrees of training in novices can often lead, during the early stages of training, to 'overuse' injury (Oja et al, 1975). Compared to non-weight bearing activities such as cycling, swimming, rowing etc., running training appears to lead to a greater incidence of skeletomuscular injury (ACSM, 1978).

The vehicle of aerobic endurance training in this study was that of preparation for the 26.2 mile marathon distance. On the basis of the above principles it was important for this program to have a number of characteristics. Firstly, the principal mode of training should be the same as that to be performed in the marathon ie. running/jogging. Second, the level of exercise training should be sufficient to ensure adequate physiological adaptation to allow marathon completion. Finally, the initial level of training prescription should be limited to reduce the risk of injury.

(b) Previous marathon training programs.

The 26.2 mile classic marathon distance involves maintenance of running effort for prolonged periods (2 hrs or more). In view of its physiological demands, the major adaptive effects of marathon training should include adequate energy production, temperature control and skeletomuscular endurance (Costill et al, 1970).

Elite marathoners maintain a regular running

background of 50 or more miles per week throughout the year and in preparation for a marathon commonly exceed 100 miles per week (Maron & Horvath, 1978). These distances although critical to the adequate overload and preparation for elite performers, are obviously impractical for the novice runner. Attempts by a novice to perform such training would certainly result in serious skeletomuscular injury and physical exhaustion.

The marathon distance has traditionally been the domain of the experienced runner only. Most if not all previously published marathon training programs have been aimed only toward these individuals. With the recent advent of mass marathon running by novice runners, the requirement for less demanding training programs has grown. These novice training programs appear to depend on the balance of 2 factors ie. sufficient running ('mileage') to allow completion of the marathon distance but not so much running that may result in overuse injury.

There is much debate as to what is sufficient mileage for marathon performance. Young (1978) has coined 'the collapse point theory' which predicts a dramatic slow-down in the latter part of the marathon if there has been insufficient training distance. Young hypothesized that a runner must cover at least 63 miles per week over the eight weeks prior to the marathon to avoid the collapse point. Other authors have suggested that lower training distances such as 40 to 45 miles

per week may be sufficient (Glover & Shephard, 1977, Shelley & Donavan, 1982).

Many authors have suggested that marathon training for the novice should be preceded by at least 12 months running experience. However, the majority of individuals competing in their first marathon want to do so in less than a year. Indeed one of the programs most commonly followed by novice marathoners has been the 21-week program of Cliff Temple aimed at 'Sunday Times' readers preparing for the first London Marathon in 1981.

Previous novice marathon training programs have usually been based on either the personal running or coaching experience of their authors. Few if any of these programs have been evaluated as to their adequacy or success. However, one such marathon training program for beginners, the 30 week program of the Department of Physical Education and Recreation at Glasgow University (Grant et al, 1984), has received such evaluation.

Preceding the 1982 Glasgow Marathon, a seminar on training for first-time marathoners was held at Glasgow University. Of the 225 people who attended, a total of 88 men and women agreed to follow the discussed program of training and provide information on their training progress and marathon performance. The 88 volunteers were self-selected in as much as they were seminar delegates who decided to take up the challenge of the marathon and also keep a precise record of their

training and marathon times. All these volunteers were novice marthoners and the majority were also novice runners with only limited previous regular exercise experience. The age of the group ranged from 18 to 70 years (36.9+9.7 yrs.).

Grant and his colleagues evaluated this training program on the basis of this group based on 2 criteria:
1. The incidence of serious skeletomuscular injury - enough to cause drop-out.
2. The ability to complete the marathon distance without a significant slow-down. This was assessed by the relative time taken to cover the last 6.2 miles.

On both these criteria this program appeared to be successful and although some 74% of the group complained of undiagnosed skeletomuscular injury during training only 10 individuals (12%) required to drop out through injury. Of the remaining 72 individuals who completed the 1982 Glasgow Marathon (mean time: 250+35 mins) the last 6.2 miles took on average 64.3 mins which accounted for some 25.6 % of total running time. 6.2 miles is 23.7% of the marathon distance. Clearly there was little evidence of a decrease in pace over the last 6.2 miles.

In view of the successful evaluation of the marathon training program of Grant et al, their program was chosen for this particular study (see Table 2). The details of this program are discussed below.

TABLE 2

Summary of prescribed training schedule (all sessions in minutes)

Week no.	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday
1		20		20		20
2		20		20		20
3		30		20		20
4		30		20		30
5		30		30		30
6		30		30		30
7	30	20		30		30
8	40	20		30		30
9	40	30		30		30
10	50	30		30		30
11	50	30		40		30
12	60	30		40		30
13	70	30		40		30
14	70	30	30	30		30
15	80	30	30	40		30
16	90	30	30	50		30
17	100	30	40	50		30
18	110	30	40	50	30	30
19	120	30	40	60	30	30
20	130	30	40	70	40	30
21	140	40	40	80	40	40
22	150	40	40	90	40	50
23	160	40	40	90	40	50
24	160	40	40	90	40	60
25	160	40	40	90	40	70
26	160	40	40	90	40	80
27	160	40	40	90	40	90
28	180	40	40	90	40	90
29	120	40	40	60	40	60
30	60	40	40	20	20	

(c) Features of the present marathon training program.

(i) Program Duration.

The longer the overall duration of a marathon training program for a novice runner the more gradual the increase in training load thus ensuring sufficient time for an adequate training adaptation to occur and also reducing the risk of injury. However, the majority of novice marathoners only take up the commitment to run this event when the entry forms become available. In Glasgow, like many other city marathons, this is only 8 months prior to the event. The 30 week duration of this study in just within this period.

(ii) Units of Training Prescription.

The majority of studies that have investigated the changes in physiological function with physical training have prescribed training to their participants in terms of duration, frequency and intensity (Pollock, 1973). Running training programs often prescribe running training on the basis of miles. In the present study, training prescription was given in terms of the frequency and duration of the sessions but not intensity or mileage.

Mileage is a training unit that is applicable to the conditioned runner. However, in the case of a group of novice runners often with widely different initial levels of fitness, prescription in terms of miles can often lead to a substantial variation in the time spent running by different individuals. This would presumably

lead to a considerable discrepancy in individual training load. Moreover in intially unconditioned novices with slow paced running, to quantify their training progress in terms of distance when they can only manage short distances initially can be psychologically demoralising.

The intensity of training was not prescribed in this study for practical reasons. Training intensity is usually prescibed either in terms of percent VO2max or percent heart rate reserve (HRR) (ie. HR rest + X(HR max - HRrest) (Karvonen et al, 1957). Values of training intensity are generally set at 50 to 60 percent VO2Tmax which corresponds to 60 to 70 percent of heart rate reserve (ACSM, 1978). During training within the laboratory environment, using either treadmill running or bicycle ergometer cycling, the maintenance of a given work intensity is relatively easy to ensure. However, when training outwith the laboratory, the intensity is generally set by asking the subjects to work within a heart rate band set by the heart rate reserve method. Many of these studies validate this training intensity by the subject monitoring their heart rate immediately on termination of exercise by palpation of the carotid or brachial pulse.

The majority of previous training studies have assessed training intensity by asking their subjects to perform post-exercise pulse assessment. However, there is considerable debate as to the accuracy of this

method of intensity estimation. McArdle and his colleagues (1969) reported a 13.5% error at moderate workloads when comparing telemetered exercise heart rate to that measured by the subjects via palpation 10 seconds post exercise. Pollock et al (1972) observed an error of only 2% in comparing palpation to ECG. However, the error of Pollock's study was achieved only after a great deal of practice (8 practice sessions). In addition this later evaluation was performed by comparison of ECG and palpation during during exercise. Thus although pulse self monitoring can be accurate it does demand considerable practice and is more reflective of true exercise intensity if actually performed during exercise itself. However, monitoring during exercise, although more indicative of true exercise intensity is often impratical for many subjects.

It was concluded that if accurate prescription of training intensity on the basis of heart rate was to be made in this study, then direct heart rate assessment during exercise would be required. Unfortunately access to a large number of such light weight heart rate recorders was not possible. In view of this training intensity prescription was given in this study. However the participants were asked to maintain a subjectively "comfortable" intensity during training. This intensity was described as one at which the exerciser should be working but not so intensely "that they could not carry

out a conversation with a training partner".

In an attempt to gain some indication of the training intensity of the study participants, a limited ammount of heart rate monitoring was performed using the PE-3000 Sports Tester monitor (Polar Electro PE-3000, Finland).

(iii) Nature of training.

Although previous training studies have often used more than one mode of exercise in their training programs, in the present study the only exercise prescribed was running/jogging. As discussed above, one of the most important adaptations necessary for successful marathon completion is the familiarity of the individual with prolonged periods of running. This total emphasis on running as the mode of training was intended to maximise such running experience.

In addition to long slow distance, elite marathoners may use interval running and fartlek in an attempt to improve their running speed and anaerobic metabolism prior to a marathon. In view of the limited duration of the present training program and the increased risk of injury with such types of training, these forms of training were considered inappropriate for this study. It was outlined that all training runs be performed at continuous steady submaximal running pace.

(iv) Training progression.

As mentioned above, the majority of previously

reported aerobic training programs were of a fixed session duration, frequency and intensity (Pollock, 1973). However a fixed training approach was not appropriate in this present study.

Intially the duration and frequency of training were maintained at a minimum to ensure a training effect and to minimize the risk of injury. Once this initial training load was experienced the duration and frequency of training increased progressively over the 30 weeks. The increment in training workload increase was minimized to again reduce the risk of injury but ensure adequate training progression to enable successfull marathon completion.

Marathon programs are commonly designed to reach a training peak prior to the event and training load is greatly reduced in the immediate build up to this event. This pattern of 'tapering' is believed to be crucial in enabling adequate physical and mental recovery from previous training demands. Two weeks 'tapering' was prescribed for this study.

(v). Training supervision.

The majority of the prescribed training was carried out independantly by the subject himself. However, some supervised group training sessions were performed. Evidence has shown that such supervised group training can improve motivation and enhance training compliance (Dishman, 1982). During the first 10 weeks of the program, 1 supervised evening training

session was carried out every 2 weeks. To aid the group in performing their 'long runs' sessions, from week 10 supervised group sessions were carried out every every sunday morning.

(vi) Warm Up & Cool Down.

In addition to the actual time prescibed for running/jogging, the participants were also asked to follow a 5 minute procedure of stretching exercises and easy jogging both prior to and after their training run.

Evidence, although conflicting, has shown that 'warm-up' exercises prior to training can improve performance by increasing muscle temperature and blood flow and also enhance oxygen consumption kinetics as well as diminish the risk of injury by increasing muscle elasticity as the result of temperature increase (deVries , 1980).

The post exercise 'cool-down' was included to reduce the risk of venous pooling and also lessen post-training muscle soreness (Pollock et al, 1984).

(vii) Training record.

Each participant was asked to maintain throughout their training a comprehensive training record so that both they and the observer could monitor their progress. A copy of one weeks training record is shown in the appendix I.

The subjects were asked to record their nude body weight and resting heart rate first each Sunday morning

immediately on rising and after emptying their bladder. It has hoped that this provide the subjects to self-monitor changes in body composition between testing.

Investigations have shown that resting heart rates can also act as an index of training fatigue. Overtraining can lead to an increase in resting heart rate of some 5 to 10 beats/min. While training the subjects were asked to estimate the length of their run as accurately as possible and this, together with the time, allowed determination of their running pace. Cumulative totals of distance and mileage were made on a weekly basis. Illnesses and weather conditions during the run were recorded to explain possible day-to-day variations in running performance. Finally in order to assess the paticipant's motivation and state of mind, a record was kept of their feelings during and immediately after each training run.

The diaries of each subject were retrieved by the author every 2 weeks and feedback to the subject on training modifications or motivation given if necessary. It was stressed that the information recorded should be as accurate as possible.

2.6.5 Statistical Methods

The analysis of the present training program results was performed only on those 39 individuals who completed the 30 weeks of training. The study group

values are analysed terms of the mean, sample standard deviation (SD) and standard error of the mean (SEM).

The effectiveness of the training program was assessed for the complete duration of training by determining the change in value of physiological variables prior to and post training (ie. T1-T3). In addition the change with training was assessed seperately in terms of the initial (T1-T2) and final (T2-T3) 15 weeks of training. The significance of these changes was determined by a Student's paired t-test.

The inter-relationship in the value or change in value of physiological variables was determined by Pearsons Correlation Test. Linear regression analysis was performed to determine the equation of a straight line in terms of $y = ax + b$. F-statistics are applied in section 4.5.2 to compare he slope and intersection of regression equations.

Statistical significance was taken at the traditional level of $P = 0.05$ or greater.

CHAPTER 3

RESULTS

3.1 SELECTION PROCEDURES

3.1.1 Initial Response

The newspaper article seeking subjects for this study engendered 215 telephone responses. The telephone questionnaires resulting from these respondents were examined and assessed on the basis of age, sex, physical activity, medical background and entry for the 1984 Glasgow Marathon.

Suitable individuals numbered 61 and these were recontacted. All 61 attended the initial meeting where the training program and testing procedures were outlined. None of the group wished to drop out at this stage despite the emphasis by the author on the demanding nature of the study and the motivation necessary to complete it.

3.1.2 Physical Activity Screening

The findings of the recreational and occupational physical activity questionnaire are summarized in Figures 5 and 6 respectively.

It is noticeable that the level of physical activity performed occupationally is relatively low with 42% of the group spending their entire working

FIGURE 5

Proportion of time spent in occupational physical activity prior to training

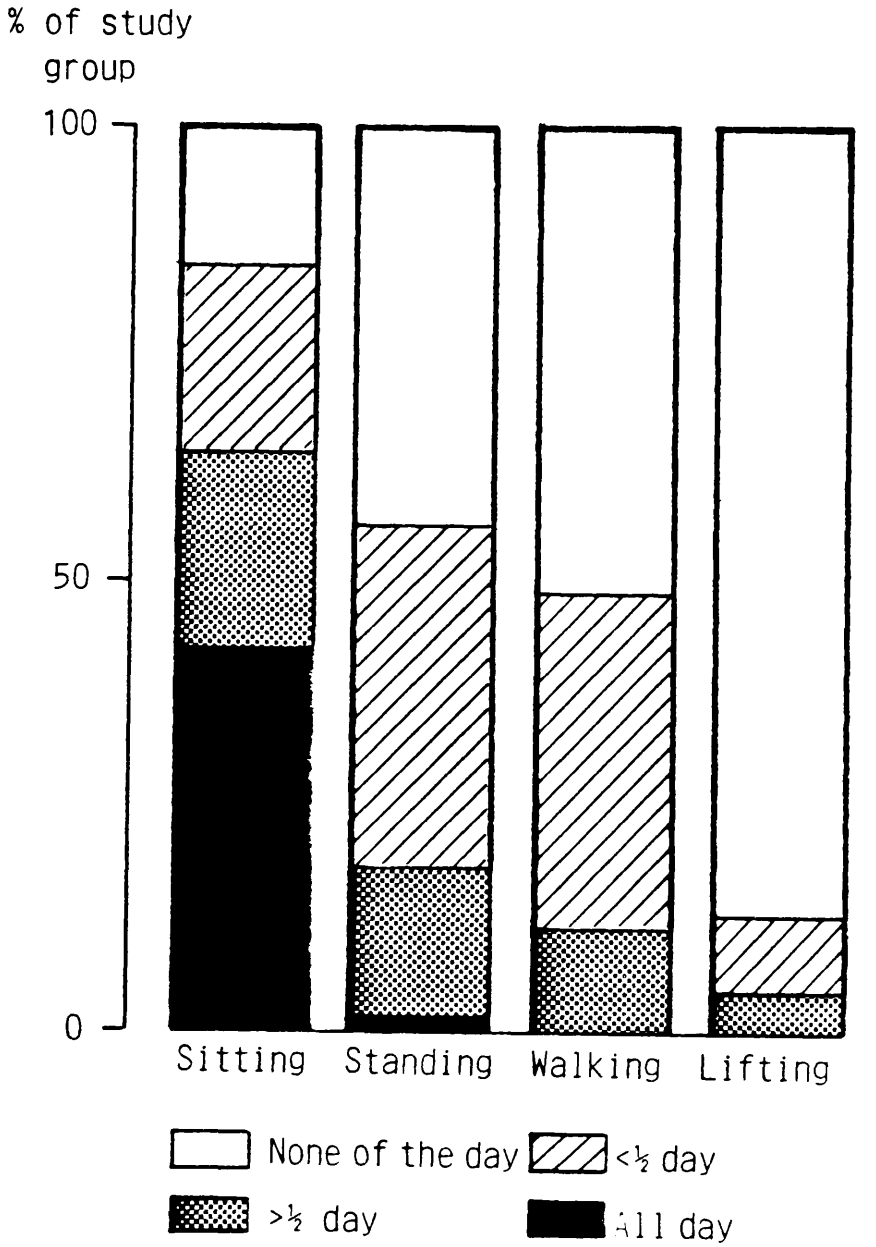
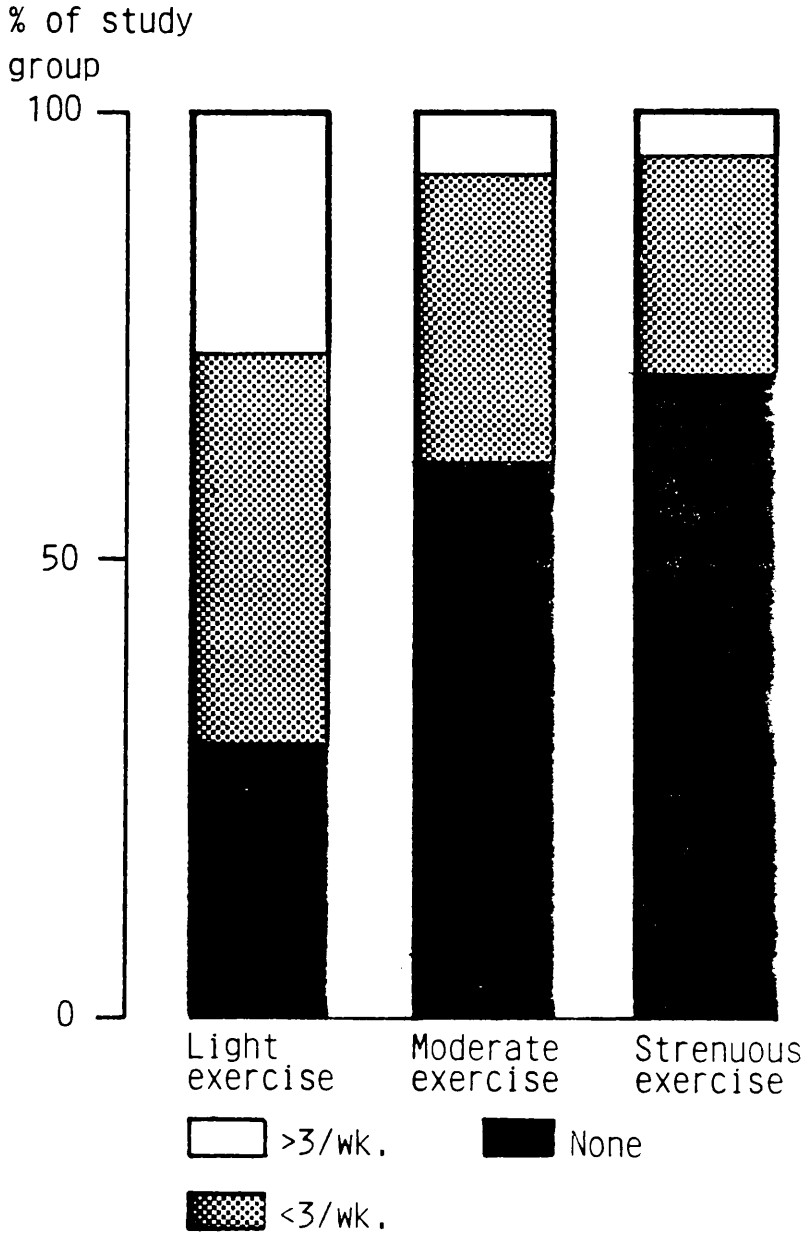


FIGURE 6

Proportion of time spent performing recreational physical activity prior to training



time in a sitting position, 44% and 49% of the group respectively having occupations that involved no walking or standing throughout the working day and only 7% of the group regularly involved in lifting at work.

As expected, a large proportion (70.5%) of the group participated in some form of light recreational activity, with more than half these individuals performing this activity 3 or more times per week. The most common of these light activities were golf, bowling and fishing. Many of the participants reported that this activity was seasonal and greatly reduced during the winter months. Only 34% and 5% respectively of the individuals in the group regularly participated in moderate or strenuous recreational activities on a weekly basis.

All of the selected study participants were classified as appropriately inactive from the telephone questionnaire. However, three individuals (5% of the total study group) admitted in the physical activity questionnaire to participating in strenuous activity, squash, more than 3 times per week. This level of activity was considered unsuitable for this study and these 3 individuals were excluded. A fourth individual, although recreationally inactive for the last 3 years, was also excluded when it was found that he had previously completed the marathon.

3.1.3 Medical Screening

The physician who performed the initial medical

examination excluded four individuals from the study on medical grounds.

Two of these individuals did not admit in the initial telephone enquiry to be being diagnosed hypertensives, controlled by anti-hypertensive drug treatment. The other two individuals were diagnosed hypertensive as their resting blood pressure was equal to or in excess of 160/95 mmHg.

All other individuals in the study were found to be in good medical health.

3.1.4 Final Study Group

As a result of the 8 rejections on the basis of medical or physical activity backgrounds, the number of individuals who finally entered the study and began the training program was 53.

3.2 TRAINING PROGRAM

3.2.1 Training Compliance

Of the original 53 subjects in the study, 39 (ie. 74%) completed the 30 weeks of exercise training. 5 participants withdrew during the first 15 weeks of training and 9 during the last 15 weeks. All 14 were questioned in detail to determine the reasons for withdrawal. The majority of these were either due to lack of time and/or loss of motivation. Only 5 individuals withdrew due to injury and only did so after seeking medical advice and generally after long periods of unsuccessful medical treatment.

The attrition rate of 26 percent over the 30 weeks of this study compares favourably with the attrition rates reported in previous training studies of a similar duration (35 to 45 percent) (Massie, 1971). Millesis and his colleagues (1976), reported attrition rates of 19.2, 29.6 and 46.4 percent for groups who trained over a 20 week period, 3 times per week for a duration of 15, 30 and 45 minutes per session respectively. The average training duration of this study was approxmately 60 minutes. In comparison to the figures of Millesis a consid erably higher attrition was to be expected in the present study.

The low attrition rate in this study depsite its demanding nature is probably reflective of a number of factors such as the motivating effect of preparing for a marathon, the comradeship of group runs and progressive nature of training presciption.

Although actual figures were not recorded, the majority of the study group suffered from some form of injury during training. In no case however was more than 2 weeks training lost. Lower limb orthopedic trauma is the most common reason for drop-out in previous training studies (Millesis et al, 1976; ACSM, 1978).

3.2.2 Assessment of Training

(a) Training distance, frequency, duration and speed.

Of the 39 participants who completed the marathon training program, 28 kept what were assessed to

maintain adequate training records. The remaining diaries were either incomplete or did not appear to accurately report training information.

The assessment of training was performed on these 28 accurately completed diaries. Summaries of training distance, time, frequency and speed are shown in Table 3. These values are plotted in Figure 8. This figure illustrates that during the final 15 weeks of training, the mean training time and frequency were considerably less than were prescribed in the training program.

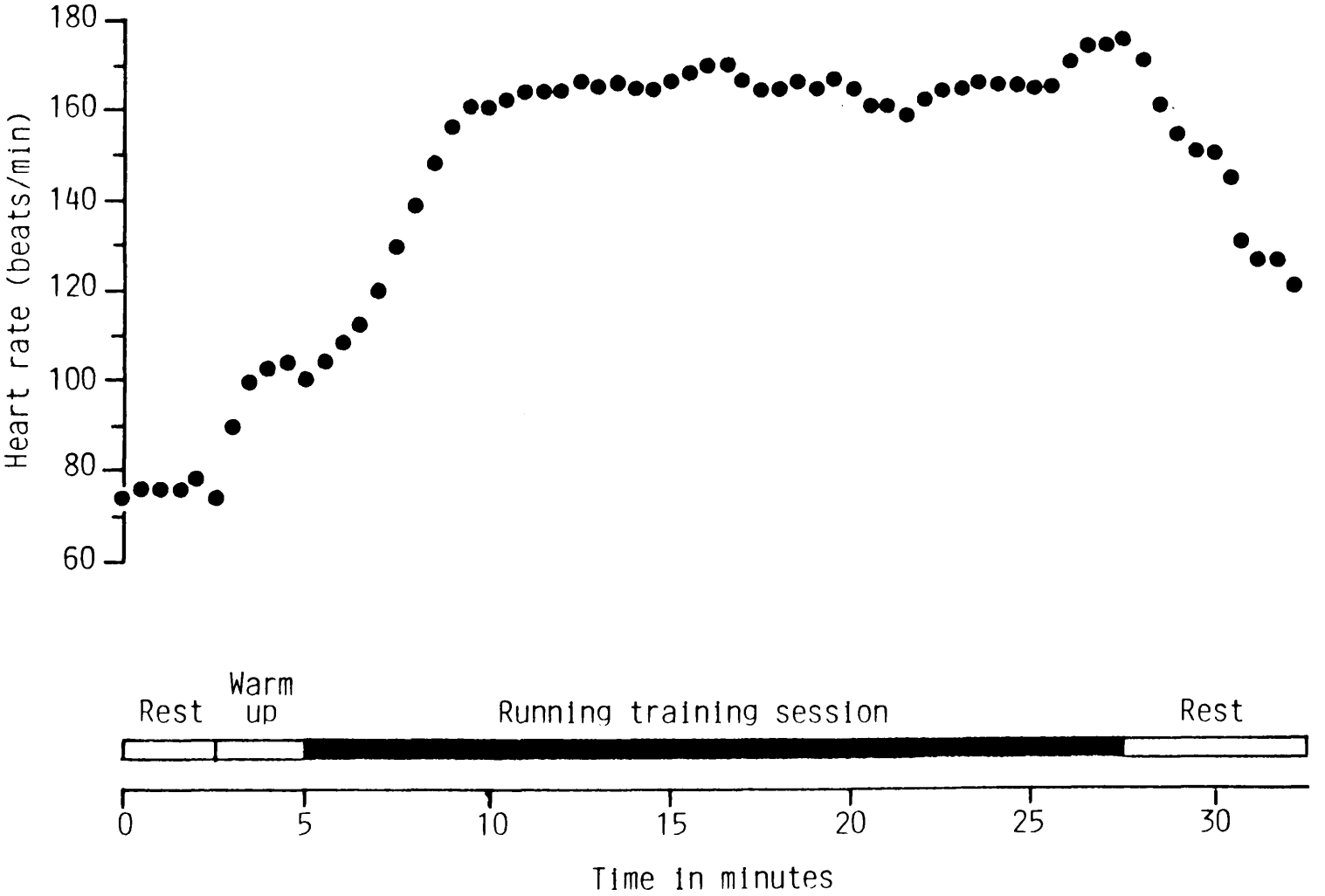
(b) Training Intensity

Training intensity, unlike training time and frequency was not prescribed in this study. However, the monitoring of training heart rate in a limited number of training sessions allowed examination of the training intensity in this small sample.

Training intensity was assessed in 8 individuals, 4 times at different stages of the 30 weeks. This monitoring was limited to runs of less than 80 minutes as this was the full extent of the monitor's recording memory. A heart rate recording obtained from a training run of one of the study participants is shown in Figure 7. This trace is typical of those obtained in that it demonstrates an immediate rise in heart rate at the onset of exercise rising to a value which appears to be steady state and is maintained throughout the training run. This value then steadily falls back to rest after exercise. The analysis of these records was performed

FIGURE 7

Heart rate response to a training session from a single individual



Comparison of actual training performed and training prescribed

FIGURE 8

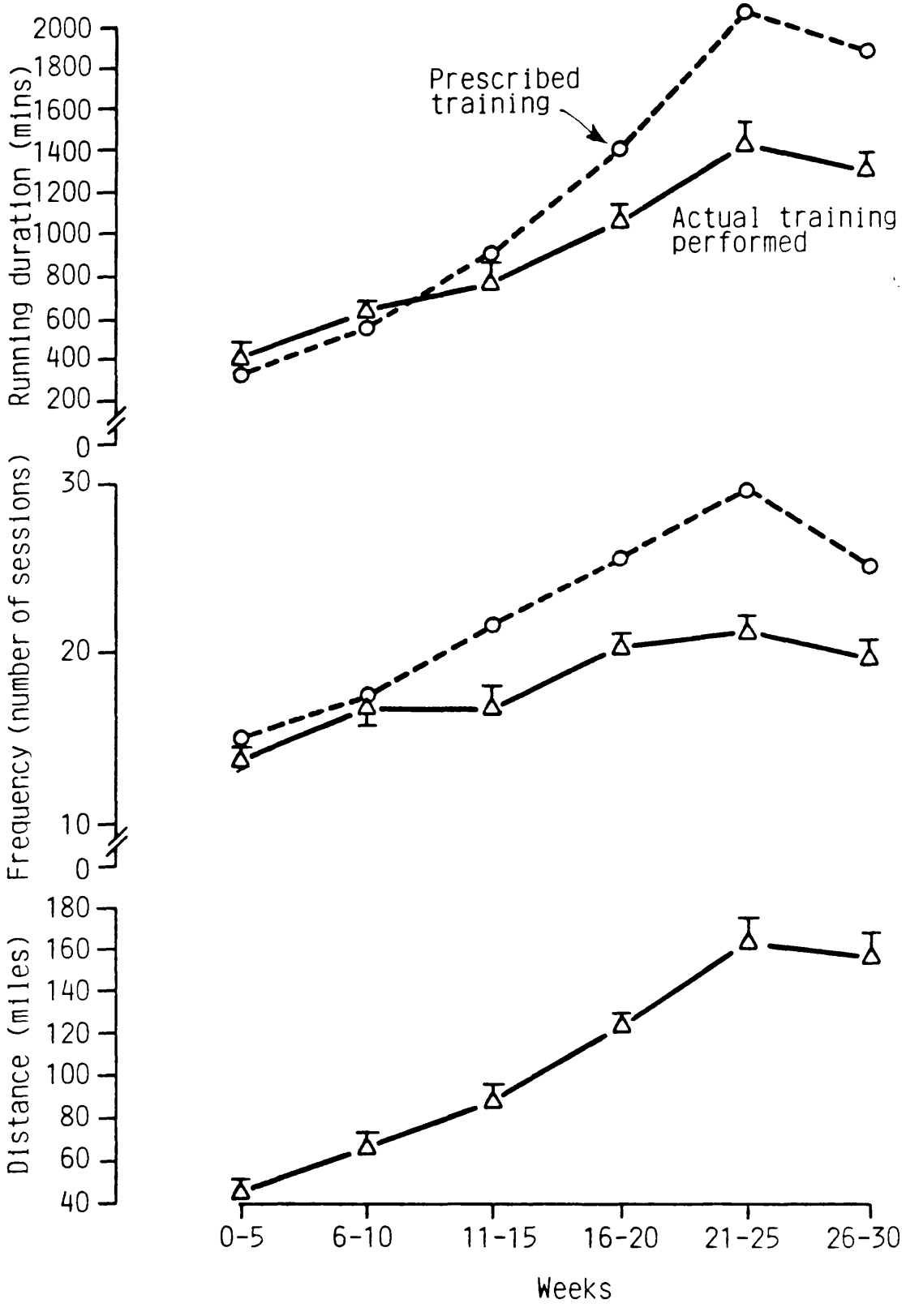


TABLE 3

Summary of training variables at various stages of training program

(means and SEM's)

Stage of training	Weeks 0-5	Weeks 6-10	Weeks 11-15	Weeks 16-20	Weeks 21-25	Weeks 25-30
Running duration (mins)	409 ± 37	616 ± 41	794 ± 58	1080 ± 61	1420 ± 93	1344 ± 76
Frequency (number of sessions)	13.9 ± 0.6	17.2 ± 0.9	17.1 ± 1.0	20.5 ± 0.8	21.5 ± 1.1	20.0 ± 1.0
Distance (miles)	45.3 ± 4.5	71.1 ± 5.2	91.2 ± 7.1	126.6 ± 5.7	164.3 ± 12.0	159.8 ± 10.0
Pace (mph)	6.6 ± 0.3	6.8 ± 0.1	6.9 ± 0.1	7.0 ± 0.1	7.0 ± 0.1	7.1 ± 0.8

by utilizing this steady state heart rate.

The steady state training heart rate, the value of these heart rates as expressed as percentage of the maximum heart rate and as a percentage of heart rate reserve are shown in Figure 9. An error of ± 10 beats/min reported with age predicted maximal heart rate (Astrand & Rodahl, 1978). Thus the highest recorded value of each subject that was attained during maximal bicycle ergometer testing was taken as maximal heart rate for intensity determination. Resting heart rate was taken as the value recorded in the subject's training diary for that week. Self-assessed heart rates were chosen instead of laboratory measured values. As this self-monitored value was taken prior to rising it was expected that it would be more reflective of true resting values.

The training heart rate of two individuals was assessed at various stages of the study (Table 4). Interestingly, this longitudinal data reveals that despite an increase in distance run, pace and time of running in these runs as training progressed, the subjects were able to maintain a constant training heart rate.

(c) Marathon Perfomance

Despite completion of the training program, 2 individuals did not run the marathon. This was due to ill-health and a family berevement.

From this training group 38 individuals started

Training heart rate and relative intensity at various stages of training program FIGURE 9

(Means and SEM's plotted)

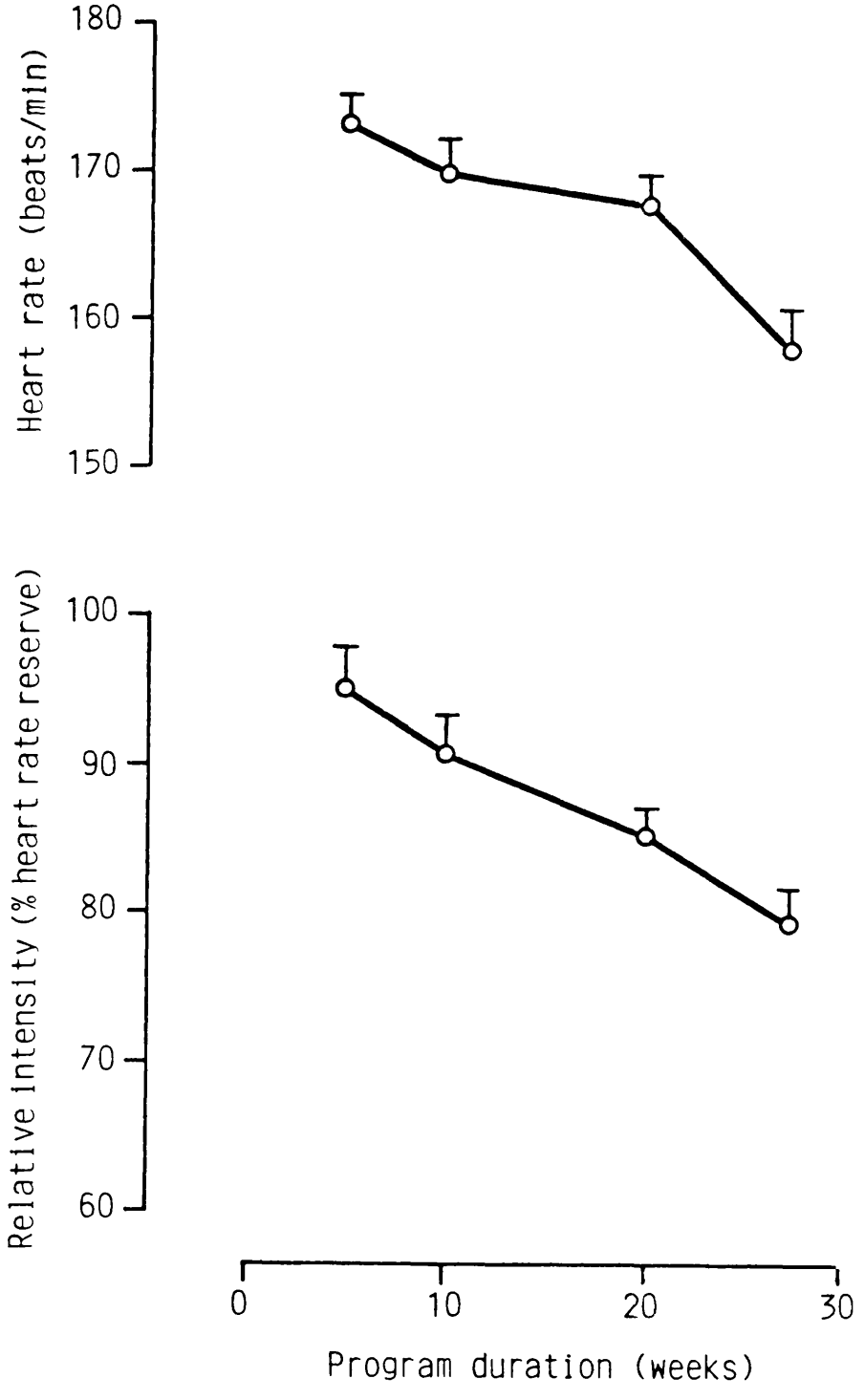


TABLE 4

Training performance of two individuals at various stages of training

Individual 1

Stage of training	Week 5	Week 10	Week 20	Week 27
Distance (miles)	2.5	4.0	4.0	5.0
Duration (mins)	25	32	30	42
Pace (mph)	6.0	7.5	8.0	7.1
Steady state heart rate (beats/min)	168	167	164	159
Relative intensity (% heart rate reserve)	98	97	95	91

Individual 2

Stage of training	Week 5	Week 10	Week 27
Distance (miles)	2.0	4.0	10.0
Duration (mins)	20	32	74
Pace (mph)	6.0	7.5	8.1
Steady state heart rate (beats/min)	170	165	160
Relative intensity (% heart rate reserve)	90	88	85

the 1984 Glasgow Marathon on 30/09/85. The 26.2 mile distance was completed by 37 of these. One individual dropped out midway through the event due to a knee injury. A mean marathon completion time for the present study subjects was 295 mins (SD: 35 mins) with a range of 202 to 327 mins.

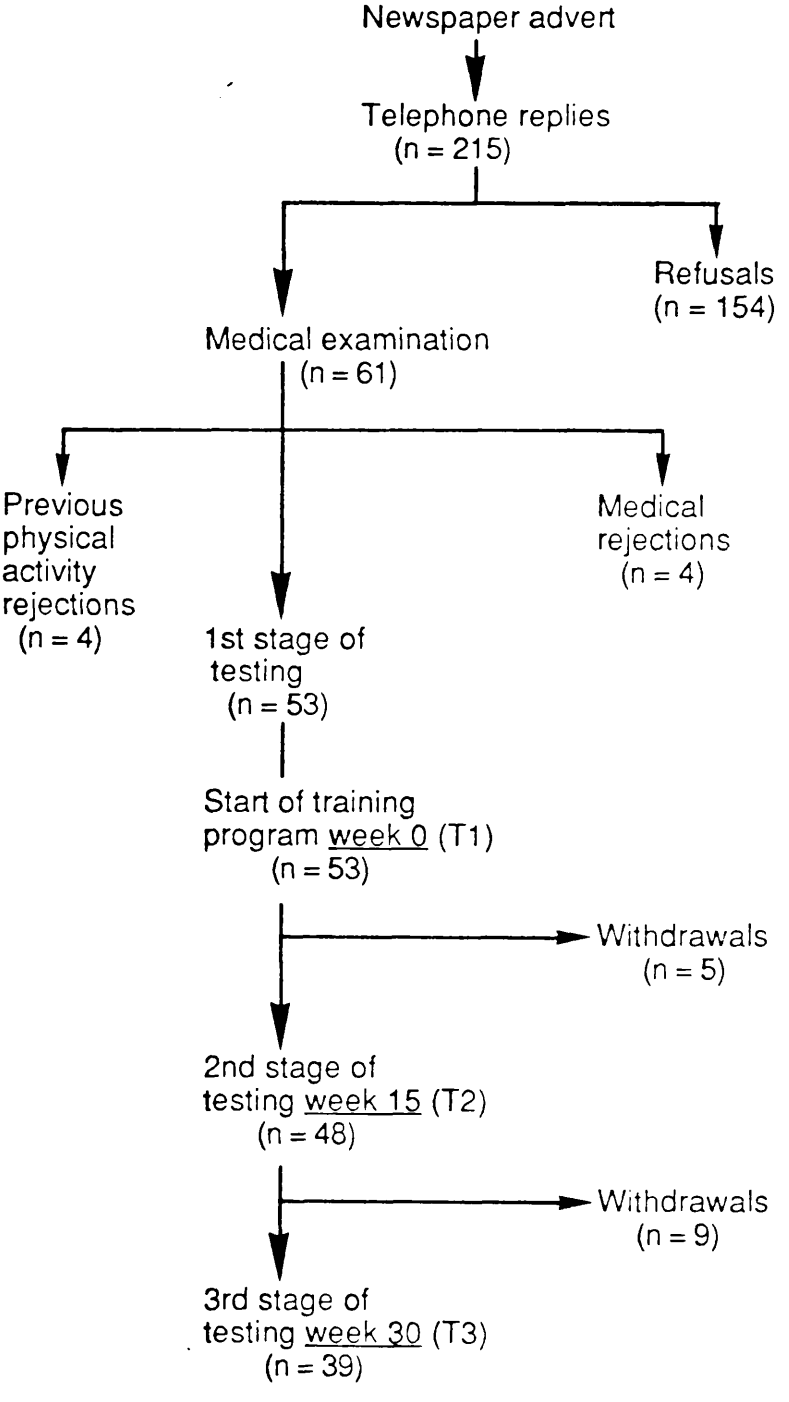
3.3 TESTING PROCEDURES

The 53 suitable volunteers obtained from the publicity and selection procedures was greater than initially expected. The maximal bicycle exercise test required the use of equipment normally used for everyday cardiological patient assessment. It was therefore only possible to perform this test on 35 individuals. All the other testing procedures were carried out on all 53 study participants. As was described above, during the course of the training program 14 study participants withdrew leaving 39 subjects who had completed the training program.

As a result of the above factors, 28 subjects performed a maximal exercise test at all 3 stages of testing, 20 attaining what were regarded as technically adequate RNVG scans. 39 individuals performed all the other testing procedures. The following are the findings of these tests based on these subject numbers. The number and relative timing of drop-outs is summarised in figure 10.

FIGURE 10

Summary of selection procedures and study numbers



3.3.2 Maximal Bicycle Ergometer Test

The following are a summary of the resting, submaximal and maximal exercise results obtained from the bicycle ergometer test at each stage of training.

(a) Pre-exercise

The values obtained after 15 minutes supine rest are summarized in Table 5. A significant reduction was observed in resting heart rate, systolic and diastolic blood pressure (all $P < 0.001$) after 30 weeks of training. In the case of heart rate and and systolic blood pressure this reduction was significantly greater ($P < 0.001$) during the first 15 weeks than the final 15 weeks.

No alteration was observed in resting blood lactate concentration with training.

Cardiac output and cardiac volume values were determined at rest while sitting upright on the bicycle ergometer. These values are summarized in Table 6. In agreemant with the supine rest, there was an average reduction in resting heart rate of some 12 beats/min with 30 weeks training ($P < 0.001$). A significant increase in end-diastolic volume and stroke volume (both $P < 0.001$) was observed. The majority of these changes appeared to take place during the first 15 weeks of training. There was no significant change in resting end-systolic volume, ejection fraction or cardiac output.

TABLE 5

Heart rate, blood pressure and blood lactate after supine rest

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Heart rate (beats/min)	68 ± 12	56 ± 8	57 ± 7	- 12 ±11 P<0.001	+ 1 ± 6 NS	- 11 ±12 P<0.001
Systolic blood pressure (mmHg)	133 ± 13	126 ± 12	122 ± 11	- 7 ±13 P<0.005	- 4 ±10 P<0.025	-11 ±12 P<0.001
Diastolic blood pressure (mmHg)	84 ± 9	82 ± 8	77 ± 10	- 2 ± 6 NS	- 5 ±15 P<0.05	- 7 ± 10 P<0.001
plasma lactate (mmol/l)	0.8 ± 0.5	1.0 ± 0.4	0.9 ± 0.3	+ 0.2 ± 0.6 NS	- 0.1 ± 0.5 NS	+ 0.1 ± 0.5 NS

TABLE 6

Cardiac parameters at rest sitting on bicyle ergometer prior to exercise

(means ± SD's)

Table 5

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Left ventricular end diastolic volume (ml)	144 ± 27	158 ± 30	160 ± 33	+ 14 ± 31 P<0.01	+ 2 ± 37 NS	+ 16 ± 22 P<0.001
Left ventricular end systolic volume (ml)	59 ± 18	59 ± 16	64 ± 21	0 ± 18 NS	+ 5 ± 18 NS	+ 5 ± 14 NS
Ejection fraction %	59 ± 8	62 ± 7	60 ± 8	+ 3 ± 9 NS	- 2 ± 6 NS	+ 1 ± 6 NS
Stroke volume (ml)	85 ± 13	99 ± 22	96 ± 18	+ 14 ± 21 P<0.001	- 3 ± 25 NS	- 11 ± 14 P<0.001
Heart rate (beats/min)	78 ± 14	64 ± 11	66 ± 8	- 14 ± 7 P<0.001	_ 2 ± 8 NS	- 12 ± 13 P<0.001
Cardiac output (litres/min)	6.6 ± 1.2	6.2 ± 1.3	6.4 ± 1.8	- 0.4 ± 1.4 NS	+ 0.2 ± 1.0 NS	- 0.2 ± 1.6 NS

(b) Submaximal exercise

In this section the value of various parameters during submaximal bicycle ergometer exercise are quoted. The number of data points at higher workloads is not 39 due to the inability of some individuals to reach this workload, achieving peak effort at a lower workload. Although the majority of individuals increased the level to which they were able to exercise maximally over the 30 weeks of training, the data reported here is the paired data on the basis of the pre-training test numbers. This was done for statistical purposes (see section 2.6.5).

(i) Oxygen Uptake

The change in absolute and weight corrected oxygen uptake with workload are summarized in Tables 7 and 8 and Figures 11 and 12 respectively.

Absolute and weight corrected oxygen uptake versus workload appeared to a relatively linear at all stages of training.

A significant reduction was observed in absolute and relative oxygen uptake at virtually all submaximal workloads after 30 weeks training (all $P < 0.001$). This reduction when expressed corrected for weight, appeared to occur mainly in the first 15 weeks of training.

(ii) Heart rate

The relationship of heart rate with submaximal workload and the change in this relationship with training is shown in Table 9 and Figure 13. At all

TABLE 7

Absolute oxygen uptake (l/min) during submaximal bicycle ergometer exercise at each stage of training

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n=39)	1.32 ± 0.27	1.14 ± 0.21	1.22 ± 0.09	- 0.18 ± 0.36 P<0.01	+ 0.08 ± 0.28 NS	- 0.10 ± 0.29 P<0.05
100 watts (n=39)	1.62 ± 0.22	1.48 ± 0.18	1.46 ± 0.13	- 0.14 ± 0.26 P<0.01	- 0.02 ± 0.17 NS	- 0.16 ± 0.22 P<0.001
125 watts (n=39)	1.96 ± 0.21	1.86 ± 0.18	1.74 ± 0.16	- 0.10 ± 0.26 P<0.01	- 0.12 ± 0.20 P<0.001	- 0.22 ± 0.20 P<0.001
150 watts (n=36)	2.25 ± 0.18	2.20 ± 0.16	1.98 ± 0.16	- 0.05 ± 0.23 NS	- 0.22 ± 0.20 P<0.001	- 0.27 ± 0.20 P<0.001
175 watts (n=31)	2.53 ± 0.19	2.49 ± 0.14	2.32 ± 0.14	- 0.04 ± 0.23 NS	- 0.17 ± 0.20 P<0.001	- 0.21 ± 0.26 P<0.001
200 watts (n=25)	2.97 ± 0.21	2.84 ± 0.17	2.64 ± 0.11	- 0.13 ± 0.28 P<0.01	- 0.20 ± 0.22 P<0.001	- 0.33 ± 0.21 P<0.001

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 11

Absolute oxygen uptake during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)

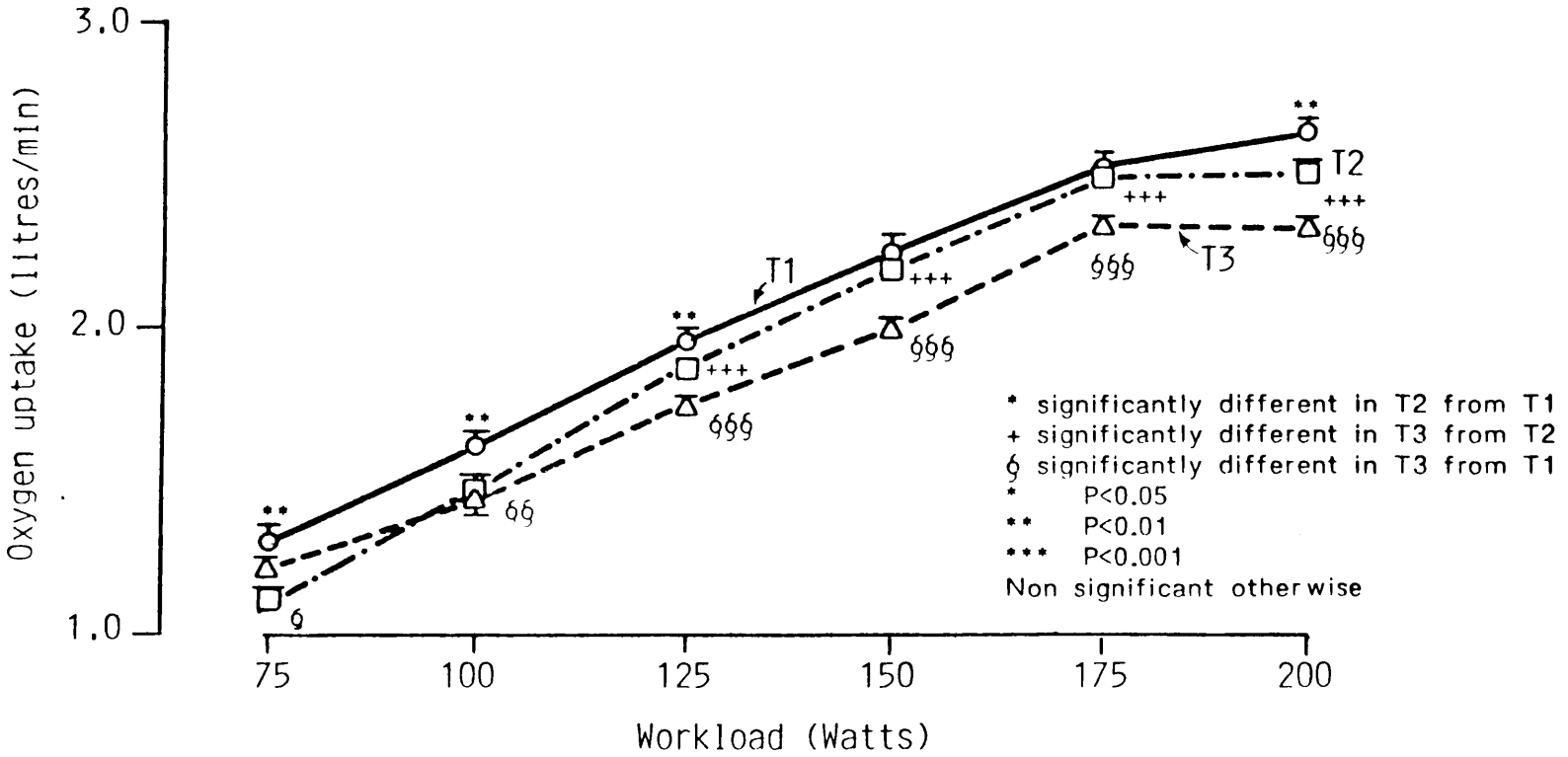


TABLE 8

Weight corrected oxygen uptake (ml/kg/min) during submaximal bicycle ergometer exercise at each stage of training

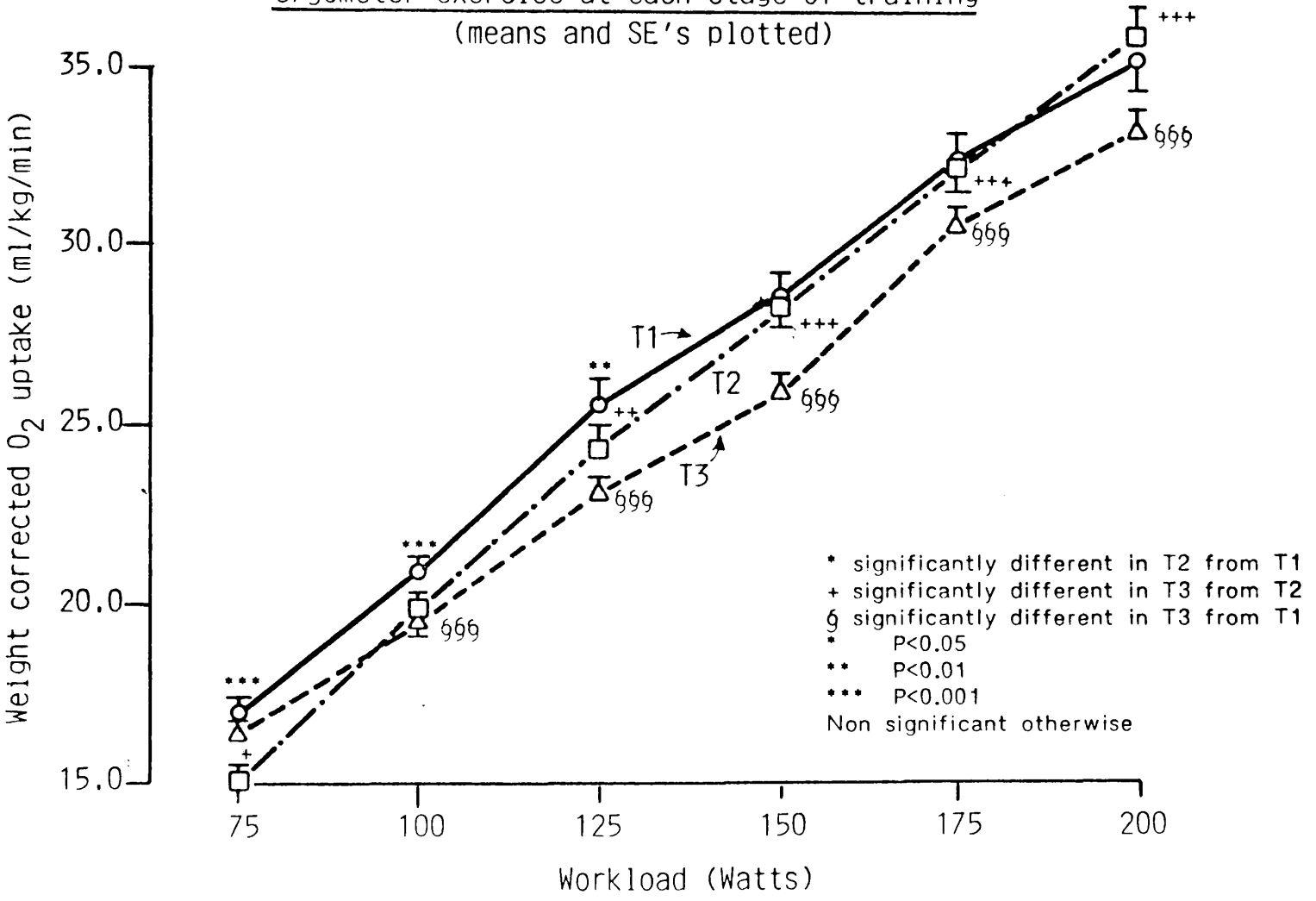
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n=39)	17.0 ± 3.7	14.9 ± 2.4	16.4 ± 2.5	- 2.2 ± 3.7 P<0.001	+ 1.5 ± 4.4 P<0.05	- 0.6 ± 3.0 NS
100 watts (n=39)	21.1 ± 3.4	19.4 ± 3.2	19.2 ± 2.4	- 1.7 ± 2.5 P<0.001	- 0.2 ± 2.6 NS	- 1.9 ± 2.5 P<0.001
125 watts (n=39)	25.6 ± 4.3	24.3 ± 3.8	23.1 ± 3.2	- 1.3 ± 3.2 P<0.01	- 1.2 ± 2.8 P<0.01	- 2.5 ± 3.0 P<0.001
150 watts (n=36)	28.6 ± 3.8	28.2 ± 3.6	26.0 ± 3.0	- 0.4 ± 2.5 NS	- 2.2 ± 2.4 P<0.001	- 2.6 ± 3.5 P<0.001
175 watts (n=31)	32.3 ± 4.3	32.1 ± 4.1	30.5 ± 4.1	- 0.2 ± 2.6 NS	- 1.6 ± 2.7 P<0.001	- 1.8 ± 2.8 P<0.001
200 watts (n=25)	35.1 ± 5.0	35.9 ± 6.2	33.2 ± 4.2	+ 0.8 ± 4.1 NS	- 2.7 ± 3.1 P<0.001	- 1.9 ± 2.4 P<0.001

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 12

Weight corrected oxygen uptake during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)



stages of training, heart rate versus bicycle workload appeared to be relatively linear.

The slope of the heart rate - workload relationship did not appear to change significantly with training, remaining at approximatly an increase of 12 beats/min for each 25 Watt increase in workload. However the position of the heart rate - workload line appeared to change with with training. A fall in heart rate of some 16 to 22 beats/min (10 to 16%) was observed at each given submaximal workload. This decrease in heart rate appeared to occur during the first 15 weeks of training with little or no change in the final 15 weeks.

(iii) Pulmonary Ventilation

The relationship of pulmonary ventilation versus submaximal bicycle workload and the changes in this relationship with training are summarized in Table 10 and Figure 14.

The relationship between submaximal workload and ventilation was again found to to be fairly linear one. At higher workloads the gradient of this line increased from about 7.5 l/min to about 15 l/min of ventilation per 25 Watts.

Although this pattern of ventilation versus workload did not appear to change with training, a reduction was observed in the value ventilation at a given submaximal workload. In 4 out of the 6 submaximal loads tested, this decrease was highly significant ($P <$

TABLE 9

Heart rate (beats/min) during submaximal bicycle ergometer exercise at each stage of training

(means and SD's)

Table 6

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n =39)	120 ± 22	106 ± 17	104 ± 17	- 14 ± 16 P<0.001	- 2 ± 10 NS	- 16 ± 12 P<0.001
100 watts (n=39)	139 ± 22	119 ± 18	117 ± 18	- 20 ± 15 P<0.001	- 2 ± 10 NS	- 22 ± 12 P<0.001
125 watts (n = 39)	152 ± 20	132 ± 17	133 ± 18	- 20 ± 15 P<0.001	+ 1 ± 10 NS	- 19 ± 12 P<0.001
150 watts (n=36)	160 ± 17	143 ± 14	144 ± 15	- 17 ± 17 P<0.001	+ 1 ± 10 NS	- 16 ± 14 P<0.001
175 watts (n-31)	170 ± 16	152 ± 13	152 ± 13	- 18 ± 12 P<0.001	0 ± 8 NS	- 18 ± 12 P<0.001
200 watts (n=25)	175 ± 15	161 ± 14	161 ± 12	- 14 ± 8 P<0.001	0 ± 9 NS	- 14 ± 10 P<0.001

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 13

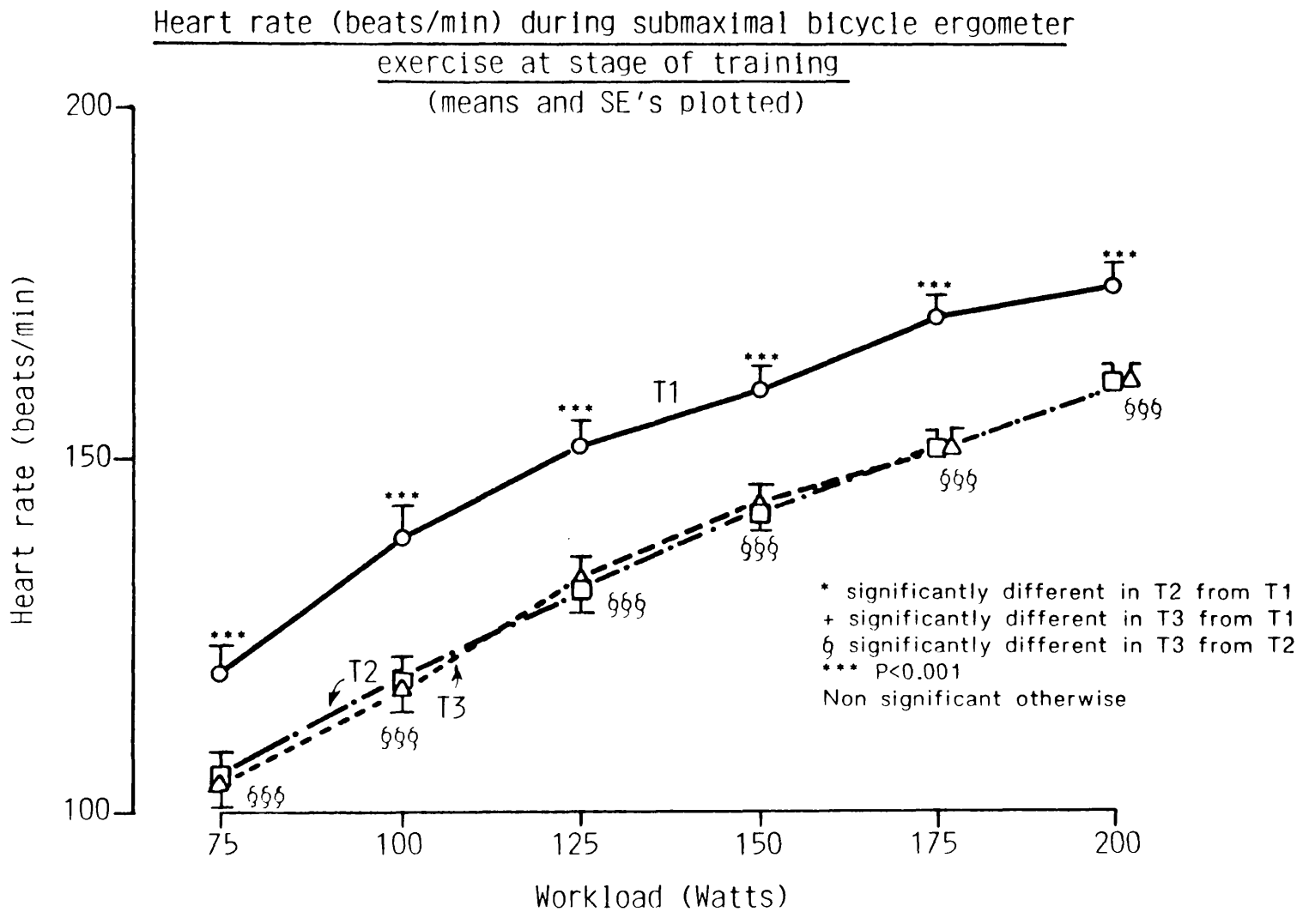

Heart rate (beats/min) during submaximal bicycle ergometer exercise at stage of training
(means and SE's plotted)
Heart rate (beats/min)
200
150
100
T1
T2
T3
***
ϕϕϕ
75
100
125
150
175
200
Workload (Watts)
* significantly different in T2 from T1
+ significantly different in T3 from T1
ϕ significantly different in T3 from T2
*** P<0.001
Non significant otherwise

TABLE 10

Pulmonary ventilation (l/min) during submaximal bicycle ergometer exercise at each stage of training

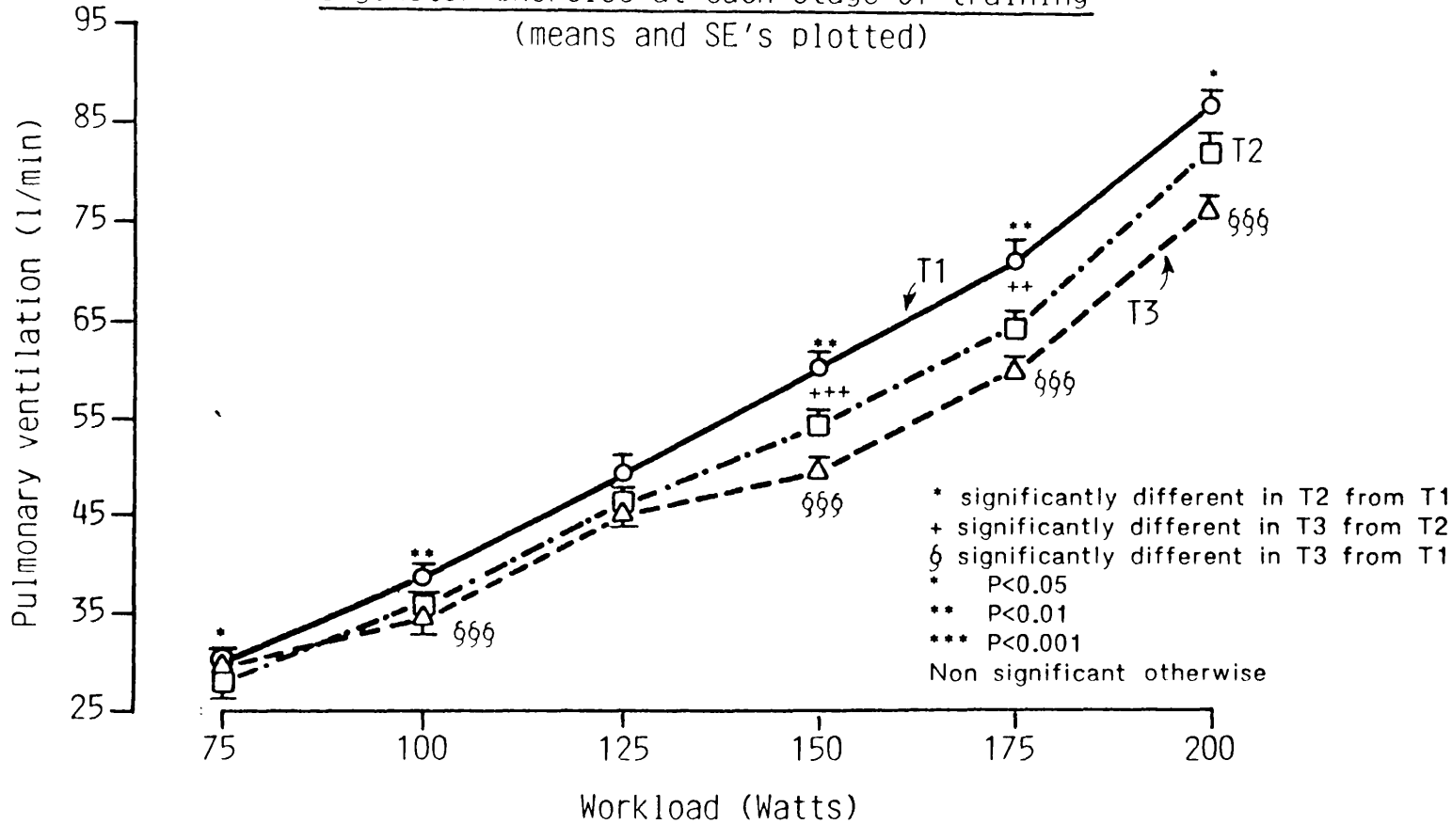
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT2-T3
75 watts (n=39)	30.3 ± 7.7	27.8 ± 6.3	29.6 ± 6.7	- 2.5 ± 7.4 P<0.05	+ 1.8 ± 8.0 NS	- 0.7 ± 5.8 NS
100 watts (n=39)	39.1 ± 9.5	36.1 ± 7.5	34.8 ± 6.9	- 3.0 ± 7.6 P<0.01	- 1.3 ± 9.3 NS	- 4.3 ± 7.0 P<0.001
125 watts (n=30)	49.2 ± 15.4	46.9 ± 12.8	45.3 ± 9.5	- 2.3 ± 13.4 NS	- 1.6 ± 8.4 NS	- 3.9 ± 12.6 NS
150 watts (n=36)	60.5 ± 16.6	53.7 ± 11.4	49.5 ± 8.7	- 6.8 ± 12.1 P<0.01	- 4.2 ± 6.6 P<0.001	- 11.0 ± 12.9 P<0.001
175 watts (n=31)	73.0 ± 20.8	64.8 ± 13.7	60.6 ± 10.7	- 8.2 ± 15.0 P<0.01	- 4.2 ± 10.5 P<0.01	- 12.5 ± 14.1 P<0.001
200 watts (n=21)	87.7 ± 13.7	82.6 ± 17.5	78.9 ± 13.4	- 5.1 ± 13.9 P<0.05	- 3.7 ± 13.9 NS	- 8.8 ± 10.9 P<0.001

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 14

Pulmonary ventilation (l/min) during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)



0.001). The magnitude of this reduction, both absolute and relative, appeared to be greater at higher workloads.

(iv) Plasma Lactate

The change in submaximal plasma lactate concentration is shown in Table 11 and Figure 15. The relationship of lactate with increasing workload was curvilinear, the increase in lactate concentration becoming greater at higher workloads.

The pattern of change in the plasma lactate versus workload with training was not consistent over the 30 weeks of training. During initial 15 weeks of training, a reduction in lactate concentration at a number of submaximal workloads was observed, the majority of these being statistically significant ($0.05 < P < 0.001$). However, during the later 15 weeks of training this pattern of chage was apparently reversed and the lactate concentration at a given workload increased and moved to that observed prior training values. As the result of these opposite directions of change in lactate levels at the initial and final stages of training, little or no change was observed overall.

A significant ($P < 0.001$) increase in the workload at which 4mmol/l lactate (OBLA.Watts) was achieved occured after 15 weeks training (Table 12). During the final 15 weeks, this direction of change was reversed although the workload at 4 mmol/l was slightly greater

TABLE 11

plasma lactate concentration (mmol/l) during submaximal bicycle ergometer exercise at each stage of training

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n=39)	1.44 ± 0.48	1.44 ± 0.86	1.64 ± 0.45	0 ± 0.76 NS	+ 0.20 ± 0.77 NS	+ 0.20 ± 0.78 NS
100 watts (n=39)	2.60 ± 1.65	2.18 ± 1.28	2.58 ± 1.21	- 0.42 ± 1.06 P<0.01	+ 0.40 ± 1.06 P<0.01	- 0.02 ± 1.05 NS
125 watts (n=39)	3.89 ± 2.27	3.03 ± 1.35	3.77 ± 1.90	- 0.86 ± 1.54 P<0.01	+ 0.74 ± 1.32 P<0.01	- 0.12 ± 1.76 NS
150 watts (n=36)	4.91 ± 1.80	3.83 ± 1.53	4.59 ± 2.20	- 1.08 ± 1.91 P<0.01	+ 0.76 ± 1.56 P<0.01	- 0.32 ± 2.71 NS
175 watts (n=31)	6.53 ± 2.38	5.18 ± 2.34	5.84 ± 2.63	- 1.36 ± 1.76 P<0.001	+ 0.68 ± 1.92 P<0.05	- 0.69 ± 2.95 NS
200 watts (n=21)	7.53 ± 2.48	7.28 ± 2.82	6.89 ± 2.75	- 0.25 ± 4.77 NS	- 0.64 ± 3.89 NS	- 0.39 ± 2.95 NS

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 15

Blood lactate during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)

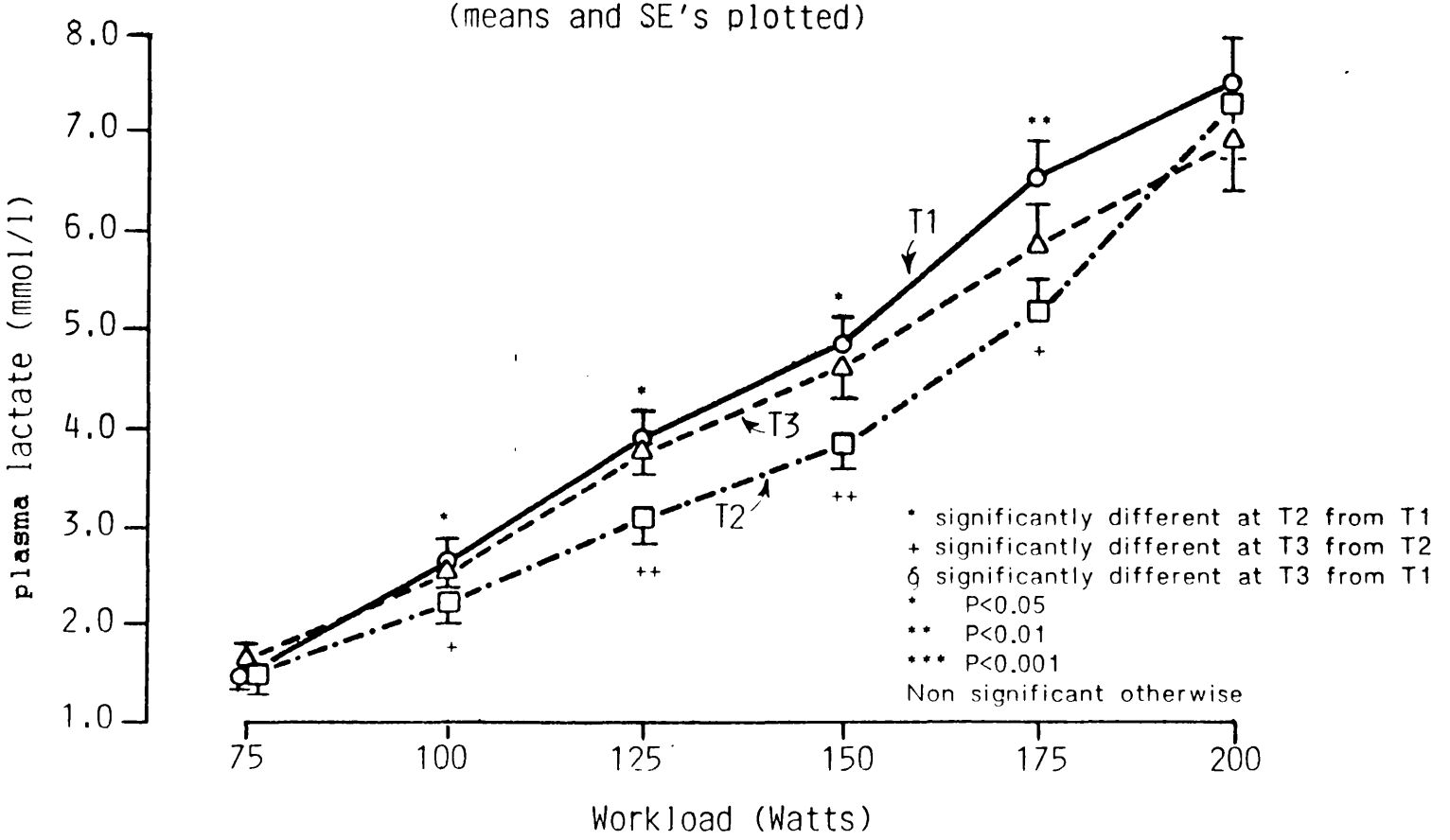


TABLE 12

Bicycle ergometer workload, oxygen uptake and relative work intensity (% VO_2 max)) at onset of blood lactate accumulation (OBLA) 4 mmol/l

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Work load (watts)	126 ± 30	152 ± 28	132 ± 34	+ 26 ± 25 P<0.001	- 20 ± 22 P<0.001	+ 6 ± 15 P<0.05
Oxygen uptake (ml/kg/min)	25.5 ± 5.3	29.0 ± 5.2	26.9 ± 4.0	+ 3.5 ± 5.4 P<0.001	- 2.6 ± 4.1 P<0.001	+ 0.9 ± 4.0 NS
Relative intensity (% VO_2 max)	75 ± 12	74 ± 11	69 ± 10	- 1 ± 13 NS	- 5 ± 14 P<0.001	- 6 ± 17 P<0.001

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

(NS) after 30 weeks than before training.

The change in lactate versus weight corrected oxygen uptake with training is shown in Figure 14. As with the workload plot, this relationship appeared to be curvilinear, there being a greater increase in lactate levels at higher values of oxygen uptake. In addition the change in lactate versus oxygen uptake was reversed with training. There being a reduction in lactate concentration at a given oxygen uptake after 15 weeks training, while after 30 weeks training this decrease was reversed.

The oxygen uptake at which a lactate concentration of 4mmol/l (OBLA.VO2) increased after 15 weeks but dcreased to a value of oxygen uptake less than pre-training after 30 weeks (Table 11). These changes were statistically significant ($P < 0.005$ and $P < 0.001$ respectively).

The change in plasma lactate versus relative proportion of aerobic capacity (ie. %VO2max) is shown in Figure 15. Once again this plot was apperently curvilinear at all stages of training. Interestingly the %VO2max at which a standard value of lactate was achieved appeared to fall with training. This reduction was significant ($P < 0.001$) at 4 mmol/l (OBLA.%VO2max) after 15 and 30 weeks weeks training (Table 12).

(v) Respiratory Exchange Ratio

The relationship between the respiratory exchange ratio and submaximal bicycle ergometer workload at the

TABLE 13

Respiratory exchange ratio (RER) during submaximal bicycle ergometer exercise at each stage of training

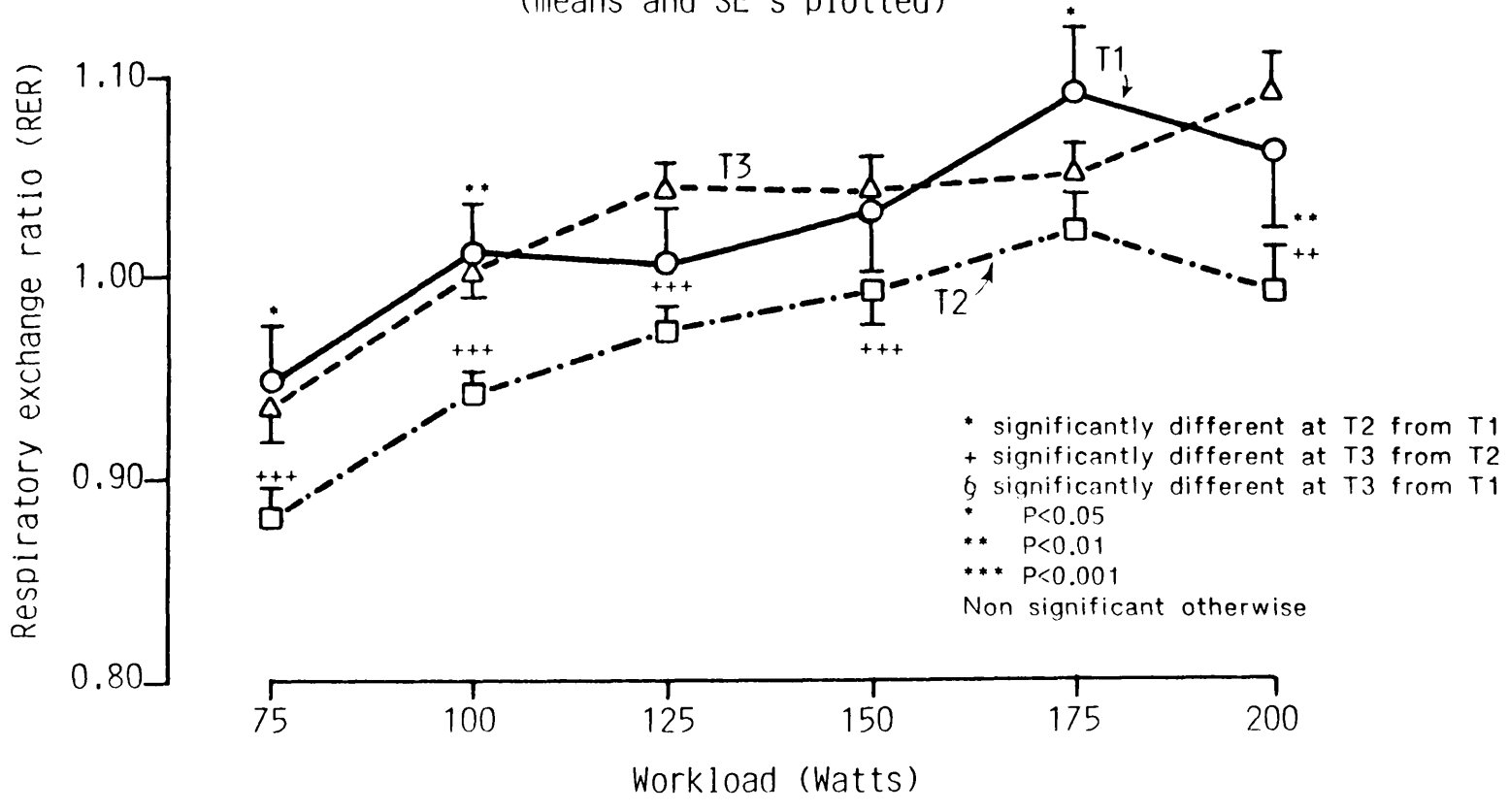
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n=39)	0.95 ± 0.16	0.88 ± 0.10	0.96 ± 0.12	- 0.07 ± 0.19 P<0.05	+ 0.08 ± 0.11 P<0.001	+ 0.01 ± 0.15 NS
100 watts (n=39)	1.02 ± 0.15	0.94 ± 0.08	1.00 ± 0.08	- 0.08 ± 0.14 P<0.001	+ 0.06 ± 0.08 P<0.001	+ 0.02 ± 0.14 NS
125 watts (n=39)	1.01 ± 0.17	0.97 ± 0.08	1.04 ± 0.08	- 0.04 ± 0.17 NS	+ 0.07 ± 0.10 P<0.001	+ 0.03 ± 0.16 NS
150 watts (n=36)	1.03 ± 0.18	0.98 ± 0.10	1.04 ± 0.08	- 0.05 ± 0.17 NS	+ 0.06 ± 0.10 P<0.001	+ 0.01 ± 0.18 NS
175 watts (n=31)	1.09 ± 0.18	1.02 ± 0.08	1.05 ± 0.06	- 0.07 ± 0.18 P<0.05	+ 0.03 ± 0.19 NS	- 0.04 ± 0.16 NS
200 watts (n=21)	1.06 ± 0.20	0.99 ± 0.08	1.09 ± 0.06	- 0.07 ± 0.18 P<0.01	+ 0.10 ± 0.19 P<0.01	+ 0.03 ± 0.19 NS

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 16

Respiratory exchange ratio during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)



various stages of training is shown in Table 13 and Figure 16.

Respiratory exchange ratio increased progressively with increasing workload at all stages of training. After 15 weeks of training there was a reduction ($0.05 < P < 0.005$) in the ratio at a standard workload. During the final 15 weeks of training this decrease was reversed ($0.005 < P < 0.001$) and respiratory exchange ratio values returned to pre-training levels.

(vi) Systolic Blood Pressure

The relationship of systolic blood pressure and workload and the change in this relationship with training is shown in Table 14 and Figure 17.

A steady increase in systolic pressure with increasing workload was observed at all stages of training. A significant decrease ($0.005 < P < 0.001$) in pressure at standard workload occured with training. On average, over the submaximal range, the reduction in systolic pressure was the same during the initial and final 15 weeks. The magnitude of pressure fall, both in absolute and relative terms, was greater during the lower workloads.

(c) Maximal Exercise

(i) Time to peak exercise

The average values of maximal workload achieved and the time to this maximal workload are shown in Table 15.

A highly significant ($P < 0.001$) increase in

TABLE 14

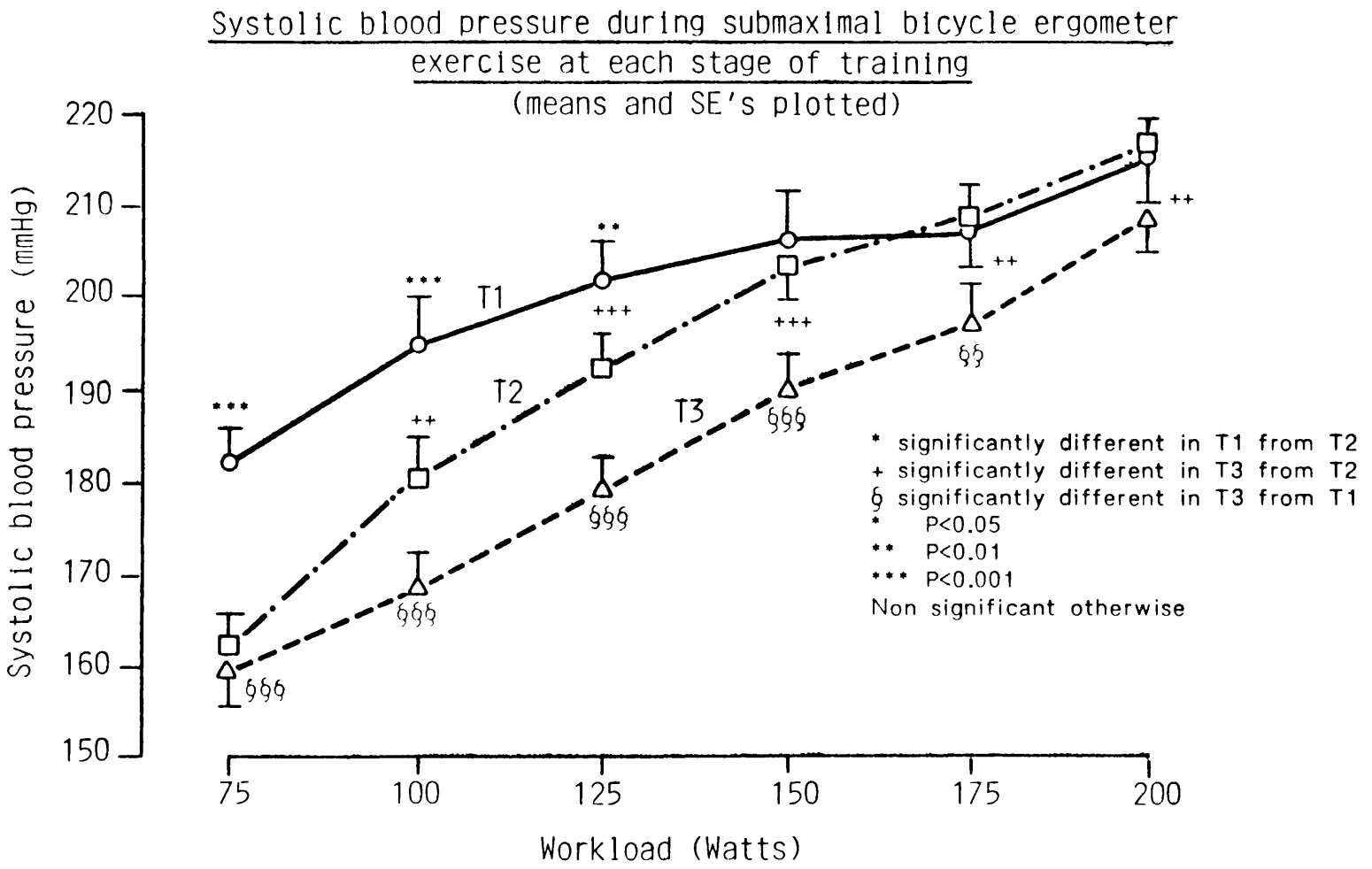
Systolic blood pressure (mmHg) during submaximal bicycle ergometer exercise at each stage of training

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
75 watts (n=39)	182 ± 22	162 ± 24	159 ± 21	- 20 ± 16 P<0.001	- 3 ± 23 NS	- 23 ± 22 P<0.001
100 watts (n=39)	195 ± 30	180 ± 23	168 ± 21	- 15 ± 21 P<0.001	- 12 ± 21 P<0.01	- 27 ± 27 P<0.001
125 watts (n=39)	202 ± 26	192 ± 22	179 ± 16	- 10 ± 21 P<0.01	- 13 ± 19 P<0.001	- 23 ± 22 P<0.001
150 watts (n=36)	206 ± 28	203 ± 21	190 ± 20	- 3 ± 24 NS	- 13 ± 21 P<0.001	- 16 ± 22 P<0.001
175 watts (n=31)	207 ± 24	208 ± 21	197 ± 20	+ 1 ± 24 NS	- 11 ± 22 P<0.01	- 10 ± 23 P<0.01
200 watts (n=21)	215 ± 27	216 ± 11	208 ± 16	+ 1 ± 22 NS	- 8 ± 11 P<0.01	- 7 ± 21 NS

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

FIGURE 17


Systolic blood pressure during submaximal bicycle ergometer exercise at each stage of training
(means and SE's plotted)
Systolic blood pressure (mmHg)
220
210
200
190
180
170
160
150
T1
T2
T3
75
100
125
150
175
200
Workload (Watts)
* significantly different in T1 from T2
+ significantly different in T3 from T2
§ significantly different in T3 from T1
* P<0.05
** P<0.01
*** P<0.001
Non significant otherwise

MAXIMAL BICYCLE ERGOMETER WORKLOAD AND EXERCISE AT EACH STAGE OF TRAINING
(means and SD's)

TABLE 15

	T1	T2	T3	⊿ T1-T2	⊿ T2-T3	⊿ T1-T3
Maximal Workload (watts)	180	209	212	+ 29	+ 3	+ 32
	± 36	± 32	+ 28	± 26	± 10	± 24
				P<0.001	NS	P<0.001
Exercise Time (mins)	18.2	20.7	21.2	+ 2.5	+ 0.5	+ 3.0
	± 5.0	± 4.7	± 4.2	± 1.5	± 1.5	± 1.8
				P<0.001	NS	P<0.001

T1 - prior to training

T2 - after 15 weeks training

T3 - after 30 we3eks training

workload and exercise time was observed with training. This increase occured during the initial 15 weeks of training, with no significant change during the later 15 weeks.

(ii) Oxygen Uptake (VO2max)

The training changes in absolute and weight-corrected VO2max for the group are summarized in Table 16.

Absolute and weight corrected VO2max increased significantly ($P < 0.05$, $P < 0.001$ respectively) after 30 weeks training. This change in VO2max appeared to occur during the first 15 weeks of training (both $P < 0.001$), with no significant change during the final 15 weeks of training.

(iii) Heart rate

The changes in maximal heart rate with training are shown in Table 16.

A significant reduction ($P < 0.01$) in heart rate was observed after the initial 15 weeks and the final 30 weeks of training. No significant change was observed during the final 15 weeks.

(iv) Pulmonary Ventilation

The changes in maximal ventilation with training are summarized in Table 16.

Maximal ventilation increased substantially over the 30 weeks of training, although this change was not statistically significant. This increase in ventilation was entirely attributable to the increase in the first

15 weeks of training ($P < 0.01$). No significant change was observed during the final 15 weeks of training.

(v) Respiratory Exchange Ratio

The changes in maximal respiratory exchange ratio with training are summarized in Table 16.

No significant change was observed in the ratio over the 30 weeks of training. However, a decrease was observed after 15 weeks. This change was reversed during the final 15 weeks of training ($P < 0.05$).

(vi) Plasma lactate

The changes in maximal plasma lactate with training are summarized in Table 16.

An increase in the peak lactate was noted at all stages of training. This increase was significant during the final 15 weeks ($P < 0.05$) and overall 30 weeks ($P < 0.001$) of training.

(vii) Systolic Blood Pressure

The changes in maximal systolic blood presuure with training are summarized in Table 16.

Although the mean value of peak systolic pressure increased after 15 weeks training, no significant change in was found over the 30 weeks.

(viii) Cardiac Volumes

The changes in maximal end-diastolic volume, end-systolic volume, ejection fraction, stroke volume and cardiac output are shown in Table 17.

End-diastolic volume was found to increase significantly ($P < 0.025$) over the 30 weeks of

MAXIMAL BICYCLE ERGOMETER CARDIORESPIRATORY AND METABOLIC PARAMETERS AT EACH STAGE OF TRAINING
(means and SD's)

TABLE 16

	T1	T2	T3	Δ T1-T2	Δ T2-T3	Δ T1-T3
Heart Rate (beats/min.)	177 ± 13	169 ± 11	172 ± 10	- 8 ± 11 P< 0.01	+ 3 ± 9 NS	- 5 ± 7 P<0.05
Absolute Oxygen (Uptake l/mn)	2.70 ± 0.53	2.99 ± 0.42	2.91 ± 0.37	+0. 29 ± 0.21 P<0.001	- 0.08 ± 0.51 NS	+ 0.21 ± 0.50 P<0.05
Weight corrected Oxygen uptake (ml/kg/mn)	33.9 ± 6.0	39.0 ± 5.6	38.8 ± 5.2	+ 5.1 ± 0.001 P<0.001	- 0.2 ± 2.9 NS	+ 4.9 ± 3.2 P<0.001
Pulmonary Ventilation (l/mn)	88.2 ± 25.2	101.4 ± 18.9	100.6 ± 20.3	+ 13.2 ± 12.1 P<0.001	- 0.8 ± 6.4 NS	+ 12.4 ± 18.1 NS
Plasma Lactate (mmol/l)	8.7 ± 1.9	10.0 ± 2.9	11.7 ± 3.1	+ 1.3 ± 4.3 NS	+ 1.7 ± 2.2 P<0.05	+ 3.0 ± 2.1 P<0.001
Systolic blood pressure (mm hg)	212 ± 24	221 ± 20	211 ± 17	+ 9 ± 16 NS	- 10 ± 18 NS	- 1 ± 4 NS

training. This increase occuring entirely the first 15 weeks of training ($P < 0.01$).

End-systolic volume did not change significantly with training. A significant increase was observed in stroke volume over the 30 weeks, this increase occuring in the initial stages of training (both $P < 0.001$). Maximal ejection fraction demonstrated small non-significant increase in value with training.

After 30 weeks, output had increased significantly from it's pre-training values ($P < 0.05$). This increase was due to an increase in the first 15 weeks of training ($P < 0.001$).

(ix) Arteriovenous oxygen difference

No siginificant alteration in the mean value of arteriovenous oxygen difference occured with training. The value of VO2 max in thissection is not the same as in section (c)(ii) above as the number of subjects in each case differed.

(c) PostExercise

(i) Heart Rate

The change in mean value of heart rate recorded post-exercise are shown in Table 18

A similar pattern of heart rate recovery versus time was observed at all stages of training. The longer the period of time post exercise, the lower the rate of recovery. With 30 weeks training it was found that the heart rate values at all times post exercise were significantly reduced (all $P < 0.001$). The majority of

TABLE 18

Heart rate and systolic blood pressure recovery post maximal bicycle ergometer exercise at each stage of training

(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Heart rate 1 minute post exercise (beats/min)	148 ± 19	134 ± 12	128 ± 16	- 14 ± 16 P<0.001	- 6 ± 14 P<0.0	- 20 ± 16 P<0.001
Heart rate 2 minutes post exercise (beats/min)	129 ± 18	116 ± 11	112 ± 14	- 13 ± 16 P<0.001	- 4 ± 12 P<0.05	- 17 ± 14 P<0.001
Heart rate 3 minutes post exercise (beats/min)	116 ± 15	105 ± 12	100 ± 12	- 11 ± 18 P<0.001	- 5 ± 15 P<0.05	- 16 ± 15 P<0.001
Systolic blood pressure 3 minutes post exercise (mmHg)	139 ± 16	137 ± 20	128 ± 22	- 2 ± 21 NS	- 9 ± 25 P<0.05	- 11 ± 26 P<0.01

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

this change appeared to occur during the initial 15 weeks of training.

(ii) Systolic Blood Pressure

The pattern of change in systolic blood pressure measured post exercise is shown in Table 18. A significant reduction ($p < 0.05$) in pressure 3 minutes post-exercise was observed. The majority of this reduction appeared to occur during the final 15 weeks of training ($P < 0.05$).

3.3.3 Treadmill Exercise Test

(a) Pre-exercise

The mean values of resting heart rate recorded while sitting prior to treadmill exercise are shown in Table 19.

A highly significant ($P < 0.001$) reduction was observed in resting rate after 30 weeks training. This reduction was entirely the result of the changes during the initial 15 weeks of training.

(b) Submaximal Exercise

(i) Oxygen Uptake

The alteration with training in absolute and weight corrected submaximal oxygen uptake are shown in Tables 20 and 21 and Figures 18 and 19 respectively.

A significant reduction ($P < 0.001$) occurred in both absolute and weight corrected values at all levels of testing after 30 weeks training. The majority of

TABLE 19

Heart rate (beats/min.) sitting prior to treadmill exercise at each stageof training
(means and SD's)

T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
71 ±16	61 ±11	62 ±10	-10 ±15 P<0.001	+1 ±5 NS	-9 ±13 P<0.001

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

TABLE 20

Absolute oxygen uptake (l/min.) during submaximal treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
3mph 0% grade	1.13 ±0.22	1.06 ±0.13	1.01 ±0.14	-0.07 ±0.23 NS	-0.05 ±0.29 NS	-0.12 ±0.20 P<0.001
4mph 0% grade	1.76 ±0.33	1.60 ±0.20	1.51 ±0.24	-0.16 ±0.26 P<0.01	-0.09 ±0.29 P<0.05	-0.25 ±0.20 P<0.001
4mph 5% grade	2.33 ±0.39	2.06 ±0.27	1.93 ±0.26	-0.27 ±0.21 P<0.001	-0.13 ±0.26 P<0.05	-0.40 ±0.22 P<0.01
4mph 10% grade	-	-	2.63 ±0.35	-	-	-

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 18

Absolute oxygen uptake during submaximal treadmill exercise at each stage of training
(means and SE's plotted)

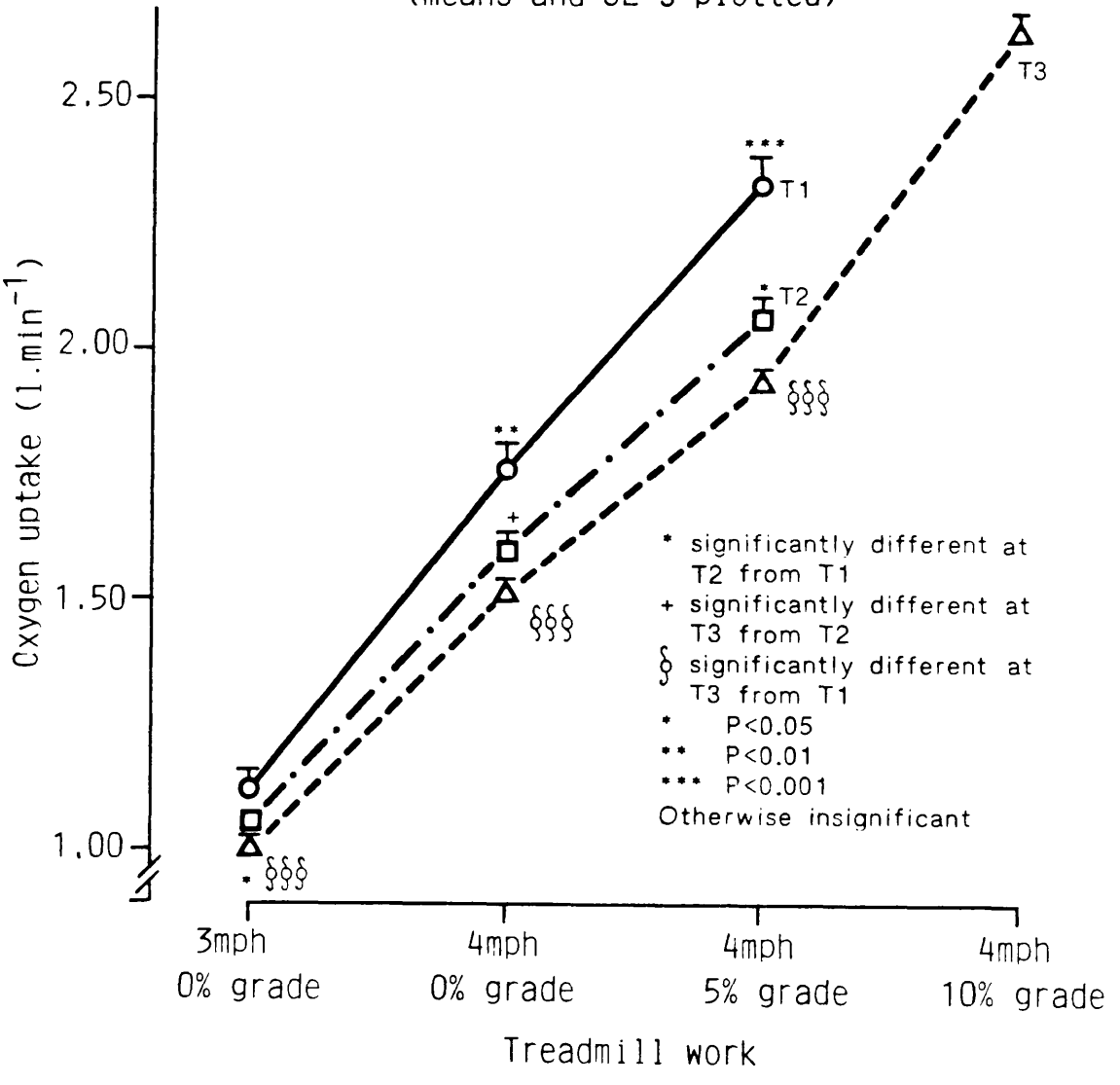


TABLE 21

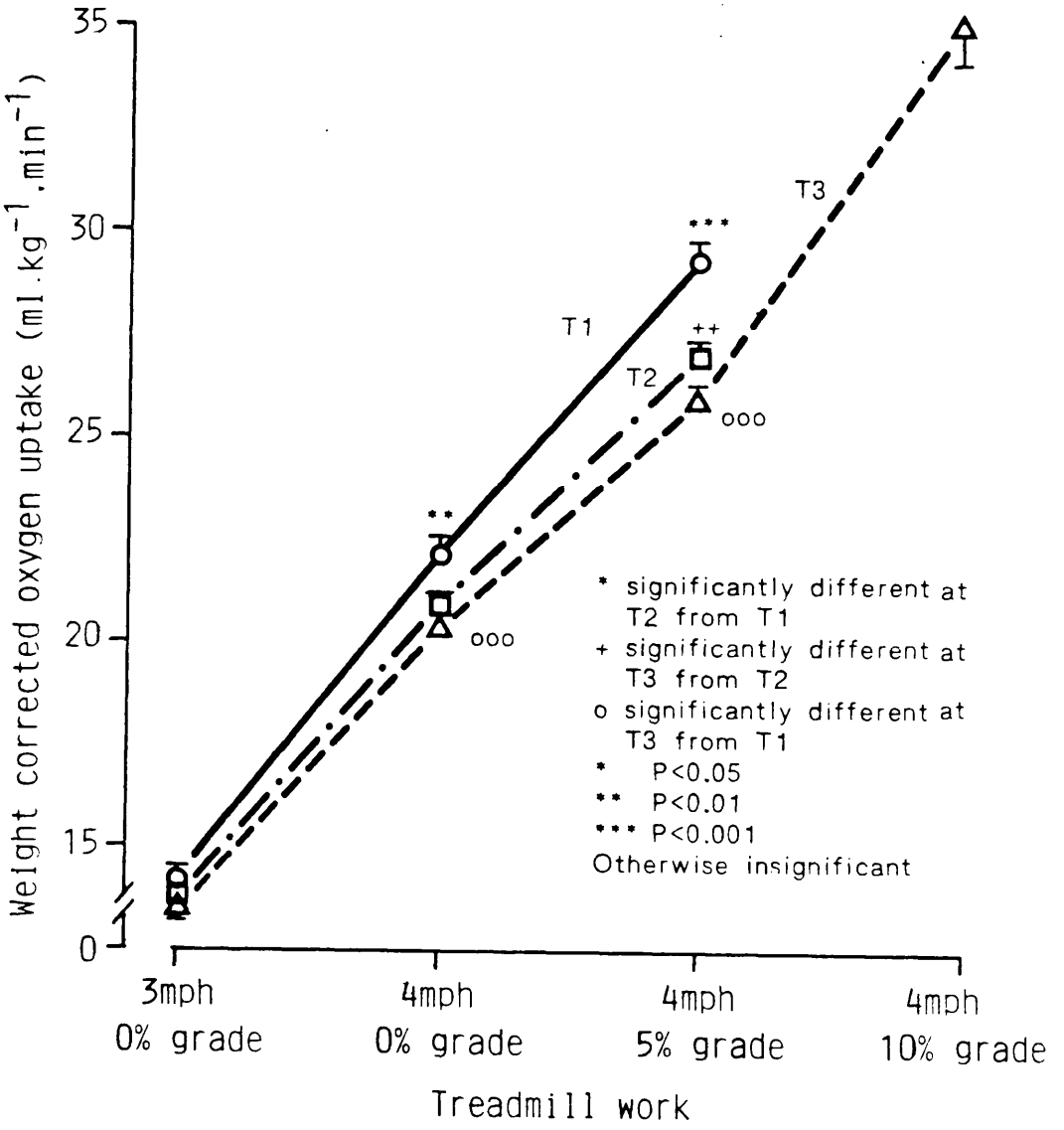
Weight corrected oxygen uptake (ml/kg/min.) during submaximal treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
3mph 0% grade	14.2 ±1.9	13.8 ±1.0	13.4 ±1.0	-0.4 ±2.1 NS	-0.4 ±2.0 NS	-0.8 ±3.1 NS
4mph 0% grade	22.0 ±2.6	20.9 ±1.7	20.3 ±1.9	-1.1 ±2.8 P<0.01	-0.6 ±3.0 NS	-1.7 ±2.8 P<0.001
4nph 5% grade	29.2 ±3.4	26.9 ±2.0	25.8 ±2.0	-2.3 ±2.4 P<0.001	-1.1 ±2.7 P<0.01	-3.4 ±3.2 P<0.001
4mph 10% grade	-	-	34.8 ±4.6	-	-	-

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 19

Weight corrected oxygen uptake during submaximal treadmill exercise at each stage of training
(means and SE's plotted)



this reduction was not found to be significantly different to that of the final 15 weeks of training.

A significant increase ($0.01 < P < 0.001$) in the absolute magnitude of oxygen uptake reduction with training was noted with increasing work level. This difference was retained ($0.05 < P < 0.01$) after correcting this change for pre-training values.

(ii) Heart Rate

The pattern of change in submaximal heart rate with increasing submaximal treadmill work and the change in this pattern with training is shown in Table 22 and Figure 20.

A highly significant ($P < 0.001$) reduction at all submaximal workloads was noted after 30 weeks training. This reduction occurred during both the initial and final 15 week periods of training (both $P < 0.001$). However, the reduction after the initial 15 weeks was greater (all $P < 0.01$) than after the final 30 weeks.

(iii) Pulmonary Ventilation

Submaximal ventilation values during treadmill exercise at each of the 3 stages of testing are shown in Table 23 and Figure 21.

Ventilation decreased significantly ($P < 0.001$) after 30 weeks training. The change was higher ($P < 0.001$) during the initial 15 weeks of training than the final 15 weeks. No significant change in value was

TABLE 22

Heart rate (beats/min.) during submaximal treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
3mph 0% grade	108 ±16	94 ±12	87 ±13	-14 ±10 P<0.001	-7 ±7 P<0.001	-21 ±11 P<0.001
4mph 0% grade	134 ±18	116 ±13	108 ±10	-18 ±11 P<0.001	-8 ±8 P<0.001	-26 ±13 P<0.001
4mph 5% grade	158 ±19	133 ±14	123 ±12	-25 ±12 P<0.001	-10 ±6 P0.001	-35 ±15 P<0.001
4mph 10% grade	-	-	146 ±120	-	-	-

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 20

Heart rate during submaximal treadmill exercise
at each stage of training
(means and SE's plotted)

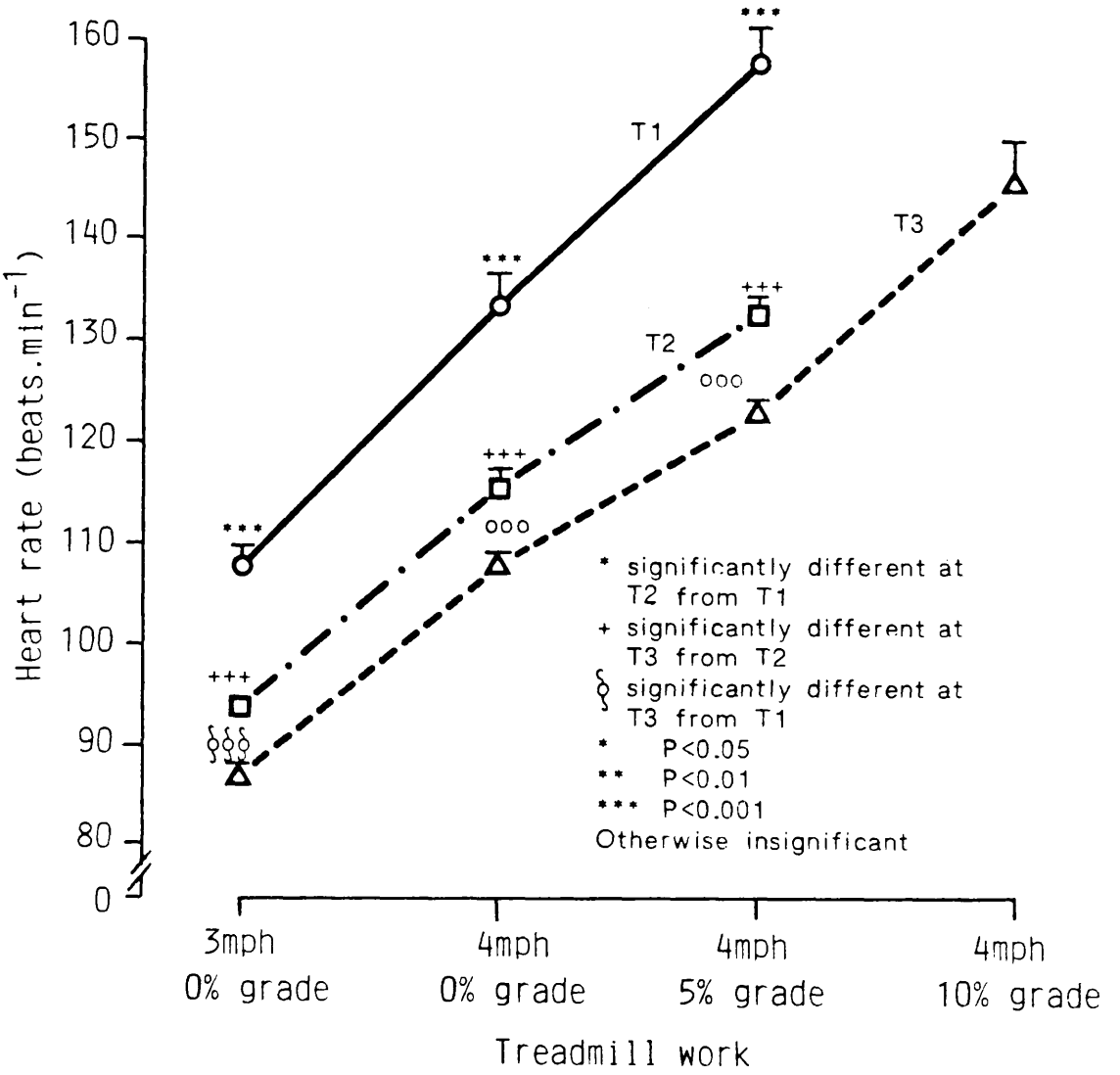


TABLE 23

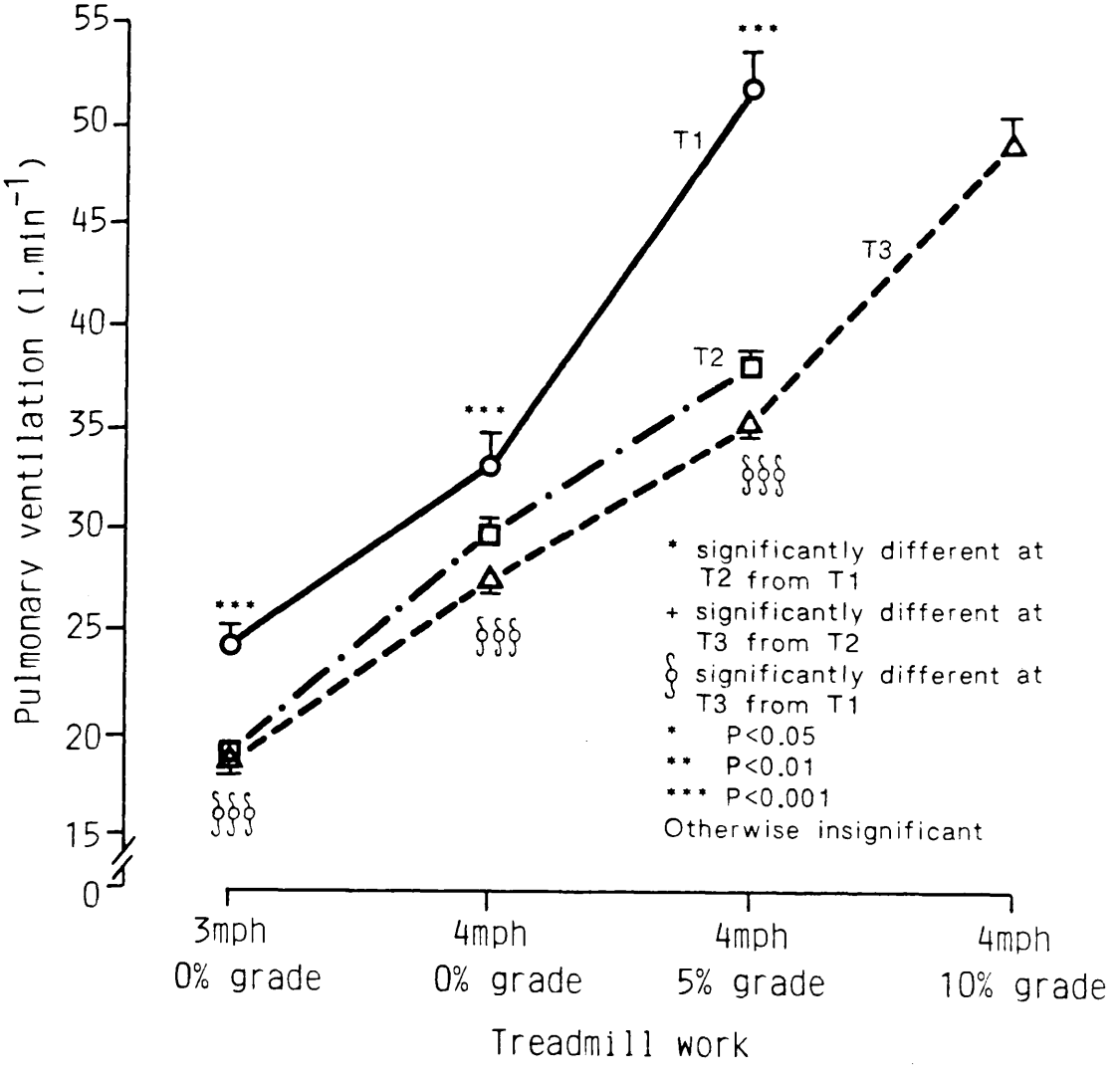
Pulmonary ventilation (l/min.) during treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
3mph 0% grade	24.5 ±7.2	19.3 ±3.8	19.5 ±3.5	-5.2 ±4.6 P<0.001	+0.2 ±2.7 NS	-5.3 ±3.8 P<0.001
4mph 0% grade	38.2 ±12.2	29.8 ±5.8	27.6 ±4.6	-8.4 ±9.5 P<0.001	+2.2 ±8.5 NS	-10.6 ±9.9 P<0.001
4mph 5% grade	51.7 ±13.7	38.0 ±7.6	35.2 ±6.0	-13.6 ±7.5 P<0.001	-2.8 ±8.6 NS	-16.5 ±10.1 P<0.001
4mph 10% grade	-	-	48.8 ±9.0	-	-	-

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 21

Pulmonary ventilation during submaximal treadmill exercise at each stage of training
(means and SE's plotted)



recorded during the final 15 weeks of training.

(iv) Respiratory Exchange Ratio

A summary of changes in exchange ratio versus treadmill workload with training are shown in Table 24 and Figure 22.

A reduction was observed in value at all levels of treadmill exercise, although this was only significant (both $P < 0.001$) at 4 mph 0% grade and 4 mph 5% grade. The majority of this reduction took place during the first 15 weeks of training with no significant change during the final 15 weeks.

(c) Post Exercise

(i) Heart Rate

The changes in post exercise heart rate at the 3 stages of testing are summarized in Table 25 and Figure 23. A significant reduction in post exercise heart rate occurred after 30 weeks training. This reduction was significantly greater ($P < 0.001$) during the initial 15 weeks compared to the final 15 weeks.

3.3.4 Body Composition and Energy Metabolism

(a) Body Composition

(i) Total body weight

A highly significant ($P < 0.001$) reduction in the group's total body weight was observed during the period of training (Table 26). Although the the fall throughout the first 15 weeks exceeded that of the last 15 weeks (ie. 2.7 ± 2.8 kg vs 1.8 ± 2.1), this difference was found to be statistically insignificant.

TABLE 24

Respiratory exchange ratio (RER) during submaximal treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
3mph 0% grade	0.88 ±0.14	0.81 ±0.05	0.84 ±0.10	-0.07 ±0.16 P<0.01	+0.03 ±0.13 NS	-0.04 ±0.14 NS
4mph 0% grade	0.93 ±0.12	0.87 ±0.05	0.87 ±0.06	-0.06 ±0.16 P<0.05	0.00 ±0.09 NS	-0.06 ±0.09 P<0.001
4mph 5% grade	0.97 ±0.09	0.91 ±0.06	0.89 ±0.05	-0.06 ±0.18 P<0.05	-0.02 ±0.10 NS	-0.08 ±0.09 P<0.001
4mph 10% grade	-	-	0.95 ±0.07	-	-	-

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 22

Respiratory exchange ratio during submaximal treadmill exercise at each stage of training
(means and SE's plotted)

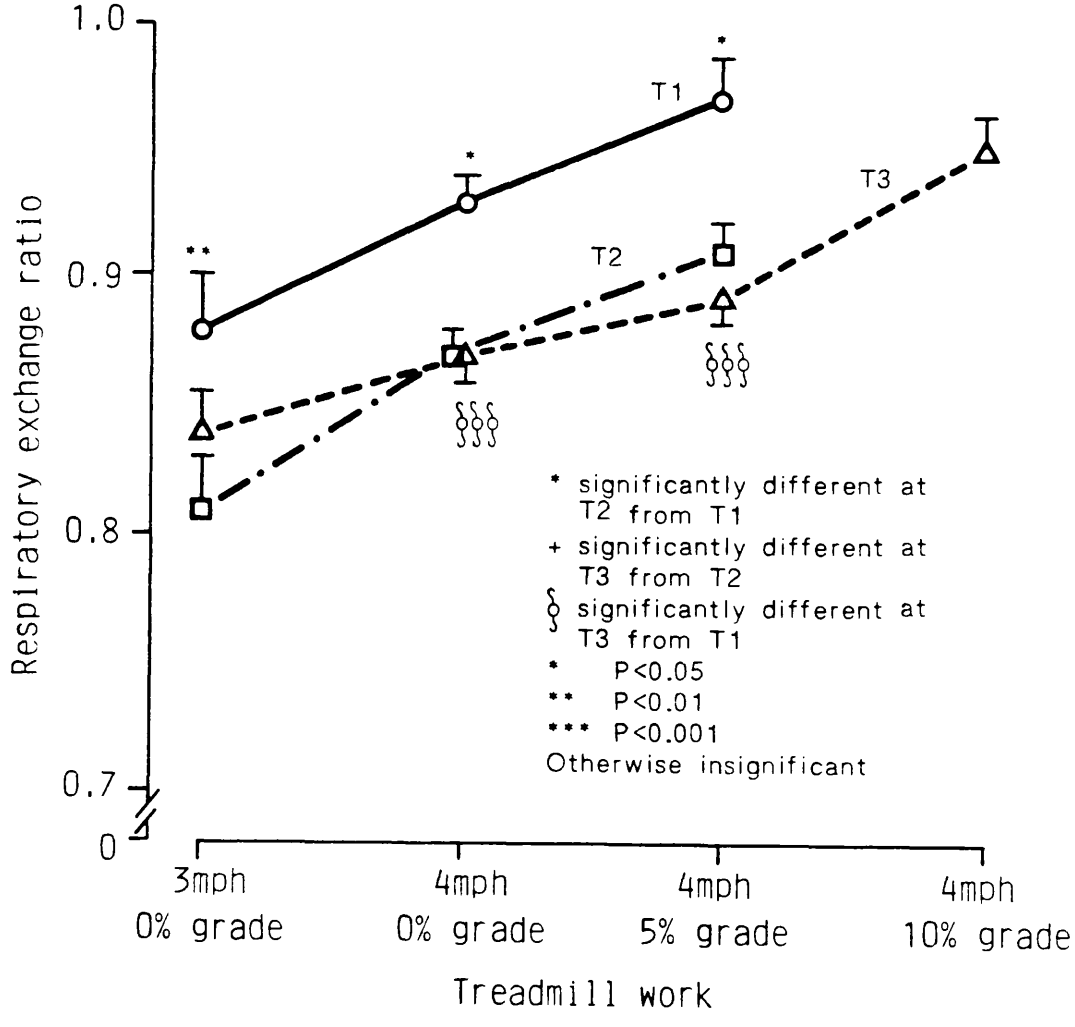


TABLE 25

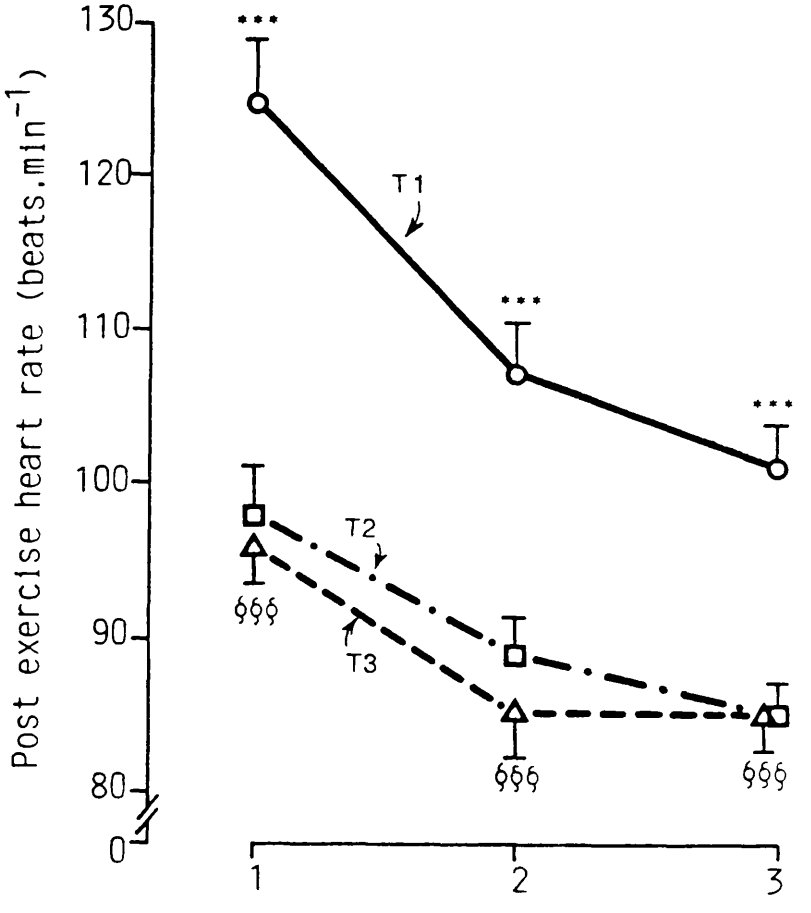
Heart rate (beats/min.) post submaximal treadmill exercise at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
1 minute post exercise	125 ±23	98 ±18	96 ±16	-27 ±21 P<0.001	-2 ±13 NS	-29 ±23 P<0.001
2 minutes post exercise	107 ±20	89 ±15	85 ±17	-18 ±17 P<0.001	-4 13 NS	-22 ±17 P<0.001
3 minute post exercise	101 ±19	82 ±14	82 ±15	-19 ±19 P<0.001	0 ±12 NS	-19 ±20 P<0.001

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

FIGURE 23

Heart rate post submaximal treadmill exercise at each stage of training (means and SE's plotted)



* significantly different at T2 from T1
\+ significantly different at T3 from T2
ϕ significantly different at T3 from T1
* P<0.05
** P<0.01
*** P<0.001
Otherwise insignificant

TABLE 26

Total body weight and skinfold values at each stage of training
(mean and SD's)

	T1	T2	T3	ΔT1-T2*	ΔT2-T3*	ΔT1-T3*
Height(cm)	176.6 ±7.6					
Total body weight (kg)	79.9 ±10.1	77.2 ±10.7	75.4 ±10.1	-2.7 ±2.8	-1.8 ±2.1	-4.5 ±3.3
Biceps skinfold (mm)	6.1 ±1.7	5.2 ±1.4	4.4 ±1.2	-0.9 ±0.8	-0.8 ±0.8	-1.7 ±1.0
Triceps skinfold (mm)	9.6 ±2.7	8.8 ±2.2	8.0 ±2.1	-0.8 ±1.5	-0.8 ±1.0	-1.6 ±1.8
Subscapular skinfold (mm)	17.4 ±4.3	15.5 ±4.2	13.7 ±3.7	-1.9 ±1.7	-1.8 ±1.7	-3.7 ±2.1
Supra-ilac skinfold (mm)	23.2 ±6.0	18.8 ±5.7	15.3 ±5.5	-4.4 ±4.4	3.5 ±3.3	-7.9 ±5.5
Sum of skinfolds (mm)	56.0 ±11.8	48.1 ±11.1	41.4 ±7.4	-7.9 ±6.0	-6.7 +5.0	-14.6 ±7.4

* all $P<0.001$

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

(ii) Skinfold values

The sum of 4 skinfolds revealed a reduction of some 27% in magnitude during the 30 weeks of training ($P < 0.001$) (Table 26). Although the fall in magnitude during the first 15 weeks was slighly greater than the last 15 weeks, this difference was not significant.

Each of the 4 individual skinfold values was reduced with training (all $P < 0.001$) (Table 26). The absolute reduction in each of the 4 skinfolds with training was, in order of location, supra-iliac then subscapular then biceps and finally triceps (Figure 24). Expressing these changes as a percentage of the pre-training value, alters the order of decrement (Figure 25). Supra-iliac then biceps then subscapular then triceps (for T1-T3: -34% vs -28% vs -21% vs -18%).

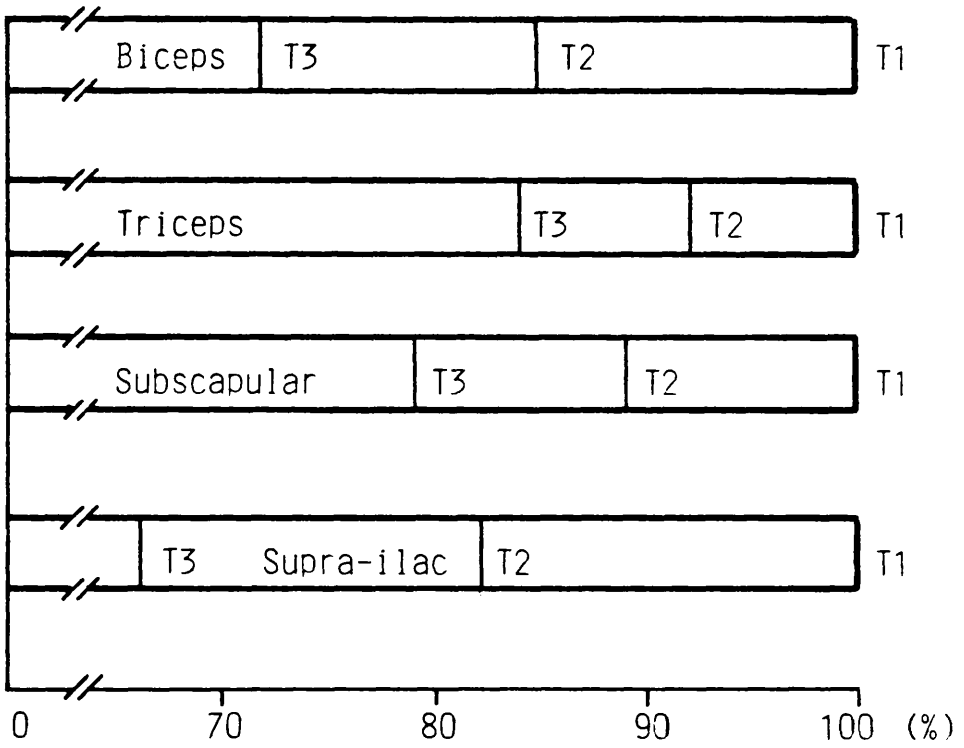
(iii) Body density and body fat content.

Body density increased significantly ($P < 0.001$) with training (Table 27). This increase was observed at both stages of training ($P < 0.001$) and was not signficantly different.

Body fat content was determined from density (Table 30). An average reduction of fat content of 5.0% and fat mass of 4.8kg was observed during the 30 weeks of training (both $P < 0.001$). The fall in fat content was no different at any stage of training (ie. T1-T2: -2.7 ± 1.6% vs T2-T3: -2.1 ± 1.8%). The fall in fat mass was greater ($P < 0.01$) during the initial stages of training than in comparison to the later stages (ie.

FIGURE 24

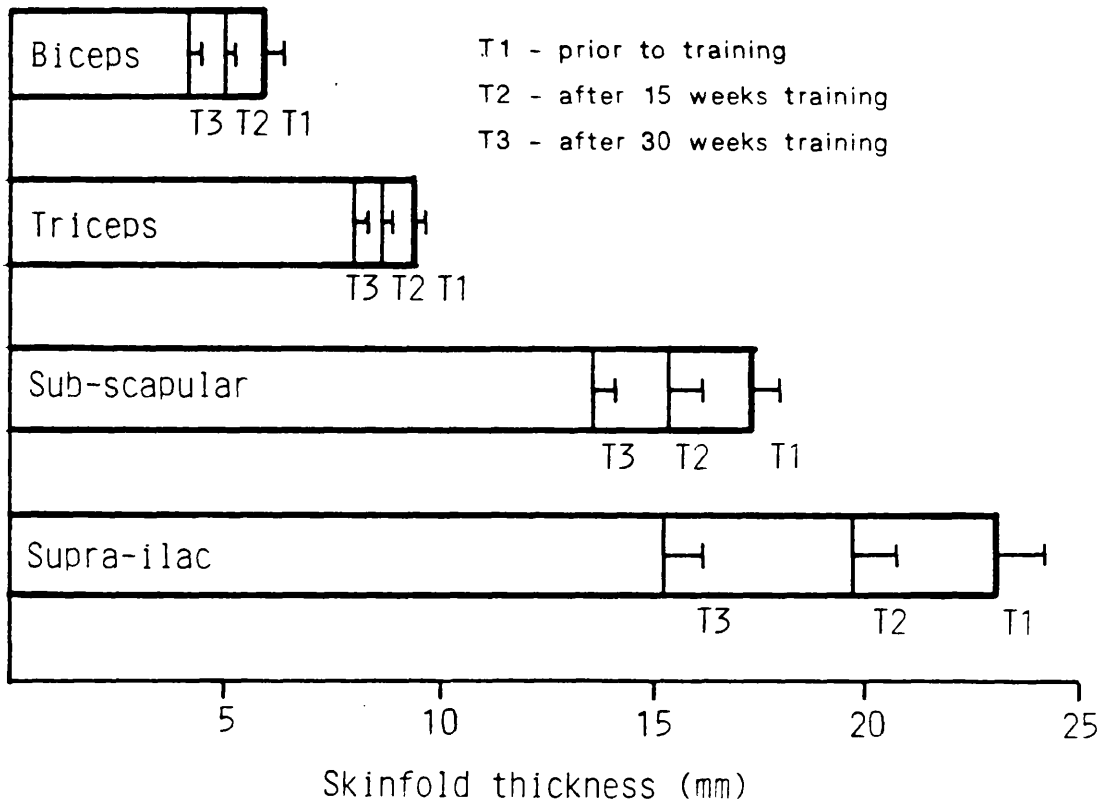
Percentage changes in skinfolds with training
(means and SD's)



T1-prior to training
T2-after 15 weeks training
T3-after 30 weeks training

FIGURE 25

Absolute changes in individual skinfold values with training
(means and SD's)



T1-T2: -2.6±1.6kg vs T2-T3: -4.5±2.6kg)

No change in fat free mass was observed in the initial 15 weeks of training. A slight increase (+0.5±3.8 kg) occurred during the final 15 weeks but this was statistically insignificant.

These changes in total body, fat and fat free mass are summarized in Figure 26.

(iv) Limb circumferences

A significant ($P < 0.001$) reduction in upper arm, gluteal and thigh circumference was observed during the 30 weeks of training (Table 28). In each case the decrement was greater in the first 15 weeks compared to the second 15 weeks although this was only significant in the case of the thigh. The calf circumference rose slightly with training but this was not significant.

(b) Energy Metabolism

(i) Exercise energy cost

As outlined in section 2.6.3, exercise energy cost was calculated for each training session based on running speed, training duration and body weight. The mean change in exercise energy cost during the first and final 15 weeks of training are summarized in Table 29.

The increase in calculated energy cost of the final 15 weeks (ie. T_2-T_3) was significantly greater ($P < 0.001$) than that of the initial 15 weeks (ie. T_1-T_2).

(ii) Body energy store

The changes in the body energy store were

TABLE 27

Body density,determined by underwater weighing body fat content and fat free mass at each stage of training
(means and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Body density ($kg/m^3 10^3$)	1.047 ±0.008	1.053 ±0.009	1.058 ±0.009	+0.006 ±0.004 P<0.001	+0.005 ±0.006 P<0.001	+0.011 ±0.0075 P<0.001
Fat content* %	22.7 ±3.7	20.0 ±4.0	17.7 ±4.7	-2.7 ±2.0 P<0.001	-2.3 ±2.2 P<0.001	-5.0 ±3.2 P<0.001
Fat weight (kg)	18.3 ±4.9	15.6 ±4.7	13.5 ±4.9	-2.7 ±1.6 P<0.001	-2.1 ±1.8 P<0.001	-4.8 ±2.6 P<0.001
Fat free mass (kg)	61.4 ±8.6	61.4 ±8.9	61.9 ±8.2	0.0 ±2.7 NS	+0.5 ±3.8 NS	+0.5 ±3.4 NS

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

* % of total body weight

FIGURE 26

Summary of body composition changes with training

(means and SD's plotted)

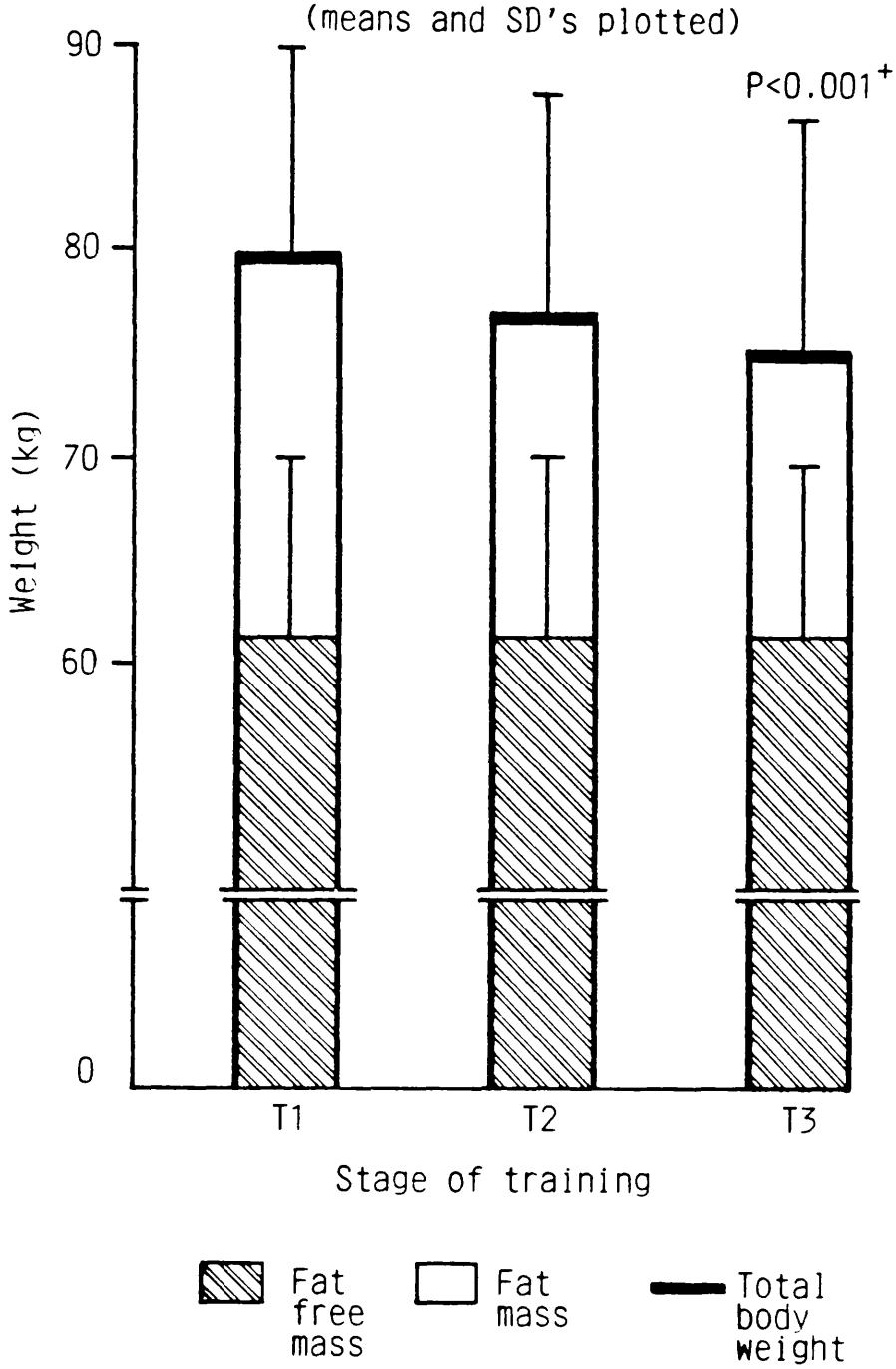


TABLE 28

Changes in the value of limb circumferences with training
(means and SD's)

	T1	T2	T3	T1-T2	T2-T3	T1-T3
Upper Arm Circumference (cm)	31.5 ±2.4	30.2 ±2.3	29.3 ±2.5	-1.3 ± P<0.001	-0.9 ±0.95 P<0.001	-2.2 ± P<0.001
Gluteal Circumference (cm)	98.0 ±5.4	94.8 ±4.9	92.6 ±4.9	-3.2 ±2.3 P<0.001	-2.2 ±2.1 P<0.001	-5.4 ±3.6 P<0.001
Thigh Circumference (cm)	57.5 ±4.4	56.4 ±3.6	56.2 ±3.6	-1.1 ±1.2 P<0.001	-0.2 ±1.0 NS	-1.3 ±1.6 P<0.001
Calf Circumference (cm)	38.4 ±2.6	38.5 ±2.4	38.5 ±2.4	+0.1 ±0.5 NS	0.0 ±0.7 NS	+0.1 ±0.9 NS

T1- prior to training
T1- after 15 weeks training
T3- after 30 weeks training

TABLE 29

Changes in energy metabolism values at each stage of training
(means and SD's)

	ΔT1-T2	ΔT2-T3	ΔT1-T3
Change in gross energy expenditure (kcal/min)	28548 ±12657	58691 ±15806	87235 ±24435
Change in net energy expenditure (kcal/min)	26365 ±12444	54463 ±15021	80828 ±24132
Change in body energy (kcal/min)	21038 ±16307	16170 ±16318	37208 ±23340
Change in energy intake (kcal/min)	+5327 ±13093 P<0.05	+38293 ±20047 P<0.001	+47751 ±25021 P<0.01

T1-prior to training
T2-after 15 weeks training
T3-after 30 weeks training

calculated for the changes in body fat by hydrostatic weighing reported in section 3.3.4 (Table 29). The change in the body's energy store during the first phase of training was not significantly different to that of the final phase of training.

(iii) Energy intake

The changes in energy intake of the subjects during the training program were determined by the difference of the increase in the exercise energy cost and the change in body energy store (Table 29). The calculated increases in energy intake at both stages of trainimg were determined to be statistically significant (T1-T2: $P < 0.01$, T2-T3: $P<0.001$). The increase in energy intake during the later 15 weeks of training was greater ($P < 0.001$) than that of the initial 15 weeks.

CHAPTER 4

DISCUSSION

4.1 AEROBIC POWER (VO_2max)

As outlined in the review of literature, VO_2max has for a considerable number of years been regarded as the gold standard index of aerobic endurance. It is therefore appropriate that this discussion chapter begins by considering the findings of this present study with regard to this parameter.

4.1.1 Alterations in Aerobic Power (VO2max) with Training

(a) Pattern of change

Pollock (1973) reviewed the change in VO_2max with training of 31 studies, involving some 450 male subjects aged 11 to 75 years. Only two of these failed to demonstrate a significant increase in VO_2max. One of these studies was performed on a highly trained group of athletes with an average initial VO_2max of 60.1 ml/kg/min (Daniels & Oldridge, 1971) while the other had a duration of some 6 weeks (Benestad, 1965). Both these factors have been implicated as potential factors reducing the magnitude of VO_2max change with training.

Thus the pattern of VO_2max increase observed in the present study is entirley in accordance with previous training studies.

(b) Magnitude of change

The magnitude of increase in VO_2max observed with training in Pollock's review revealed a range 1.5 to 23.7 ml/kg/min which corresponds to a 3 to 97% increase. However, the majority of studies in this review and since reported an increase in VO_2max most commonly ranging from 16 to 20% (Pollock, 1973; ACSM, 1978). Indeed the usually large imrpovment of 93% observd in the study by Cureton and Philips (1964) was believed to reflect the initally low value of VO_2max (26.5 ml/kg/min) of their middle aged subjects. This low initial value was thought to have resulted from the subjects not achieving a true VO_2max at initial testing (Pollock, 1973).

A significant increase in direct VO_2max from bicycle ergometry on average of 0.21 l/min and 4.9 ml/kg/min was observed after the 30 weeks of present aerobic endurance training program (see Table 16). These correspond to proportional increases in direct VO_2max of 8 and 15% respectively. This improvement was observed to occur entirely during the initial 15 weeks of training with no change in the average value of VO_2max in the later 15 weeks.

Thus the magnitude of VO_2max is no greater than that typically reported by previous aerobic endurance training programs. This comparative finding was slightly surprising in view of the substantially more demanding nature of the present program in terms of

duration and frequency of training. Indeed on the basis that the improvement in VO_2max is dependent on the training program one may have expected an average increase in VO_2max in the present study in excess of that typically reported, ie. greater than 20%.

There are a number of possible mechanisms that may account for a smaller than expected increase in VO_2max with training.

(c) Mechanism of change

The precise physiological mechanisms responsible for the increase in VO_2max in this study will be discussed in section 4.2. The significance of the various mechanisms responsible for a reduced magnitude of VO_2max change with training will be reviewed with regard to the present study.

(i) Experimental factors

It is possible that the magnitude of VO_2max increase in the present study is lowered because of experimental error.

The reproducibility of VO_2max measurement was not assessed in this study. This was not done because of substantial evidence from other laboratories of a high value of direct VO_2max assessment reliability. A recent review of 9 exercise laboratories reliability quoted a "reliability (or correlation) coefficient" for these studies ranging from 0.74 to 0.98 with an average coefficient of variation of 9.1 percent (Mellerowicz & Smodlaka, 1981). To ensure a similar high value of

reproducibility, care was taken in expeirmental detail such as calibration of gas analysers, maintainance of a standard testing environment and precise timing of expired gas sampling. The experimental proceedure for VO_2max assessment in this study is discussed in detail in section 2.6.1 (b)(i).

It therefore appears that inadequate VO_2max repoducibility is not responsible for the observed pattern or magnitude of VO_2max response in this study.

(ii) Subject age

The few studies that have considered the effect of aerobic endurance training on older age groups have reported a reduced magnitude of training effect, in terms of absolute change in VO_2max, in comparison to their younger counterparts (Pollock, 1973; Astrand & Rodahl, 1977). As was outlined in the introduction, it has been suggested that this discrepancy is reflective not of an aging effect per se but a reduced magnitue of training performed by the older groups (Lamb, 1984).

Comparison of previous studies of similar training effort on different age groups may then aid the evaluation of an age factor in this study. To the author's knowledge only two studies to date examining the effect of marathon training on VO_2max have been published. The first by Kavanagh and Shephard (1967) examined the effect of a marathon training program on a middle-aged group of post myocardial infarction

patients. The second and more recent by Martin et al (1986), reported on the effects of a competitive marathon season on a group of 5 elite distance runners. Improvements in VO_2max of 55% and 0.5% respectively were observed over the period of training in these studies. Although also considering marathon training the experimetal design of these two previous studies makes comparsion difficult. The study of Kavanagh and his colleagues assessed VO_2max changes on the basis of prediction from submaximal exercise observations. Predicted VO_2max has been reported to consistently overpredict VO_2max improvement in previously unconditioned groups (Astrand & Rodahl, 1977). In addition because of the cardiac status of this group it may not be valid to compare the results to a healthy population. In the study by Martin, the subjects initial conditioning was very high and a mean initial value of VO_2max was reported as 75.8 ml/kg/min. It is not surprising that such a high initial value of VO_2max should not increase significantly with training. Thus this comparision neither confirms or refutes the ageing hypothesis.

Although studies have observed a discrepancy between the absolute change when comparing young and older groups, when expressed as a relative change, an age effect is no longer apparent (Saltin et al 1968). Indeed re-analysis of the review study of Pollock (1973) reveals that there was no relationship (r =

-0.12, NS) between age and the magnitude of VO2max change. This later finding may reflect the lack of standardiation of these studies in terms of training load and initial value of VO_2max. However, there was no evidence of any relationship between the individual magnitude of VO_2max and age at any stage of the present study (T1-T2: r = +0.23, T2-T3: r = -0.12, T1-T3: r= +0.20, all NS).

Thus in view of this above evidence it appears unlikely that age of has reduced the magnitude of VO_2max adapation in the present study.

(iii) Initial VO_2max of study group

Reviews of aerobic training studies have clearly shown that there is an inverse relationship between the pre-training value of VO_2max and the change in VO_2max observed with training (Pollock, 1973; ACSM, 1978) ie. the greater the initial VO2max the smaller the change with training. Reanalysis of the review study of Pollock (1973) demonstrated a significant relationship ($r = -0.47$, $P<0.05$) between initial VO_2max and the change in magnitude of VO_2max with training. Bed ridden patients demonstrate an increase of up to 30% in VO_2max (Saltin et al, 1968) while fit endurance athletes often demonstrate no increase despite an increase in training effort (Daniels et al, 1978). As might be expected the study that reports the 93% increase in VO2 max with training also reports the lowest initial values of VO_2max (Cureton & Philips, 1964). It may therefore be

hypothesized that the increase in VO_2max with the present training study is reduced on account of a high initial VO_2max.

To evaluate the present study initial value of VO_2max, the expected 'normal' values of VO_2max for this group must be obtained. Two potential sources of comparison values for the initial VO_2max value can be used: population values and the values reported in previous training studies.

There a few carefully selected population studies examining the values of VO_2max over a wide range of age and activity backgrounds. In 1966 Shephard reviewed the world's literature on aerobic power of population samples. Acknowledging the difficulty in interpreting and comparing individual studies which used different methodologies and small sample sizes, Shephard concluded that there was a striking difference between Scandinavian values and those from the remainder of Europe and the North America. As evidence he cited reports of the VO_2max of 63.7 ml/kg/min for Swedish soldiers and 36.5 ml/kg/min for US servicemen. He concluded that this difference reflected the more active life led by the Scandinavians in comparison to other countries rather than genetic differences. However, a recent large population study comprising 1,514 male US serviceman aged 17 to 55 years found a mean value of VO_2max of 51 ml/kg/min (Vogel et al, 1986). These later figures are considered to be the

more reflective of the above two US studies, as the values for US servicemen outlined by Shephard were based in part on indirect, predictive techniques.

In the present study a mean direct VO_2max of 2.70±0.52 l/min or 33.9±6.0 ml/kg/min was obtained prior to training. This value is considerably lower than that of the Scandinavians and recent US values. However, the method of assessing VO_2max in these previous studies has primarily been treadmill assessment. The method of direct VO_2max assessment in this study was bicycle ergometry. As has been outlined previously, the VO_2max value from maximal treadmill exercise consistently exceeds that of maximal bicycle ergometer exercise by about 7%. Applying this correction to the pre-training maximal bicycle ergometer exercise values of VO_2max in this study increases the mean values to 2.89 l/min and 36.3 ml/kg/min.

Comparison of these corrected direct VO_2max figures with those above still shows those of the present study to be considerably lower. The explanation for this difference presumably reflects the decrement in VO_2max with increasing age which occurs regardless of activity background (Astrand et al, 1973), while the above population studies considered both young and older individuals alike. Indeed in the study by Vogel and his colleagues average VO_2max values of 38.7 ml/kg/min are quoted for servicemen in a similar age

range to the present study.

It thus appears that the pre-training value of weight corrected VO_2max is similar to that obtained in other similarly aged population studies reported in the literature. This finding is perhaps slightly surprising in view of the sedentary background of the present group. Agreement in values probably reflects the general sedentary background of the population at large, of which this study group is therefore typical.

To compare the initial value of VO_2max in this st udy to previous training studies, re-analysis of the review study of Pollock (1973) was performed. This revealed an average initial VO_2max for these previous studies of 38.7 ml/kg/min. This value appears to be higher than the inital value observed in this study.

For the initial VO_2max to account for a reduced magnitude of VO_2max increase with training it would be expected that the initial value of VO_2max of the present study would be greater than that typically reported in population or previous studies. Comparison demonstrated that the present initial VO_2max value was the same or lower than that expected for the group. A significant relationship was observed between individual initial VO_2max values and the change in VO_2max at each stage of the present training study (T1 vs T1-T2: $r = -0.47$, $P < 0.05$ & $r = -0.60$, $P < 0.01$; T1 vs T1-T3: $r = -0.54$, $P < 0.01$ & $r = -0.72$, $P < 0.001$ respectively).

Thus although the initial value of VO_2max did appear to be an important factor in determining the individual magnitude of change in VO_2max with training in the present study it did not appear to be responsible for the an overall reduced magnitude of VO_2max improvement in comaparison to previous studies.

(iv) Testing Protocol

There are a number of potential problems associated with the exercise testing protocol that could potentially reduce the magnitude of increase in VO_2max with training.

Exercise Specificity

Exercise specificity is the term applied to the observation of a smaller magnitude or absence of a training effect when the testing and training modes of exercise are disimilar. In this study the method of exercise testing to determine VO_2max was bicycle ergometry while the mode of exercice training was running. According to the exercise specifity hypothesis this difference in exercise modes may lead to a reduced magnitude of VO_2max adaption.

However, it is interesting to note that not all previous training studies have been able to demonstrate the exercise specificity effect. Pechar et al (1974) observed a reduced magnitude of VO_2max increase after 8 weeks cycle training when comparing treadmill to bicycle ergometer testing. However, a study by Wilmore and his colleagues (1980) found no evidence of training

specificity after 20 weeks running or bicycle training when compared by bicycle and treadmill testing. This discrepency of finding with regard to training specifity was, according to Wilmore, a reflection of the insufficient training time employed by Pechar and his colleagues to reveal changes by bicycle ergometer testing.

It is possible to test the hypothesis of training specifity in this stduy as both bicycle ergometer and treadmill testing wer performed. Treadmill testing was onlt performed submaximally and this hypothesis was tested by comparing the change in submaximal heart rate reponse with training. The average observed magnitude submaximal heart rate reduction after 30 weeks training was greater for treadmill testing (average: 27 beats/min) than bicycle ergometer testing (average: 18 beats/min) (see tables 9 and 22). This evidence is highly suggestive that the magnitude cardiovascular training effect in this study is exercise mode dependant. However, caution should be applied when extrapolating this submaximal finding to VO_2max as it has been shown that there is often a poor relationship between submaximal heart rate and VO_2max changes wih training (Fox, 1973; Fox, 1975). Indeed a fall in submaximal heart rate can occur after training despite no change in VO_2max (Pollock, 1973).

Thus taking into account the potential problems of submaximal observation, it appears that training

specificity may have been reponsible in the present study for a reduced of adaption.

Failure to achieve a maximal testing effort

A commonly quoted limitation of direct VO_2max assessment is the failure of the subject to perform maximally (Shephard, 1984). This particuarly the case in peak bicycle ergometer exercise were local leg fatigue can ensue before central fatigue (Shephard et al, 1968a). It is therefore possible in this present study that the subjects failed to achieve a maximal effort at this phase of testing.

To examine this hypothesis consideration needs to be taken of the criteria of maximal effort. As was mentioned in the review of literature, by definition VO_2max is the maximum amount of oxygen the body can use despite further increase in work load. Thus the absolute criteria for achievement of VO_2max is the 'plateau' of oxygen uptake at peak exercise (Taylor et al, 1955). The plateau documents that VO_2max has been achieved and that the final work level was accomplished largely through the contribution of anaerobic energy sources (Stamford, 1975). The definition of this plateau has been suggested to be the difference between the oxygen cost of two successive work loads of less than 150 ml/min (Taylor et al, 1955) or less than 54 ml/min (Mitchell et al, 1958).

The plot of the overall oxygen uptake response

(see Figures 11 and 12), both absolute and weight corrected, versus the bicycle work load revealed no evidence of a plateau at any stage of training in this study. It appeared that there was an almost linear increase in VO2 from the lowest work loads to peak. With regard to individual responses only 4 (14%), 5 (18%) and 4 (14%) individuals at T1, T2 and T3 respectively demonstrated an increase in oxygen uptake of 150 ml or less between the peak bicycle load and the load immediatley before. Thus despite the achievement by all the subjets in this study of subjective fatigue, exhuation and an inability to continue exercise, on the basis of the 'oxygen plateau' at peak exercise it appears that the majority of this group would be regarded as having not achieved VO_2max.

Despite its theoretical basis, the oxygen plateau has been criticized as difficult to obtain in practice and appears to be dependant on both age, cardiorespiratory fitness and the mode of testing (Shephard, 1971). Indeed it is often very difficult to observe an oxygen plateau (Cumming and Freisen, 1967). Virtually all the examples of plateau have been observed in well-trained individuals who are used to exercising at high intensities for a prolonged period of time. These studies may not be transferable to poorly conditioned middle-aged adults where muscle factors are liable to limit exercise capacity (Shephard, 1971). Studies on bicycle ergometers

frequently demonstrate, and confirm the results of the present study, that oxygen uptake continues to increase with increasing work load as long as the subjects can maintain cycling speed (Wyndham et al, 1959; Niemela et al, 1980). Finally even though plateauing of VO_2 is felt to be the most valid indicator of a maximal effort, a number of studies have demonstarted that VO_2max values were not the same when a plateau ws obtained and when it was not (Shephard et al, 1968a; Davies et al, 1984).

In view of the practical difficulties of a plateau, other criteria for VO_2max have been suggested. These include a respiratory exchange ratio greater than or equal to 1.15 (Issekutz et al, 1962), post exercise blood lactate in excess of a particular value: 8 mmol/l (Astrand & Rodahl, 1977) or 5.5 mmol/l (Von Doben et al, 1967) and finally an increase in heart rate to within 5 beats of that estimated for age (Maritz et al, 1961). Although average peak heart rate was significantly lower after training than before (see Table 15), there was no significant alteration in the value of respiratory exchange ratio at maximal exercise and a significant increase in the peak concentration of blood lactate.

All these criteria of peak effort have certain limitations attached to them. The assumption behind the exchange ratio is that at this value the build-up of lactate and other acid metabolites in the blood are sufficient to displace enough carbon dioxide to

increase the ratio above 1.0. However, the ratio may also be affected by overventilation. Indeed Cummings and Borysyk (1972) could demonstrate no correlation between blood lactate and the respiratory exchange ratio at peak exercise. With regard to blood lactate, a single post exercise lactate value fails to take into account the volume of distribution, variable time relationships in peak blood lactate concentrations and individual variations in the production of blood lactate in relation to VO_2max (Cummings and Borysyk, 1972). Finally, the prediction of maximal heart rate has many pitfalls. The standard deviation for maximal heart rate during exercise is ±10 beats/min (Astrand and Rodahl, 1977). There is a gradual decline in maximal heart rate with age (Robinson , 1938; Astrand, 1960). Furthermore, longitudinal studies have shown a wide scatter in the decline of maximal heart rate with age (Astrand, 1973).

Despite the limitations of these three criteria, Cummings and Borysyk have suggested that for the testing of middle-aged subjects, if two of the three criteria are exceeded, a true value of VO_2max will have been obtained. To assess this in the present study the maximal exercise tests of each subject at each stage of testing was evaluated for achievement of these 3 criteria. The results of this analysis are summarized in Table 30. In summary it was observed that 68% (n = 19), 68% (n = 19) and 78% (n = 22) achieved two of

TABLE 30

Criteria for achievement of VO_2 max at each stage of training

	Number of subjects achieving criteria		
	T1	T2	T3
Peak plasma lactate (>8mmol)	16	22	23
Peak RER (>1.15)	19	20	24
Peak heart rate (to within 5 beats of predicted peak)	22	14	18
2 of the 3 criteria	19(68%)	19(68%)	22(78%)
all 3 criteria	14(50%)	10(32%)	16(57%)
Oxygen uptake plateau (<150ml)	4(14%)	5(18%)	4(14%)

T1-prior to training
T2-after 15 weeks training
T3-after 30 weeks training

these three criteria at T1, T2 and T3 respectively. The main reason for a proportion of the group not exceeding the 2 of 3 criteria is apparently due to a common failure to increase respiratory exchange ratio above 1.15. Niemela et al (1980) also reported the failure to regularly observe such a change in middle-aged men performing progressive bicycle exercise to maximum. The use of exchange ratio as a criterion of having attained VO_2max has been justified only in exercise lasting 3-4 min (Issekutz et al, 1962). Such short supramaximal exercise is likely to lead to significant overventilation which could increase the value of the ratio to over 1.0. No significant relationship was observed between maximal respiratory exchange ratio and maximal blood lactate at any stage of testing (T1: r = +0.31, T2: r = -0.01, T3 : r = +0.14, all NS), confirming the problem of the theoretical premise upon which the peak respiratory exchange ratio is based.

The complete magnitide of improvement in VO_2max was observed in the first 15 weeks of training in this study. Therefore it is perhaps surprising that the lowest proportion of individuals not achieving the criteria of maximal effort occured at T2. In view of the continuing training effort of another 15 weeks resulting in no further change in VO_2max it might of been expected that a lower proportion of individuals would have achieved the criteria of maximal effort at T3. However, the highest proportion of subjects

achieieving these criteria as observed at T3. Thus it appears unlikely that the magitude of change in VO_2max in this study is the result of failure to achieve true maximal effort.

Increased exercise time

A potential limitation of the bicycle exercise protocol in this study, was the time required to achieve maximal effort. Prior to training the average maximal exercise time was 18.2 mins with an average 3 minute increase after 30 weeks training to 21.2 mins (see Table 15).

It has been demonstrated that an exercise duration of 5 minutes or less will give insufficient time for the oxygen transport system to reach peak (Astrand and Saltin, 1961). Thus it generally recommended that an exercise test used to assess VO_2max directly select an exercise protocol to ellicit maximal effort in 10 to 15 minutes (Shephard, 1984; Lamb, 1978). The problem of a protocol that greatly exceeds this time span being that the subject may loose concentration (Shephard, 1971) or that the individual's core temperature rises to an extent leading to a shunting of blood to the cutaneous vasculature thereby reducing the supply to the muscle and thus reduing the arterio-venous oxygen difference and value of VO_2max.

In an attempt to reduce the possibility of such an over-heating effect the testing environment was maintained in a 'thermal comfort' zone throughout the period of the

study. However, it is not inconcievable that the prolonged nature of initial bicycle ergometer test in this study may have resulted in such a temperature regulating effect which would have in turn reduced the pre-training value of VO_2max. However, it is difficult to evaluate the contribution of such a temperature effect to the values of VO_2max in this study. However, if over-heating was a significant effect it would have been expected that the maximal arterio-venous oxygen difference would display such an effect. However, the value of arterio-venous oxygen difference at peak exercise in this study demonstrated no change at each testing stage (see table 17).

Thus it appears unlikely that a temperature effect compromised the value of VO_2max in this study.

(iv) Training program

Insufficient Training Load

A set of guidelines have been set aside by the American College of Sports Medicine, based on a review of a number of previous training studies, suggesting the minimum aerobic endurance training effort to ellicit a training effect. (ACSM, 1978). These guidelines suggest a minimum training effort of 15 minutes, 3 times per week at 60% or 50% of maximal heart rate or VO_2max respectively. The average training effort achieved in this study clearly exceeds these minimum guidelines (see tables 3 & 4 and figures 8 & 9).

Some aerobic training studies based on the ACSM minimum guidelines involving previously trained individuals fail to demonstrate an increase in VO_2max despite an increase in endurance performance (Daniels et al, 1978). This observation is usually explained on the basis that the training threshold required to ellicit an improvement in VO_2max increases with increasing previous conditioning (Pollock, 1973, ACSM, 1978). It is therefore possible that the failure to increase VO_2max during the later 15 weeks of training in this study is reflective of an insufficient training load.

To test this hypothesis reference must made to the actual training levels achieved by this group during the study (see Table 3 and Figure 8). From this information it can be seen that although the subjects were on average consistently below the prescribed training levels during last 15 weeks of training the mean duration and frequency of the study did increase during this time: from 180 mins/week to 280 mins/week and 3.4 sessions/week to 4.2 sessions/week (from the end of the 15th week to the peak of the second 15 week period). In view of this progression in training load in terms of duration and frequency it seems unlikly that a minimum aerobic endurance training effect was not being achieved. It is more difficult to include in this conclusion training intensity as this parameter was not consistently assessed for practical reasons in

this study.

The figures on training intensity obtained from a limited number of subjects via exercise heart rate recording (see Figure 9) reveals a steady reduction in training intensity as the study continued, although at no time did it drop below 80% maximal heart rate. Monitoring was only performed during shorter (80 mins or less), and therefore probably more intense, training sessions. The overall training intensity for the present training program may have been expected to be somewhat less. Therefore it is possible that inadequate training intensity may have reduced the magnitude of VO_2max training effect.

However, studies have shown that the intensity and duration of duration are interrelated and it is the total amount of work accomplished that is the important factor with regard to an increase in VO_2max (Burke and Frank, 1975; Pollock, 1973). The training intensity of this study fell as the training duration increased. Indeed calculation of average energy expenditure demonstrated that the total amonunt of work during the latter half of this present training program was significantly greater than that of the first 15 weeks (see Table 32). Thus on the basis of this concept of total energy expended there also appears to be little evidence of insufficient training.

Insufficient overall training duration

Some aerobic endurance training have been found to

be of insufficient duration to allow for any observable improvement in VO_2max (Fox et al, 1975). Indeed the American College of Sports Medicine have suggested that the minimum duration of aerobic endurance programs be 15 to 20 weeks (ACSM, 1978). This question of training program is thought to be particularly important in studies involving older individuals as they reportedly take longer to demonstate a training effect than their younger counterparts (Pollock, 1973).

The 30 week duration of this present training program study clearly exceeds ACSM recommendations and for this reason might be expected to be of sufficient duration to allow for VO_2max adaption. However, a running training program observed by Kasch and his colleagues that continued over a 24 month period revealed that 12 months training were required for VO_2max to level off (Kasch et al, 1973). This latter study suggests that, despite the appearance of a levelling off in VO_2max in this study after 4 months training, continued training may have resulted in further increases. This hypothesis was difficult to evaluate in the present study due to the completion of the main motivation for training ie. the marathon.

Insufficient Recovery Time.

Studies have recently shown that prolonged or very intense exercise can affect the nutritional status of the muscle by depleting its stores of glycogen. The marathon distance of 26.2 miles has been shown to

result in a specific glycogen depletion of the type I and type IIa muscle fibres in non-elite runners (187+24 mins) (Sherman et al, 1983). Moreover if running is performed over a shorter distance (10 miles) on 3 consecutive days a simliar pattern of glycogen depletion will occur. Recovery of the glycogen stores in both these studies was observed to take about 3 days. The performance of further exercise while glycogen levels are depleted will result in a reduction in performance as assessed by exercise time (Berstrom et al, 1967).

During the latter stages of this present study it was not an uncommon finding from the subject's training diaries running on 5 to 6 consecutive days. Thus in accordance with the above studies it is feasible that glycogen depletion in the present study may have resulted in a reduction in performance as assessed by VO2max. However, it is difficult to evaluate such an effect because of no control group for comparison. Moreover it is believed that such depletion of glycogen reserves also requires a reduction in carbohydrate intake (Sherman et al, 1981). No such alteration was noted subjectively by the study participants. Indeed many noted an increase in carbohydrate intake during the later stages of training.

Muscle glycogen depletion studies have observed a reduction in submaximal blood lactate concentration (Henriksson, 1977). However, in the later 15 week of

training in this study the opposite pattern of lactate reponse observed (see table 11 and figure 15). Although this finding appears to rule out the glycogen depletion hypothesis, the present training program may result in specific depletion of the type I and type IIb fibre depletion. Thus IIa fibres would be preferentially recruite during testing and thereby increase rather decrease blood lactate oncentraion in comparsion to a non-depelted state. This specific pattern of glycogen depletion is discussed in more detail in section 4.4.2 (b).

(v) Achievement of functional VO_2max limits

The final and most obvious hypothesis for the failure of this present study to observe an increase in VO_2max during the later 15 weeks of training is that the majority of individuals have achieved their true limit of VO_2max improvement.

The absolute value of VO_2max one can achieve is considered to be substantially genetically determined (Klissouras et al, 1971). There is considerable debate as to the extent of improvement that demonstrated in VO_2max with training. A number of authors have suggested that an initally sedentary individual can expect a proportional increase in VO_2max with training of up to 30% (Astrand & Rodahl, 1977; Lamb, 1978).

Few previous training studies on previously sedentary individuals have demonstrated a magnitude of VO_2max increase of 30%. This value of 30% is

considerably in excess of average increase in the group VO_2max in the present study. Indeed the maximum individual increase in VO_2max observed in this study was 24%. Thus it appears unlikely that this achievement a genetically determined limit of VO_2max improvement occured in this study.

(d) Physiological significance

VO_2max is probably regarded as the gold standard measure of aerobic endurance. The increase in VO_2max of 8% observed over the 30 weeks of the present training is therefore stongly suggestive that the subjects in the pesent study significantly increased their aerobic endurance capacity over the period of this study.

Surprisingly the magnitude of VO_2max improvement in this study was no greater than previously reported by less demanding training programs. Thus in terms of VO_2max assessment, it appears that this study does not support the hypothesis that increased aerobic endurance training effort will result in an increased magnitude of training adapation. Never-the-less the failure to demonstrate such an effect may have reflected the experimental design limitations of training specifity and inadequate recovery time prior to testing.

4.2 HAEMODYNAMIC FACTORS CONTRIBUTING TOWARDS AEROBIC POWER

The purpose of this section is to examine the haemodynamic factors responsible for the increase in aerobic power observed with training in this study.

As has been already discussed in the review of literature (section 1.3), VO_2max is apparently not limited by pulmonary factors but by the factors of oxygen transport and oxygen utilization by the working skeletal muscles. In accordance with the Fick Principle, VO_2max can be defined as the product of maximal cardiac output and maximal arteriovenous oxygen difference. As the result of this equation there is a dual potential basis for an increase in VO_2max with training ie. an increase in maximal cardiac output, an increase in maximal arteriovenous oxygen difference or both.

To the author's knowledge only 5 studies, involving only some 52 individuals in total, have investigated the relative contribution of maximal cardiac output and maximal arteriovenous oxygen difference to an increase in VO2max with aerobic endurance training (Saltin et al, 1968; Hartley et al, 1969; Rowell, 1974; Ekblom et al, 1968; Rerych et al, 1980). The small number of reported studies probably reflects the practical difficulties involved in

TABLE 31

Review of previous training studies investigating the haemodynamic factors contributing toward changes in VO_2 max.

(mean values quoted)

Author		VO_2 max (l/min)	CO max (l/min)	SV max (ml)	HR max (beats/min)	A-V.dO_2 max (mlO_2/100ml)
Young sedentary males						
Saltin et al	T1	2.52	17.2	90	192	14.6
(1968)	T2	3.41	20.0	105	191	17.0
[n = 3]	Δ	+0.89	+2.8	+15	-1	+2.4
	%	+35	+16	+17	-1	+16
Ekblom et al	T1	3.10	22.4	112	200	13.8
(1969)	T2	3.44	24.2	127	192	14.3
[n = 8]		+0.34	+1.8	+15	-8	+0.5
	%	+11	+8	+13	-4	+4
Rowell (1963)	T1	3.42	22.8	118	193	15.1
[n = 6]	T2	3.87	23.8	128	186	16.3
	Δ	+0.45	+1.0	+10	-7	+1.2
	%	+13	+4	+8	-4	+8
Mean**	T1	3.11	21.6	114	196	14.4
	T2	3.59	233	123	190	15.4
	Δ	+0.49	+1.7	+9	-6	+1.0
	%	+16	+8	+8	-3	+7
Young active males						
Saltin et al	T1	4.54	24.3	126	193	18.4
(1968)	T2	4.75	26.8	141	190	17.3
[n = 2]	Δ	+0.21	+2.5	+15	-3	-1.1
	%	+5	+10	+12	-2	-6
Rerych et al	T1	*	25.5	138	185	*
(1980)	T2	*	32.0	176	181	*
[n = 18]	Δ	*	+6.5	+38	-4	*
	%	*	+25	+28	-2	*
Mean**	T1	-	25.8	137	186	-
	T2	-	31.5	172	182	-
	Δ	-	+5.7	+35	-4	-
	%	-	+22	+26	-2	-
Sedentary middle-aged men						
Hartley et al	T1	2.68	18.7	103	182	14.4
(1968)	T2	3.06	21.1	120	176	14.5
[n = 15]	Δ	+0.38	+2.4	+17	-6	+0.1
	%	+14	+12	+16	-3	+1
Present study	T1	2.64	18.4	103	179	14.4
[n = 20]	T2	3.04	22.7	131	173	13.4
	T3	3.10	22.0	127	173	14.0
Δ T1-T3		(+0.46	+3.6	+24	-6	-0.4)
	%	+17	+20	+23	-3	-3

* factor not measured

** Weighted for relative subject numbers.

assessing cardiac output, particularly during exercise. Some of the technical requisites of these methods are described in section 2.6.1(c).

Three of these studies were carried out on previously sedentary young men (18 to 24 yrs) (Ekblom et al, 1968; Saltin et al, 1968; Rowell, 1974). One study was performed on previously conditioned young athletes (Rerych et al, 1980). The final study investigated a group of middle aged men (35 to 55 yrs), (Hartley et al, 1969). The findings of these previous studies and the present are summarized in table 31.

The remainder of this section will be devoted to comparing the findings of the present study with regard to changes in maximal cardiac output and arteriovenous oxygen difference to previous studies. In addition the likely mechanisms responsible these changes will be examined.

4.2.1 Maximal Cardiac Output.

(a) Pattern of change

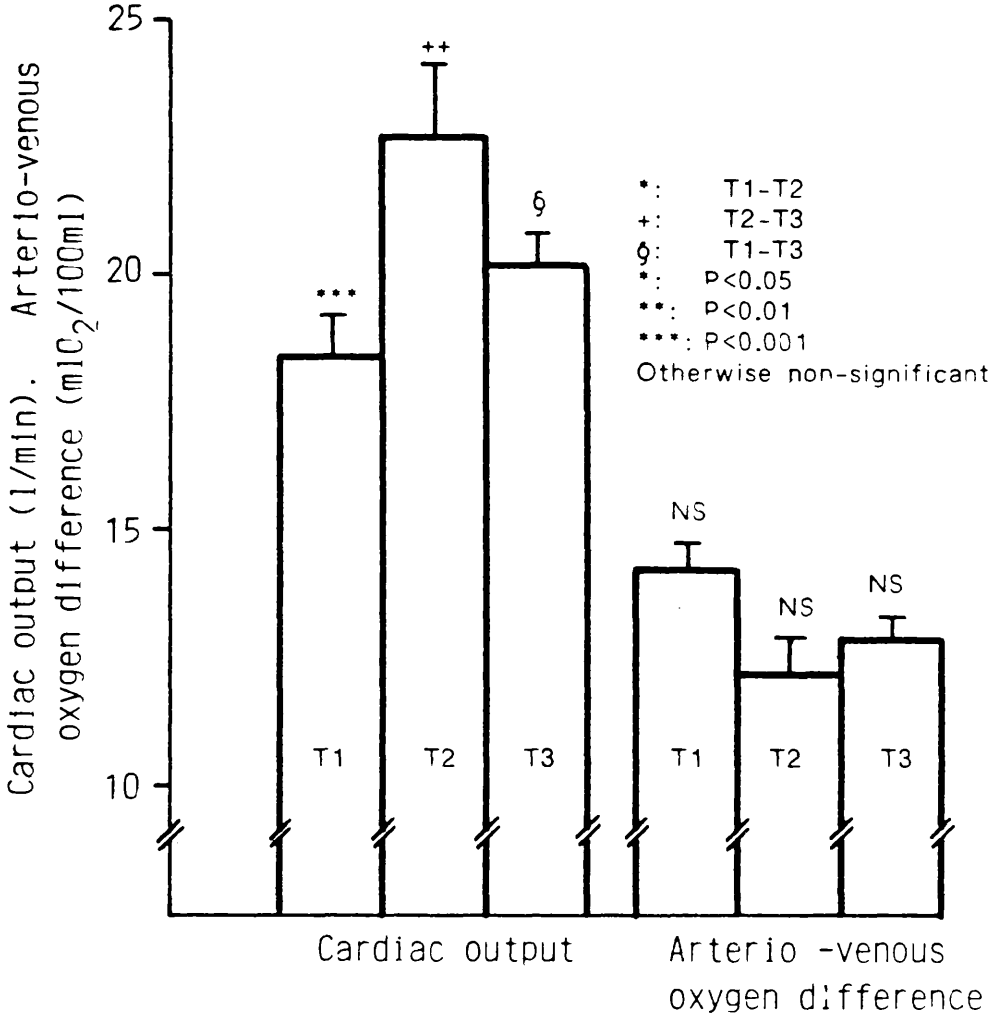
All 5 previous aerobic endurance tráining studies reviewed demonsrated an increase in maximal cardiac output with training. The increase in maximal cardiac output that was observed in this present study after both 15 and 30 weeks training is therefore entirely in accordance with previous studies. (see table 17).

(b) Magnitude of change

Over the 30 weeks of the present study an increase maximal cardiac output of on average 3.6 l/min (19.6%)

FIGURE 27

Summary of cardiac output and arterio-venous oxygen difference changes with training
(means and SE's drawn)



was observed. This aboslute and proportional increase in maximal cardiac output in greater than that typically reported by previous aerobic endurance studies.

A variety of factors may potentially determine the magnitude of increase in maximal cardiac output with training.

(i) Experimental error

It is possible that the magnitude of change in this study is a reflection of experimental error. In this study and that of Reych et al (1980), the technique of radionuclide ventriculography (RNVG) was used to assess cardiac output. Although a relatively new technique the reproducibilty values the present and other laboratories is very high (see section 2.6.1 (b)(v)). Thus it is unlikely that experimental error would contribute to the observed pattern of results.

(ii) Initial Conditioning

Saltin and his colleagues (1968) demonstrated a reduced magnitude of improvement in maximal cardiac output when comparing conditioned and unconditioned individuals, particularly in percentage terms (+ 10.3 & + 16.4% respt.). However, the study of Rerych and his colleagues demonstrated an increase in maximum cardiac output (on average + 6.5 l/min) in athletes over the competitive period. This increase is greater than any other previous study. There did not appear to be any significant relationship between the initial value of

maximal cardiac output and the change in its value with training (r = + 0.28, NS). Therefore it appears that the pre-training value of maximal cardiac output is not a determinant the change in its value with training.

(iii) Subject age

The magnitude of maximal cardiac output change observed in sedentary midddle-age studies appears to similar to that observed in young sedentary male studies. Age, is is apparently not detrminant of the magnitude of cardic output change.

(iv) Training program

Ekblom et al (1968) reported a smaller magnitude of increase in maximal cardiac output in comparison with Saltin's group despite both the studies considering a young sedentary male subject group. This discrepancy was considered reflective of the more strenuous aerobic endurance training program employed by Saltin and his colleagues. However, when Rowell (1974) employed essentailly the same training program as that of Saltin, he reported a smaller magnitude of cardiac output increase. Reviewing previous training studies there did not appear to straight forward relationship between training program characteristics and the magnitude of peak cardiac output change.

From the above evidence, no one single factor is responsible for detrmining the magnitude of peak cardiac output training adaption. However, it is possible that the magnitude of increase in maximal

cardiac output with training is the result of a balance between initial conditioning and the aerobic endurance training program. Thus smaller increase observed by Ekblom et al compared with Saltin et al despite the same training may be the result of the higher initial value of maximal cardiac output in Ekblom's compared to Saltin's study group.

The initial pre-training value of maximal cardiac output in this study (ie 18.4±4.0 l/min) (see Table 16) is essentially the same as has been reported for similarly aged sedentary groups (Hartley et al, 1969). The training demand of the present training program considerably outweighs that of previous studies. Thus it might of be expected that the increase in maximal cardiac output in the present study would be greater than that of previous training studies. In accordance with this hypothesis, the magnitude of maximal cardiac output increase in this study over 30 weeks of training was greater than that observed in the majority of previous studies reviewed.

(b) Mechanism of change

The factors contributing toward an increase in maximal cardiac output will be reviewed with the reference to the present study findings.

(i) Maximal Heart rate

Maximal heart rate is regarded by many as remaining essentially unaltered by aerobic endurnce training (Astrand and Rodahl, 1977). However, athletes

studies have often reported a value of peak heart rate that is less than non-athletic groups of a similar age by about 10 beats/min (Astrand and Rodahl, 1977). Although this observation is thought to be the result of constitutional factors, a training effect cannot be ruled out. Indeed the present study and the previous haemodyn ic studies reviewed in this study all report a fall in maximal heart rate with training.

The peak exercise workload often increases considerably with training. It is therefore doubtful that a drop in maximal heart rate is simply the result of reduced exercise effort. Never-the-less the mechanisms for this reduction are not well established. A reduction in submaximal and resting heart rate with training is considered to be the result of an increase in parasympathetic activity (Lamb, 1984). It is possible that a similar increase in vagal activity is reponsible for the observed reduction in maximal heart rate (Mountcastle, 1980). Ekblom (1967) has suggested that an increase in heart volume at peak exercise may be responsible for this change in autonimic activity. However, experiments have shown that blocking the vagi with atropine does not increase maximal heart rate (Robinson et al, 1953). Thus although aerobic endurance training appears to reduce peak heart rate, the mechanism of this change remains unclear.

As cardiac ouput is the product of heart rate and stroke volume and as the value of peak heart rate with

training is reduced, the increase in maximal cardiac output with training must be the entirely the result of an increased maximal stroke volume.

(ii) Maximal stroke volume

A significant increase was observed in stroke volume with the present training program. This finding agrees with the previous investigative groups reviewed. Despite this agreement the increase in maximal stroke volume with aerobic endurance training, the mechanism of this change remains unclear.

The majority of previous training studies that have assessed the effects of training on cardiac function have used the indicator-dilution technique from which stroke volume is determined by division of cardiac output by heart rate. The cardiac function assessment technique of RNVG was used in the present study and the previous study of Rerych et al. RNVG allows measurement not only of cardiac ouput and stroke volume but also end-systolic and end-diastolic volumes. Thus it is possible with the use of RNVG to assess the relative importance of end-systolic and end-diastolic volumes to an increase in maximal stroke volume.

Stroke volume has been shown to be dependant on preload (loading on the heart prior to contraction), afterload (loading on the heart after contraction) and myocardial contractility (usually dependant on the degree of sympathetic activity) (Berne & Levy, 1974). The potential role of these 3 factors in increasing

peak stroke volume will be examined below.

Preload

Preload is the loading on the heart prior to contraction and therefore reflected by the end-diastolic volume. Provided other factors remain constant, an increased preload will result in an increased stroke volume.

An increase in maximal end-diastolic volume was observed with training in this study while maximal end-systolic volume remained relatively unaltered during this time (see Table 17). This pattern of cardiac volume change is in agreement with that observed by Rerych and his co-workers (1980). This later study was performed on young previously conditioned college swimmers during a competitive season. In view of the difference in age and fitness characteristics of the the present and that of Reych, this training effect is apparently independant of age or initial condiitoning.

The increase in end diastolic volume with training is reflective of an increase in preload. Both the filling time of the heart and rate off venous return may effect these values. As the peak heart rate was slightly reduced with training it is possible that diastolic volume may have increased as the result of an increasd filling time.

A number of studies have shown that aerobic endurance training can lead to a substantial increase

in plasma volume (Saltin et al, 1968; Astrand & Rodahl, 1977). This would contribute towards this increased venous filling with training. Plasma volume was not measured in this study. It has been known for some years that the venous return is affected by the degree of skeletal and respiratory muscle activity, ie. the skeletal/respiratory venous pump. The increase in peak work rate and minute ventilation with training in this study is indicative of an increase in the activity of the venous skeletal/respiratory pump.

Thus the enhanced end-diastolic volume observed in the present study may be the result of increased filling time and increased efficiency of venous return. An increase in end-diastolic volume will lead to an increase in stroke volume presumably by the Frank-Starling mechanism - an increase in diastolic volume increasing the force of contraction of the ventricle so as to maintain a relatively constant systolic volume (Mountcastle, 1980).

Afterload

A reduction in afterload with training may also contribute to this increase in maximal stroke volume with training. Afterload on the myocardium is essentially determined by peripheral vascular resistance, an increase in vascular resistance resulting in a fall in stroke volume. It is possible to determine peripheral vascular resistance indirectly from mean blood pressure and cardiac output.

PVRmax = COmax/MBPmax

Where PVRmax is the peak peripheral vascular resistance

COmax is the maximal cardiac output

MBP is the mean arterial blood pressure.

In this present study although the value of maximal cardiac output increased with training, maximal systolic blood pressure appeared to remain relatively unaltered (see Table 15). Diastolic blood pressure is considered to remain relatively unaltered during exercise in healthy individuals (AHA, 1972). These results are suggestive of that a decrease in maximal peripheral vascular resistance with training may also have enhanced stroke volume. This hypothesis is supported by a similar finding by a review of six previous training studies (Rowell, 1974).

A fall in resistance with training is probably the result of a redistribution of a greater proportion of the total cardiac output through the vasodilated vascular beds of the working skeletal muscle.

Myocardial contractility

An increase in myocardial contractility has been shown in exercise in hypertrophied rats (Froelicher, 1972). Increased contractility will result in a more efficient emptying of the heart, resulting in a reduction in end systolic volume and increased stroke volume. However, the observed failure to reduce end-systolic volume with training, in both the present study and that of Reych et al, is suggestive of no such

increase in myocardial contractility.

Myocardial contractility is a notoriously difficult factor to assess in humans. A parameter that is reported to be reflective of its value is the ejection fraction value (Froelicher, 1983). An increased ejection fraction value is associated with increased myocardial contractility. Ejection fraction is calculated as follows:

$$EF(\%) = ESV/(EDV - ESV) \times 100$$

where LVEF is the left ventricualr ejection fraction

ESV is the end systolic volume

EDV is the end diastolic volume

When ejection fraction values are calculated for this study a slight fall in its value over the 30 weeks of training was observed. (T1: 46±7 %, T2: 45±9 %, T3: 43±5 %). Rerych and his colleagues also found no change in left ventricular ejection fraction with training. This failure of both studies to observe an increase in ejection fraction with training is suggestive that enhanced myocardial contractility was not responsible for the increase in peak stroke volume with training.

4.2.2 Arteriovenous Oxygen Difference.

(a) Pattern of change

The majority of previous training studies assessing the alterations in arteriovenous oxygen difference with training have assessed this value directly by measurement of oxygen content of the arterial and mixed venous blood from catheters in

brachial artery and deep cubital veins respectively (Saltin et al, 1969; Hartley et al, 1968; Ekblom et al, 1968). In this study such direct assessment was not possible and oxygen difference was determined indirectly by the Fick Principle. This indirect method of assessment has also been used by Varnauskas et al. (1966) in their training studies of cardiac patients.

Examination of the review table of previous haemodynamic studies reveals that an increase in arteriovenous oxygen difference with training can contribute upto fifty percent of the observed increase in maximal oxygen uptake. However, some of these studies observed no change in peak arteriovenous oxygen difference over the period of training. A number of factors may be responsible for this differing pattern of training reponse.

(i) Initial conditioning

A widening in peak arteriovenous oxygen difference with training was observed in all three studies involving intially sedentary young men (Saltin et al, 1968; Rowell, 1974; Ekblom et al, 1969). In the case of previously conditioned young men (Saltin et al, 1968), no change in maximal oxygen difference was observed with training. Pre-conditioning apparently prevents an enhancement of peak oxygen difference with training. This inability to increase peak arteriovenous oxygen difference with training in initially conditioned individuals appears to be due to having already reached

optimized values of oxygen extraction prior to training.

(ii) Subject age

Despite the initally sedentary nature of the middle-aged participants of both the present study subjects and that of Hartley et al (1968) no change in maximal oxygen difference was observed with training. Thus it appears that age, like pre-conditioning, may also prevent an increased arteriovenous oxygen difference with training. Hartley and his colleagues proposed that aging is associated reduced trainability of the peripheral oxygen transport system. However, they did not suggest what factors would be responsible for such a change.

(iii) Exercise specificity.

Changes in muscle with aerobic endurance training, such as increase capillarity and oxidative enzyme capacity, are only local (Lamb, 1984). Indeed the the one-leg training studies of Saltin have reported an increase in VO2max with training when the trained leg is exercised and no change in VO2max when the non-trained leg is exercised (Saltin 1983). Therefore it appears that using different muscles reduces the chance of observing an increase in arteriovenous oxygen difference with training.

In both the present study and that of Hartley, the subjects trained by running but were assessed at peak effort on a bicycle ergometer. A situation of exercise

specificity may therefore apply. The study that revealed the greatest widening in oxygen difference with training used the same form of exercise assessment as that carried out in training (Saltin et al, 1968).

Thus it appears that a determinant magnitude of increase in arteriovenous difference with training is the result of the pre-conditioning levels of th subjects. The failure of middle-aged populations, including the present study, is probably not reflective of aging but training specifity. It is probable that if the mode of exercise testing and training were the same in this study, the magnitude of VO2max increase would have risen as the result of an increase peak arteriovenous oxygn difference.

The factors contributing to the increase in arteriovenous oxygen difference with training are considered below.

(b) Mechanism of change

The increase in arteriovenous oxygen difference observed with training in young sedentary groups is reflective of either an increase in the oxygen content of blood or a fall in venous oxygen content.

Only one study has observed an increase in the oxygen carrying capability of arterial blood with training (Detry et al, 1971). This study was carried out on cardiac patients and is therefore probably reflective of the limited pulmonary function, developed as the result of impaired heart pump. The findings of

this study are therefore unlikely to apply to a healthy population. Indeed training studies in healthy individuals have observed no change in peak arterial oxygen content and a fall in venous oxygen content (Saltin et al, 1968; Ekblom et al, 1969).

Thus the increase in maximal oxygen difference with training appears to be the result of a reduction in venous oxygen content. Such an adaption could be the result of more complete extraction of the oxygenated blood in the working muscle or more effective regulation of cardiac output in that less blood is distributed to the the inactive areas of the body such as the splachnic region. In a review of the importance of these two factors by Rowell (1974) concluded that alterations in blood distribution could be ruled out since during exercise in non-conditioned individuals the splachnic beds are already highly constricted. Thus the widening of oxygen difference with training can be attributed to a increase rate of oxygen extraction by the working muscle. Indeed in support of this hypothesis a number of investigators have observed a fall in femoral venous oxygen content (- 5 ml/litre on average) with training (Saltin et al, 1968; Ekblom et al, 1969; Shappel, 1971).

The precise mechanism by which this increased oxygen extraction is accomplished remains unkown. A number potential mechanisms have been suggested: 1) an enhanced diffusion of oxygen from capillary to muscle

because of greater stores of myoglobin in trained muscles, 2) increased capillary density so that oxygen can diffuse more readily into working fibres, 3) a greater ability of skeletal muscle mitochondria to make use of delivered oxygen (Rowell, 1975).

There is evidence that aerobic endurance training does result in greater myoglobin concentrations in trained muscles (Hollosзy & Booth, 1973). However, the role of myoglobin in promoting the diffusion of oxygen during exercise is not proved.

Studies have observed an increased capillary/fibre ratio with training (Brodal et al, 1982). Despite this observation there is not necessarily any relationship between greater capillarity and greater blood flow or oxygen uptake (Maxwell et al, 1980).

It is well documented that many mitochondrial adaptions accompany aerobic endurance training, in particular an increased mass of mitochondria and activities and amounts of the enzymes involved in the Krebs cycle (Hollosзy & Booth, 1976). Calculations by Hollosзy have shown that even though blood flow to the working muscle may ultimately limit maximal oxygen uptake, it is possible that mitochondrial adaptions can allow greater extraction of the delivered oxygen in trained muscles to increase maximal oxygen uptake (Hollosзy, 1973).

4.3 SUBMAXIMAL CARDIORESPIRATORY FUNCTION

Although $\dot{V}O_2$max is often regarded as the most effective index of aerobic endurance function, a number of training program studies have also assessed submaximal cardiorespiratory parameters (Pollock, 1973; Astrand & Rodahl, 1977).

Submaximal measures have the advantage that their assessment does not demand the subject motivation and effort required by maximal parameters. In addition submaximal tests often take less time to perform and do not require such strict medical supervision; practical considerations which are important when testing large groups of individuals. Probably the greatest problem with submaximal exercise data is that it is more influenced by factors such as unfamiliarity with the testing environment, emotion, subject condition and time of day (Shephard et al, 1968b). However, the same studies have shown that if these factors are carefully controlled, the reproducibility of submaximal cardiorespiratory measures can be high.

In this section the changes observed during the present training program in the submaximal cardiorespiratory parameters of heart rate, oxygen uptake and pulmonary ventilation will be considered. The possible mechanisms responsible for these changes and their physiological significance will also be

discussed.

4.3.1 Submaximal Heart Rate

(a) Pattern of change

A significant reduction in heart rate was observed in the present study during both treadmill and bicycle ergometer exercise at a standard exercise workload. This 'training bradycardia' confirms what is probably one of the most classically reported effects of both cross-sectional and longitudinal aerobic endurance training studies (Pollock, 1973, ASCM, 1978). Indeed the response of submaximal heart rate, together with VO_2max, is often used as a measure of the efficacy of an aerobic endurance program (Astrand & Rodahl, 1978).

(b) Magnitude of change

The magnitude of submaximal heart rate reduction reported in previous training, like the changes in VO_2max, can vary significantly. However, most aerobic endurance training programs report some degree of submaximal exercise bradycardia. Indeed a review by Clausen (1977), noted that it was not unusual to observe a 20 to 40 beat/minute decrease in submaximal heart rate following a 6 month aerobic endurance program of moderate intensity.

In the present study a range of reduction in submaximal treadmill and bicyle ergometer exercise heart rate of 14 to 35 beats/minute was observed after 30 weeks training. The magnitude of this reduction during treadmill exercise (22 to 35 beats/min) was

greater than that of bicycle ergometer (14 to 22 beats/min). It appears this magnitude of reduction submaximal exercise heart rate is of a similar order to that quoted earlier by Clausen. Indeed when assessing the training bradycardia effect of the present program by bicycle ergometer exercise, the magnitude of change was on average less than previous training studies.

The factors responisble for determining the magnitude of submaximal heart rate adaption with training will be discussed below.

(i) Experimental error

Shephard (1970) observed reduction in heart rate with repetition of the same absolute submaximal workload, despite no change in VO_2max. This decline in heart rate is accounted for by a reduction in subject anxiety and an increase in mechanical efficiency. This effect is termed "familiarization". It was discussed earlier that a variety of factors, such as laboratory temperature and subject familarisation, can influence submaximal heart rate. It is therefore possible that the reduction in heart rate observed in this study is not a training effect per se but the result of expermental errors. Never-the-less as has been discussed in section 2.5.3, laboratory conditions were maintained constant throughout testing and in addition all subjects were given a period of familiarisation with the testing equipiment.

(ii) Aerobic efficiency

Prediction of VO_2max from submaximal heart rate values is dependant on the assumption of a linear relationship between heart rate and oxygen uptake (Astrand & Rodahl, 1977). Therefore it has been suggested that training bradycardia is secondary to an increase in mechanical efficiency or aerobic efficiency rather than a direct effect of training on heart rate itself (Cotes & Mead, 1959).

A significant reduction was observed in oxygen uptake during standard submaximal exercise on both bicycle ergometer and treadmill. It is therefore plausible that this increase in aerobic efficiency may be responsible for the bradycardia observed in this study. To examine this hypothesis treadmill and bicycle ergometer submaximal heart rate was plotted versus oxygen uptake at each stage of training (see figure 28). Although these figures demonstrate a reduction in the magnitude of heart rate response when expressed relative to oxygen cost of exercise, reveals that there is still a reduction in heart rate that is unaccounted for. Indeed expressing submaximal heart rate and oxygen uptake jointly as a value of oxygen pulse, reveals that there is a significant increase in this value with training (see figure 29).

Thus although a proportion of submaximal exercise training bradycardia in this study is secondary to a reduction in submaximal oxygen uptake other factors

FIGURE 28

Submaximal heart rate - absolute oxygen uptake relationship for bicycle ergometer and treadmill exercise

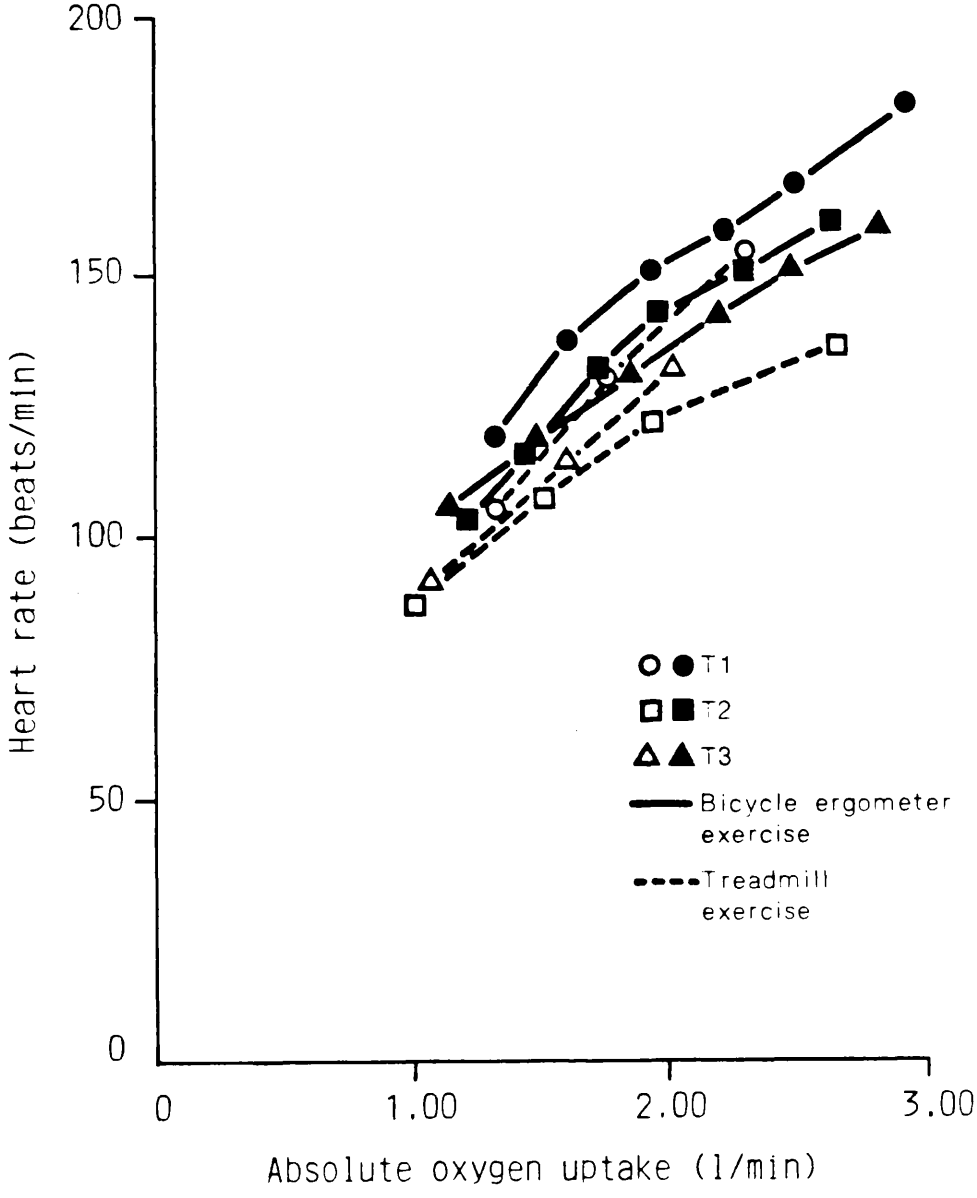
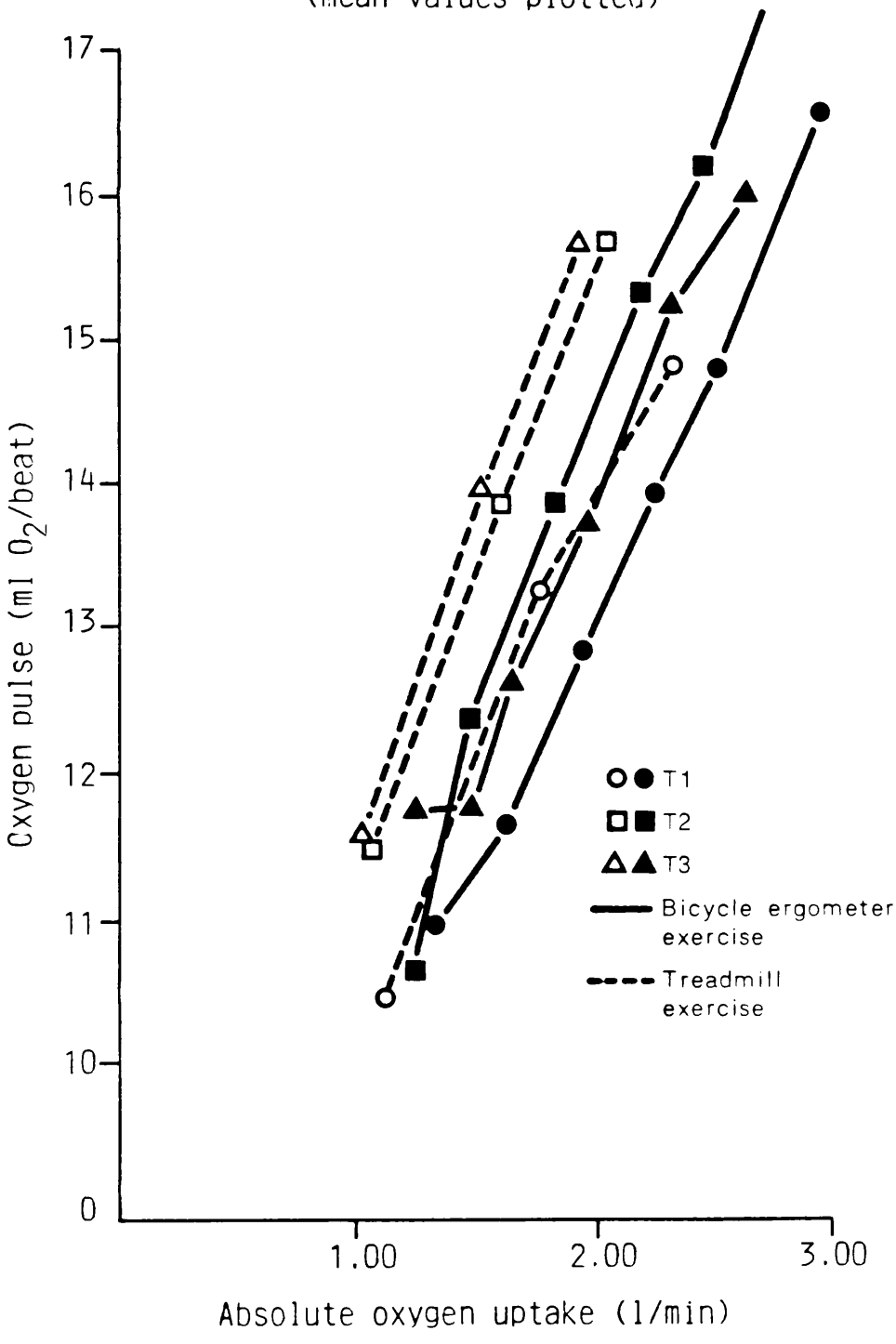


FIGURE 29

Submaximal oxygen pulse for bicycle ergometer and treadmill exercise
(mean values plotted)



must also be responsible.

(iii) Training program

Like VO_2max, the relative intensity (Pollock et al, 1969), duration and frequency (Fox et al, 1975) of the training program has been shown to be an important factor in determining the degree of training bradycardia. In general the greater the demand of the program, the greater the degree of bradycardia.

Despite the demanding nature of the present training program the magnitude of training bradycardia was not greater than that observed in previous typically less demanding studies. However, recent evidence that of the various indices of training effort, training intensity is the most important in determining the magnitude of reduction in submaximal heart rate (Mellorowicz & Smodlaka, 1981).

(iv) Initial fitness

The initial value of submaximal heart rate also appears to determine the relative magnitude of training bradycardia (Astrand and Rodahl, 1977). The greater the initial value, the greater the magnitude of change. When comparing the response of a previously trained population to an untrained population the magnitude of reduction is in general greater in the untrained group (Pollock, 1973). The higher the initial submaximal heart rates, the greater the magnitude of training induced bradycardia.

It is difficult to determine the potential effect

of initial submaximal heart rate values in the present study because of the lack of comparitive heart rate values for the present exercise tests. However, the pre-training VO_2max values for the present study group indicated a initial value of aerobic endurance fitness that is similar to previous training studies. Thus the level of initial fitness of the present study subjects probably did not influence the magitude of training bradycardia.

(v) Training specifity

Figure 26 demonstrates that over the range of submaximal oxygen uptake the value of heart rate during treadmill exercise was less than that of bicycle ergometer exercise. This finding is in agreement with previous studies (Hermansen & Saltin, 1969). It is proposed that this discrepancy is reflective of the higher intramuscular pressure at a given oxygen uptake value during bicycle exercise, with this pressure in turn reducing the return of blood to the heart and thus the stroke volume. To compensate for this the heart rate rises.

In addition figure 26 also demonstrates that the magnitude of training bradycardia observed during treadmill exercise was greater than that of bicycle ergometer exercise. This finding of exercise specifity with regard to submaximal heart rate is in accordance with the previous studies that have used different modes of exercise testing (Pechar et al, 1974; Wilmore

et al, 1980). The mechanism of this exercise specific effect will be discussed below.

From the above review the magnitude of training bradycardia in the present study might of been expected to be greater than previous aerobic endurance studies which are typically of a less demanding training effort. Taking into account the other potential contributory factors it is not clear why such a increased magnitude of training bradycardia not observed in this study.

(c) Mechanism of change

It is commonly accepted that the reduction in submaximal heart rate with training is the result of a change in autonomic balance. The sympathetic and parasympathetic balance alters so that there is a greater dominance of the parasympathetic component (Ekblom et al, 1967). However, there is no completely satisfactory explanation as to the precise mechanism of this change (Astrand & Rodahl, 1978). A review of the possible mechanisms by Clausen (1977) has proposed two potential mechanisms for training induced submaximal exercise bradycardia. The first is a central mechanism in which intra-cardiac factors are responsible and the second a peripheral mechanism in which extra-cardiac factors are responsible. The importance of these two mechanisms in the present study will be discussed below.

The central hypothesis of submaximal bradycardia

has been suggested since the early training studies of Christensen (1931) who observed that the fall in exercise heart rate was accompanied by a rise in stroke volume and he therefore concluded that the bradycardia was secondary to an increase in stroke volume. An increase in stroke volume would presumably reduce sympathetic drive to the heart which will in turn reduce heart rate. Possible explanations for this increase in stroke volume were in turn thought to be the result of an increase in both blood volume and myocardial contractility (Clausen, 1977). Indeed recent studies have demonstrated an increase in left ventricular end-diastolic volume and myocardial hypertrophy with training thus suggesting increases in heart filling and myocardial contractility respectively (Perrault et al, 1982). According to the Frank Starling mechanism of the heart, such an increase in heart filling will increase the contractile properties of the heart and thereby improve stroke volume (Mountcastle, 1980). A reduction in the submaximal exercise circulating catecholamine levels has been reported with training (Winder et al, 1978). Such a reduction could also be a mechanism for bradycardia due to a reduced rate of sinoatrial activity. (Smith & El-Hage, 1978). A final potential mechanism is that of stroke volume increase due to a decrease in cardiac afterload. In support of this a fall was observed in the present study in the systolic blood pressure response to bicycle ergometer exercise with

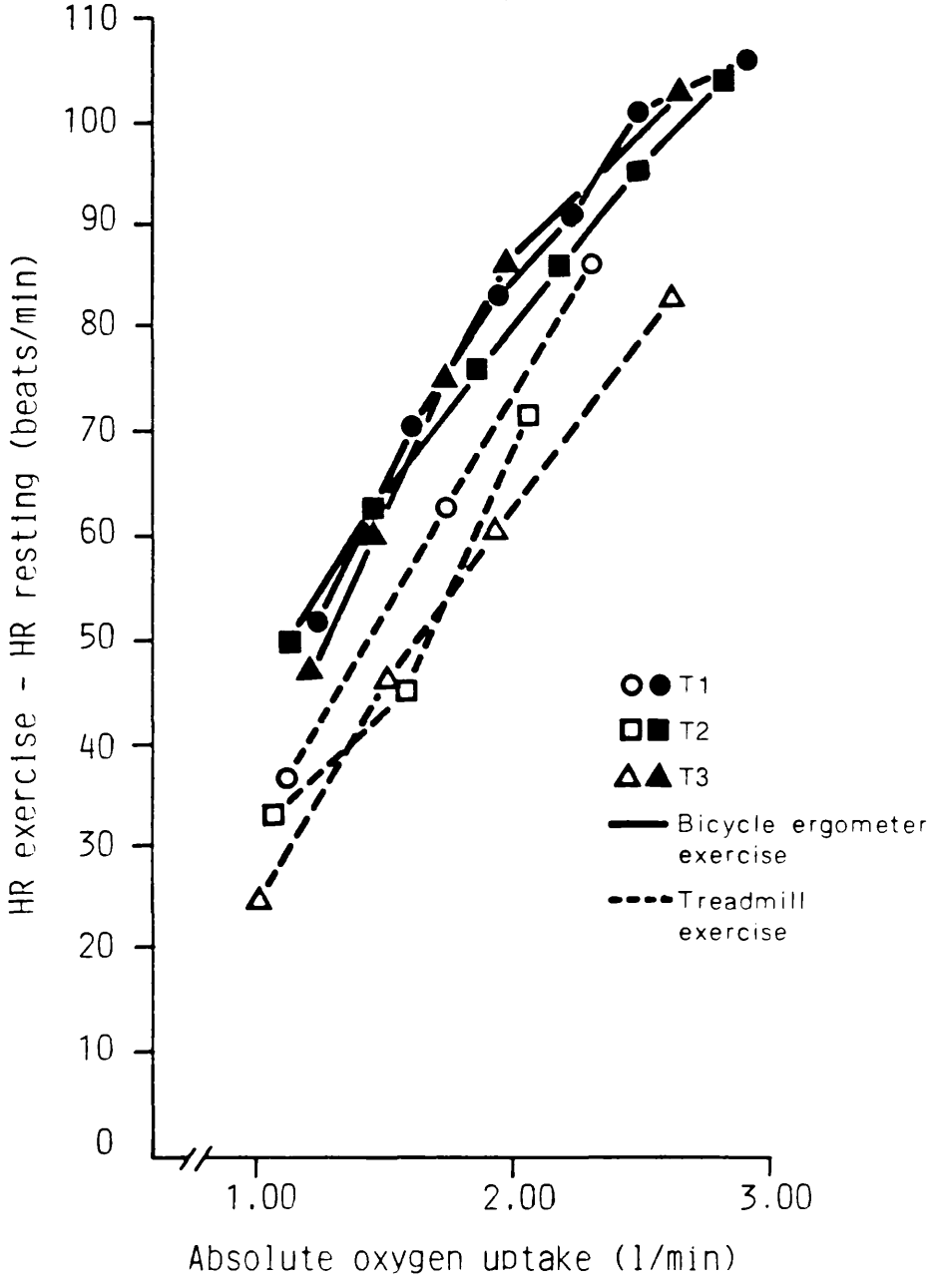
training (see table 14 and figure 17). Although diastolic blood pressure was not assessed during exercise in this study previous work suggests that its value changes very little from rest to peak exercise and if anything may slighlty fall (Astrand & Rodahl, 1977). A fall in exercise systolic blood pressure in combination with little change in diastolic blood pressure indicates a fall in mean arterial blood pressure with training. Such a reduction in mean blood pressure would reduce the cardiac afterload thereby allowing for an enhanced submaximal stroke volume.

In his review, Clausen suggests that if an intra-cardiac mechanism of submaximal exercise bradycardia were to be true then it should be demonstrable not only in submaximal exercise heart rate but also at resting heart rate. Indeed if the intracardiac factors were the only ones modified by aerobic endurance training then the submaximal exercise bradycardia would of a similar magnitude to resting bradycardia. Although a reduction in resting heart rate was observed after training and prior to both treadmill and bicycle ergometer exercise, correction of the submaximal exercise heart rate for this change in rest did not completely take account of the bradycardia effect (see figure 30).

It therefore appears that intra-cardiac or central factors are not the only ones responsible for observed reductions in submaximal heart rate with training. The

FIGURE 30

Submaximal heart rate corrected for resting heart rate changes with heart rate
(mean values plotted)



suggestion of the involvement of a peripheral mechanism supports the findings of Saltin and his colleagues (1977) which were derived from single leg or arm training studies. They observed a decrease in submaximal heart rate when exercising the trained limb but not the untrained limb. This latter finding suggested that peripheral factors arising from the actual trained muscle were important in determining the training induced bradycardia.

In the present study the average magnitude of submaximal exercise bradycardia observed with training, taking account of reductions in both submaximal oxygen uptake and resting heart rate during treadmill exercise, was significantly greater ($P < 0.001$) in comparison with those observed during bicycle ergometer exercise. In view of the mode of training of the present training program, this discrepancy suggests that a proportion of the observed bradycardia was the direct result of changes within the trained muscle groups. It has been proposed that improvement in the efficiency of muscle metabolism may modify afferent nervous discharge from working muscles, reducing sympathetic drive from the cortex which will in turn reduce exercise heart rate (Clausen , 1977). In addition it has also been proposed that an improvement in muscle oxidation will increase the ability to extract oxygen and thereby increase the arterio-venous oxygen difference which may also contribute towards

bradycardia (Froelicher, 1983).

Fox and his colleagues (1975) have suggested that an extracardiac mechanism of exercise bradycardia is reflective of a relationship between the reduction in submaximal heart rate and the increase in VO_2max with training. This observation is presumably based on the assumption that the two parameters are interrelated by the arteriovenous oxygen difference. In agreement with Fox's group , a poor correlation was observed between the change in submaximal bicycle ergometer heart rate and VO_2max with training (75 watts: r=-0.11, 100 watts: r = -0.12, 125 watts: -0.20, 150 watts: r = -0.25 , all NS). This finding is suggestive that no bradycardial response with training is accounted for by an increase in arteriovenous oxygen difference. However, this does not exclude the possibility of other extracardiac mechanisms contributing towards exercise bradycardia in this study.

A reduction in post training submaximal heart rate was observed in the present study during both bicycle ergometer and treadmill exercise. Although this bicycle ergometer reduction was less than that of treadmill it does suggest that there was transfer of the peripheral training effects of the present running program to the bicycle ergometer. Although this finding appears to contradict the one limb training study of Saltin and his colleagues in which no bradycardia was observed when the non-trained muscles were exercised, the

findings of this study have been observed before (Roberts and Aspaugh 1972; McArdle et al, 1978) have both observed reductions in submaximal heart rate post running training when testing with bicycle ergometer and tethered swimming. A possible explanation for the discrepancy between the present and above studies versus that of Saltin is that running, which activates a relatively large muscle mass, modifies submaximal heart via either intra-cardiac or extra-cardiac factors while smaller muscle mass activities, such as a single limb, are more extra-cardiac in nature.

Thus it appears that in this present study the observed training induced submaximal exercise bradycardia is the result of what appears to be both central and peripheral mechanisms. This finding of both intra-cardiac and extra-cardiac mechanisms of submaximal exercise heart rate reduction with training is in agreement with the conclusions of Clausen (1977).

(d) Physiological significance

The reduction in submaximal heart rate with training is often considered to be one of the most important physiological benefits to be gained from aerobic endurance training. A reduction in submaximal heart rate, particularly if there is also a reduction in submaximal blood pressure, indicates a reduction in rate/pressure product of the heart and is suggestive of a reduction in myocardial oxygen demand (Froelicher, 1983). Such an adaptation can be particularly important

to individuals with exercise induced myocardial ischemia as the result of coronary heart disease.

The prediction of VO_2max is based on the assumption that a lower heart rate at a particular workload after training is indicative of an improvement in VO2max (Astrand & Rodahl, 1977). Because of exercise specifity, the magnitude of heart rate reduction observed with training during treadmill and bicycle ergometer exercise were not the same in this study. Thus the magnitude of VO_2max prediction from the two forms of exercise would also not be the same. Because of the greater extent of training bradycardia during treadmill exercise, the predicted treadmill VO_2max value would outweigh the predicted value from bicycle ergometer exercise.

The value of VO_2max predicted from submaximal values at each stage of the present training study is shown in Figure 31. The 33% increase in treadmill predicted VO2max over 30 weeks is considerably greater than may be expected from the prediction of submaximal bicycle ergometer values.

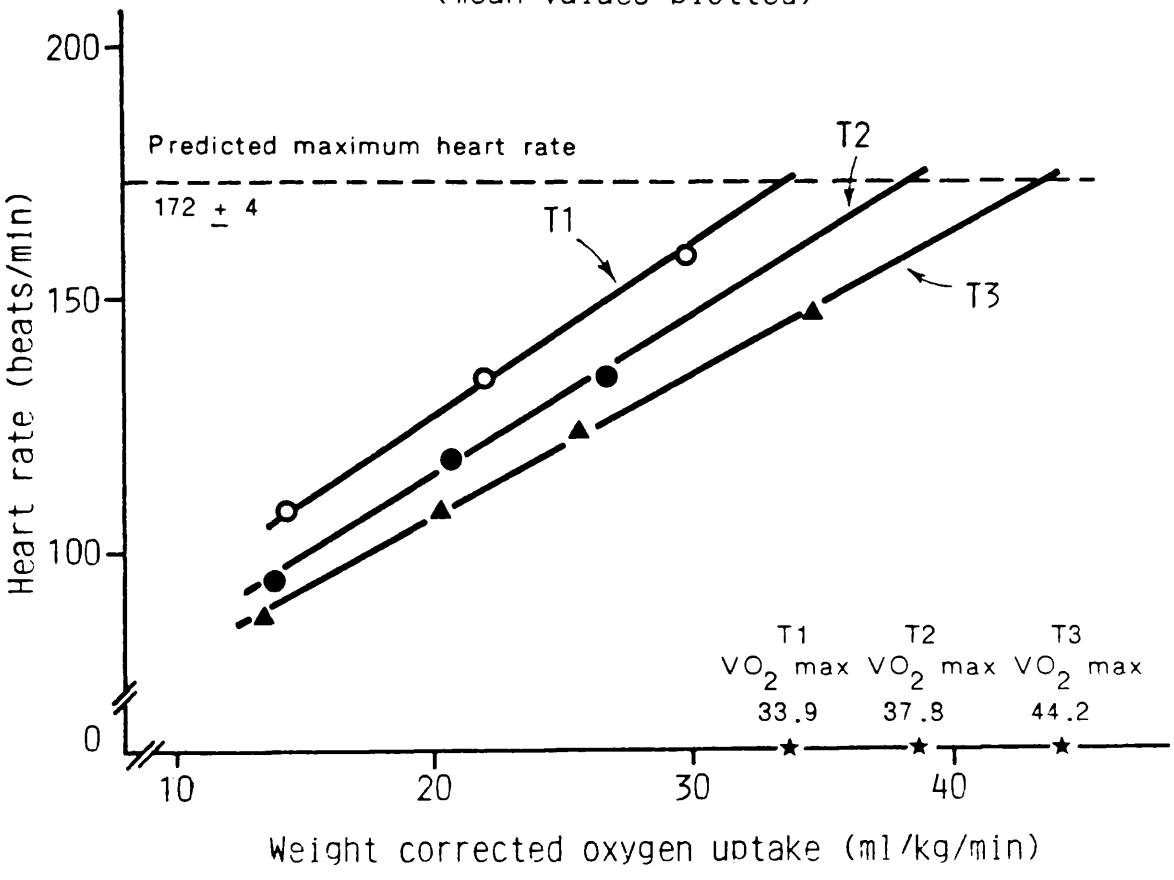
It should be noted that because of differences in the type of exercise the values of predicted VO2max for bicycle ergometer and treadmill exercise may not be the same anyway.

4.3.2 Submaximal oxygen uptake

Cross sectional studies have shown that athletes

FIGURE 31

Treadmill predicted VO_2 max values from extrapolation of treadmill exercise heart rate versus oxygen uptake
(mean values plotted)



tend to consume less oxygen at a given treadmill workload than their non athletic counterparts (Dill et al, 1965). Indeed aerobic efficiency is thought to be an important factor in determining distance running performance (Davies & Jakeman, 1982). However, it is difficult to separate from cross-sectional studies the effect of inheritance and the effect of training per se.

Longitudinal studies are less conclusive than cross-sectional, the majority revealing little or no change in the value of oxygen uptake at standard submaximal workload (Astrand & Rodahl, 1977). Indeed those that have shown a decrease in submaximal oxygen uptake with training have also observed a similar response in the control group (Patton & Vogel, 1977). Such a control reponse is suggestive that the decrease in oxygen uptake is a practice effect rather than a training effect per se. Shephard (1970) has clearly demonstrated, with both bicycle ergometer and treadmill exercise, this improvement in aerobic efficiency with familiarization. He also observed that repeated practice of these testing procedures stabilized the value of submaximal oxygen uptake.

It has also been suggested that the incidence of training induced improvements in aerobic efficiency increases with activities that demand increasing skill. McArdle and his colleagues (1978) observed that in a group of jogging trained athletes there was no significant change in pre and post training treadmill

submaximal oxygen uptake but there was a significant decrease in tethered swimming oxygen uptake. This effect presumably illustrates the increased skill demand of the latter form of activity.

The effect of the present training study on submaximal oxygen uptake will be discussed below.

(a) Pattern of change

Contrary to previous studies a substantial and statistically significant reduction in oxygen uptake during standard treadmill and bicycle ergometer exercise was observed in this study (see tables 7,8,20 & 21 figures 11,12,20 & 21).

(b) Magnitude of change

The relative magnitude of this improvement in aerobic efficiency in this study appeared to be dependant on two factors - the initial value of oxygen uptake for each subject and the level of exercise being performed. The reduction in treadmill submaximal oxygen uptake increased the greater the value of initial oxygen uptake ($r = +0.60$, $P < 0.01$). A smaller but statistically significant relationship was observed during bicycle ergometer exercise ($r = +0.22$, NS). In addition the absolute and relative magnitude in oxygen uptake decrement with training appeared to increase with increasing treadmill and bicycle ergometer work load (see Figures 32 & 33).

It is difficult to evaluate the magnitude of

FIGURE 32

Comparison of change in submaximal oxygen uptake of treadmill and bicycle ergometer exercise with training
(mean values plotted)

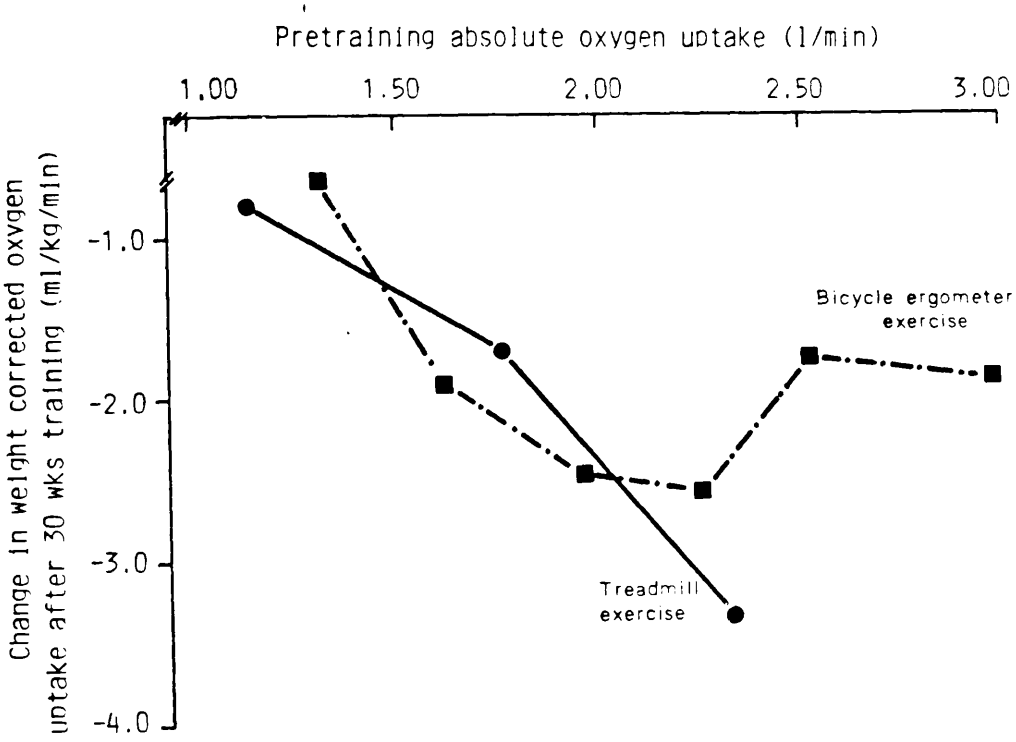
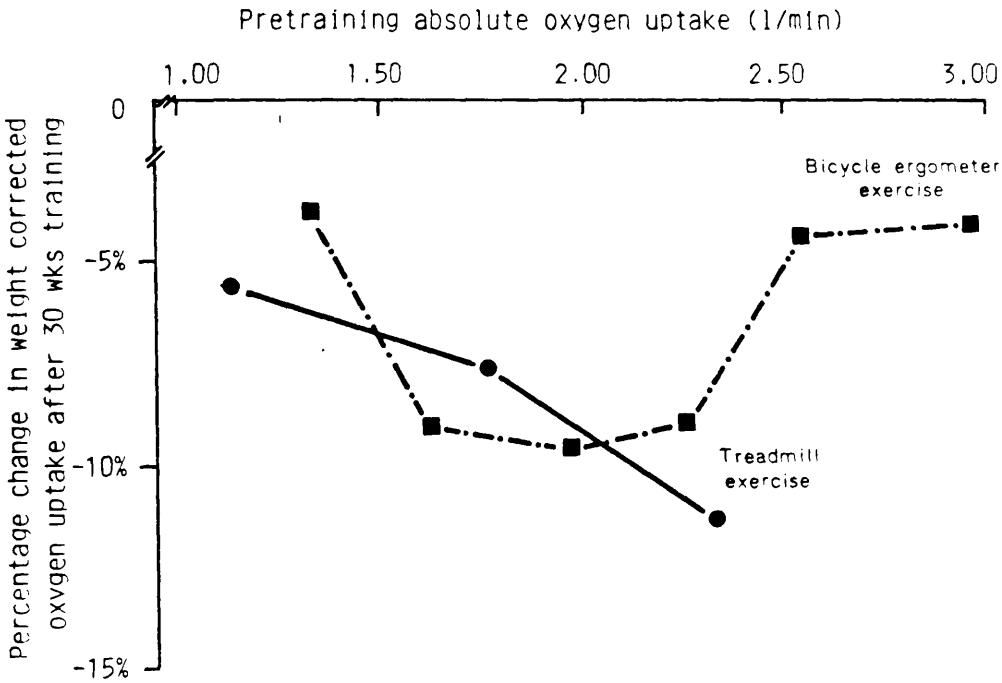


FIGURE 33

Comparison of percentage change in submaximal oxygen uptake of treadmill and bicycle ergometer exercise with training
(mean values plotted)



change in aerobic efficiency because of the small numbers of longitudinal training studies that reported a change in this parameter. Cross-sectonal studies comparing athlete to their non-athletic counterparts report differences of 17 to 20 percent (Daniels, 1985). Although the 5 to 15 percent range of reduction in submaximal oxygen cost in this study is less than that of athletes studies it does take into acount possible consitutional differences.

(c) Mechanism of change

In view of the lack of studies which have observed any change in submaximal oxygen uptake with training there are no commonly accepted mechanisms for the training induced increase in aerobic efficiency observed in this study. A number of potential mechanisms will be reviewed below with regard to the findings of the present study.

(i) Experimental factors

It is plausible that the reduction in oxygen uptake in this study was the result of experimental factors such changes in the testing environment or errors in metabolic analysis. However as described in sections 2.5 and 2.6.1, attempts were made to reduce these factors to a minimum.

(ii) Familiarisation

To reduce the likelihood of a decrease in submaximal oxygen uptake as the result of a practice effect, the subjects in this study were allowed to

practice the activities of treadmill and bicycle ergometer exercise prior to testing. As no control group participated in this study, it is difficult to evaluate the effectiveness of this familiarisation.

(ii) Reduction in body weight

It has been shown that the oxygen uptake in weight bearing exercise, such as walking, is directly proportional to total body weight, the greater the weight the more oxygen that is required to perform the exercise (Durnin & Passmore, 1967)

A significant reduction in mean body weight was observed post training in this study. Thus it might be expected that the reduction in absolute value of treadmill oxygen uptake was the secondary to this change in body composition. Expressing treadmill oxygen uptake as weight corrected values reduced the relative magnitude of decrease in oxygen uptake training. Thus although changes in body weight in this study contributed toward the reduction in treadmill oxygen cost it does not completely account for the observed magnitude of change.

(iii) Oxygen uptake kinetics

It has been shown that training can increase the rate at which the submaximal oxygen uptake steady state is achieved (Whip & Wassmerman, 1972; Hagberg et al, 1980). This is suggestive of a reduction in the degree of anaerobesis during the early stages of exercise as the result of an increase in oxidative activity of the

working muscles.

This increase in non steady state oxygen uptake tends to suggest an increase in overall submaximal oxygen uptake. However this need not be so. A shift towards oxidative metabolism with training would increase the extent of aerobic fat metabolism, a process that is known to produce a much more improved level of ATP than anaerobic glycolysis. Moreover, although initial lactate production may not involve oxygen its metabolism and breakdown does. Thus in view of the greater energy efficiency of aerobic metabolism it is feasible that an increase in skeletal muscle oxidative metabolism would lead to a reduction in overall oxygen uptake by reducing the level of oxygen extraction.

(iv) Reduced basal oxygen uptake

If the level of resting oxygen uptake was reduced it might be expected that the submaximal oxygen uptake would decrease to a similar level so that the increase in oxygen uptake with exercise remains constant. In support of this notion Simmons and Shephard (1975) have observed equivalent reductions in resting and submaximal oxygen uptake post training. This reduction in resting oxygen uptake was attributed to a reduced postural tension while sitting on the ergometer. Although no measurement of resting oxygen uptake was made in this study, the method of bicycle ergometer exercise demanded that the subjects hold their chest

against a gamma camera at times during the test, a situation that may have increased postural tension but which could, with practice, be more easily accomodated.

(vi) Exercise specifity

Despite taking the factors of body weight change, oxygen kinetics and basal oxygen uptake into account the magnitude (both absolute and relative) of reduction in oxygen uptake during submaximal treadmill exercise exceeded that of bicycle ergometer exercise. This discrepancy is possibly indicative of exercise specific adaptions biomechanical efficency.

Training is known to result in a more effective pattern of neural recruitment of skeletal muscle in which the inhibition of anatagonists and recruitment of agonists is effectively increased resulting in a more effective biomechanical pattern of upper and lower limb usage eg. a reduction of arm swing and choice of effective stride length during running (Astrand & Rodahl, 1977). Such an increased biomechanical efficiency would lead to a reduction in the oxygen cost of exercise. It would be expected that this neuromuscular adaption would be exercise specific and therefore only be revealed in testing using the same or a similar mode of exercise to that of training.

(c) Physiological significance

As discussed above, studies on endurance athletes have shown that a reduced oxygen cost of running is a important factor in enhancing endurance running

performance. This presumably reflects the fact that a reduction in submaximal oxygen uptake will, like an increase in VO_2max, reduce the relative intensity of effort as defined by %VO_2max. The time for which aerobic endurance exercise time can be sustained is inversely related to the relative intensity at which exercise is performed (Pacy et al, 1986).

Daniels and his colleagues illustrated the importance of aerobic efficiency by considering two female athletes (Daniels et al, 1985). Although one female had a considerably higher VO_2max (73.3 vs 60.4 ml/kg/min) than the other they produced similar running performance. This apparent discrepancy was resolved by the athlete with the lower aerobic power having a aerobic efficiency while running that outweighed the other so that at race speed both performed at the same relative intensity. It would therefore be expected that improvement in aerobic efficiency with the present training would explain an enhancement in running performance, in particular in the later 15 weeks of training when aerobic power failed to change.

The prediction of VO_2max in based on the assumption that training only modifies the heart response to exercise while the energy cost of submaximal exercise remains constant (Astrand & Rodahl, 1977). If a method of VO_2max prediction is used where submaximal oxygen cost is not assessed such as the Astrand-Rhyming nomogram (Astrand & Rhyming, 1954) an

improvement in aerobic efficiency result in overprediction.

This error can be illustrated in the present study using the average submaximal treadmill exercise heart rate and oxygen uptake data for the group. VO_2max value can be predicted from these values using linear extrapolation (Maritz et al, 1961). Figure 34 illustrates the extrapolation of the actual oxygen uptake values obtained at each stage of training as well as the extrapolation based on the assumption of a constant aerobic efficiency. It can be seen that this assumption leads to a considerable (15%) over-prediction of VO_2max value for the group. This error illustrates the importance when predicting VO_2max in actually measuring the value of exercise oxygen cost before and after training.

4.3.3 Submaximal Pulmonary ventilation

The assessment of oxygen uptake involves determination of minute ventilation (Consolazio et al, 1963). Despite this pumonary ventilation is a seldomly reported physiological parameter in training studies. This omission probably reflects three factors.

First, it is usually considered that submaximal ventilation is unaltered by training and that if it does change then the magnitude of this change is usually small. Indeed the majority of training studies that do report decreases in ventilation with training are cross-sectional and compare highly trained

FIGURE 34A

Illustration of error in prediction of change in predicted VO_2 max assuming a constant mechanical efficiency

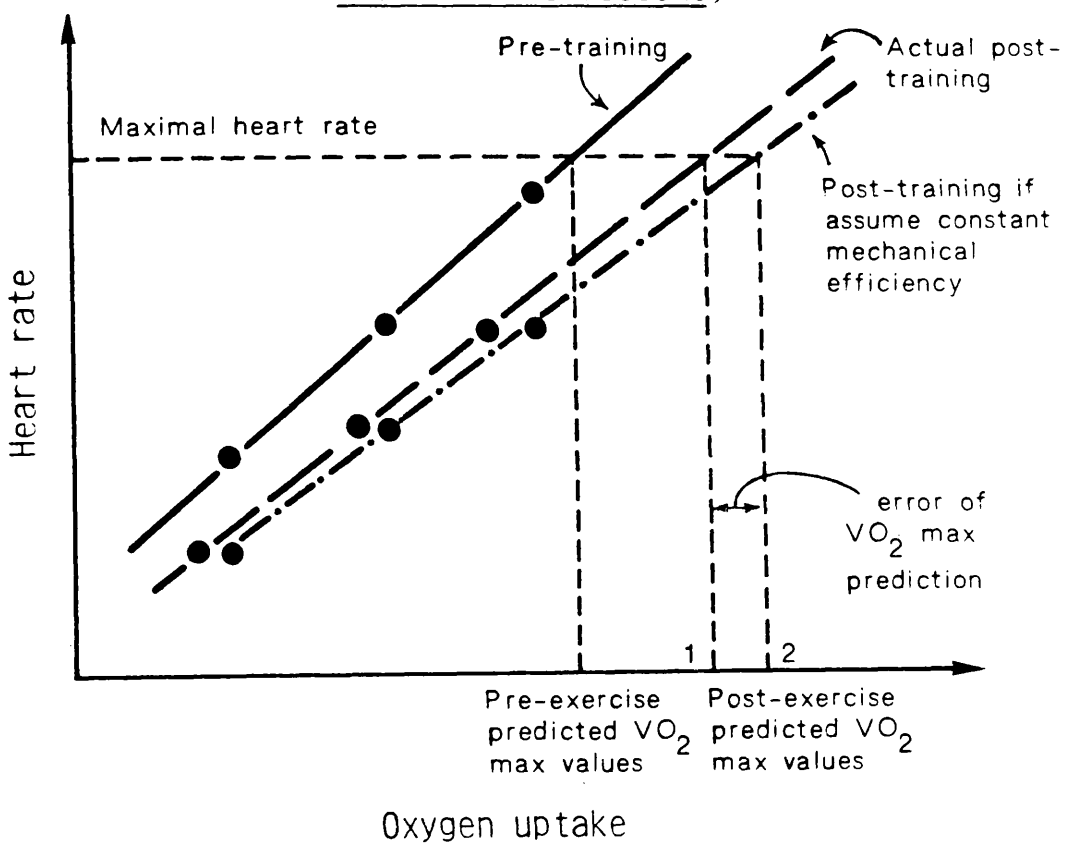
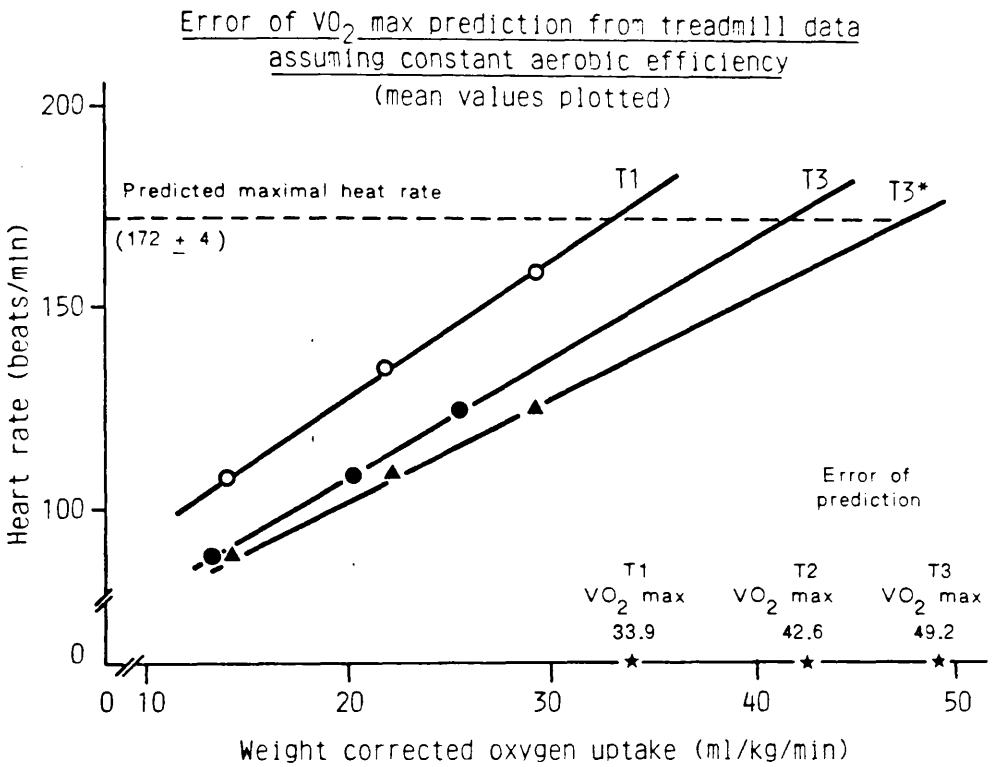


FIGURE 34B



T1 - Actual T1 values

T3 - Actual T3 values based on no 4mph 10% gradient value

T3* - T3 values assuming constent aerobic efficiency

endurance athletes with non athletes (Martin et al, 1979). It is therefore difficult to determine if this difference is a measure of training or genetic factors. Secondly, the significance of a change in submaximal ventilation on aerobic endurance performance has not been determined. However, that the above studies have shown that athletes do in fact appear to have a lower submaximal ventilation suggests that this factor may in some way be associated with an increase in aerobic endurance performance. The final factor that may have reduced the number of training studies examining the respiratory effects of training, as has been discussed in a previus section, is that aerobic performance is limited by cardiovascular and not respiratory factors. However, the fact that respiratoy factors are not limiting does not necessarily dictate that training cannot result in respiratory adaptation. In view of the potential short-comings of each of the above three factors the present section reports on the changes in submaximal ventilation observed with this present training study and discusses the mechanism and physiological significance of such changes.

(a) Pattern of change

In this present study a decrease was observed in submaximal ventilation during the period of training. A significant decrease was observed in submaximal ventilation during both bicycle ergometer and treadmill exercise (see tables 10 & 23 and figures 14 & 23). This

reduction in ventilation is in agreement with the limited number of training studies that have reported this variable (Ekblom et al, 1968; Patton & Vogel, 1977). However, in view of the lack of previous training studies which have reported changes in submaximal ventilation it is not possible to compare the magnitude of change of the present study with previous training studies.

(b) Mechanism of change

(i) Experimental error

Submaximal ventilation like other submaximal cardiorespiratory factors is prone to variation as the result of subject familiarisation (Patton & Vogel, 1977) and environmental change, such as increased temperature (Astrand & Rodahl, 1977). However, as has been discussed in the above section every effort was made in the present study to lessen the effects of such factors. It can therefore be assumed that the changes in submaximal ventilation are the result of the present program of training.

(ii) Aerobic Efficiency

As described in section 4.3.2, submaximal oxygen uptake decreased with training. It is therefore plausible that the observed reduction in submaximal ventilation in this study was secondary to an improvement in mechanical efficiency. To test this hypothesis the values of minute ventilation during treadmill and bicycle ergomter exercise were plotted

against oxygen uptake (see Figure 35). This figure reveals that although there ventilation decreased with training during treadmill but not bicycle ergometer exercise. Expressing the values of ventilation as ventilatory equivalents (ie. Ventilation/Oxygen uptake) also revealed a change in ventilatory function only during treadmill ventilation exercise (see Figure 36).

Ventilatory equivalent is considered an indicator of ventilatory efficiency and its reduction suggests an ability to uptake the same amount of oxygen into the body despite a reduced inspiration volume of air. Factors must reponsible for this training adaption ar considered below.

(iii) Exercise specifity

A decrease in ventilatory equivalent was only observed during treadmill exercise and not during bicycle ergometer exercise. This finding is suggestive that the mechanism of the training induced reduction in ventilation is specific to the utilization of trained muscle groups and therefore local muscle adaptation.

This hypothesis of a local muscle contribution to submaximal ventilation has been confirmed by other studies (Rasmussen et al, 1975; Pannier et al, 1980). Rasmussen and his colleagues observed a reduction in submaximal ventilation only when the same limb was both exercised and trained. They suggested that this local muscle adaptation was the result of reduced sympathetic activity, as the result of a reduction in afferent

FIGURE 35

Change in pulmonary ventilation versus absolute oxygen uptake with training (mean value plotted)

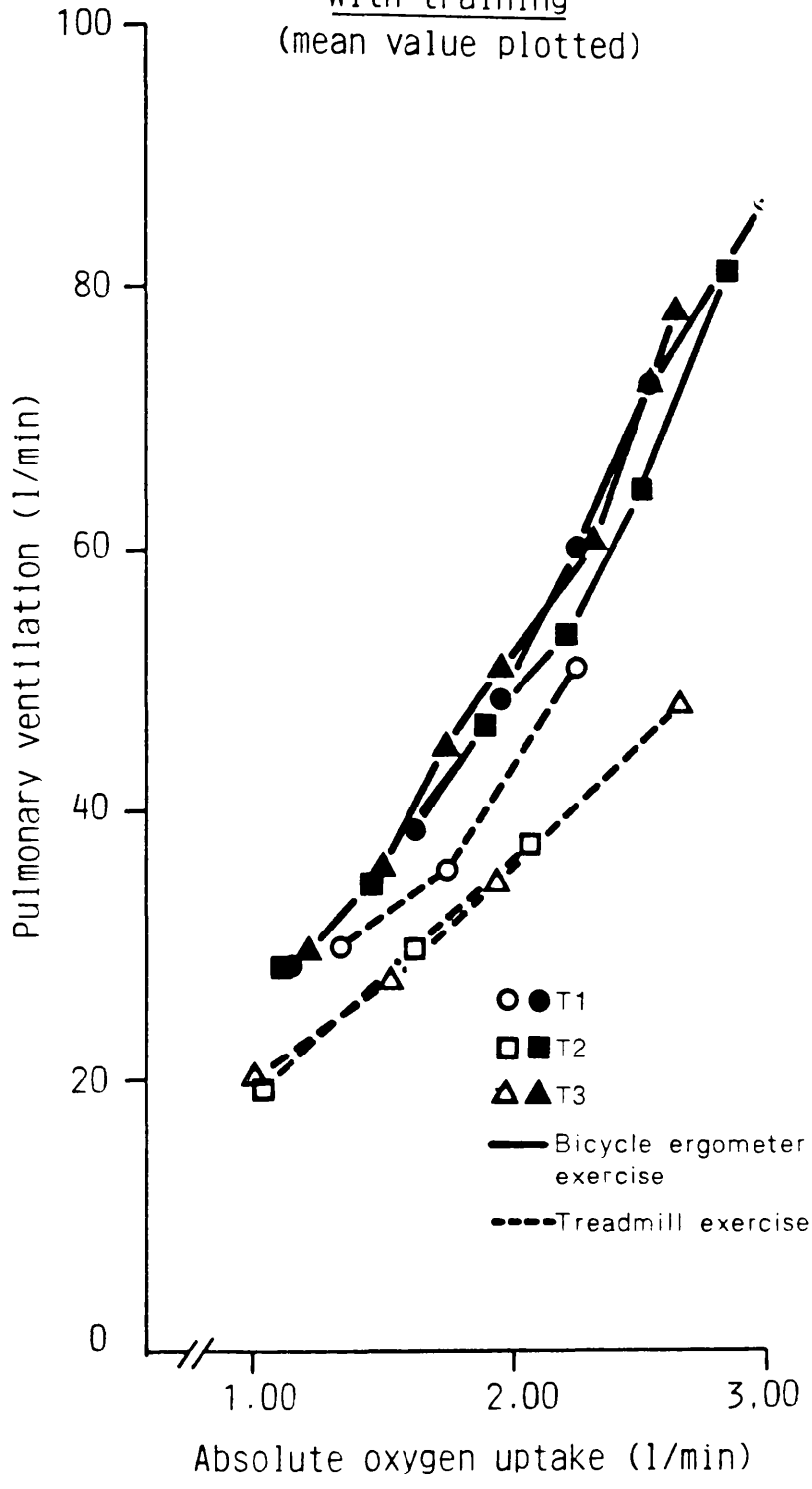
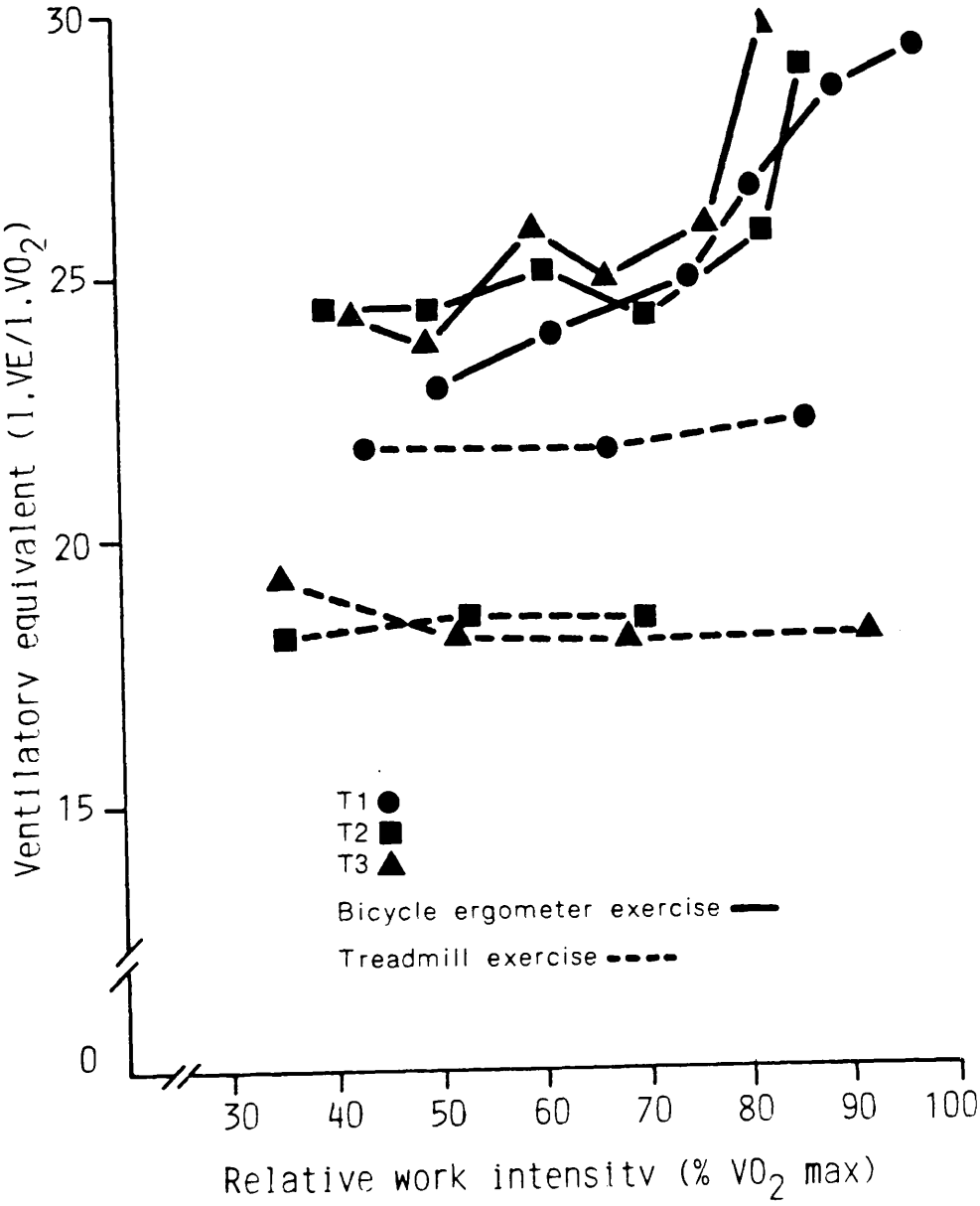


FIGURE 36

Change in ventilatory equivalent versus relative work intensity with training (mean value plotted)



impulses from exercising muscles, and a reduction in blood lactate concentration. In support of this reduction in sympathetic activity with training, a significant correlation was observed in this study between the change in submaximal heart rate and change in ventilation ($r = +0.67$, $P < 0.01$). Although blood lactate was not measured during treadmill exercise in this study, the significant fall in submaximal blood lactate levels during bicycle ergometer exercise did not appear to ellicit a significant reduction in ventilation. However, as the reduction in submaximal blood lactate is also reported to have an element of training specificity in its magnitude, it might be argued that if blood lactate had been measured in treadmill exercise they would have demonstrated a significant decrease at all stages of training. A reduction in blood lactate would then reduce ventilation by reducing peripheral or central chemorecepetor drive as the result of an increase in arterial pH and reduction in arterial carbon dioxide tension respectively (Sutton and Jones, 1979).

(iv) Reduced anatomic dead space

Although tidal volume and breathing frequency were not separately assessed in this study, comparative studies have shown that at a standard value of submaximal ventilation, athletes have a lower breathing frequency and a higher tidal volume than non-athletes (Sutton & Jones, 1979). If such a change in breathing

pattern were also true in this present training study there would be a decreased anatomic dead space per minute that would enable the maintenance of a constant alveolar ventilation despite a reduced overall minute ventilation.

(v) Reduced physiological dead space

It is feasible that training could reduce the physiological dead space of the subjects by improving the delivery of alveolar air to well prefused areas of the lung and/or by improving the perfusion of the lung to well ventilated areas. In support of this notion Carlsten and Grimby (1966) have shown that aerobic endurance training improves pulmonary blood flow particularly to the upper regions of the lung.

(vi) Chemosensitivity

It has been demonstrated that athletes have a reduced ventilatory response to hypercapnia and hypoxia relative to non-athltes (Martin et al, 1979). This finding is suggestive of a reduced ventilatory chemosensitivity although it is not resolved as to whether this effect is the result of endowment or training.

(c) Physiological significance

As outlined in the introduction to this discussion section, the role of reduced submaximal ventilation on aerobic endurance performance has yet to be clarified. Two possible areas of physiological importance relative to this training adaptation are breathing and perceived exertion.

Training was observed in this study to improve the submaximal ventilatory equivalent. Thus training enables a given value of overall oxygen uptake to be maintained with reduced minute ventilation. This reduction in ventilation will in turn reduce the work of breathing. Studies have shown that the oxygen cost of breathing during exercise is small (Shephard, 1966) thus suggesting that such a training effect is minimal. However, during prolonged ventilatory effort such as marathon running this reduction in oxygen cost may prove beneficial.

A close correlation has been shown to exist between ventilation and the perception of exercise effort (Wigertz, 1970). Thus it is possible that a reduction in submaximal exercise ventilation may improve aerobic endurance performance via a reduction in percieved exertion.

4.4 PLASMA LACTATE RESPONSE

In recent years submaximal plasma lactate response has become an increasingly reported variable when assessing exercise capacity. This rising popularity is probably reflective of the success of lactate as a predictive and evaluative factor in aerobic endurance exercise performance (Farrell et al, 1979). A recent review (Jacobs, 1986) of a number of studies has shown that lactate related variables account for a larger proportion of the variation in aerobic endurance

exercise performance in athletic groups than the other variables traditionally determined in the exercise laboratory, including in particular, VO_2max.

Aerobic endurance training studies have traditionally employed VO_2max as the measure of change in exercise capacity (Pollock, 1973). However, in view of its predictability it has been increasingly suggested that submaximal exercise blood lactate may be a more valid indicator of training status than VO_2max (Daniels et al, 1978). The criticism of VO_2max centres around the inverse relationship between the initial VO_2max and the magnitude of change induced by training (Saltin et al, 1968). Additionally, for a reliable and reproduceable value of VO_2max to be obtained, it is critical that the subject achieves central fatigue and volitional exhaustion (Shephard et al, 1968a). The submaximal nature of the lactate response does not demand either such strenuous effort on the part of the subject or the medical precautions that such effort necessitates. Moreover it will be seen below that there appears to be no evidence to show that the initial blood lactate response dictates the magnitude of the training induced change. Thus the lactate response to submaximal exercise is being used increasingly as a longitudinal marker of adaptation to aerobic endurance exercise.

In this section, comparison will be made between the training induced change in lactate response

observed in the present training study with previous aerobic endurance training studies. The mechanisms responsible for such change will also be examined. As the change in lactate response in the present study after the first 15 weeks training appeared to be different to that of the second 15 week period of training, the two 15 week periods of this study will be discussed separately.

4.4.1 Initial 15 weeks training

(a) Pattern of change

After 15 weeks of training a reduction in the lactate concentration over the range of submaximal workloads and oxygen uptake was observed (see Table 11 & figures 15 & 26). Moreover the lactate concentration of 4 mmol/l was achieved at a value of submaximal workload and oxygen uptake of 26 watts (26%) and 3.5 ml/kg/min (14%) respectively, higher than before training. Table 12 demonstrates the insignificant change in lactate response versus relative work intensity (ie. %VO2max).

This pattern of lactate response change is entirely in agreement with previous aerobic endurance training studies (Williams et al, 1967, Ekblom et al, 1968, Saltin et al, 1969, Karlsson et al, 1972, Denis et al, 1983, Sjodin et al, 1982; Yoshida et al, 1982, Hurley et al, 1984, Denis et al, 1984).

(b) Magnitude of change

Comparison of the magnitude of reduction in

lactate concentration observed in the present study to that observed in previous studies is not as straight forward as one might expect. This is due to the different way in which previous studies have reported their lactate response. Early studies reported the lactate response by plotting lactate concentration versus a range of submaximal workloads, oxygen uptake values or percent VO_2max values (Saltin et al, 1969,; Ekblom et al, 1968; Williams et al, 1967). More recently, lactate response has been reported as a single value, this value being the workload, oxygen uptake or percent VO_2max at which a given lactate concentration is achieved (Denis et al, 1982 & 1984; Hurley et al, 1984; Yoshida et al, 1982; Sjodin et al, 1982) This later index, the onset of blood lactate accumulation (OBLA), is referred to as the workload, VO2 or %VO2max at which blood lactate passes the threshold 4 mmol/l.

The study of the kinetics of lactate accumulation has justified the use of a single value of lactate concentration when evaluating the submaximal blood response to exercise (Tesch et al, 1983). Therefore to compare the change in lactate of the present training program with the above training studies all the above studies have been reanalysed and their lactate response expressed in terms of OBLA. (see Table 32).

It can be seen from this review, that all the previous endurance training studies considered revealed an increase in the value of OBLA at a given oxygen

TABLE 32

Review of previous studies examining training adaption in lactate response as expressed - OBLA. 4mmol

Author	n*	OBLA. VO_2+ (ml/kg/min) T1	T2	Δ	OBLA %. VO_2 max+ T1	T2	Δ
Sedentary young adults							
Ekblom et al (1968)	8	26.1	32.4	+6.3	57	60	+3
Karlsson et al (1977)	14	29.2	41.3	12.1	62	70	+8
Hurley et al (1984)	8	33.7	45.4	+11.7	78	83	+5
Denis et al (1984)	6	34.8	42.9	+8.1	70	73	+3
	Mean	30.9	40.5	+9.6	67	72	+5
Young athletes							
Sjodin et al (1982)	8	58.6	60.6	+2.0	85	87	+2
Sedentary middle-aged men							
Saltin et al (1968)	8	23.1	25.6	+2.5	62	68	+6
Denis et al (1982)	5	30.4	40.2	+9.3	72	83	+11
Denis et al (1984)	6	34.9	43.8	+8.9	72	84	+12
	Mean	29.5	36.5	+7.0	69	78	+9
Present study	28	25.5	29.0	+3.5	75	74	-1

*number of subjects in each study
+ mean values of OBLA reported

uptake (ie. OBLA.VO_2). All these studies also reported an increase in OBLA at a relative work intensity (OBLA.%VO_2max), although in a number of these studies this later change was not statistically significant (Denis et al, 1984; Saltin et al, 1968; Sjodin et al 1982). It is apparent, from this review, that the magnitude of OBLA.VO_2 change in this study was somewhat less than the average change observed in previous studies (+3.5 versus +7.6 ml/kg/min). A number of factors could, in theory, be responsible for a change in the magnitude of OBLA change with training. The contribution of these factors in the present study will be discussed below.

(i) Experimental factors

It is possible that the reduced magnitude of lactate change in the present study reflects experimental limitations.

An important experimental factor in the evaluation of change in lactate response with training is the reproducibility of lactate measurement. Although the reproducibility data for this study is not reported, evaluation of the technique by other laboratories has revealed very high coefficients of reproducibility (Karlsson et al, 1983). Indeed Sjodin and his colleagues quoted test-retest correlation coefficients of 0.90 an 0.89 (Sjodin et al, 1982).

Experimental conditions are important when assessing resting or submaximal exercise blood lactate

concentrations. Both heat and cold can elevate lactate levels (Fink et al, 1975). The mechanism for change in lactate concentration with differing environmental conditions is not well understood but appears to reflect changes in hepatic and skeletal blood flows. As previously outlined in the methods section, constant thermo-neutral temperature and humidity conditions during testing were maintained throughout the study.

A final experimental consideration when evaluating blood lactate response is the exercise protocol. Since time is required for lactate to be released from the muscle to the circulation, it is important that the duration of each increase in exercise increment is sufficient to allow achievement of adequate steady state. Moreover it has also been shown that changing the time of workload increment progression can alter the pattern of blood lactate response (Yoshida, 1984; McLellan, 1985).

Yoshida (1984) found that submaximal blood lactate concentration observed during an exercise protocol involving 25 watt increases in workload every 1 minute was less than the lactate concentration observed with 4 minute increments at the same workload. This pattern of lactate response led to an overestimation of OBLA during the 1 minute protocol in comparison with the 4 minute protocol (177.5±5.5 watts vs. 144.0±7.0 watts). Thus it appears from this study that a time of 1 minute is insufficient to allow blood lactate to accumulate to

steady state. However, as there were no intermediate workload times (ie. 2 and 3 mins.) in Yoshida's study it is difficult to determine if a duration of 4 minutes does actually allow steady state and whether time intervals of less than 4 minutes would be sufficient.

In this study the workload duration was held constant at 3 minutes. According to the above study it is possible that this workload duration is insufficient to guarantee steady state lactate concentration. As exercise time was kept constant throughout the different stages of the study then comparison of the lactate response should be valid despite the possibility of not obtaining steady state conditions.

Thus although not formally assessed, the maintenance of a thermo-neutral testing environment and constant workload duration suggests that the reproduceability of the lactate measurement in this study was sufficient not to effect the magnitude of lactate change with training.

(ii) Pre-training OBLA

A specific criticism of VO_2max centres around the inverse relationship between initial VO_2max and the magnitude of change with training. The importance of initial VO2max has been demonstrated for both individual and group training response comparisons (Saltin et al, 1968; Pollock, 1973).

Correlation analysis of the change in individual OBLA.VO_2 versus initial OBLA.VO2 in this study

demonstrated no significant relationship (r = +0.23, NS). The review of previous studies did reveal that the lowest increase in OBLA.VO_2 value corresponded to the athletic population with highest initial OBLA.VO_2 value. However, overall there did not appear to be any consistent relationship between the initial value of OBLA.VO_2 and its change in these studies. Thus it appears that the initial value of lactate response is not a primary determinant of the magnitude of lactate training adaptation.

(iii) Subject age

A review of the changes in absolute VO_2 max with aerobic endurance training reveals a reduced magnitude of increase in middle-aged groups in comparison to their younger counterparts (Pollock, 1973). It might therefore be expected that the age of the present group could account for the reduced magnitude of OBLA.$\dot{V}O_2$ change.

From the review table of previous studies it can be seen that the magnitude of change in both OBLA.VO_2 and OBLA.%VO_2 max was slightly greater on average for sedentary young male groups than middle-aged male goups. However, a recent study by Denis et al (1984) comparing middle aged men and younger men with the same initial VO_2 max and OBLA.$\dot{W}$atts over the same 20 week training program found no significant difference in the change in OBLA.Watts of the two groups. Finally no significant relationship was observed between the change in OBLA.VO_2 value and age for the individuals of the

present study over the first 15 weeks of training.

Thus it appears from this analysis that the reduced magnitude of OBLA$^{\dot{V}O_2}$ change is not a direct result of the age of the present study group.

(iv) Training program

In accordance with the principle of training overload, it is to be expected that the characteristics of the training program will be a major determinant in the magnitude of lactate training response.

The similarity of the exercise programs of the reviewed previous lactate studies makes it difficult to assess such an effect. The average training duration and frequency of the present study during the initial 15 weeks of training was similar to these previous studies, suggesting that these training factors could be ignored. It is noticeable that a training intensity of 75% VO_2max or more is maintained in all these studies. Indeed it has been suggested that to ellicit an improvement in lactate response, a particular critical intensity of aerobic endurance training is necessary (Hollman et al, 1981; Sjodin et al, 1982). The importance of training intensity in altering lactate response will be discussed in more detail in section 4.4.2 (b)(ii).

Although training intensity was not monitored comprehensively in this study, the limited exercise intensity monitoring performed suggests that an intensity of approximately 85 to 95 percent heart rate

reserve was maintained during the initial 15 weeks of training (see figure 9). This intensity is at least equivalent to the 75% VO_2max of previous training studies, and it thus appears that the present training program is not responsible for the magnitude of OBLA change.

(v) Exercise specificity

Exercise specifity studies that have the compared the adaptions to training using different modes of exercise have not considered submaximal blood lactate (Pecher et al, 1974; Verstappen et al, 1978; Wilmore et al, 1980). Never-the-less one legged training studies (Saltin et al, 1976; Henricksson, 1977) have clearly demonstrated that the magnitude of blood lactate adaptation is greatest when the same limb is trained and tested. It might also be expected that the training adaptation of blood lactate is also exercise specific, particularly in view of blood lactate being largely dependant on the amount of muscle lactate production and therefore dependent on the pattern of muscle recruitment.

From the review of previous lactate training studies it appears that endurance training programs using the same mode of exercise when training and testing reported a greater magnitude of OBLA.VO2 increase (Hurley et al, 1984; Denis et al, 1982 & 1984, Karlsson et al, 1974) than studies tending to use different modes of exercise (Saltin et al, 1968; Ekblom

et al, 1968). In the present study the bicycle ergometer was used as the mode of exercise testing while running was used as the mode of exercise training.

On the basis of the evidence reviewed, it is probable that training specificity was responsible for the reduced magnitude of lactate improvement observed in the first 15 weeks of training in the present study.

(b) Mechanism of change

Although a reduction in the submaximal blood lactate response is a commonly reported adaptation with aerobic endurance programs the exact nature of this change still remains relatively unclear and indeed a number of potential mechanisms have been proposed. These mechanisms will be reviewed below with regard to the present study.

(i) Increased oxygen delivery

For some time it has been believed that a reduction in lactate concentration with training reflects an increased oxygen delivery to the exercising muscles due to cardiovascular adaptations caused by training.

It has been suggested that no change with training in the blood lactate response to a given relative intensity of effort (ie. percent VO2max) is indicative that improvements in blood lactate response observed at standard submaximal workloads are due to the same

adaptations responsible for the increase in VO2max (Hurley et al, 1984). Expressing the lactate response of the present study relative to percent VO2max revealed that there was essentially no change in lactate response with training. This finding supports the previous training studies by Saltin et al (1969) and Ekblom et al (1968). Thus it appears that the reduction in lactate concentration in this study is the result of the same changes that are responsible for the increase in VO2max. As discussed in an earlier section one of the chief factors responsible for the improvement in aerobic power in this study was an increase in peak cardiac output. Thus it is possible that such a cardiovascular adaptation could account for the reduction in lactate response.

Never-the-less against this hypothesis is the finding that blood flow per kg of exercising muscle is actually lower in trained than in untrained subjects at the same absolute, submaximal workload (Hubbard 1973). Indeed there is information to suggest that lactate accumulation is not necessarily a phenomena associated only with a lack of oxygen and that lactate can be produced in completely aerobic conditions if NADH and pyruvate are available to lactate dehydrogenase (Karlsson & Jacobs, 1982).

(ii) Reduction in oxygen deficit

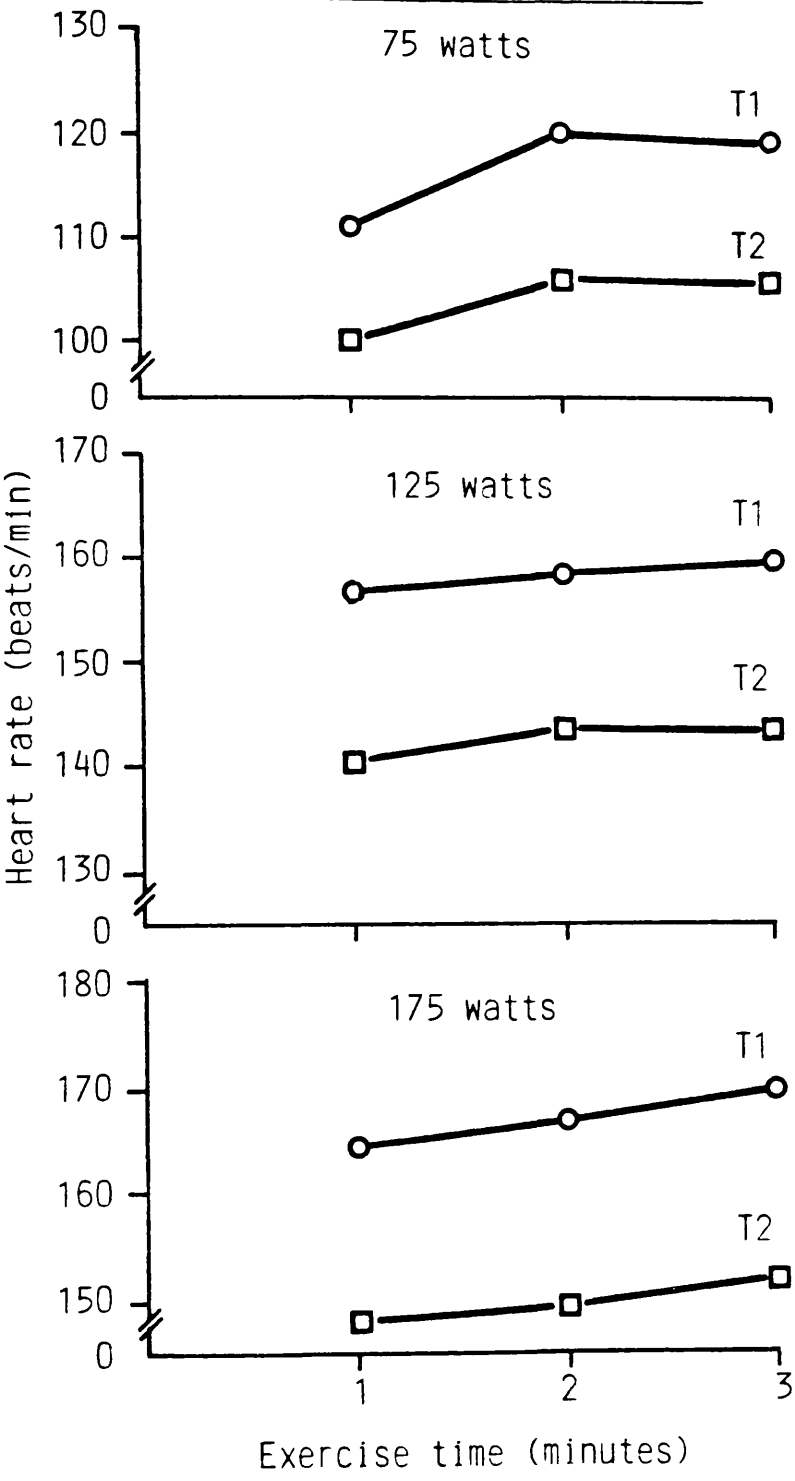
Lactate is not just the product of steady state metabolism. It is rarely taken into consideration that

lactate is formed at the onset of exercise when the circulatory apparatus is in the acceleration phase and steady state not yet established. With this in mind Karlsson et al (1972) have proposed that training results in a faster cardiovascular response to exercise and a lower oxygen deficit is incurred thereby reducing lactate levels early in exercise which may therefore reduce the cummulative effect on lactate release at higher work rates. This oxygen deficit hypothesis has been confirmed by the observation of a faster rate of adjustment of heart rate, minute ventilation and oxygen uptake from rest to steady state exercise with aerobic endurance training (Hickson et al, 1978; Hagberg et al, 1980). Therefore it is plausible that the fall in blood lactate levels observed in this study after 15 weeks is the result of incurring a reduced oxygen deficit. As this hypothesis refers to the oxygen demand during the early non steady state stages of exercise it would agree with the fact that there was no change or even a fall in steady state oxygen uptake with the present study after training.

Heart rate was assessed in this study every 60 seconds during each 3 minute bicycle exercise work load. No obvious change in the rate of heart rate adjustment to steady state, suggestive of a fall in oxygen deficit, was observed in this study (see Figure 37).

However, the previous investigative groups, by

Rate of heart rate adaption during bicycle ergometer exercise at T1 and T2 (mean values plotted)



using breath-by-breath analysis, were able to assess the metabolic changes to exercise over 5 second intervals. Moreover the changes in oxygen deficit occurred mainly in the first minute of a workload increment. The 60 second intervals of heart rate assessment of the present study are probably too great to allow observation of such an effect. Therefore it is not possible to either confirm or refute this hypothesis of a decrease in oxygen deficit with training as a possible mechanism of decreasing submaximal blood lactate in the present study. However it is interesting to note that Hagberg and his colleagues (1980) suggest that this reduction is the result of a decrease in blood lactate rather than the converse arguement of Karlsson et al (1972) ie a reduced oxygen deficit will result in a reduction in blood lactate. The former argument seems intuitively more acceptable.

(iii) Metabolic changes within the skeletal muscle.

Since the hypothesis of increased oxygen delivery for the reduction in blood lactate has gone out of favour the explanation of metabolic adaptation within aerobic endurance trained muscles being responsible for this change has been preferred (Holloszy and Coyle, 1984).

Well trained males and females have been demonstrated to have an increase in the size number of mitochondria in their aerobic endurance trained muscle

groups (Henricksson, 1977). These mitochondria have increased levels of the marker enzymes for the pathways of free fatty acid oxidation (Costill et al, 1979, Janssen and Kaijser, 1977), citric acid cycle (Schantz et al, 1983; Gollnick et al, 1973) and respiratory chain (Morgan et al, 1971; Costill et al, 1979). Although there is no evidence in the human study literature of a change in type II muscle fibres reaching the oxidative profile of type I there does appear to be good evidence in endurance trained individuals that there is conversion of IIb fibres to IIa fibres (Jansson and Kaijser, 1977; Chi et al, 1978). An increase in lipolysis and an increase in citric acid activity may reduce the rate of glycolysis, and hence lactate production, by citrate feedback on the rate-controlling enzyme phosphofructokinase (Rennie et al, 1976). An increase in the capacity of the malate-asparate shuttle (carries NADH into the mitochondria) (Hollosky, 1976) has also been demonstrated with endurance training. Such a shuttling will diminish the requirement to regenerate NAD as the result of reduction of pyruvate to lactic acid.

Aerobic endurance training appears to result in rather minor changes in glycolytic enzyme activtiy (Baldwin et al, 1972; Gollnick et al, 1972) although there is a significant increase in the proportion of heart-specific isoenzyme and a decrease in the skeletal muscle-specific isoenzyme for LDH (Sjodin, 1976). This

form of LDH favours the equilibrium reaction of lactate toward carbon dioxide and water.

In support of this hypothesis of increased fatty acid metabolism was the fall in the value of submaximal respiratory exchange ratio observed with the present training study (see figure 16 & table 13). However, this reduction may also reflect a reduced carbon dioxide output as secondary to a fall in pulmonary ventilation rather than a change in muscular metabolism (see figure 14).

Complete confirmation of this hypothesis would require that the respiratory quotient be measured directly by arterial-venous blood sampling or that the metabolic capacity of the trained muscles be assessed by biopsy. Neither of these 2 procedures were possible in this study. Never-the-less in view of the wealth of evidence from the above studies it appears highly plausible that an improvement in the oxidative enzymes and enzymes of fat metabolism within trained muscle would have at least partly contributed to the reduction in blood lactate observed in the present training study.

(v) Change in the pattern of muscle fibre recruitment

The above mechanism of an altered metabolic profile of trained skeletal muscle is based on the concept that the reduction in blood lactate concentration in this study is the result of reduced production of lactate by the trained exercising muscle.

Another mechanism that also utilises this concept of reduced muscle lactate production is one of an altered pattern of muscle fibre recuitment.

There is currently no evidence to demonstate that the pattern of motor recruitment to a given task is altered by training. In view of the glycolytic activity of these type II fibres it might be inferred that this change in pattern of muscle fibre recruitment will also lead to a reduction in muscle lactate production and thus a reduction in blood lactate level. Although there was no direct evidence for such a change in recruitment pattern with training in the present study this hypothesis cannot be discounted.

(vi) Increase in lactate uptake/metabolism

For the above proposed mechanisms of an increase in oxidative capacity or a change in the pattern of muscle fibre recruitment to be responsible for the reduction in lactate concentration in the present study, then it might also be expected that the value of submaximal oxygen uptake would increase. However, in agreement with previous training studies no such change in submaximal oxygen uptake was observed (see figures 11 & 13). Indeed in the present study the value of oxygen uptake fell over a large proportion of its submaximal range. It is plausible that any increase in oxygen uptake as the result of an increase in oxidative capacity or change in muscle fibre recruitment pattern is masked by an increase in mechanical efficiency as

the result of training. However, it is also possible that the reduction in lactate concentration may be the result of some other factor.

Blood lactate concentration is dependent not only on the rate of muscle production but also the rate of lactate diffusion or the rate of lactate uptake and metabolism. Thus the decrease in blood lactate of the present study could theoretically be the result of a reduced rate of lactate diffusion or an increase in lactate uptake and thus metabolism.

Lactate is a relatively small molecule and there is no evidence of a change in its diffusibility with training (Diamant et al, 1968). However, it has been recently demonstrated, using carbon-tracer studies in rats, that a fall in blood lactate levels with training is due to an increase in lactate uptake while muscle production is unaffected (Brookes and Fahey, 1984). In particular, observations were made of an increase in the rate of lactate uptake by the liver (for conversion to glucose monomers by gluconeogenesis) and by cardiac and oxidative skeletal muscle (for oxidation). It appears that the skeletal muscle is the main site of lactate removal during exercise (Gollnick and Hermansen, 1973).

Respiratory quotient is an indicator of the type of fuel being metabolised (value of 1.0 for carbohydrate and value of 0.7 for fat) and a reduction in blood lactate levels with training due entirely to

increased lactate uptake would not result in a change in respiratory quotient. Although in this study measurement of steady state respiratory quotient during exercise was not possible, its ventilatory equivalent, the respiratory exchange ratio, was assessed. The mean value of this ratio at standard submaximal work loads revealed a pattern of fall after 15 weeks training, although these changes were often small and statistically insignificant (see figure 16) . This finding appears to suggest that the rate of carbohydrate turnover had dropped slightly with training and that a fall in the rate of lactate production did occur in response to training. However, a fall in the value of respiratory exchange ratio with training may not be a result of change in the substrate utilization. Such a pattern of change may also result from the observed fall in pulmonary ventilation at a given submaximal work load or an alteration in the acid-base balance of the blood as a result of a fall in blood lactate.

(d) Physiological significance

In view of the strong relationship between blood lactate response and exercise performance the reduction in submaximal blood lactate observed in this study is suggestive of an improvement in aerobic endurance performance of the study participants. The precise means whereby a reduction in lactate improves aerobic endurance performance is not altogether clear.

It has been suggested that lactate accumulation may cause muscle fatigue as the result of acidosis (Simonsen, 1971). In prolonged exercise associated with a substantial aerobic contribution this degree of acidosis would not occur. During such prolonged aerobic endurance exercise fat becomes an increasingly important substrate and it has been shown that acidosis inhibits fat mobilization (Farrell et al, 1979). Thus a fall in lactate accumulation reduces the possible inhibition of fat metabolism with consequent greater dependence on carbohydrates. This increased carbohydrate utilization may result in glycogen depletion which has been associated with exhaustion during aerobic endurance exercise (Costill et al, 1973).

4.4.2 Final 15 weeks training

(a) Pattern of change

In contrast to the lowering of submaximal lactate concentration observed after the first 15 weeks training a reversal in lactate response occurred during the second 15 weeks of training. Thus after 30 weeks training the lactate concentration at a standard submaximal bicycle workload was essentially the same as prior to training (see figure 15).

(b) Mechanism of change

The pattern of lactate response during the later 15 weeks of training was entirely unexpected. No

previous aerobic endurance training studies have reported such a lactate worsening.

In an incremental exercise test to exhaustion, the ventilation first increases linearly with respect to oxygen uptake then increases more sharply. The point at which this rapid increase in ventilation occurs has become known as the 'anaerobic threshold' and is thought to reflect the point at which metabolic acidosis occurs (Wasserman et al, 1973) on account of increasing blood lactate concentration (Brookes & Fahey, 1984)..

Ready and Quinney (1982), observed a decrease in anaerobic threshold, assessed the workload, VO2 or %VO2max as the point at which there is a systematic increase in ventilatory equivalent for oxygen without an increase in ventilatory equivalent for carbon dioxide, as the result of 6 to 9 week period of de-training. On first impressions this finding is suggestive that the reversal in plasma lactate response in the later 15 weeks of the present training study may have been reflective of a de-training effect. However, caution should be applied in comparing the results Ready and Quinney (1982) to the present study. Firstly the method of de-training of Ready and Quinney was to completely cease the training of their subjects. No such stoppage of training occured in the later 15 weeks this study (see Table 3). Secondly there is currently

considerable debate as to validity of anaerobic threshold being reflective of plasma lactate changes (Brookes & Fahey, 1984). Thus changes in ventilatory threshold with aerobic endurance training may bear no direct relation to the changes in plasma lactate response with training.

The possible factors responsible for the change in plasma lactate reponse during the later 15 weeks of training are discussed below.

(i) Experimental factors

As previously discussed in section 4.4.1 (b)(i), environmental factors were standardized as much as possible and are therefore unlikely to be a significant cause of error.

(ii) Increased resting plasma lactate concentration

It is plausible that the reversal in plasma lactate reponse during the later 15 weeks of training was the result not of increase in lactate production by exercising muscle but the result of an initially raised plasma lactate concentration.

prior to exercise. However, the mean value of resting lactate revealed no significant change during any stage of training in this study (see table 5).

(iii) Dietary changes

The consumption of a high carbohydrate diet, a relatively common dietary regime prior to marathon participation, has been shown to elevate lactate during submaximal exercise (Yoshida, 1984). Increased carbohydrate intake ('carbohydrate loading') is a common regime of dietary manipulation prior to long duration endurance performance, such as marathon running (Costill et al, 1973). Although no dietary analysis was performed in this study a number of the study participants did report an increase in carbohydrate intake during the later stages of training.

(iv) Exercise specificity

It has already been discussed in section 4.4.1(a)(iii) that the differing modes of exercise training and testing in this study introduce the problem of training specifity. Although exercise specificity might be expected to reduce the magnitude of lactate change with training it is very unlikely that it would account for the increase in plasma lactate concentration observed during the later 15 weeks of training.

(v) Nature of training program

It has been suggested that, to ellicit an

improvement in submaximal lactate response, a certain critical intensity of aerobic endurance training is necessary (Hollman et al, 1981; Sjodin et al, 1982). However, there is some debate as to the magnitude of this threshold value. Some investigative groups recommend an exercise intensity corresponding to a steady state exercise lactate concentration of approximately 4 mmol/l (Hollman et al, 1981; Sjodin et al, 1982). Although training at this lactate concentration has been demonstrated to be effective in improving blood lactate response, the actual measurement of lactate levels during a training study presents practical difficulties. Sady and his colleagues (1980) have demonstrated that training at 80% VO_2max improves anaerobic threshold while training at 40% VO_2max does not. This study appears to be supported by the previous lactate endurance training study which consistently employed a training intensity of 75% VO_2max or more.

A recent study has reported findings in opposition to this suggested need for a threshold of aerobic endurance training intensity for improvement in blood lactate response. Poole and Gasser (1985) examined the improvement in lactate response in 3 groups of previously inactive young men with similar physical characteristics who trained for 7 weeks at high intensity (105% VO_2max), moderate intensity (70% VO_2max) or light intensity (50% VO_2max). No significant

difference was observed in the magnitude of lactate improvement across the 3 groups. Interestingly, Poole and Gasser also observed, in agreement with Sady and his colleagues (1980), that anaerobic threshold improved in relation to the intensity of training. Thus it appears to be inappropriate to extrapolate from anaerobic threshold studies to lactate response in that the anaerobic threshold and blood lactate concentrations are not affected by training in the same manner.

Although training intensity was not consistently assessed in this study the above evidence suggests that it is unlikely that insufficient training intensity is responsible for the pattern of lactate reverse. However, there are difficulties in directly comparing the study of Poole and Gasser with the present study. Firstly the subjects of Poole and Gasser were untrained whereas at T2 the subjects in the present study had undergone 15 weeks of training. Thus it is feasible that an intensity of 50% VO_2max or more for the subjects in the present study would have been insufficient to ellicit a reduction in lactate concentration. In addition the 7 week duration of Poole and Gasser's study is fairly short in relative training study terms. It is possible that prolonging this study beyond its 7 week duration may have led to a set of findings in which those individuals training at the low intensity experienced a reduced magnitude of lactate

change.

In the present study the intensity of training was found to be as high as 95% HRmax in the initial stages of training although in later stages of training this value fell to 79% HRmax. These values correspond approximately to 85% and 69% VO_2max (ACSM, 1980). Therefore it is clear that the intensity of training in the later stages of this study did not achieve the threshold of 75% VO_2max typically reported in previous lactate training studies. As the intensity of training in the later 15 weeks of training was lower than that of the earlier stages of the training program it is feasible that this intensity may have been insufficient to prevent a detraining effect and thus a return to pre-training lactate concentration.

(vi) Increased muscle glycogen

Sherman et al (1981) recently investigated the effect of a 7 day tapering period in which training load was reduced in comparison to typical training level. Three subjects groups were examined: a control group (non-tapering), a tapering group on a high carbohydrate diet and finally tapering group on a normal diet. They observed that in both the normal and high carbohydate diet tapering groups there was a significant increase in exercising muscle glycogen content in comparison to the control groups.

In the present training study there was quite clearly a reduction in the average training load, as assessed by training duration and frequency, during the

last 5 weeks of training (see Figure 15). Although the dietary intake of subjects in this study was not measured, a number of subjects remarked on a subjective increase in carbohydrate content of their diet. According to to the data of Sherman et al (1981) the training taper observed in this study, with or without dietary change, would lead to an increase in trained muscle glycogen content. Thus at the final stage of testing the levels of muscle glycogen of the subjects in this study would have exceeded both the pre-training and 15 week training levels.

Ivy et al (1981) observed an increase in blood lactate at given standard submaximal workloads in a high pre-test glucose intake group in comparison to a normal diet group. The investigators attributed this increase in blood lactate in glucose fed subjects to an increase in carbohydrate utilization that was in excess of the rate of oxidation.

It is probable that the increase in trained muscle glycogen concentration at the final testing phase of the present study, like increased glucose ingestion, would lead to an increase in the rate of carbohydrate utilization beyond that of oxidation. This would in turn increase the rate of muscle lactate production and thereby reverse the submaximal exercise plasma lactate response at the final stage of testing to that observed prior to training.

(c) Physiological significance

This finding, although unexpected, may have

important implications to future lactate training studies. Firstly more research is required to assess the minimum training intensity necessary for lactate improvement. In addition to prevent the effects of training glycogen depletion on blood lactate during testing , sufficient time must be allocated for recovery between training and testing. This might involve a recovery time of 2 days or more.

4.5 BODY COMPOSITION AND ENERGY METABOLISM

4.5.1 Body Composition

(a). Pattern of change

Reviews of the body composition changes with aerobic endurance type training have shown that in general there is a significant reduction in body fat together with either a slight increase or no change at all in fat free mass (Pollock et al, 1971; Wilmore, 1983; Pacy et al, 1986). In view of this it can be concluded that the reduction in total body weight is due entirely to a reduction in fat content. These exercise study findings contrast with typical patterns of body composition changes associated with dieting which generally show reductions in body weight resulting from decreases in both fat and fat free mass.

Observations in this study after 15 and 30 weeks of training (see Table 26 & Figure 32) showed significant reductions in total body weight and fat

content as determined by both skinfold and densitometric techniques. Although a slight alteration in fat free mass was observed this change was not statistically significant. It thus appears that the body composition findings of the present study are typical of previous aerobic endurance training studies.

Some previous training studies report an increase in fat free mass with training such that it will offset the reduction in body fat. This increase in fat free mass results from an increase in muscle mass and is generally associated with previously sedentary groups (McArdle et al, 1981). Despite the initial sedentary nature of the subjects involved in the present training study the virtually constant value of fat free mass suggests there was no such increase in muscle mass. An increase in calf muscle bulk might be expected in the present study since these muscles were highly activated during running. However, no significant increase was observed in calf circumference (see Table 28). Never-the-less it is feasible that a change in the composition of the fat free mass may have occurred during the present training study.

For a number of years it was thought that exercising a specific area would result in a selective utilization of fat from this area, thus reducing the mass of locally stored fat. This phenomena is termed 'spot reduction' (Mayer, 1968). However, no scientific

evidence has been presented to support this hypothesis. Indeed, studies up to now have obtained contrary findings. Gwinup and his colleagues (1971) demonstrated that the dominant arm of professional tennis players had a greater arm cirumference than the non-playing arm. This discrepancy was not the result of fat content, as the skinfold mesurements of the 2 arms showed, but was due to the greater muscle mass of the dominant arm and this pattern of change has been confirmed (Krotkiews et al, 1979).

The four skinfold sites assessed in this study revealed an absolute and relative magnitude of loss that was in accordance with the initial size of the skinfold. The greater the initial size of the fat depot the higher the loss in fat from this area (see Figures 24 & 25). Thus, in this study, subscapular losses were the highest followed by supra-iliac, triceps and then biceps. As no skinfold estimation was performed on the exercising lower limb in this study it is not possible from these values to either confirm or refute the spot reduction hypothesis. As outlined above, the calf circumference was observed to increase slightly over the 30 weeks of training although this change was not statistically significant (see Figure 28). This slight increase in calf circumference is likely to be the result of an increase in muscle mass which would outweigh the effect of any fat loss in this area. Calf skinfold required to be estimated to predict any such

regional fat change.

Although training does not appear to result in spot reduction of fat, it is possible that it may have a differential effect on the fat patterning of different body segments. Despres et al (1985) observed that after a period of aerobic training the reduction in the skinfold thicknesses of the extremities, the limbs, was less than the reduction in the skinfolds of the trunk. The present study confirmed this observation as the extremities skinfolds (biceps and triceps) demonstrated a reduction of 20.5 % versus the 29.2 % reduction in the trunk skinfolds (supra-iliac and subscapular) (see TABLE 33). These findings may reflect the greater initial fat deposit in the trunk although it is conceivable that training has different regional effects on adipose tissue metabolism.

Smith and his associates (1979) noted that fat cells from the subcutaneous abdominal deposit are more sensitive than extremity fat to the effect of catecholamines. Wilcox (1981) has found from biopsy that the more centrally located abdominal fat cells of athletes are smaller than their gluteal fat cells, suggesting that the former are preferentially depleted. It is difficult to interpret from this later study whether this is the result of training or genetic selection.

Thus it appears that there is little evidence of a spot reduction pattern of fat loss in this study

TABLE 33

Regional pattern of skinfold change with training (means and SD's)

				Absolute (mm)			Percentage (%)		
	T1	T2	T3	T1-T2	T2-T3	T1-T3	T1-T2	T2-T3	T1-T3
Extremity Skinfolds (mm)	15.6 ±3.7	14.0 ±3.0	12.4 ±2.8	-1.6 ±1.9 P<0.001	-1.6 ±1.3 P<0.001	-3.2 ±2.3 P<0.001	-10.2 ±9.8 P<0.001	-11.4 ±8.1 P<0.001	-20.5 ±14.3 P<0.001
				P<0.01	P<0.001	P<0.001	P<0.05	P<0.025	P<0.01
Trunk Skinfolds (mm)	40.7 ±9.5	34.6 ±9.0	28.8 ±8.5	-6.1 ±5.9 P<0.001	-5.8 ±3.9 P<0.001	-11.9 ±6.7 P<0.001	-15.0 ±11.9 P<0.001	-16.8 ±10.2 P<0.001	-29.2 ±13.9 P<0.001
Trunk / Extremity	2.6 ±0.6	2.5 ±0.6	2.3 ±0.6	-0.1 ±0.5 NS	-0.2 ±0.4 P<0.005	-0.3 ±0.6 P<0.005	-	-	-

Extremity = biceps + tricpes
Trunk = suprailiac + subscapular

T1 - prior to training
T2 - after 15 weeks training
T3 - after 30 weeks training

although there does appear to be a regional difference.

(b) Magnitude of change

Wilmore recently reviewed 40 studies examining the effect of aerobic endurance training on body composition of non-obese males aged 15 to 59 years (Wilmore, 1983). A summary of the results of this review are reported in Table 34.

In comparing the results of the present training study with those of the above review, considerable differences become obvious. The average 4.5 kg loss in total body weight over the 30 weeks of the present study was considerably greater than the mean value of all the reviewed studies and indeed was greater than any of the individual studies reported. The loss in body fat content observed in the present study, both densitometric and skinfold, was also considerably greater than the mean value of the previous studies. Indeed the reduction in both body weight and fat mass was greater than any of the 40 aerobic training studies reviewed by Wilmore.

Despite the large proportion of fat and weight loss over a relatively short period of time as shown in the present study, the rate of fat loss is not outwith the loss threshold of 1 kg of fat per week as recommended by the American College of Sports Medicine in their guidelines on weight loss (ACSM, 1983). Studies have shown that a too rapid weight loss is associated with medical risks such as myocardial

TABLE 34

Reanalysis of body composition training studies reviewed by Wilmore (1983)

(Mean SD's and range)

Subject age (years)	Program duration (weeks)	Session duration (minutes)	Session frequency (days/week)
23 ± 7	18 ± 16	34 ± 12	3 ± 1

Total body weight (kg)			Fat content(%)			Fat free mass (kg)		
Pre	Post	Δ	Pre	Post	Δ	Pre	Post	Δ
82.6 ±10.4	81.3 ±10.4	-1.3 ±1.2 (+1.0 to -3.3)	20.5 ±6.3	18.9 ±6.1	-1.6 ±1.1 (+0.9 to -3.3)	64.8 ±4.8	65.3 ±5.3	+0.5 ±1.2 (+3.1 to -1.6)

atrophy and sudden death (Sours et al, 1981).

(c) Mechanism of change

There are 4 main factors that may have been responsible for the marked alteration in body composition observed in this study: experimental error, the initial body composition characteristics of the study group, changes in the subject's lifestyle outwith the training program and finally the nature of the training program. The possible contribution of each of these factors in the present study will be evaluated below.

(i) Experimental error

It is feasible that the experimental errors could account for the pattern of results in this study. Body fat was assessed by the skinfold technique and underwater weighing. Both these techniques are reported to only have an accuracy in predicting body fat to within plus or minus 3 to 4 per cent of total body weight (Durnin & Rahaman, 1967; Round table, 1986). This error in body fat prediction is of a similar order to that of body fat change reported with training. However, efforts were made to minimize this prediction error in this study by selecting appropriate skinfold regression equations, using the same trained observer throughout the study and regularly calibrating the experimental equipment such as the skinfold calipers and the load cell for underwater weighing.

The error of body fat prediction from skinfolds is

dependent on the regression equation used and that error of prediction is increased when a regression equation is applied to individuals different to that from which the equation was derived (Wilmore, 1973). To obviate this problem of skinfold equation specificity, non population specific equations have been devised for application to a variety of populations based on age and sex (Durnin & Womersley, 1974; Jackson & Pollock, 1978). Although the Jackson and Pollock equations are probably the most popular of present day skinfold equations, the Durnin and Womersley equations were used in this study not only because they are non-population specific, having been successfully evaluated in a variety of different population groups (Wilmore, 1983), but also because these equations were initially derived in the laboratory used for the present study and the procedure was therefore more familiar to the author.

The reproducibility of the skinfold technique and underwater weighing is reported to be high. Durnin and Taylor (1960) assessed the body density of 3 individuals by underwater weighing on several occasions over a period of 12 months. They found a standard deviation of assessment of only 0.0008 g/ml. A recent study has also demonstrated high correlation coefficients (0.94 to 0.96) of repeated skinfold assessment by the same observer (Shaw, 1986). These figures were derived from both experienced and inexperienced observers. The reproducibility of the

skinfold technique has been shown to be dependant on the skinfold site (Womersley & Durnin, 1973). The biceps, triceps, subscapular and supra-iliac sites have been consistently found to have higher values of reproducibility than sites, such as thigh and calf, used in other regression equations. The Jackson and Pollock equations include the lower limb skinfolds while the Durnin and Womersley equations do not. It was therefore anticipated that the Jackson and Pollock estimate of fat content would be less reliable than that of the Durnin and Womersley equations.

An experimental consideration that could potentially effect the validity of the results of the present study is the physical condition of the subjects at each stage of testing. Dehydration will result in a reduction in total body weight. Moreover dehdyration may alter the fat free mass density such that the assumption of constant fat free mass density implicit in underwater weighing would be violated, leading to an error in total body density determination. As discussed in section 2.5.1, the subjects were requested at all stages of testing to avoid procedures that would effect their levels of hydration. All subjects complied with this request.

Although the question of experimental error with regard to the skinfold and body densitometry techniques can be ruled out to a large extent at present, this matter will be returned to in section 4.5.2.

(ii) Initial body composition charcteristics

That the initial characteristics of the subjects would be important in determining the magnitude of body composition change with training is suggested by two bodies of evidence. Firstly, analysis performed on the review studies reported by Wilmore demonstrates that there is a significant correlation between both the initial values of total body weight and body fat versus the value of change in these parameters with training ($r = +0.44$ and 0.38 respt., both $P < 0.05$). A second, and more convincing, body of evidence comes from a meta-analysis study of Epstein and Wing (1980). They report that in aerobic endurance studies on subjects with an initial body fat in excess of 20 per cent, they observed a significantly greater magnitude of body fat loss than those studies with an initial body fat of less than 20 per cent (ie.0.30 lb/week versus 0.10 lb/week). This difference presumably reflects the increased oxygen cost and therefore increased energy expenditure of weight supported exercise, such as running, of heavier individuals (ACSM, 1980).

In view of this evidence, it may therefore be expected that the marked reduction in body weight and fat content observed in this present study could be a consequence of the pre-training values of body fat being consistently greater than in previous studies. However, a comparision of the initial values of both body fat content and total body weight in the present

study with the mean initial values of the previous studies reviewed by Wilmore reveals little difference. Thus it appears that contrary to expectations, the pre-training body composition characteristics of the present study do not account for the magnitude of body composition change with training.

(iii) Alteration in subject's lifestyle

It is plausible that the marked reduction in body composition observed during the 30 weeks of training was not the result of the training program but changes in the lifestyle outwith the the exercise program.

Although all subjects were advised to maintain a similar pattern of lifestyle throughout the training program, two patterns of lifestyle change could potentially account for the observed pattern of body composition change: an increase in energy expenditure as the result of increased physical activity (recreational or occupational) and/or a decrease in energy intake.

Although activity patterns and food intake were not measured, the subjects were asked at each stage of testing if they considered that either of these parameters had altered since the last testing stage. The only change that was consistently reported was that during the later weeks of the program the subjects consciously increased their energy intake, particularly in terms of carbohydrate, in an attempt to build up their body reserves for the marathon. This pattern of

increased energy intake would tend to reduce the magnitude of fat and weight loss observed.

Although no measurement of daily food intake or physical activity patterns outwith training of the subjects in this study were performed, the subjective evidence of the subjects appears to suggest that it is unlikely that such changes would account for the observed alterations in body composition.

(iv) Nature of the training program

The final factor that may be responsible for the marked alteration of body composition in this study is the magnitude of training effort.

Comparing the present training program to the programs reviewed by Wilmore it is clear that the training effort associated with the present study is greater than typical previous aerobic endurance programs in terms of overall program duration and training duration and frequency. As the degree of training effort determines the magnitude of energy expenditure it might be expected that this increased training effort would result in an increased loss in body fat content and weight. Unlike maximal oxygen uptake, the precise effect of the different components of training effort on the magnitude of change of body fat content and weight has not been evaluated. Never-the-less there are some aerobic endurance training studies that throw light on this matter and these studies are reviewed below.

To determine the importance of training frequency, Pollock and his colleagues (1975) examined the effects of 30 to 45 minutes running at 80 to 95 percent maximal heart rate for four, three, and two days per week for a period of 20 weeks. A significantly greater skinfold loss was observed after running on 3 and 4 days/week compared to 2 days/week. It is difficult to conclude from this study if an increase in training frequency is responsible for the increase fat loss since the total number of sessions in each group was not the same. However, in support of the importance of training frequency, the meta-analysis study of Wing and Epstein (1980) reported an average loss in fat mass of 0.08, 0.22 and 0.45 lbs/week for training frequencies of 1, 3 and 4/5 days/week respectively.

To examine the effect of training intensity and duration, Girandola (1976) compared the training responses of young women to high intensity training of short duration with low intensity training of long duration. It was found that there was no significant difference in the magnitude of fat loss in the two groups. As the energy expenditures of these two groups were the same it appears that the critical factor in evaluating the training response to a training program is the overall energy cost of the program. Indeed it has been suggested by the American College of Sports Medicine (ACSM, 1978), that provided the frequency and duration of training be at least 3 days per week and 20

minutes per session then the critical determinant of body fat loss is an energy expenditure of at least 300 kcal per session. In support of this notion, the study of Epstein and Wing (1980) demonstrated a rate of fat loss of 0.20 and 0.35 lbs/week for training programs involving expenditures of less than 1000 and greater than 1000 kcal/week respectively.

From figure 8 it can be seen that the present study group did not comply fully with the prescribed training program, particularly during the later stages of training. Despite this the actual training effort still represented a considerably more demanding aerobic endurance training program, in terms of overall duration, session duration and session frequency, than has been typically performed in previous studies. The increased magnitude of both fat and weight losses, reported in the previous study, is likely to be the result of the greater energy expenditure of that training program. The energy expenditure of the present training study will be discussed in section 4.5.3. It is not possible to determine from the present study whether a particular training parameter (ie. session frequency, duration or intensity) is more important in determining the magnitude of body composition change than any other.

The hypothesis that increased energy expenditure resulting from increased training effort enhances fat weight loss is confirmed by the evidence of 2 previous

aerobic endurance training studies. Leon et al (1979) had six obese male subjects walk vigorously for a period of 90 minutes for 5 days per week for 16 weeks. Work output increased progressively each week to an energy exenditure of between 1000 and 2000 kcal/session. At the end of the 16 weeks, subjects averaged 5.7 kg of weight loss with a 5.9 kg loss of fat weight. Similarly, Zuti and Golding (1976) followed the progress of adult women who expended 500 kcal per session, 5 days per week for 16 weeks. At the end of the 16 weeks the women had lost 5.8 kg of fat.

(d) Physiological significance

Exercise is often dismissed as an effective means of losing body weight because of evidence from previous studies that total body weight changes little over the period of exercise training (Wilmore, 1983). Dieting is usually suggested as the alternative. The increased weight loss associated with dieting programs is quite clearly the result of both fat free and fat loss, particularly in the early stages (Lamb, 1978). However, weight loss with exercise training can often be underestimated as the result of an increase in fat free mass, offseting the effect of a reduction in fat mass. The results of the present study are particularly significant with regard to body composition change, as they clearly demonstrate that aerobic endurance training programs can lead to significant reductions in total body weight over a relatively short period in

time. Moreover this loss is entirely the result of fat mass, the fat free portion remaining relatively constant. In view of its influence on fat free mass on resting metabolic rate, prevention of fat free mass loss is considered a crucial factor in the long term success of weight reduction programs (ACSM, 1983).

Increased body fatness has been reported to have an adverse effect on both mortality and morbidity. It has been implicated in the development of diseases such as hypertension, coronary heart disease and diabetes. Longitudinal epidemiological studies have demonstrated that reducing body fat content can lead to a reduction in the incidence of such diseases (Pacy et al, 1986). The literature is unclear as to the level of body fatness that must be attained to avoid increased medical risk, although a recent round table meeting of experts in this area agreed a value of 10-25 percent fat as "optimal for health" in males regardless of age (Round table, 1986). With the present study a reduction was observed in average fat content from an intial value in the high part of this range to an average value in the middle.

Studies have shown that aerobic endurance athletes such as marathoners have extremely low values of body fat content (4-6 percent fat) that just exceed the minimal recommended levels (Costill et al, 1970). The energy cost of exercise, particularly a weight supported activity such as running, has been

demonstrated to be directly proportional to body weight (Passmore & Durnin, 1967). Thus low body fat and its influence on the total body weight of aerobic athletes will reduce the oxygen cost of running and thereby increase aerobic efficiency. Such an increased aerobic efficiency, like aerobic power, is considered to be one of factors that improves aerobic endurance performance. Thus the pattern of body composition change of the present study, regardless of changes in cardiovascular function, may be associated with the increase in running performance assessed in small numbers of subjects during the training study (see Table 4).

4.5.2 Skinfold - body density relationship

Numerous studies have examined the effect of aerobic endurance training programs on body fat content (Wilmore, 1983; Pacy et al, 1986; Pollock, 1973). Assessment of body fat content is invariably performed by either the densitometric or skinfold methods, the former generally being regarded as the gold standard technique. The majority of previous training studies have assessed body fat content by the skinfold technique as opposed to body densitometry.

According to a recent review, more than 40 different studies since 1950 have produced over 100 equations to predict body fat from skinfold measurements (Lohman, 1981). The popularity of skinfold techniques appears to be due not only to the technical

demands of direct body density assessment but also to the questionable validity of some of the assumptions of the body density technique. The skinfold method is also based on a number of assumptions but these assumptions frequently go unchallenged. The skinfold method has the advantage of its technical simplicity.

The skinfold technique allows determination of body fat by the use of regression equations which are based on the skinfold and total body density relationship. A number of studies in the scientific literature have examined the change in the body density and skinfold relationship in populations of different age and sex (Durnin and Womersley, 1974; Jackson and Pollock, 1978). However, few if any reported studies have assessed possible changes in skinfold-body density relationships associated with aerobic endurance training.

As neither the skinfold nor body densitometric methods are considered the gold standard method of body fat content a comparative study of the change in body fat with training as assessed by the two methods would provide valuable data on the extent of their compatibility.

(a) Pattern of change

In agreement with observations made in previous studies the relationship between total body density and the sum of four skinfolds in this present study did not appear to be a linear one at any stage of training (see

Figure 38). Previous investigators have suggested that the relationship is either a quadratic or logarithmic one (Durnin and Womersley, 1974). In accordance with the study of Durnin and Womersley (1974), from which the four skinfold sites were taken, it was assumed in that a logarithmic relationship existed and a best-fit logarthmic line was fitted to the data (see Figures 39 & 40).

The regression coefficient between the log of the sum of skinfolds and total body density prior to training ($r = -0.62$) is slightly lower than has been previously reported in the literature for sedentary populations (Table 35). Durnin and Womersley reported a range of r-values of -0.7 to -0.9. The lower correlation value observed in this study probably reflects the more homogenous nature of the skinfold values and smaller size of the present group rather than a break-down in the skinfold-body density relationship. In support of this supposition the coefficient of variation of the sum of skinfolds in the present study was calculated to be 21.1%, well below the value of 40.3% to 43.6% reported by Durnin and Womersley (1970).

A fall in correlation value was observed with training ($r = -0.62$ to -0.53 to -0.48) while the coefficient of variation of the sum of skinfold values stayed relatively constant with training. Although findings are suggestive of a breakdown in the skinfold

Individual values of body density and sum of four skinfolds

FIGURE 38

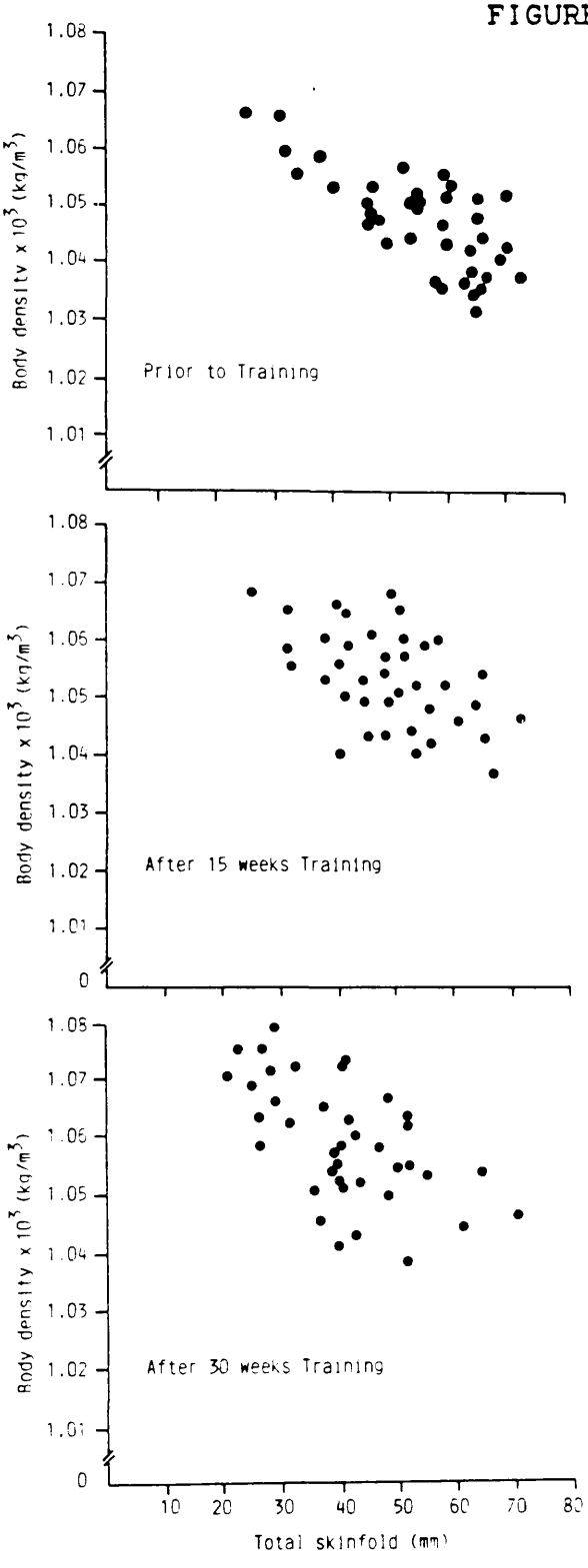


FIGURE 39

Alteration in total body density - sum of skinfold regression relationship with training

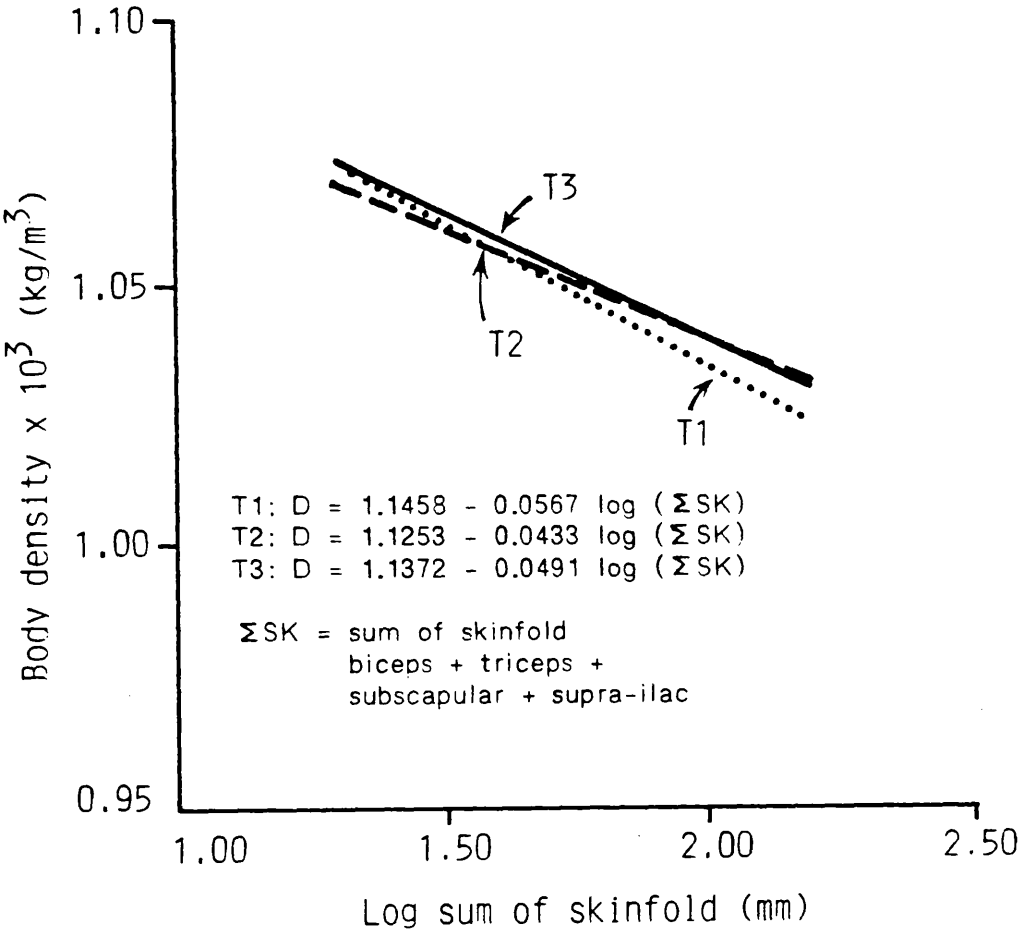


FIGURE 40

Individual skinfold regression equation plots at each stage of training

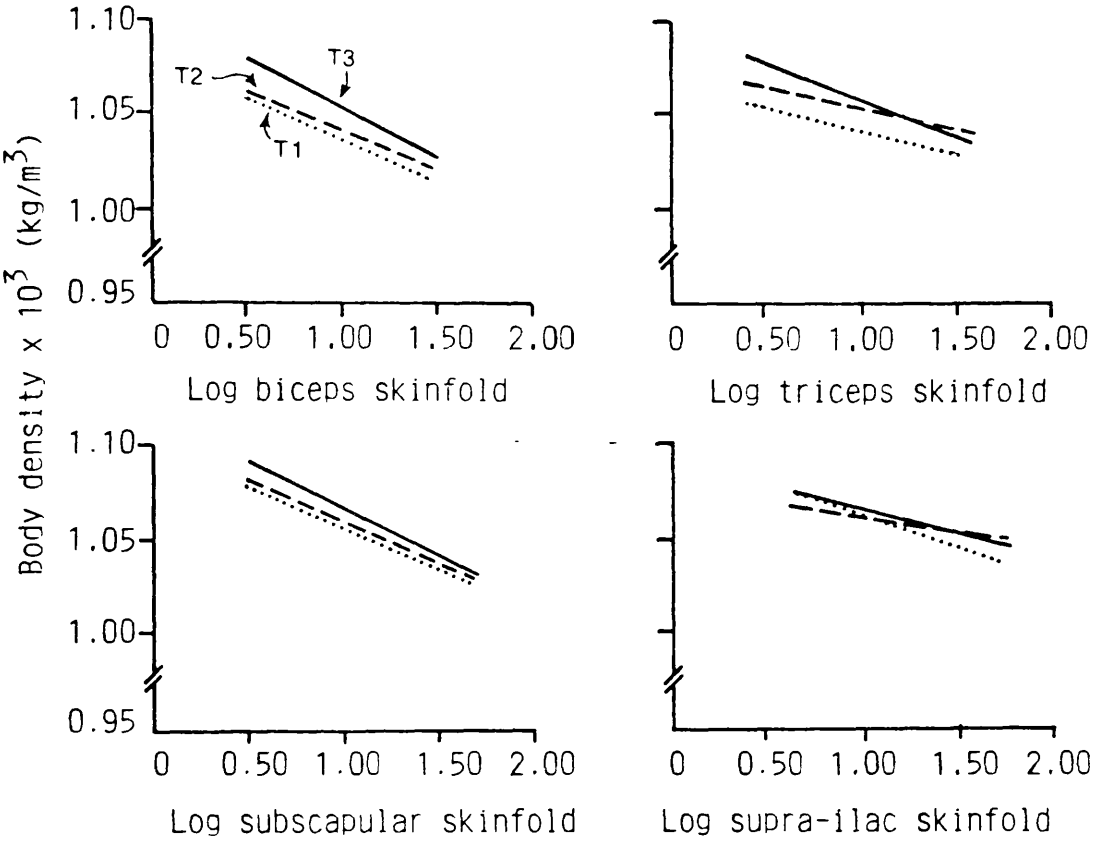


TABLE 35

Correlation coefficients (r) of body density obtained from regression analysis on log of skinfold measurements at each stage of training

	r-values		
Skinfold	T1	T2	T3
Biceps	-0.64	-0.54	-0.37
Triceps	-0.16	-0.26	0.26
Subscapular	-0.52	-0.49	-0.52
Supra-ilac	-0.59	-0.41	-0.52
Sum of 4 skinfolds	-0.62	-0.48	0.52

Linear regression equations for the estimation of body density from the log of the skinfold thickness; density = c-m.log skinfold

	T1		T2		T3	
	c	m	c	m	c	m
Biceps	1.080	0.044	1.082	0.041	1.081	0.036
Triceps	1.058	0.012	1.075	0.024	1.080	0.025
Subscapular	1.099	0.042	1.089	0.036	1.115	0.050
Supra-ilac	1.100	0.039	1.084	0.025	1.099	0.055
Sum of 4 skinfolds	1.145	0.057	1.125	0.043	1.422	0.052

T1 - prior to training
T2 - 15 weeks post training
T3 - 30 weeks post training

- body density relationship with training, the reduction in the regression coefficient values with training was not statistically significant (see Table 36). The intercepts of these regression equations were estimated assuming a common slope. There was a consistent trend for the intercept to become greater with training. These regression lines for which a common slope was determined were found to be significantly different ($P < 0.001$) at each of the testing stages (see Table 36).

Thus it appears that with the present training program there was a significant change in the skinfold-body density relationship. This change in relationship was such that: (1). after training a given sum of skinfolds predicts a higher value of body density and thus a lower value of body fat than before training. (2). after training a given value of body density corresponds to a higher sum of skinfolds value and thus higher value of body fat than before training. The effect of the present training study on the skinfold-body density relationship is displayed diagramatically in Figure 41.

(b) Magnitude of change

Assuming a common regression coefficient for the regression equations at each stage of training, the intercept estimate of the skinfold-body density linear log. relationship increased by $3.1 \times (10)^{-3}$ kg/m^3 over the 30 weeks of training (see Table 36). This value of

TABLE 36

F- statistics for testing equality sum of regression coefficients for the regression of skinfold thickness on body density at each stage of training

Degrees of freedom: 2 and 29

F- value: 0.31
NS

Common slope and intercept estimates from instances where regression coefficients are not significantly different

Common slope: 0.0586

Intercepts T1: 1.1491
T2: 1.1508
T3: 1.1522

$\Delta 3.1 \times 10^{3} kg.m^{-3}$

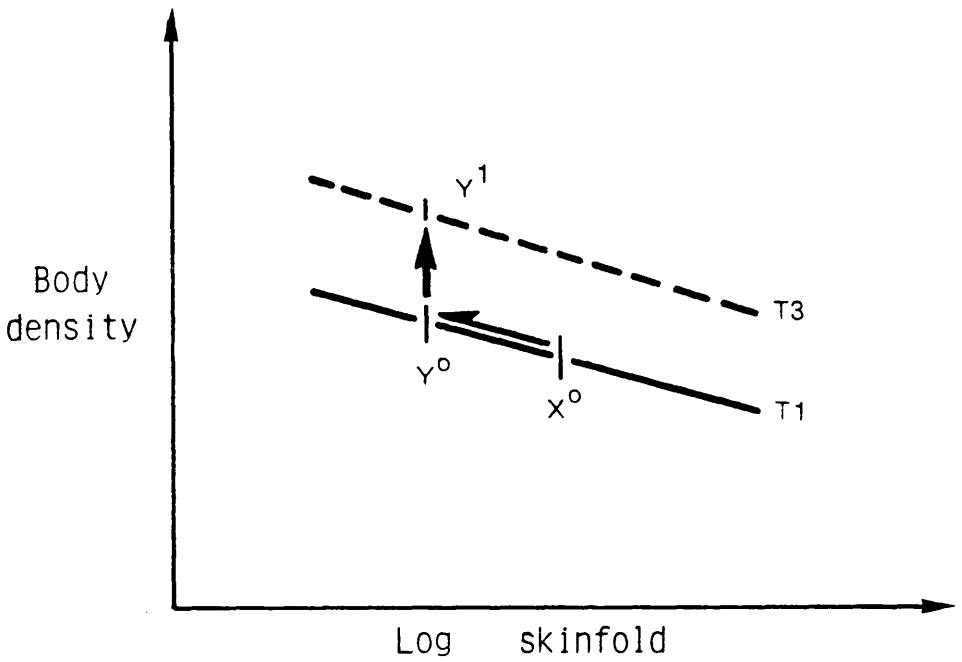
F- statistics for the analysis of covariance of the sum of skinfold thickness on body density at each stage of training

Degrees of freedom: 2 and 29

F- value: 15.8
$P<0.001$

Summary of the effect of training on the skinfold-body density regression equation

FIGURE 41



T1 - pre-training
T3 - post-training

$X^o \rightarrow Y^o$: Normal training effect ie. change in position on regression line as the result of reduction is Σ sk.

$Y^o \rightarrow Y^1$: Change in regression line as the result of alteration in skinfold-body density relationship.

Thus overall training effect $X^o \rightarrow Y^1$.

body density increase corresponds to a reduction in body fat content of some 1.2 per cent. Thus it appears that the change in skinfold-body density relationship observed during the present training program was such that it resulted in a 1.2 per cent lower estimate of body fat than that of the skinfold-body density relationship estimate before training. In other words, if the same skinfold-body density equation had been used throughout training, the change in body fat estimate from the sum of skinfold would be 1.2 per cent lower than the change in body fat content determined by body densitometry.

Another method of determining the magnitude of the alteration in the skinfold-body density relationship with training is to determine the change in body fat content from the sum of skinfolds using the Durnin and Womersley regression equations (ie. a constant skinfold-body density equation) and compare this value to the change in body fat content estimated by body densitometry. These calculations reveal that the magnitude of body fat change with training obtained from the skinfold technique was on average 1.6 % lower ($P < 0.001$) than that obtained from underwater weighing (see Table 37).

The small difference in the errors of body fat change estimated by these two methods (ie 1.2% versus 1.6%) probably reflects the difference in the present skinfold-body density regression relationship from the

TABLE 37

Changes in body fat content and fat free mass determined by skinfold method at each stage of training
(means SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Fat content (%)	23.7 ±5.7	22.2 ±2.8	20.3 ±2.9	-1.5 ±0.6 P<0.001	-1.9 ±0.5 P<0.001	-3.4 ±1.8 P<0.01
Fat weight (kg)	19.0 ±5.4	17.1 ±4.0	15.2 ±4.0	-1.9 ±1.7 P<0.001	-1.9 ±1.6 P<0.001	-3.8 ±2.0 P<0.001
Fat free mass (kg)	61.0 ±8.1	60.1 ±7.0	60.2 ±7.5	-0.9 ±3.0 NS	+0.1 ±2.0 NS	-0.7 ±3.1 NS

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

Durnin and Womersley equations.

(c) Mechanism of change

Both the skinfold and densitometric techniques of body fat content determination have methodological assumptions. Therefore the alteration in the skinfold-body density relationship observed in this study could concievably be the result of discrepancies in either of the two techniques. The relative contribution of these discrepancies to the present study are evaluated below.

(i) Skinfold technique:

Experimental error

As discussed in section 3.3.4(b), the following precautions were taken to reduce experimental error of the skinfold technique to a minimum: the same trained observer to make all assessments, all skinfold thickness assessments taken by the same standardised method throughout the study to ensure reproducibility, the four skinfold sites were chosen because of their high repoducibility values and the same regularly calibrated skinfold caliper was used throughout the study.

The skinfold technique involves the thickness measurement of a double fold of skin and compressed subcutaneous adipose tissue. To infer the mass of fat in the body from this measurement requires a series of assumptions whose validity is rarely challenged (Martin et al, 1985). The validity of these assumptions to the

present study are evaluated below:

Fat patterning

It has been accepted for some time that there are very large variations in the patterning of subcutaneous fat between different individuals (Edwards, 1951). Recent cadaver studies indicate that there are also wide variations in the amount of subcutaneous fat in different anatomical sites within the same individual (Clarys et al, 1987). Therefore the number and site of skinfold readings are critical in obtaining a representative adipose tissue pattern.

The four skinfold sites chosen in the present study are restricted to the upper body segments. Prior to the start of training in this study, good agreement was found between the average value of body fat content as estimated from the sum of skinfold thicknesses at these sites and body densitometry. (see Table 38). This finding is suggestive that prior to training, the skinfold sites selected for this study were in fact representative of overall subcutaneous fat content as well as total body fat content.

Although the skinfold sites were representative of body fat content prior to training it did not necessarily follow that these sites would be representative of the pattern of body fat loss with training. However, very little has been published in the scientific literature in this area. Despres and his colleagues (1985), in the study referred to above,

TABLE 38

Magnitude of body fat change with training and comparison of skinfold and densitometric techniques
(mean and SD's)

	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
% Fat skinfold	23.7 ±5.7	22.2 ±2.8	20.3 ±2.9	-1.5 ±0.6 P<0.001	-1.9 ±0.5 P<0.001	-3.4 ±1.8 P<0.001
	NS	P<0.001	P<0.001	P<0.05	NS	P<0.001
% Fat densitometry	22.7 ±3.7	20.0 ±4.0	17.7 ±4.7	-2.7 ±2.0 P<0.001	-2.3 ±2.2 P<0.001	-5.0 ±3.2 P<0.001

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

report on the proportion of fat loss with training from different skinfold sites on the body but note that the proportion of fat loss from the skinfold sites of the trunk exceeded that of skinfold measurements taken from the extremities. Applying an analysis, similar to that of Despres and his colleagues, to the present study confirms a similar pattern of fat loss patterning (see Table 39).

Thus, although there is very little previously published information in this area, it is quite possible that the lower estimate of body fat loss with training as obtained by the skinfold technique is the result of the four skinfold sites not being representative of total body fat loss. This hypothesis will be considered in more detail in the section 4.5.2 (c).

Proportion of body fat situated subcutaneously

A possible explanation for the change with training in the position of the skinfold-body density regression lines with training may be that a greater proportion of fat was lost internally rather than subcutaneously.

Over the 30 week training period of this study, on average 1.0 kg more of body fat was estimated as lost by using the densitometry method as compared to the skinfold method. The distribution of subcutaneous and internal fat has been a source of considerable experimental contradiction. General textbooks usually

TABLE 39

Alteration in skinfold-body fat ratio with training
(means and SD's)

Ratio	T1	T2	T3	ΔT1-T2	ΔT2-T3	ΔT1-T3
Σ4 skinfolds (mm)/% fat*	2.50 ±0.40	2.44 ±0.54	2.43 ±0.61	-0.06 ±0.31 NS	-0.01 ±0.42 NS	-0.07 ±0.50 NS
Σ4 skinfolds (mm)/fat mass* (kg)	3.20 ±0.62	3.60 ±0.84	3.12 ±0.93	-0.04 ±0.51 NS	-0.04 ±0.63 NS	-0.09 ±0.66 NS

T1- prior to training
T2- after 15 weeks training
T3- after 30 weeks training

*fat content determined by densitometry

state that about half the total body fat content is subcutaneous. However, more recent cadaver studies suggest that subcutaneous fat represents a considerably greater proportion of total body fat than does internal fat (Alexander, 1964; Martin, 1984). Alexander (1964) found that subcutaneous fat accounted for 80 percent of total body fat. Based on this estimate that 20 per cent of fat is distributed internally and 80 per cent subcutaneously, it is within theoretical limits that a subject with the average initial composition for this study (ie. total body weight: 80kg and body fat content: 22.7 per cent) could loose up to 2.9 kg of fat with training from internal sources alone (see Figure 42). Thus it is theoretically possible that 1.0 kg of body fat loss as estimated by the skinfold and body densitometry techniques could be accounted for by a loss of this same amount from internal sources with no alteration in subcutaneous fat content.

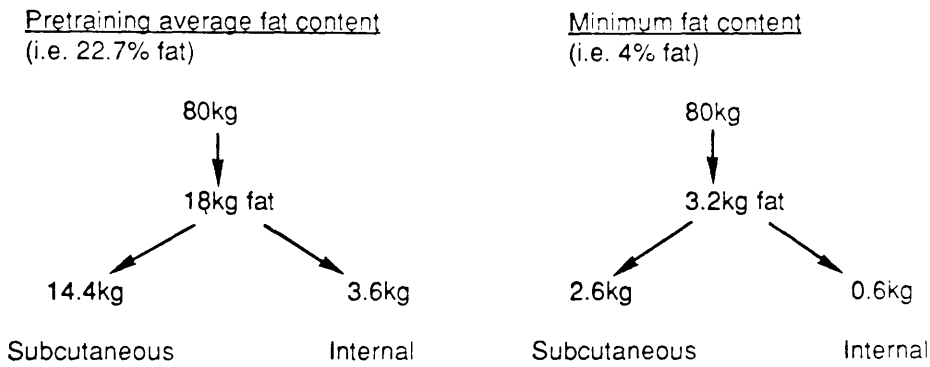
Although body fatness has been shown to decrease with exercise training, little is known about the proportions of subcutaneous and internal fat affected by this condition. Allen et al (1956) found that higher proportions of fat were stored subcutaneously with increasing values of body fatness.

The only study to address the question of the effect of training on fat distrbution was recently carried out by Despres et al (1985). Using the sum of skinfolds as an indicator of the total subcutaneous

FIGURE 42

Theoretical magnitude of internal fat loss with training

Assuming fat is distribution: 80% subcutaneous
20% internal



Thus theoretically possible to lease up to 3.0kg of fat internally with the present training.

fat, they observed that although elite distance runners have a lower proportion of subcutaneous fat, a 20 week training program did not affect the proportion of subcutaneous to total fat. The same methodology as applied by the Despres group was also applied to this present study. Although there was a tendency for the sum of skinfolds / % fat content by densitometry to fall with training (T1: 2.50±0.41, T2: 2.44±0.54, T3: 2.43±0.61), this change was not statistically significant. This analysis tends to suggest that body fat was lost with training from internal and subcutaneous sources in relatively equal proportions.

Thus it appears unlikely that the alteration in density-skinfold relationship was due to a preferential loss of body fat from internal as oppossed to subcutaneous sources.

Compressibility of skinfold

Another possible explanation for the shift in the relationship between body density and skinfold thickness with training is that skinfold compressibility may decrease with training. Such a reduction in skinfold compressibility would result in an increased skinfold thickness, even though the subcutaneous fat content had not altered.

It has been demonstrated that with increasing age there is a tendency for skinfold compressibility to decrease (Brozek & Kinsley, 1960) and that skinfold compressibility can alter. However, there has been no

reported experimental evidence from training studies to either support or refute the hypothesis of reduction in compressibility with training. Never-the-less it might be expected that as the result of training, the loss of underlying subcutaneous fat may reduce skin tension and therefore, if anything, increase compressibility. Indeed in many situations the author did observe what appeared to be a marked reduction in the skin tension with training, particularly in those individuals with large initial body fat stores.

Thus it appears unlikely that changes in skinfold compressibility would account for the altered body density and skinfold thickness relationship.

ii. Body densitometry

Experimental error

It is possible that the difference in estimates of body fat change with training could be the result of experimental errors in the underwater weighing method. However, as has already been discussed above, the reproducibility of this technique in this laboratory is high and moreover, care was taken to ensure that throughout all stages of testing the experimental procedure was rigorously adhered to and the condition of the subject was standardised as much as possible.

Thus it is probably reasonable to exclude experimental error of the densitometric method. However, the alteration in the skinfold-body density relationship with training could reflect a breakdown in

one of the assumptions implicit in the densitometric technique.

The densitometric method is based on the two compartment model and therefore regards the body as consisting of either fat mass or fat free mass. The following four assumptions are central to the method:

1. Density of fat (0.901 kg/m^3) and fat free mass (1.100 kg/m^3) are known
2. These densities are of a constant value in all individuals
3. Density of tissues that compose the fat free mass are the same for different individuals
4. Man differs from standard man only in fat content.

The main objection to the method of densitometry is the assumption that there exists a fat free mass of fixed density that remains constant and has a constant composition. Theoretical calculations demand that the fat free mass be composed of about 72% water, of density $1.00 \times (10)^3$ kg/m^3; 20% protein, of density $1.34 \times (10)^3$ kg/m^3 and about 7% mineral of density of about $3.00 \times (10)^3$ kg/m^3. Based on this assumed composition, the density of fat free mass is determined $1.100 \times (10)^3$ kg/m^3 (Siri, 1956).

Observations made during recent cadaver studies have shown that the value of fat free mass density is not constant in different individuals but has a standard error of some plus or minus 0.002 kg/m^3 (Martin et al, 1985). Although this error appears to be

relatively small, it would result in an error of fat content of approximately plus or minus 7.5 per cent fat in an individual with a body density of 1.100 kg/m^{3} and predicted body fat of 16.1 per cent fat with a fat free mass density of 1.100 kg/m^{3}.

In some studies in which the body fat of an athletic population has been assessed by densitometry, the estimate of body fat is less than 4 per cent (Pollock et al, 1977; Martin et al, 1985b). Although the amount of 'essential fat' is not accuately known, any body fat estimate less than 3 to 4 per cent is very likely to be anomalous (Wilmore, 1983). This discrepancy in fat content estimate with densitometry is generally accounted for as a violation in the constancy of the fat free mass density.

In section 4.5.1(a) it was outlined that in this study no significant change was observed in the weight of fat free mass with training. However, this maintenance of the mass of the fat free compartment does not necessarily imply that the composition of this compartment remains the same with training. It has been suggested that there is variation between normal individuals in the water, mineral and protein content, and therefore the density of fat free mass (Durnin and Womersley, 1974). Such a variation in fat free mass will influence the value for fat mass as predicted from these measurements.

It has been known for some time that aging is

associated with bone demineralisation (Sorernsen et al, 1968). Theoretically this change in bone content could reduce the density of fat free mass by up to 0.012 x $(10)^3$ kg/m^3 (Durnin & Womersley, 1974). Although aerobic endurance training is unlikely to alter the mineral content of the fat free mass, such as in situations of bone demineralisation with aging, it could possibly alter the water and protein content. There are two main theoretical situations associated with training that could result in such a change.

Changes in adipose tissue content

When body fat is lost with training it is not lost exclusively in its chemical form of fat alone but as adipose tissue. Adipose tissue comprises about 64% fat, 22% 'cell residue' and 14% extracellular water (Brozek et al, 1963). Since the density of water at 37 °C is approximately unity and that of the cell residue is about 1.078 x $(10)^3$ kg/m^3 (Brozek et al, 1963), the fat free mass loss has a relatively low density of 1.047 x $(10)^3$ kg/m^3. A decrease in body fat content would therefore also be accompanied by a rise in the density of fat free mass.

It is possible to calculate the effect of this adipose tissue effect on fat free mass density. According to the densitometric method, there was a reduction in body fat content with training in this study of some 4.8kg on average. If this loss is assumed to have the Brozek composition of adipose tissue, it

will consist of 2.6kg of fat, 0.9kg of 'cell residue' and 0.5kg of extracellular water. ie a loss in fat free mass of 1.4kg. For the sake of this calculation, the average fat free mass weight is considered to be equal to the pre-training fat free mass (ie 60.0kg) and the composition of this fat free mass is intially as outlined by Brozek. It was estimated that the effect of this loss in 'cell residue' and extracellular water would increase the density of fat free mass by 0.008 x $(10)^{3}$ kg/m^{3} (see Figure 43).

Thus it appears that a result of regarding fat loss as adipose tissue loss would be to increase the fat free mass density. Such an increase in fat free mass density with training could explain the greater reduction in fat mass observed with the densitometric method and therefore also possibly account for the observed change in skinfold-body density relationship with training.

Changes in muscle mass

Another means whereby the density of fat free mass may change with training would be due to an alteration in its protein and water content as the result of a change in muscle content. Recent evidence by Martin et al (1986) from their cadaver analysis, reveals that the percentage of muscle within the fat free compartment may vary from 41.9% to 59.4%. Womersley et al (1976) using total body potassium have shown that increased muscularity results in a decrease in density of the fat

FIGURE 43

Theoretical alteration in fat free mass density with training adipose tissue loss

Pretraining:-

Assume FFM composition = 72% water
21% protein
7% mineral

Thus $\frac{1}{d.ffm} = \frac{0.72}{1.000} + \frac{0.21}{1.340} + \frac{0.07}{3.000}$

$= 0.72 + 0.157 + 0.023$

$= 0.900$

$d.ffm = 1.111 \times 10^3 \text{ g/ml}^{-3}$

Post-training:-

According to densitometry = 4.8kg adipose tissue loss with training.
Change in ffm weight = 61.0 - 1.7 = 59.3kg
Assume adipose tissue content = 64% fat (chemical) = 3.07kg
22% 'cell residue' = 1.06kg
14% extracellular water = 0.67kg

ie Water mass: 43.9 - 0.67 = 43.2kg (72.8%)
Protein mass: 12.6kg (21.2%)
Mineral mass: 4.2kg (7.1%)

Thus $\frac{1}{d.ffm} = \frac{0.728}{1.000} + \frac{0.212}{1.340} + \frac{0.071}{3.000} - \frac{0.018}{1.078}$

$= 0.894$

$d.ffm = 1.119 \times 10^3 \text{ kg.m}^{-3}$

As the result of adipose tissue loss with training; d.ffm would theoretically increase by 0.008×10^3 g/ml.

fat free mass. Mean values which have been quoted for the density of skeletal muscle at 37°C are 1.064 kg/m³ (Gersh I. et al, 1944) an 1.066 and 1.064 kg/m³ calculated from data on two reasonably normal human cadavers (Forbes and Lewis, 1953). Thus an increase in muscle alone would tend to reduce the density of fat free mass.

The pattern of skinfold-body density relationship change with training is suggestive of an increase in fat free mass density rather than a reduction as outlined above. A reduction in fat free mass density with training would imply a reduction as opposed to an increase in body muscle. There was no evidence of such a change with training in this study from calf circumference, although this is a relatively crude measure of the muscle mass in one body area. Never-the-less a reduction in skeletal muscle mass seems an unlikely pattern of body composition change with aerobic endurance training.

(d) Athlete studies

It appears from the analysis in the previous section that a breakdown in both the assumptions of the densitometric and skinfold methods could account for the change in the skinfold-body density relationship observed in the present training study. Thus the discrepancy in estimates of fat loss with training could be accounted for by either of the two techniques.

Two groups of experimental evidence from body composition assessment of athletes have expressed conflicting findings with regard to this discrepancy. One group of athlete studies is suggestive of densitometric error while the other more recent body of evidence suggests that it is skinfold error. The findings of these two groups of opinion will be reviewed below in relation to the present study.

Although the amount of 'essential' body fat is not accurately known, any body fat estimate less than 3-4% is very liklely to be anomalous (Wilmore, 1983). However, several athletic studies have derived estimates of 2% or lower (Pollock et al, 1977; Adams et al, 1982) and some have even obtained a body density estimate that corresponds to a negative value of fat content (Micheal and Katch, 1968). It seems clear that densitometry leads to errors when predicting the body fat content of some athletic populations. This discrepancy is generally considered to be reflective of a breakdown in the the assumed value of fat free mass density (Martin et al, 1986). It therefore appears from this group of athlete studies that body densitometry is associated with a significant error in body fat prediction.

Sinning and his colleagues have performed two recent studies on male and female athletes comparing the error of prediction in the body fat estimate determined by densitometry versus that determined from

a variety of different skinfold regression equations which utilise different skinfold sites (Sinning et al, 1985). It was found that the skinfold equations of Durnin and Womersley used in this study consistently over-predict body fat content by 3.9 to 5.5 per cent fat when compared to densitometric values. However, the Jackson and Pollock equations were able to predict body fat to within 0 to 1.0 per cent fat of densitometry.

These results suggest that the Jackson and Pollock equations illustrate body fat content in a athletic population more accurately than the the equations of Durnin and Womersley. An explanation for this discrepancy can be gained by comparision of the two populations from which each set of equations were derived. A summary of the male populations considered by each of these authors is shown below.

Durnin & Womersley	Jackson & Pollock
n = 209	n = 308
17-72 yrs.	18-61 yrs.
5-50 %fat	1-33 %fat
0.990-1.082 g/ml	1.061-1.0996 g/ml

This comparison reveals that although the two populations were of a similar age range, the population of Jackson and Pollock was larger and more hetreogenous in terms of low body fat and high body density values. Durnin and Womersley described ther population as containing a "propendernce of moderately sedentry middle-aged

men" while Jackson and Pollock describe their population as "wide range of men who varied considerably in body structure, body composition and exercise habits". Thus it appears that the population of Jackson and Pollock contains more athletic subjects and may therefore be more repesentative of the body fat content such groups. However, as this population difference does not appear to that great it may that other factors may be responsible for the prediction discrespancy of the 2 set of equations.

Comparison of the two groups of equations reveals that there are no differences in the number of sites used per equation (both use four) but that the Jackson and Pollock equations appear to consider other skinfold sites: thigh, pectoris, abdomen and calf. Thus it appears that the Jackson and Pollock equations are able to predict body fat in athletes more accurately than the Durnin and Womersley equations because the former consider both upper and lower body skinfold sites while the later assesses only upper body sites. That the Durnin and Womersley equations have been successful in accurately predicting the body fat content in non-athletic populations is suggestive that athletes have an altered pattern of body fat distribution that these equations do not take into account.

It is not possible to conclude from the studies of Sinning et al whether this different distribution of subcutaneous body fat in athletes is the result of genetic factors or physical training. However, if it is assumed that part of this phenomena is the result of physical training then it is possible to extrapolate these results to the present study. Such extrapolation leads to the conclusion that the discrepancy in the body fat estimate of the densitometric and skinfold methods observed with the present training program, is the result of the skinfold sites selected in this study not being completely reflective of the pattern of body fat loss. If neglecting to assess the skinfold site from the lower limbs leads to over-estimation of the body fat content after training compared to densitomtry, it can be concluded that the present training program results in a high proportion of fat loss from the lower limbs. The only data in this study that aids comment on this hypothesis is the indirect evidence from changes in body circumference. Both gluteal and thigh circumference demonstrated significant reductions in size after the 30 weeks of training, this change presumably reflecting a marked loss in body fat from these regions (see Table 28).

In conclusion it appears from these athlete

studies, that the change in the skinfold-body density relationship with the present training program is the result of both a change in density of fat free mass as well as the selected skinfold sites not being reflective of the total body pattern of subcutaneous and total body fat loss.

(e). Physiological significance

The findings of the present study are suggestive that the magnitude of fat loss estimated in previous training studies may be inaccurate. In accordance with the pattern of results of the present study the majority of training studies will have under-estimated body fat loss because of the use of skinfolds. Never-the-less in view of the smaller overall change in body fat content of previous studies, this under-estimation would be expected to be small.

The regression equations of Durnin and Womersley consistently over-estimated body fat in both the post training situation in this study and also in athlete groups. To prevent such a continued error it is critical to use the other non specific population regression equations of Jackson and Pollock, which have no such athlete prediction error, or use a new set of regression equations based on the appropriate athletic group.

But more work is required in this area particularly with regard to longitudinal changes in fat free mass density associated with exercise training.

4.5.3 Energy metabolism

Physical exercise and dieting are often suggested as means of manipulating ones body fat content. Sceptics of the role of exercise in weight control, dismiss its potential effects by outlining that the increase in energy expenditure with exercise is offset by an increase in appetite and thus energy intake (Mayer, 1956).

Animal studies demonstrate that exercise training does not supress appetite (Wilmore, 1983) but it is difficult to extrapolate these results directly to man. However, few studies have specifically examined the effect of exercise on the relationship between the body's energy stores, energy expenditure and energy intake. The lack of scientific literature in this area probably reflects the practical difficulties of assessing both energy expenditure and energy intake together with the degree of accuracy of these methods. The best estimates of total 24-hour energy output are obtained by use of direct calorimeters or respiratory chambers. Obviously, these are inappropriate for long-term non-laboratory based exercise training regimes. Precise measurement of energy intake requires careful weighing of food and duplicate sampling.

Monitoring food intake itself may intrude so much on daily life and habits that eating behaviour becomes affected. Even if it were feasible to assess energy intake and energy expenditure directly, measurement to within ± 100 kcal/day is virtually impossible. Never-the-less such an error in energy value could result in a significant alteration in the body energy stores over a period of time. (Garrow, 1978).

In the present study, the means of energy expenditure and energy intake assessment were selected in order to overcome the practical methodological difficulties described by previous studies. The methods of assessment are outlined in detail in section 2.6.3(d). The method can be summarised in terms of the three assumptions upon which it was based. Firstly it assumed that the subjects were in relative energy balance prior to the study, so that any alteration in energy store could be accounted for by an alteration in energy metabolism occuring within the study period. Secondly, it was assumed that any change in the body's energy store with training would be entirely reflected by an alteration in body fat content. The standard energy value of 7700 kcal per kg of fat was applied to these figures (Epstein & Wing, 1980). Finally it was assumed that any imbalance between the change of energy expenditure and change in body's energy store with training, would be reflective of an alteration in energy intake. The gross energy expenditure cost of the

training program was predicted from the subject's training diary on the basis of running speed, duration and subject body weight. The net training energy expenditure was then determined by subtraction from the exercise energy cost of the resting energy value (Garrow, 1978). The validity of these assumptions will be discussed below.

(a) Pattern of change

Over the 30 weeks of the present training study, the increase in exercise energy expenditure was observed to be significantly in excess of the energy value accountable to the change in body energy store (Table 29). On the basis of the assumption of initial energy balance, a significant increase in energy intake, sufficient to balance this energy difference, must have occurred over the 30 weeks of the present study. This increase in energy intake appeared to be restricted to the final 15 weeks of training, there being little difference in the magnitude of change of energy expenditure and energy store during the initial 15 weeks of training.

The evaluation of the pattern of change in energy intake with training in this study posed some initial planning problems particularly since very few previous training studies have reported directly on this parameter. However, in a recent publication, Epstein and Wing (1980) reviewed selected aerobic training studies and predicted the change in energy intake. This

prediction of energy intake was based on a similar method to the present study ie. from the difference between the change in exercise energy expenditure and the change in energy store value. Epstein and Wing selected only those studies which considered sedentary non-obese male groups who did not participate in a planned diet during the training program. Exercise energy expenditure was determined by the same indirect method as used by the present study and therefore only included studies that specified exercise parameters sufficiently to allow calculation of energy expenditure. Although change in body energy store was determined on the basis of 7700 kcal/kg fat, body fat changes were assumed to be entirely reflected by a change in total body weight. It is feasible that the total body weight change with training may not be the same as the change in body fat store. For the purpose of comparison with the present study, this potential error was overcome by considering only those studies in which body fat changes were reported. On this basis the changes in energy intake with training were re-calculated for 15 of these studies (174 subjects). The summary values of this re-analysis are shown in figure 40.

It is clear from the mean values in this review that the energy value associated with a change in body fat with training is somewhat less than the energy cost of exercise training. This discrepancy in energy values

TABLE 40

Reanalysis of energy intake prediction from training studies reviewed by Epstein & Wing (1980)

(mean and SD's)

Subject age (years)	Program duration (weeks)	Session duration (minutes)	Session frequency (days/week)
38 ± 8	19 ± 3	35 ± 12	3 ± 1

Initial body weight (kg)	Initial body fact content (%)
81±7	20 ± 4

Total net exercise energy expenditure (kcal)	Change in energy store value (kcal)	Change in energy intake (kcal)	Per week	Per day
19317 ±16441	10530 ±1767	+8787 ±9091	+469 ±532	+67 ±76

(change in body fat -1.3 ± 1.40)

Control studies (n = 7 studies; n = 64 subjects)

Net exercise expendible (kcal)	Change in energy store (kcal)	Change in energy intake	Per week	Per day
0	1660 ± 2696	-1660 ± 2696	-83 ± 135	-12 ± 18

is suggestive of an increase in energy intake with training. When the average increase in energy intake in these reviewed studies is expressed as an overall value for the complete period of training it appears to be large (ie. 8787±9091 kcal). However, when expressed as a change in energy intake value per day (ie. 67±76 kcal) the increase is less than the ±100 kcal error of energy intake measurement. Thus it is likely that such a change in energy intake with training would not be detected by conventional assessment procedures.

The few studies that have attempted to assess the change in energy intake by direct assessment of food intake have failed to show any significant change over the period of training. Dempsey et al (1964) subjected a group of moderately obese and non obese men to an 8 week training program in which a significant reduction in body weight and body fat content was observed. Although predictive calculations suggest an increase in energy intake over the 8 weeks, the daily record of food intake showed that 1 hour of exercise per day had no effect on either appetite or energy intake. A recent study measuring the change in 7-day food intake in a group of 33 females over 10 weeks of aerobic endurance training came to a similar conclusion (Dikson-Parnell & Zeichner, 1985). This discrepancy in the predictive and direct findings is presumably reflective of the above hypothesis that the increase is insufficient to to be detected by contemporary means.

Of the 15 studies reviewed, 13 demonstrated an increase in energy intake with training while the other 2 demonstrated a reduction. Thus it appears that the pattern of energy intake increase in the present study agrees with the majority of these previous studies. However, the magnitude of the increase in energy intake in the present study is considerably greater than in the reviewed studies. Indeed, only one study of those reviewed demonstrated an increase in daily energy intake value in excess of the error associated with energy intake assessment (Leon et al, 1979). The increased magnitude of energy intake change observed in the present study will be discussed in the section below.

(b) Mechanism of change

a. Experimental error

Controls

Seven of the 15 studies reviewed above examined the change in energy intake of a non-exercising control group over the period of the study and although these controls performed no prescribed exercise training they, like the exercise group, revealed an increased energy intake over the period of the study.

This finding is perhaps suggestive that the increase in energy intake observed in the exercise group is not a result of the exercise training per se. However, the magnitude of this increase in energy intake (1660±2696 kcal) was considerably less than that

of their exercising counterparts. Indeed when expressed as a change in energy intake per day this value was virtually insignificant ie. 12±18 kcal.

Calculation of change in energy store value

In the present study it was assumed that the change in value of the body energy store resulted entirely from a change in the body fat content and a standard energy value was then applied to this change. This assumption could lead to errors in energy intake prediction in several ways.

The present pattern of results may have reflected the underestimation of body fat loss by hydrostatic weighing. However, judging from skinfold predicted body fat changes in this study this seems unlikely.

It is plausible that the change in energy store was not restricted entirely to the body fat compartment. Glycogen, like fat, also represents a considerable energy store and depletion of such carbohydrate stores could offset the change in fat store. Extra energy would be expended in protein synthesis as the result of an increase in skeletal muscle. However, it is unlikely that these latter two factors would contribute significantly. Firstly, the primary fuel during long duration moderate intensity exercise, such as the present study training program, is fat rather than carbohydrate (Astrand and Rodahl, 1977). Therefore it appears unlikely that glycogen depletion would significantly contribute to the changes

in the body energy store. Additionally, no significant change in fat free mass or calf circumference was observed over the period of training in the present study. This is suggestive of little change in skeletal muscle mass.

Van Itallie & Young (1977) have suggested that the discrepancy between the energy cost of training and the change in energy store, reflects that the standard energy value of fat is not 7700 kcal per kilogram. Epstein and Wing (1980) in evaluating their review did not disagree with this hypothesis but dismissed it as being "..impossible to attempt to take account of..". However they did "..not feel that it would lead to any systematic basis across studies". To account for the observed increase in energy intake in this study the standard energy value of fat would need to have been as great as 14000 kcal per kilogram.

Calculation of exercise training energy cost

For practical reasons exercise energy cost was predicted in this study rather than being assessed directly. The method of prediction used in this and previous studies is based on multiplying the standard energy cost of a particular activity by the number of minutes over which this activity was performed (Astrand and Rodahl, 1977). The standard exercise energy cost is usually derived from standard tables (Durnin and Passmore, 1967). In the present study the energy cost of running was obtained from the standard equation of

Shephard (1969).

It is often argued that these standard energy values are one of the main sources of error in energy expenditure prediction. To reduce such a systematic error, the standard energy cost equation of running was selected on the grounds that it was derived on a variety of subject types who were tested over a range of treadmill running speeds (see section 2.6.3(c)). To take account of the difference between treadmill and outdoor running, a correction was made for a wind resistance factor (Pugh, 1970). It was not feasible to correct for the less consistent terrain associated with outdoor running. This terrain factor would almost certainly lead to a greater estimate of exercise energy cost and thereby increase the magnitude of any discrepancy when compared to the value associated with a change in body fat content.

The variables of running speed and duration used in the prediction of exercise energy cost in this study were entirely derived from the subjects training diaries. Although they represented a possible source of experimental error the majority of these diaries were carefully and accurately maintained. Any diaries with suspect training entries were entirely ommitted from the exercise energy cost analysis.

Assumption of energy balance

The calculation of energy intake requires that prior to the study the subjects were in relative energy

balance. Although not directly assessed, the relatively constant total body weight of all the subjects in the months prior to training is suggestive that this criteria was satisfied.

The method of energy intake prediction in this and the above reviewed studies is based on the concept of energy balance ie. a discrepancy in the change in energy store value and change in energy expenditure is suggestive of a change in energy intake. However, Garrow (1970) in one of the most authoritative textbooks on energy metabolism suggests that energy balance may not be a valid concept. In support of this notion is a review of 6 energy balance studies involving some 69 individuals by Durnin (1961). Although all subjects maintained weight balance, only 2 (5.8%) subjects were in daily balance, 41 (59%) subjects in some degree of balance and the remaining 26 subjects (21%) revealed no evidence of balance what-so-ever.

Although it is possible that energy balance may not have applied during this present study it is impossible not to take its contribution into account.

b. Decrease in non exercise energy expenditure.

The greater magnitude of exercise energy cost versus the energy value associated with a change in body energy store was interpreted as indicative of an increase in energy intake of the subjects during the period of the study. However, it is possible that this

pattern of change is suggestive of a fall in non exercise energy expenditure.

It has been proposed by Epstein and Wing (1980) that during a training program subjects may reduce their activity outwith the study itself, eg by going to sleep earlier as the result of increased fatigue. The pre-training assessment of low recreational activity levels (see figure 6) suggests that such a reduction in non training activity pattern in the subjects of the present study would have little effect on the present pattern of results. In addition, subject interviews after 15 and 30 weeks training revealed no conscious change in daily activity.

In addition to recreational activity, resting metabolic rate and thermogenesis are reported to make a significant contribution to daily energy expenditure. It has been claimed that exercise can ellicit a prolonged and significant effect on post-exercise metabolism (Hermansen et al, 1984). Pacy et al (1986) recently reviewed 17 training studies which examined post exercise metabolism. They concluded that although it appears that resting metabolic rate and thermogenesis may rise following intense execise, this is only an acute effect of relatively small magnitude.

Body composition is considered to be one of the main factors determining energy expenditure, both in terms of basal and exercise metabolic rates. Dore and his colleagues (1982) have demonstrated that basal

metabolic rate is determined by both total body weight and fat free mass. Although fat free mass remained relatively constant over the 30 weeks of the present study, the marked reduction in total body weight is suggestive of a fall in basal metabolism. Literature is very divided on the effect of training on basal metabolic rate, some report an increase, others a decrease and others no change (Schneider & Forster 1931; Terjjung & Tipton 1970; Warwick & Garrow, 1981). Moreover in studies that do report a significant increase, it is difficult to determine if this is just a carry forward effect of exercise and therefore not a 'true' basal metabolic rate. However, using the prediction equation of Dore et al, the reduction in body weight observed over the 30 weeks of this study accounts for a reduction of basal metabolic rate of some 0.04 kcal/min ie. 4032 kcal over the whole 30 week period.

It has been shown that during exercise, particularly weight supported exercise such as walking, metabolic rate is proportional to body weight (Whipp & Wasserman). Therefore the change in body composition observed with the present training study would result in a decrease in energy expenditure. However, it is difficult to accurately assess the contribution of this reduction in energy expenditure to the pattern of change in this study.

c. Increase in energy intake

Reviewing both the factors of experimental error and changes in non exercise energy expenditure, it appears that these factors can to a large extent be dismissed. Therefore the pattern of energy metabolism resulting from this study is strongly suggestive of a significant increase in energy intake, particularly in the final 15 weeks of training. In support of this many of the subjects when interviewed at 30 weeks did report a subjective increase in appetite during the later stages of training. Moreoever many also voluntarily increased carbohydrate intake as part of their carbohydrate loading regime.

The mechanism of appetite regulation has been the subject of considerable investigation (Wilmore, 1983). Indeed several factors such as increased body temperature, catecholamine release and increased blood glucose have been reported to have suppressive effects on the appetite centre of the hypothalamus (Wilmore, 1973). However, the precise mechanism leading to the observed increase in appetite is not known although it probably reflects a protective response to prevent complete depletion of the body fat stores. Indeed if it were not for the increase in energy intake, a loss of some 10.1 kilograms of fat (13.1 %), more than double the actual loss, would have been observed over the 30 weeks of training.

(c) Physiological significance

Aerobic exercise is often dismissed as a difficult way to lose fat in view of both the small energy cost associated with such exercise and the potential effect of increased energy intake. However, this study has clearly demonstrated a significant and marked reduction in body fat content. This benefical effect of aerobic endurance training on body composition in this study occurred despite a significant average increase in energy intake of over 100 kcals per day. Such an increase in energy intake may be an important factor in maintaining the compliance of individuals when training to lose body fat content.

Brownell et al (1986) suggest form athlete studies that the body has a protective mechanism to 'defends' itself against excess fat depletion. The increase in energy intake observed over the training period of this study is perhaps reflective of such a defensive mechanism.

CONCLUSION

The purpose of this study was to investigate the suggestion from previous studies that there is reduction in training adaptibility with aging.

Magnitude of training effect

An average increase in weight corrected VO2max of 15 percent was observed during the period of this present training study. This increase is comparable to the 10 to 20 percent range of VO2max change quoted by the American College of Sports Medicine in a review of aerobic endurance training studies involving young male adults. In agreement with previous middle-aged training studies, this increase in VO2max was observed to be entirely the result of an increase in peak in cardiac output with no change in calculated oxygen difference. The reduction in submaximal heart rate with training observed with this study was also within the range typically reported from young adult training studies.

Severity of training programs

The severity of the present training program increased progressively upto the 28th week. Taking into account this progression, the overall severity of the present training program, in terms of training duration and frequency, is greater than that performed in

previous training studies involving both young adult and middle-aged subjects.

This increased training load is illustrated in terms of the magnitude of fat loss with training. A reduction of on average 5 percent of total body weight in body fat content was observed during this study. The magnitude of this change is considerably in excess of the range of body fat alteration reported in previous training studies of both young and older subjects. The increased magnitude of change is almost certainly reflective of the greater energy costs of the present study exercise program in comparison to previous training programs.

The results of the present study indicate that middle-aged males can achieve the same magnitude of cardiorespiratory change to that observed in previous studies involving young adults. However, in support of the hypothesis that training adaptability is reduced by aging, the severity of aerobic endurance training to achieve this change was greater in this stdudy than that reported in previous young adult training studies.

In terms of the success of the training program with regard to marathon performance, 38 of the 53 subjects who started the program, completed the marathon distance in times ranging from 3 hours 22 minutes to 5 hours 27 minutes.

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APPENDIX A

Running heart research

Volunteers are being sought for a unique investigation into coronary illness. STEWART McINTOSH explains how the Scottish People's Marathon will help study the problem.

HEART ATTACKS are a major risk for men aged between 35 and 50. But thanks to an unusual initiative involving doctors, scientists, and the Glasgow Herald, a major study is to be carried out on 40 participants in this year's Scott's Porage Marathon measuring the effects of marathon training — does it reduce body fat? How does it change the level of fitness? What is the effect on the heart?

Male volunteers between 35 and 50 who intend to run in the marathon on September 30 are being urgently sought for the experiment. The only condition is that they should have little or no running experience. Those who are physically fit need not apply. The object is to measure the effect of training on unfit people.

Volunteers should immediately contact Rod Taylor at the Institute of Physiology, Glasgow University, (telephone 041 339 8855, ext. 497 or 612, or 041 637 1321 in the evenings).

Despite the impact of the marathon boom, research on the physical effects of constant training on unfit people has been limited. The study which will be carried out by Glasgow University scientists and the Western Infirmary's cardiac unit will provide one of the most extensive studies so far.

The volunteers will be subjected to a rigorous battery of tests, including sessions on exercise bikes to measure the effects of training on the ability of the lungs to consume and process oxygen. Doctors from the cardiac unit will be monitoring changes in the strength of the subjects' hearts as their training builds up. Meanwhile, scientists from the University's physiology department will carry out a wide range of physical measurements to ascertain any changes in height, weight, limb circumference, bone diameter, and body fat.

The high incidence of cardiac problems in areas like the West of Scotland is sometimes put down to the levels of cholesterol (which comes from dairy foods and animal fats) in the bloodstream. The Herald volunteers will have their cholesterol levels monitored to find out whether increased fitness reduces the cholesterol level.

Volunteers will have to be highly motivated towards finishing the marathon and be prepared to turn up on six occasions between now and September 30 for the various tests.

Stan Grant, of Glasgow University's department of physical education, believes that the experiment will be the first of its kind. "This study represents a unique level of co-operation between doctors and scientists which will provide us with a much better picture of the effects of physical fitness than we have had previously.

APPENDIX B

PHONE CONTACT QUESTIONAIRE
MARATHON STUDY RECRUITMENT

1. Name _____

2. Age _____(MUST be 35 to 50 years)

3. Address _____

_____

_____

4. Phone Number Home _____

Work _____

5. Have you begun to train for the marathon yet?

Yes _____ No _____

Do you consider yourself very physically fit?

Yes _____ No _____

6. Have you been hospitalized recently?

Yes _____ No _____

Are you receiving regular medical treatment at the moment?

Yes _____ No _____

7. Would you be willing to be tested during the next three weeks on two separate occassions which will involve a weekday and a weekend, Saturday or Sunday, and be fairly flexible in the time you would be available during this period?

Yes_____ No _____

Thank you again for calling. Rod Taylor will be in contact by phone in the next couple of days to let you know if you have been successful. Meantime he asks if you would not start training at any cost.

APPENDIX C

Name: _____ Phone _____

Address _____

Date _____ Birth Date: _____

Personal Physician's Name: _____

MEDICAL HISTORY

(self–administered)

1. Have you ever been hospitalized? YES _____ NO _____

Hospitalized for Disease/Operation	Duration	Age When Hospitalized	Physician's Comments
I.			
II.			
III.			
IV.			

2. List any disease, illness, injury, you have had not listed in 1. above. (e.g. mumps, measles, broken bones, etc.)

I. _____

II. _____

III. _____

IV. _____

3. Do you have, or have you had, any of the following?

	YES	NO	PHYSICIAN'S COMMENT
Alteration of your ability to remember	___	___	_____
Recurring headaches	___	___	_____
Recent changes in your vision	___	___	_____
Chest Pain	___	___	_____
Cough on Exertion	___	___	_____

	YES	NO	PHYSICIAN'S COMMENTS
Numbness of an arm or leg	___	___	_____
Weakness of an arm or leg	___	___	_____
Difficulty in speaking, or slurred speech	___	___	_____
Fainting or dizziness	___	___	_____
Difficulty in walking (staggering)	___	___	_____
Shortness of breath	___	___	_____
Lung Diseases	___	___	_____
Rheumatism or arthritis	___	___	_____
Heart disease	___	___	_____
Epilepsy	___	___	_____
Tumors	___	___	_____
Mental illness	___	___	_____

4. Do you have or have you had diabetes mellitus (sugar diabetes)?
   YES _____ NO _____

   A. If yes, what kind of treatment did/do you receive:
      Insulin _____ Diet _____ Pills _____ No treatment _____

5. Have you ever had or been told you had high blood pressure:
   YES _____ NO _____

   A. If yes, when? _____

   B. Do or did you take medicine for high blood pressure?
   YES _____ NO _____

6. List all medicine (include vitamins, etc.) you are taking.

[illegible]	Cause of Medication	Physician's Comment

SMOKING AND EXERCISE

Full Name (Print) _____ Date: _____
(last name first) I.D. _____

Please circle the appropriate answer.

1. Do you smoke cigarettes at present? Yes No

2. If "yes", how many per day?
   less than ½ pack ½ pack or less than 1 pack 1–2 packs more than 2 packs

3. Do you inhale? Yes No

4. How long have you been smoking?
   less than 1 year 1–5 years more than 5 years

5. Did you smoke cigarettes in the past and quit permanently? Yes No

6. If "yes", how many did you smoke per day?
   less than ½ pack ½ to less than 1 pack 1–2 pack more than 2 packs

   Did you inhale? Yes No

7. When did you quit smoking cigarettes?
   less than 1 year ago 1–5 years ago more than 5 years ago

8. Do you smoke cigars at present? Yes No

9. If "yes", how many cigars per day?
   less than 2 2–5 more than 5

10. Do you smoke a pipe? Yes No

11. If "yes", how many pipefulls do you smoke per day?
    less than 2 2–5 more than 5

FAMILY HISTORY

Have any of your family suffered from?

	YES	NO	AGE	RELATIVE
Heart attacks.	...	...	...	.....
High Blood Pressure.	...	...	...	.....
High Cholesterol.	...	...	...	.....
Diabetes	...	...	...	.....
Congenital Heart Disease	...	...	...	.....
Heart Operations	...	...	...	.....
Other	...	...	...	.....

Explain.

.....

DIET

What is your weight now? ..... 1 year ago?..... Age 21.....

Are you dieting?..... Why? .....

APPENDIX D

PHYSICAL ACTIVITY QUESTIONNAIRE.

This questionnaire is designed to obtain some idea of your present occupational and recreational activity.

STRICTLY CONFIDENTIAL.

Question 1:
1.0 Are you employed? Yes No

(if no go straight to question 4)

1.1 What is your present occupation ? .....

	0-10	10-20	20-30	30-40	over 40
1.2 For how many years ?					

1.3 Time spent working:

	0-2	3-5	6-8	9-10	over 10
hours/day					

	1	2	3	4	5	6	7
days/week							

	Up to 30	30-40	41-44	45-47	48-49	50 and over
weeks/year						

Question 2:
Would you assess your occupation as: (please check only one)

Continuously physically strenuous

Intermittently physically strenuous

Continously of moderate physical activity

Intermittently of moderate physical activity

Physically inactive

Question 3:
3.0 At work, about how much time do you usually spend sitting ?

All or nearly all	More than half	About half	Less than half	None or very little

3.1 At work, about how much of your time do you usually spend walking ?

All or nearly all	More than half	About half	Less than half	None or very little

3.2 At work, about how much of your time do you spend standing ?

All or nearly all	More than half	About half	Less than half	None or very little

3.3 Does your occupation involve regular lifting ?

Yes No

Question 4:
Would you describe yourself during the last one or two years as :-

Recreationally very physically active

Recreationally fairly active

Recreationally inactive

Question 5:
Considering a typical week, how many times on average do you do the following exercise, for more than 15 minutes, during your leisure time.

A: Strenuous activity (heart beats rapidly)(i.e. running, fast jogging, hockey, football, vigorous swimming, vigorous long distance cycling, circuit training, mountaineering.

	Once	Twice	3-4	5-6	7-8	9-10	More than 10
Times per week							

	15-20	20-30	30-45	45-60	Over 60
Average duration of each session in minutes.					

B. Moderate activity (not exausting) (i.e. fast walking, hill walking, easy jogging, popular and folk dancing).

	Once	Twice	3-4	5-6	7-8	9-10	More than 10
Times per week							

	15-20	20-30	30-45	45-60	Over 60
Average duration of each session in minutes.					

C: Mild exercise (minimal effort) (i.e. yoga, archery, fishing from river bank, bowling, golf, easy walking).

	Once	Twice	3-4	5-6	7-8	9-10	More than 10
Times per week							

	15-20	20-30	30-45	45-60	Over 60
Average duration of each session in minutes.					

APPENDIX E

UNIVERSITY OF GLASGOW
Institute of Physiology
Exercise Laboratory
Glasgow G12 8QQ

FROM:- RODNEY TAYLOR

TO:-

DATE:-

SUBJECT:- TESTING NOTIFICATION AND INSTRUCTIONS

1. Your appointment for exercise testing is on

   Date:-

   Time:-

   Place:-

2. Avoid exposure to extremes of climate (e.g. sauna, sunbathing etc.) and physical activity (particularly intense physical training or competitive sporting activity) on the day of and the day preceding the test.

3. Ensure adequate sleep on the evening immediately prior to the test.

4. Avoid alcohol, cigarettes, drugs, food, drink and exercise immediately (one hour at least but preferably two hours) before the test.

5. Bring with you lightweight sports clothing, i.e. training shoes (or nearest equivalent), shorts, sports or light shirt and sox. A shower and changing facility is available. If you plan to shower after testing please bring a towell and swimming trunks.

6. If something prevents you from keeping your appointment, or if you have any questions, please phone 041-339-8855 (extension 497) and ask for a member of the exercise staff.

APPENDIX F

PRE-TEST CHECKUP

Name _____

Date of Test _____

To obtain accurate test results and provide maximum safety and comfort the following points must be checked:-

1. At least 5 hours sleep the evening before testing?	Yes		No	
2. No exercise for 24 hours before test?	Yes		No	
3. Abstinence from a heavy meal at least 2 hours prior to test?	Yes		No	

4. Amount of alcohol 24 hours before testing. _____

5. Amount of coffee, tea, cocoa or carbonated drinks 8 hours before testing. _____

6. Number of cigarettes of any kind, pipes, cigars for at least 6 hours before testing. _____

APPENDIX G

RESPIRATORY CALCULATIONS

$$VE = \frac{PB - PH2\emptyset}{760(1+\emptyset.\emptyset\emptyset367T)}$$

Where VE : volume of expired air (l/min)

PB : ambient barometric pressure (mmHg)

$PH_{2\emptyset}$: vapour pressure of water at gasometer temperature (mmHg)

T : temperature of gasometer ($^{\emptyset}C$)

$$VO_2 = \frac{VE}{1\emptyset\emptyset} (\% NE \times \emptyset.265 - \%\emptyset_2E)$$

Where VO_2 = volume of oxygen consumed (l/mm)

% NE : percentage of nitrogen in expired air = 100% - ($\%\emptyset_2E + \%\emptyset_2E$)

$\%\emptyset_2E$: percentage of oxygen in expired air

$$RQ = \frac{\%CO_2E - \emptyset.\emptyset3}{(\%NE \times \emptyset.266 - \%\emptyset_2E)}$$

Where RQ = respiratory quotient.

APPENDIX H

TREADMILL RUNNING TEST RESULTS

Subject: A SUBJECT Date: 01/01/84 Time: 10/00

Weight: 70 Kg. Sex: MALE Height: 185cm. Age: 35 years

Barometric pressure (mmHg): 758 Temp.: 20 'C Humidity: 35 %

Vapour pressure: 17.5 mmHg Heart rate (resting): 65 b.p.m.

FEV1.0: 2.5 l.t.s. VC: 5 litres. % FEV1.0: 75 %

Gas number: 1

Treadmill speed: 4 m.p.h. Treadmill gradient: 0%

Exercise time: 6 mins. Collection time: 3 mins.

Air meter - Initial: 434 litres. Final: 492.9 litres.

Volume difference: 59.4 litres.

Expired air temperature: 22 'C

Atmospheric correction factor: .902

VE STPD: 19.8 l.min. VE STP: 17.851 l.min.

FI Oxygen: 15.4 % FE Carbon dioxide: 4.52 %

True oxygen: 5.715

R.Q.: .858

Calorific value of expired air: .277

Energy expenditure: 4.936 Cals/min.

Oxygen consumption: 1.02 l.min.

VO2: 14.574 ml.kg-1.min-1 VCO2: .873 l.min-1.

VE B.F.: 17.497

Subjective rating (6-20): 6

Heart rate: 100 beats. min-1. Oxygen pulse: 10

% Rise in heart rate: 53 %

Comments:

Name_____ Week Commencing Sunday___/___/____ Week No._____

Body Weight (lbs)_____ Resting Heart Rate (beats per minute)_____

	Distance	Time Taken	Average Pace	Illness/Injury	Feelings (Elation/Depression etc)	Weather Conditions
Sunday (/ /84)						
Monday (/ /84)						
Tuesday (/ /84)						
Wednesday (/ /84)						
Thursday (/ /84)						
Friday (/ /84)						
Saturday (/ /84)						
WEEK'S TOTALS	Distance	Time	No. of Runs			

CUMULATIVE TOTALS No. of Weeks _____

No. of Miles _____

No. of Hrs/Mins_____

WEEK'S RACES

Name of Race_____ Distance_____ Time_____

APPENDIX I

APPENDIX J

Calibration of the electrically braked bicycle ergometer

Calibration of the Siemens electrically braked bicycle ergometer was performed prior to and immediately after the study. The calibration proceedure was performed by a Siemens electrical engineer using a calibration method specific to this bicycle ergometer and for which details are not available. No change in bicycle ergometer settings was reported between these two calibrations, suggesting that the calibration had remained unaltered during this time.