THE EFFECTS OF A UNIVERSITY FITNESS PROGRAMME ON HEALTH RELATED VARIABLES IN PREVIOUSLY SEDENTARY MALES

by

STANLEY GRANT

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SUMMARY

There is an increasing body of evidence which suggests that regular physical activity and fairly high levels of aerobic fitness provide a degree of protection against coronary heart disease. In addition, appropriate exercise may reduce the incidence of low back pain by improving local muscular endurance and flexibility in certain areas.

This study reports on the effects of a 10 week university fitness programme (which consisted of 20 minutes of aerobic activity, 5 minutes of muscle conditioning exercises and 5 minutes of flexibility exercises three times per week) on health related fitness variables.

Twenty one exercisers, age 37.0 ± 10.24 years (range 21-58) and 22 controls, age 38.6 ± 7.85 years (range 17-54) volunteered to take part in the study. Assessment was carried out before training commenced and 10 weeks later at the conclusion of training.

Two sample t-tests were used to determine if the exercise group demonstrated a greater average improvement than the control group. Ninety five percent confidence intervals (95% CI) indicate the average range of improvement. The exercise group

showed a greater average improvement over the controls from test 1 to test 2 in the following: Resting heart rate 95% CI (-2.4,-12.8) beats/minute; Steady state heart rate 95% CI (-7.8, -16.2) beats/minute; Predicted Vo2 Max 95% CI (3.2, 6.7) ml kg-1 min-1; Sit ups 95% CI (3.1, 7.0,); Flexibiliity 95% CI (3.3, 6.9) centimetres.

A paired t-test showed that total cholesterol and high density lipoprotein demonstrated a significant improvement for both the exercise and control groups. However, there was no significant difference between the exercise and control group changes. In the exercise group total cholesterol fell from 6.5 ± 1.2 to 5.6 ± 1.1 mmol/litre and high density lipoprotein rose from 1.12 ± 0.22 to 1.29 ± 0.24 mmol/litre. The control group total cholesterol fell from 6.2 ± 1.20 to 5.5 ± 0.84 mmol/litre and high density lipoprotein rose from 1.16 ± 0.2 to 1.26 ± 0.27 mmol/litre. There were no significant changes in the exercise group or the control group in body weight, bicep skinfold, tricep skinfold, suprailiac skinfold, total skinfold, percentage body fat, systolic blood pressure, diastolic blood pressure, composition of the diet, energy intake of the diet or triglycerides.

This study demonstrated one of the accepted indications of an improvement in aerobic fitness, a sub maximal bradycardia. A decrease in heart rate at a given sub

maximal workload increases the efficiency of the heart and reduces the possibilty of ischaemia. Although aerobic fitness in the exercise group improved, there was no beneficial effect on body weight, body fat, blood pressure, or the lipid profile. Diet did not change and energy expenditure increased. It is therefore rather surprising that there was no measured significant change in body composition.

By contrast, the absence of an effect on blood pressure when baseline levels were within normal range agrees with previous studies which have shown that "normal" blood pressure will show a small decrease or is unlikely to change as a result of aerobic exercise. Both groups showed a significant decrease in total cholesterol and a significant increase in high density lipoprotein but no significant difference between groups. Clearly, there is some underlying factor other than exercise which has caused these changes. Triglycerides were within normal range and previous studies have shown that aerobic exercise is unlikely to reduce triglycerides which were not previously elevated. The exercise programme increased local muscular endurance in the abdominal region and flexibility in the lower back and hamstrings. These improvements may make the subjects more resistant to lower back problems.

Thus, the exercise programme was effective in improving

some health related fitness variables. Perhaps a programme of a longer duration would have favourably influenced some other aspects of health related fitness.

CHAPTER 1

SECTION ONE

INTRODUCTION

1.1.1 Coronary Heart Disease

The increasingly sedentary nature of the western lifestyle is considered to pose a threat to the body and may in fact contribute to a deterioration in body functions. Coronary heart disease (CHD) is a major cause of death and disability in the western world and is particularly prevalent in Scotland which has one of the highest incidence rates in the world (Kannel et al; 1984; Smith, 1987). Not only is CHD responsible for over 50% of death in certain age categories, it produces devastating effects on the lives of many families.

CHD is a result of the interaction of a multiplicity of factors. Narrowing of the arteries (atherosclerosis) over a period of time is due to a depositing of plaque in the arteries which become lined with plaque which is composed of lipids and scar tissue. As the build-up of plaque progresses blood flow through the arteries is impeded. Arteries which supply the brain and heart are particularly vulnerable. The basic etiology of

atherosclerosis is not completely understood. Changes in the arteries' smooth muscle wall may trigger the disease. Atherosclerosis is believed to be initiated by environmental factors including smoking and high blood pressure. High cholesterol diets and saturated fats are often implicated with the disease. However, high concentrations of low density lipoprotein are linked with the development of plaque while high levels of HDL appear to inhibit the formation of plaque (Mc Ardle et al; 1986).

1.1.2 Coronary Heart Disease Risk Factors

Numerous population studies over the years have identified certain characteristics which make the individual more susceptible to CHD (Kannel et al; 1976). Some of these factors cannot be influenced - heredity, sex, race and age. However, other risk factors can be altered. Cigarette smoking and other tobacco related activities, hypertension, elevated blood cholesterol and triglycerides, high levels of low density lipoprotein, obesity, physical inactivty, diabetes, and emotional stress are all risk factors which are amenable to change (Lipid Research Clinic, 1984; Kuller, 1984; Pollock et al; 1984; Hubert et al; 1983). Recent evidence indicates that a decrease in some of the risk factors results in a lowering of CHD mortality rate (Frohlich et al; 1987); Consensus Conference, 1985). The three primary risk factors are

high levels of blood cholesterol, hypertension and cigarette smoking. However, these risk factors and others such as diet and heredity are interrelated. Thus it is difficult to quantify the importance of one factor considered in isolation (Pollock et al;1984). Pollock et al; (1984) concede that CHD risk profiles should only be used for descriptive and educational purposes. However, they highlight the fact that the chances of developing CHD increases from onefold to almost fourfold when the individual increases the primary risk factors from one to three. It should be noted however, that most of the association is correlational, and does not necessarily imply causality. It seems logical that a reduction in risk factors will lead to a lowering in the incidence of CHD. On page 570, Blair, (1980) states that, " Regardless of the basic mechanism -development of atherosclerosis - much evidence supports the risk factor hypothesis".

Most epidemiological studies show that the relationship between most risk factor levels and CHD is continuous and linear or curvilinear. There does not appear to be a cut-off point or threshold level for most risk factors. The European Atherosclerosis Society, (EAS) (1987) states that the absence of a cut-off point does not interfere with the strategy of moving the population distribution to a more favourable distribution. The EAS stresses however, that clinical

practice should make use of cut-off points or action limits. These guidelines would make assessment of risk and choice of subsequent action easier for the practitioner.

Although the recent decrease in CHD mortality rates in the United States has been greater than would have been expected from the observed changes in CHD risk factors alone, a decrease in CHD has been associated with a reduction in the three primary risk factors (Borhani, 1985; Kuller, 1984).

1.1.3 Physical Activity and Coronary Heart Disease

As western society becomes more automated, the number of manual tasks man needs to perform is reduced. In the U.K. and the U.S. the adult population is being encouraged to exercise to offset the reduction in occupational energy expenditure and to promote health through exercise. This advice appears to have some foundation. Many epidemiological studies have demonstrated that those who are physically active appear to gain some form of protection from CHD.

La Porte et al; (1984) suggest that there are two mechanisms by which physical fitness can reduce CHD. The first mechanism relates to the efficient pumping action of the heart. Aerobic training normally improves cardiovascular functional capacity in

previously sedentary subjects. For a standardised level of activity myocardial oxygen consumption is decreased after training (Clausen, 1977). The second mechanism is the influence of activity/ fitness on CHD risk factors. The fact that changes in CHD risk factors are associated with a reduction in CHD makes the prospect of physical activity/ fitness an appealing one for the modification of CHD risk factors.

However, is the improvement in fitness a pre-requisite for the modification of CHD risk factors? Haskell, (1985); and La Porte et al; (1985); agree that the health benefits of exercise may not occur simultaneously with an improvement in physical work capacity and that the development of aerobic fitness may not be essential for the promotion/maintenance of health. They stress that the stimuli for health benefits are not well defined. However, there is consensus that physical activity appears to lower the risk of many types of disorders (Haskell, 1987; La Porte et al; 1985). Regular low level activity which does not promote aerobic fitness may be as important or more important than high intensity aerobic training where favourable alterations in lipid levels are concerned (La Porte et al; 1984; Cook et al; (1986). Thus, it may be important to distinguish between physical fitness and physical activity. Several landmark studies have established a link between occupational and recreational activity and CHD (Morris

et al; 1953; Morris et al; 1958; Morris et al;1973; Paffenberger et al; 1977; Paffenberger et al;1978a). These studies suggested that protection from CHD was offered by moderate or vigorous activity and was only evident in those individuals who were currently active. Previously active people or those involved in athletic pursuits in earlier life did not gain protection from CHD (Paffenberger et al; 1978b). Although these studies provide strong evidence that exercise has a role in the prevention of CHD, much of the evidence is circumstantial. While some epidemiological literature on the relationship between CHD and physical activity suggests that there is an inverse relationship between CHD and physical activity, the evidence is not conclusive (Leon et al; 1977). An explanation for some of the conflicting evidence may be the variety of research design and the methodology employed. For example, the measurement of physical activity has often relied on self-reporting of activity or assumptions have been made that a particular job entails the same amount of energy expenditure for everyone (Solboski et al; 1987; Kannel et al; 1985). La Porte et al; (1984) discuss the problems of physical activity assessment and highlight the difficulties of self selection and the problems of measurement of activity. Clearly it is difficult to determine if those who exercise become healthy or healthy individuals are predisposed to exercise. However, researchers concur that individuals who have been identified as having CHD risk factors

should be given appropriate counselling to improve aerobic fitness, slowly and safely to minimise CHD risk (Cooper et al; 1976; Poole, 1984).

1.1.4 Exercise and Coronary Heart Disease Risk Factors

Several studies have attempted to ascertain the effect of exercise on CHD risk factors. The results of those studies have been equivocal. Differences in initial baseline levels and confounding factors such as changes in body weight, or body fat, or in dietary intake, alcohol consumption, and tobacco usage have had a bearing on the results (Seals et al; 1984a; Goldberg et al; 1985).

If physical activity influences CHD risk factors, what kind of exercise is important in the modification of these factors? A link has been established between aerobic fitness and a reduced level of CHD risk (Cooper et al; 1976; Peters et al; 1983; Gibbons et al; 1983; Poole et al; 1984). Is this connection a function of genetic endowment as well as training programmes? If exercise studies can demonstrate that physical activity has a bearing on CHD , it will be difficult to apply these findings to the general population unless the appropriate exercise prescription can be accurately defined. The mode, frequency, duration, and intensity of the exercise must be accurately quantified. Some studies relating physical exercise to CHD have failed

to define with any precision the exercise prescription adopted (Seals et al; 1984a; Goldberg et al; 1985). Longitudinal studies have attempted to examine the role of exercise in CHD risk factors. However, detailed investigations into the apparent protective effect of aerobic exercise on CHD will help explain any mechanisms involved and provide clarification of the stimulus which is necessary to improve the CHD risk profle.

1.1.5 Flexibility, Local Muscular Endurance and Low Back Pain

Other variables are considered to contribute to optimal health and physiological function. Low levels of strength, local muscular endurance and flexibility can impose limitations on individuals. It is suggested that 80% of lower back pain cases are caused by muscular deficiency (Kraus et al, 1961). The qualiity of life is often reduced in people who have low back pain. Moreover, chronic back pain can lead to loss of income as well as the inability to carry out daily tasks and even premature retirement. The scale of the problem is highlighted by Hicks, (1988) who states that back pain causes 30 million working days to be lost each year in the UK as well as costing the National Health Service 156 million pounds per annum. Increases in local muscular endurance in the abdominal area and/or an improvement in the flexibility of the lower

back and hamstrings have the potential for decreasing the incidence of back pain (Pollock et al; 1984).

1.1.6 Department of Physical Education and Sports Science Fitness Sessions

The Department of Physical Education and Sports Science offers a wide range of mass fitness sessions to a large section of the University community. It is believed that these fitness programmes make a contribution to health related fitness. These fitness sessions are thought to improve aerobic power, local muscular endurance and flexibility and may influence CHD risk factors.

Some attempt has been made to investigate the effects of the training programmes offered by the Department of Physical Education and Sports Science. Unpublished evidence has shown that a high intensity aerobic programme produced an improvement in aerobic fitness. Regular fitness tests undertaken by members of the University community also suggest that there is improvement in some further aspects of fitness. However, no statistics on these tests or a systematic monitoring of the exercise prescription have been undertaken.

Studies of the effects of training programmes will provide information on the direction and magnitude of

various physiological and health related changes. Such information can be of assistance in evaluating the potential benefits of a structured training programme and the information gained will provide guidance on the suitability of specific exercise prescriptions for promoting and maintaining health.

1.1.7 Aim of Study

The aim of this study is to evaluate the effectiveness of an exercise session carried out three times per week, 30 minutes per session over a 10 week period on CHD risk factors and other health related variables. It is important to assess the effect of group exercise programmes as more information is required concerning the changes which may occur in people who undertake these programmes. If the results are favourable, these programmes could be extended into the community. This development would be important as it may result in a considerable impact being made in the West of Scotland population which is renowned for its ill-health.

CHAPTER 2

REVIEW OF LITERATURE

SECTION ONE

AEROBIC POWER

Physical fitness is not a unitary measure, but is composed of different parts. Researchers do not always concur as to what the component parts of physical fitness are (Burke et al; 1982). Nevertheless, several authorities consider that aerobic power, strength, local muscular endurance, flexibility, and body composition are the constituent parts of physical fitness (Sharkey, 1984). As the components are separate, it is not surprising that training for each component involves different regimes and is assessed in various ways. However, different weighting to fitness components has been given by several writers. Aerobic power is regarded as the most important aspect of physical fitness for the general population by the connection with the prevention of heart disease (Shephard, 1971; Hockey, 1973; Fentham & Bassey, 1979).

2.1.1 Aerobic Power

Aerobic power or Vo2 Max is the maximum amount of

oxygen per minute that a subject can extract from the inspired air during exercise (Astrand and Rodahl, 1977). The capacity to perform heavy prolonged muscular work is dependent on the supply of oxygen to the working muscles which is limited by the combined capacity of the respiritory and cardiovascular systems to transport oxygen to the working muscles. Vo2 Max = stroke volume * heart rate * Av-o2 Diff. The functional ability of the heart is reflected by the cardiac output(CO). The CO is the volume of blood pumped by the right or left ventricle in one minute. The CO is determined by multiplying the heart rate and the stroke volume (SV). The SV is the volume of blood ejected by each ventricle during each beat. Once the oxygen has been carried by the blood to the cells, the ability of the cells to extract oxygen from the blood is reflected by the difference in the content of oxygen between the arterial and venous blood (A-Vo2 Diff). The greater the volume of oxygen extracted by the muscles, the greater the A-Vo2 difference becomes (Mathews and Fox, 1976). A more detailed account of the components of the oxygen transport system is given in Astrand and Rodahl, (1977) and White, (1978).

A high Vo2 Max will result from a high cardiac output and A-Vo2 Diff. However, Mathews and Fox, (1976) consider the stroke volume to be the most important factor as trained endurance athletes have been shown to have stroke volumes 70% higher than the untrained.

Clearly the characteristics of the skeletal muscle as well as the state of training will influence Vo2 Max. Other factors which influence Vo2 Max include blood volume, capillary density and haemoglobin levels. It should be noted that the lungs are not a limiting factor in Vo2 Max unless there is lung disease or the test is conducted at altitude (Pollock et al; 1984). Discussion of the limiting factors in the oxygen transport system is to be found in Shephard, (1982) who concluded that peripheral factors were not the limiting factors in the oxygen transport system. Vo2 Max is probably limited by the arterial oxygen transport system (Brooks et al; 1984; Shephard, (1982).

2.1.2 Measurement of the Oxygen Transport System

As most people are very rarely involved in maximum aerobic effort, a measure of the subjects physiological response at sub maximal intensities has more relevance to everyday life. However, greater maximal capability will be beneficial and will probably result in a reduced level of effort at standardised sub maximal work loads (Saltin et al; 1969). If one wishes to evaluate the efficiency of the oxygen transport system, Lamb, (1978) suggests that a test which measures the ability of the lungs, heart and circulation to supply oxygen to the muscles should be used. Pollock et al; (1978) state that a maximum oxygen uptake test is used

as a measure of the oxygen transport system because it summarises what is happening in the different elements of the oxygen transport system.

2.1.2(a) Direct Measurement of Maximal Oxygen Uptake

Direct measurement of oxygen uptake requires expensive laboratory facilities as analysis of expired air using elaborate apparatus is necessary. Lamb, (1984) states that the reproducability of direct tests of Vo2 Max is good. He suggests that one would expect differences to vary between tests of about 2-4%. However, direct measurement of Vo2 Max has its limitations as it is exhausting, and can endanger life. Lamb, (1978) notes that to find the "real" Vo2 Max, subjects must be highly motivated and that in some cases, especially in the first test, exceptionally low values may be found with unfit subjects. One of the criteria for the attainment of Vo2 Max is the establishment of a plateau. This criterion is often difficult to achieve especially with non athletic subjects (Hammond et al; 1984).

2.1.2(b) Prediction of Maximal Oxygen Uptake

For these reasons and the fact that expensive equipment is necessary, indirect measures of Vo2 Max have been developed. Most of these predictive tests are based on the assumption that there is a linear relationship

between heart rate and work intensity. As the work intensity is increased, the oxygen uptake increases in a linear manner (Astrand et al, 1986). Heart rate and oxygen uptake are also linear over a wide range of heart rates between about 125 to 170 beats per minute in young males (Astrand et al; 1954). It is possible to predict the oxygen cost of exercise levels and using one steady state or several steady state heart rates extrapolate to the subject's maximum heart rate can to give a prediction of Vo2 Max. If the subject's maximum heart rate is unknown, an estimate of maximum heart rate must be made. Normally the formula 220 minus age is used to predict maximum heart rate but maximum heart rate could deviate from this estimated value by ± 20 beats per minute (Wilmore, 1977).

Predictive methods of Vo2 Max using the linear relationship principle rely on the subject attaining a steady state where the oxygen uptake equals the oxygen requirement of the body.

2.1.2(c) Steady State

Various researchers have given advice on the time to steady state. The fitness of the individual and relative intensity will influence the speed of approach to steady state (Whipp et al; 1972; Hickson et al; 1978). Continuous protocols have demonstrated that steady state can be achieved rapidly i.e. in two or

three minutes for heart rate and Vo2 plateau when energy cost increases are not large (Nagle et al; 1971; Shephard, 1982). Continuous steady state protocols can have shorter stage durations than discontinous protocols as the subject does not need to warm-up to each work load. Complete stabilisation of heart rate is difficult to achieve as thermoregulatory changes and diversion of blood flow to the skin can result in small increases in heart rate. However, Shephard et al; (1978) has shown that 3 minute stages allow the subject to come close to or attain steady state values for Vo2 and heart rate for a given work load and quote heart rates between 98% and 101% of the steady state heart rates obtained during 5 minutes of work. Vo2 ranged from 97% to 100% of the values obtained during 5 minutes work. Shepard, (1971) concedes however that 5 minutes may be more desirable for a "true" steady state. Furthermore, Whipp et al; (1972) have shown that steady state can be achieved in 3 minutes during lower work loads though at higher work loads 5 minutes are necessary.

2.1.2(d) Methods of Predicting Maximal Oxygen Uptake

A series of studies have investigated the accuracy of various methods of predicting Vo2 Max. Maritz et al; (1961) consider their method of prediction of Vo2 Max to be more accurrate than the Astrand Ryhming

Nomogram, (1954) as the Maritz method uses 4 sub maximal measurements whereas the Astrand Ryhming Nomogram relies on one sub maximal measurement which could be more liable to produce a spurious result. While it is possible that one sub maximal heart rate is unrepresentative of linearity, measurement of 4 points over a wide range of sub maximal values will produce a good estimate of Vo2 Max.

Some researchers suggest a wide range of sub- maximal heart rates (100-160 beats per minute) because they showed that a more accurate prediction of Vo2 Max was obtained when a wide spread of heart rate values were used (Wyndham et al; 1967). Astrand and Ryhming, (1954) found a range of 125-170 beats per minute to be the most favourable bandwidth for the most accurate prediction. However, Montoye et al; (1986) reported that the exclusion of exercise heart rates below 120 beats per minute did not improve estimates of Vo2 Max while Davis et al; (1968) have shown that predictions of Vo2 Max using the Astrand Ryhming Nomogram were much better with heart rates between 140 and 180 beats/ minute.

Comparison of various predictive methods indicates that the Maritz method appears to be the most favourable (Washburn et al; 1984; Wright et al; 1978). However, Shephard et al; (1968) have demonstrated in their comparison of the Astrand Ryhming Nomogram, Margaria et

al; (1965), and Maritz et al; (1961) methods that there is little difference between the Maritz method and Astrand Ryhming methods. Studies have reported an error up to ± 15% in the prediction of Vo2 Max using the Astrand Ryhming Nomogram (De Vries, 1966; Astrand and Rodahl, 1986). Other researchers who have compared different predictive methods of Vo2 Max with direct measurement have found a correlation of around 0.83 for the Maritz method and 0.80 for the Astrand Ryhming Nomogram (Washburn et al; 1984; Shephard, 1982).

There is agreement that the fewer the physiological variables that are known, the poorer the prediction of Vo2 Max (Montoye et al; 1986; Wyndham 1967; Maritz et al; 1961). Thus, sub maximal oxygen costs should be measured when possible. The attempt to improve the prediction by measuring maximal heart rate is clearly undesirable with an "unscreened" population. However, even when only sub-maximal heart rates and work loads are known predicted Vo2 Max is useful for comparing groups over a period of time (Wright et al; 1978; Durnin, 1981; Shephard, 1982). Despite the errors in prediction of Vo2 Max, sub maximal tests can provide "a legitimate basis" for monitoring variations in fitness levels (Wright et al; 1978). Tests of predicted Vo2 Max will demonstrate trends if used to evaluate training programmes. Although Wright et al; (1978) found the error of prediction was between 1 and 16% in two subjects over a four month period, using the

Astrand Ryhming Nomogram, this error was constant and changes in Vo2 Max were demonstrated using the the Astrand Ryhming Nomogram. Moreover, Saltin et al; (1969) in their study of middle aged men over a 10 week period found that the group mean Vo2 Max values of the predicted and directly measured scores were identical.

Although indirect measurement of Vo2 Max has problems which limit the accuracy of prediction, these difficulties are insuficient to eliminate this type of procedure from being of value in assessing changes in groups which undertake training programmes.

2.1.3 Development of Aerobic Power

Several factors are involved when the improvement of aerobic power is considered.

2.1.3(a) INTENSITY

There is agreement that there is a minimum stimulus required to promote improvement in aerobic power. However, research in the area has produced varying training thresholds. In their policy statement The American College of Sports Medicine, (ACSM; 1978) consider 50% of Vo2 Max (60% heart rate reserve) to be the minimum threshold for the improvement of Vo2 Max in healthy adults. Other studies have advocated minimum training thresholds beteen 45% and 60% of Vo2 Max. Ribisl, (1980) recommends from his review that 60% of Vo2 Max is the minimum threshold for the development of aerobic power whereas Mc Ardle and Katch, (1986) after their scan of the literature suggest 50% to 55% of Vo2 Max as the lowest level of intensity that will provide a training effect. It is accepted that the lower the fitness level, the lower the training stimulus necessary to improve aerobic power. Recent studies have demonstrated that unfit, middle aged men improved their aerobic power while they trained at around or under 45% of Vo2 Max (Badenhop et al; 1983; Gossard et al; 1986).

Many studies have used the Karvonen formula to determine training intensity. Karvonen et al;(1957) found that to improve aerobic power, the heart rate during the exercise session must be increased to at least 60% of the heart rate reserve (HRR). The HRR is determined by establishing the resting heart rate and maximum heart rate. Subtract the resting heart rate from the maximum heart rate. Take 60% of this figure and add this value to your resting heart rate to establish 60% of HRR. Davis & Convertino, (1975) confirm that there is a good relationship between the percentage of HRR and the corresponding percentage of Vo2 Max. Obviously a heart rate guide is of greater practical value to individuals who do not have access to oxygen consumption measurement and therefore has greater applicability to the general population.

The intensity of effort can be measured/prescribed using a percentage of maximum aerobic power or heart rate reserve. It is essential that intensity is prescribed correctly as too low a stimulus can result in little or no fitness gain while too high a training intensity can also be ineffective. High intensity training for sedentary individuals often results in pain and distress and injury which can lead to a reduction in compliance with the training programme (Pollock, 1978). Unfit individuals can only sustain high intensity work for short periods as they fatigue

quickly. Therefore, the duration of the training period will be reduced and will be less effective. Cardiac events are more likely to occur at a high level of work. Thus, if the training programme is to promote an improvement in aerobic power, it is essential that an appropriate training zone is established. Reliable resting heart rates are often difficult to establish as resting heart rates are influenced by several factors including emotion, posture, time after a meal and the time of day (Pollock et al; 1984). However, the findings of Davis et al;(1975) suggest that variation in the resting heart rate has little or no effect on the HRR. Davis et al; (1975) measured resting heart rates in 5 different stuations and found that the differences in resting heart rate did not dramatically alter the HRR figure.

2.1.3(b) Duration

From the evidence available, ACSM; (1978) conclude that intensity and duration are interrelated. Present research suggests that so long as the training intensity is above the minimum necessary to elicit a training effect, the magnitude of improvement is dependent on the total work or energy cost of the activity. Improvement will be similar if the energy cost of the training is the same ACSM; (1978).

When an upper and lower training heart rate has been

established, the individual must attempt to train within these limits for a designated time. This range is called the training zone. Exercise reviews conducted by ACSM; (1978) and Wenger et al; (1986) reveal that sessions of 15 minutes will promote improvement in aerobic power and that the longer the duration the greater the improvement.

Pollock and his colleagues, (1978) suggest that most beginners should be able to train within the range of 60%-75% of HRR for 20-30 minutes. They refer to their experience when they say that sedentary individuals can tolerate this intensity and duration. It should be noted that Millesis et al; (1976) have shown that the incidence of injury is much greater with beginners who train for longer than 30 minutes per session.

2.1.3(c) Frequency

Researchers have found gains in Vo2 Max to be just as great with a frequency of two days per week as with a frequency of four days per week (Fox, 1979). Although frequency of training is an important variable where changes in Vo2 Max are concerned Jackson et al; (1968) found that 2-3 training sessions per week were enough to promote improvement in low fitness groups. As fitness levels improve, longer and more frequent sessions are both possible and necessary to produce further improvement. Wenger et al; (1986) refer to

over 100 studies when they suggest that individuals who are in a low fitness category can expect improvement with training frequencies from 2 - 6 times per week. However, those in high fitness categories will not improve with only 2 sessions per week (Wenger et al; 1986). A higher attrition rate through injury is to be expected with sedentary subjects who train five times per week (Pollock et al; 1978).

2.1.3(d) Mode of Activity

ACSM; (1978) suggest that activities which involve large muscle groups in rhythmical continuous movement are best for the development of aerobic power. Running, hiking, swimming, bicycling, cross-country ski-ing are examples of activities which are considered to be appropriate modes of exercise for the enhancement of aerobic power. It should be noted that some activities. e.g. running, jumping are more likely to produce injury than non weight bearing activities.

2.1.3(e) Exercise Prescription for Sedentary Individuals

It is not enough to prescribe exercise and leave the individual (s) to embark on a training programme. Pollock et al; (1984) outline what they believe to be criteria for a successful introduction to a fitness programme. They stress that education, motivation and

leadership are the keys to a successful exercise programme. Individuals who are embarking on an exercise programme should be given realistic goals and be informed on how best to monitor exercise. Pollock and his colleagues, (1984) suggest that adequate leadership and guidance in the early stages of an exercise programme will enhance the chances of proper implementation and progress.

It is recommended that a gradual approach to an exercise programme should be adopted (Sharkey, 1984). High intensity or frequent sessions can lead to distress, muscle soreness, muscle strain injury and unnecessary fatigue (Wenger et al; 1986).

2.1.3(f) Measurement of Exercise Heart Rate

Obviously, it is desirable to monitor exercise heart rate during the training session. However, there are logistical problems in attempting to monitor fairly large numbers of exercisers. An option is to monitor heart rates during pauses in the exercise session. While training heart rates can decrease fairly fast after the cessation of a training session, Pollock et al; (1984) concluded that training heart rates of 80% to 90% of maximum heart rate can be accurately measured by middle aged men. The subjects in the Pollock study took a 10 second pulse count within 12 to 14 seconds of cessation of exercise. The authors found that the

exercise heart rate was underestimated by only 2% in 152 trials. Mc Ardale et al; (1969) allowed 4 seconds for the exercising subjects to find their pulse for a 10 second count at the end of the exercise period. In heart rates of 180 beats per minute a 2.7% underestimate was reported while at heart rates of 140 beats per minute this underestimate increased to 7.6%. Pollock et al; (1984) concede that a 15 second count may result in a greater decrease in exercise heart rate (of around 5%) but suggest that a 15 second count may in fact be more accurate as a 10 second count multiplied by six may in fact magnify an error in the monitoring of the heart rate.

2.1.4(a) Initial Levels of Fitness and Expected Improvement in aerobic Power

Improvement in aerobic power is inversely related to fitness levels (Sharkey, 1975). Pollock's 1973 review agrees with this statement but indicates that there are inconsistencies to the general pattern.

2.1.4(b) The Effect of the Programme Length on Aerobic Power

The majority of the studies investigating training and aerobic power have not extended beyond 20 weeks. As changes are going on over a prolonged period it is reasonable to say that studies conducted over a short

period of time may have limitations as some groups may take some weeks to" break in " to the exercise programme. Thus middle aged sedentary individuals may need longer to adapt to the exercise programme before they gain benefit from the programme ACSM; (1978).

2.1.4(c) Expected Improvement in Aerobic Power

The degree of improvement in Vo2 Max has ranged from 0% to 93%. Most studies have shown an increase of between 10% and 20% Pollock, (1973).

2.1.5 Summary

Changes in aerobic power are dependant on the interaction of the intensity, duration and frequency of the training sessions. The magnitude of improvement is influenced by the initial level of fitness as well as the the length of the training programme. Comparison of the numerous studies in the area is made difficult by the lack of standardisation of testing protocols, the variety of mode of exercise in the training regimes, and the details of the records relating to the exercise performed. A summary of the conclusions of researchers would suggest that if the type of exercise involves large muscle groups (e. g., running, cycling) in continuous activity at an intensity which elevates the heart rate to the appropriate "training level", and if this exercise is

performed two to three times per week for a minimum of 15 minutes, the Vo2 Max of most sedentary people is likely to improve, but little or no improvement would be expected if one of the three basic elements (intensity, frequency, duration) is not included in the training regime.

BLOOD PRESSURE

2.2.1 Blood Pressure

Mean arterial blood pressure is a function of the cardiac output multiplied by the total peripheral resistance. Blood pressure varies depending on the sex, age and population studied. Intra-individual variation may be caused by posture, time of day, ambient temperature and bladder tension (Armitage et al; 1966; Heller et al; 1978). "Normal" blood pressure is determined by randomly selecting a group from a set population and establishing statistics from the measurements from the group. A normal blood pressure range is usually ascertained by arbritarily choosing a percentage above and below the mean of the population group (Evans et al; 1971).

2.2.2 High Blood Pressure

High blood pressure, or hypertension is considered to be the upper end of a normal distribution of a selected group. Evans and Rose; (1971) state that the operational definition of hypertension should be flexible as ranges applicable to patients may not be appropriate for other sections of the population.

There is not agreement on the exact definition of hypertension. The World Health Organisation (WHO) (O'Brien et al; 1982) has defined normal blood pressure as systolic blood pressure equal or less than 140 mmHg, together with a diastolic value equal or less than 90 mmHg. WHO (O'Brien et al; 1982) defines high blood pressure as systolic blood pressure equal to or greater than 160 mmHg and diastolic blood pressure equal to or greater than 95 mmHg.

It should be stressed that "average" blood pressures for a particular group may not be optimal for longevity. Lew, (1973) cites a vast amount of North American evidence using insurance company figures when he states that mortality was lowest among individuals who had below average blood pressure. In this case optimal blood pressure for longevity was below 110/70 mmHg.

Individuals who are identified as being hypertensive are associated with higher levels of all-cause mortality (Lew, 1973). Other problems linked to hypertension are damage to arteries, stroke, left ventricular hypertrophy, and heart failure (Frohlich et al; 1987; Kannel et al, 1984). Moreover, effective control of high blood pressure has now been shown to reduce morbidity and prolong life. Treatment of high blood pressure has led to stroke prevention and a decrease in heart failure (Kannel et al; 1984).

However, a large body of evidence indicates that the side effects of drug treatment may make this mode of treatment undesirable (Medical Research Council, 1987). Thus, the possibility that increased levels of aerobic fitness/activity could decrease blood pressure has great appeal.

2.2.3 Cause of High Blood Pressure

The largest number (around 85%) of cases of high blood pressure are termed ideopathic hypertension or essential hypertension i. e. the cause of high blood pressure is unknown. Factors which can contribute to the development and maintenance of hypertension are heredity, the influence of the renin-angiotensin system, changes in sympathetic nervous system and the levels of dietary sodium uptake (Ross et al; 1976). Other nutritional factors e.g. fats and fibre are considered to influence blood pressure (Hart et al; 1986). McMahon et al; (1985) postulate that excesses in alcohol consumption and calorie intake could be a contributary factor to hypertension and adjustments to these factors could result in an amelioration in hypertension. In around 15% of subjects with hypertension, a specific disease process is identified and secondary hypertension is diagnosed. An example of secondary hypertension is renal failure. Further information on the causes of hypertension is to be found in Kenny et al; (1984).

2.2.4 Measurement of Blood Pressure

Blood pressure can be measured directly by measuring within the artery itself or noninvasively and indirectly by using a sphygmomanometer. If an intra-arterial assessment is made, normally one measure is taken. With a sphygmomanometer several observations are usually taken. Thus, it may be that several measurements using a sphygmomanometer over a period of time may be more valid than one intra-arterial measurement. There is great variability in blood pressure during the day and from day to day. Armitage and Rose, (1966) followed 10 subjects over a 6 week period during which time 40 observations were made on each subject. On page 332 they refer to the readings from these 40 observations when they state that "each subject showed a variability which was large both on an absolute scale (a range of 25-30 mmHg) and relative to the variation between individuals." Sphygmomanometry is prone to error which can be a function of the observer and/or the instrument (Evans and Rose, 1971). Inappropriate cuff size and lack of calibration are two possible causes of instrument failure. Observer errors can result from improper positioning of the cuff, a false interpretation of the Korotkoff sounds, observer bias or digit preference.

It is now possible to measure blood pressure using a

semi-automatic sphygmomanometer. This apparatus has a standard cuff with velcro fastenings. The blood pressure is recorded on a digital display after the cuff has been inflated automatically. Gillies et al; (1987) assessed the validity of a Copal semi-automatic sphygmomanometer and concluded that this instrument would be a worthwhile alternative to other types of blood pressure measurement devices. Moreover, Copal semi-automatic sphygmomanometers are used every day in the Blood Pressure Unit of the Western Infirmary, Glasgow.

2.2.5 Blood Pressure and Fitness

Several studies have shown an inverse relationship between fitness and blood pressure (Cooper et al; 1976; Gibbons et al; 1983). A well controlled example is the study of Blair et al, (1984) who assessed fitness by means of maximum treadmill test on 4820 men and 1219 women aged between 20 to 65 years. The subjects were followed for 1 to 12 years (median 4 years). After adjustments had been made for age, sex, baseline blood pressure and baseline body mass index it was found that individuals with a lower level of fitness had an increased risk of 1.52 of developing hypertension compared with highly fit people.

2.2.6 Aerobic Training and Blood Pressure - Normotensive Subjects

The longitudinal studies which have examined the effects of training on normotensives and hypertensives and have used control groups demonstrate that no change or modest reductions in blood pressure are likely with a pre training systolic blood pressure (SBP) of less than 140 mmHg and diastolic blood pressure (DBP) under 90 mmHg (see Table 1). Interpretation of changes in blood pressure after aerobic training should be treated cautiously. McCarthy et al; (1984) make sweeping statements in their review of aerobic training and normotensives. They refer to several studies which have demonstrated that normotensive subjects over 30 years of age who had low fitness levels showed decreases in blood pressure whereas younger more fit normotensive subjects had little or no change after aerobic training. These conclusions can be criticised as some of the studies did not include a control group. As it is often the case that blood pressure usually falls on repeated measurements, it seems obvious that a control group should be included during any study period (Fagard et al; 1985). Moreover, the control group should have similar characteristics to the exercise group. Those factors which could influence blood pressure changes should ideally be similar in the exercise group and control group so that a meaningful

TABLE 1.

EFFECTS OF AEROBIC TRAINING ON RESTING BLOOD PRESSURE

LONGITUDINAL CONTROLLED STUDIES IN NORMOTENSIVE STUDIES (MALES)

AUTHORS	CHARACTERISTICS OF SUBJECTS NUMBER	AGE	TRAINING PROGRAMME LENGTH	FREQ. (DAYS)	DURATION (MINS)	INTENSITY	MODE	BLOOD PRESSURE (mm.Hg). SYSTOLIC PRE	SYSTOLIC POST	DIASTOLIC PRE	DIASTOLIC POST
Mann et al 1969	T 105	38	6 months	4.5x	60	Heart Rate 160 - 190 beats/min.	Walk/Jog/Run	128	124*	80	75*
	C 28	35	6 months				Calisthenics	132	125	81	78
DeVries et al 1970	T 112	69.5	6 weeks	3 x	60	Heart Rate under 145 or 120 beats /minute.	Walk/ Run	140	136*	76	73*
	C 32	?	6 weeks					141	140	76	75
Pollock et al 1971	T 16	48.9	20 weeks	4 x	40	63-76% of Max Heart Rate	Walk	121	118	78	76*
	C 8	?	20 weeks					117	118	77	81
Bonnano et al 1974	T 8	33-58	12 weeks	3 x	50	70-85% of Max Heart Rate	Walk/Jog	123	126	84	78*
	C 4	30-53	12 weeks				Calisthenics	135	132	87	78*
Millesis et al 1976	T 17	28	20 weeks	3 x	30	85-90% of Max Heart Rate	Walk/ Run	130	123	88	87
	C 16	28	20 weeks					125	125	86	86
Gettman et al 1976	T 20	20-35	20 weeks	3 x	30	85-90% of Max Heart Rate	Walk/ Run	114	117	76	80
	C 11	20-35	20 weeks					118	115	73	77
Wolfe et al 1979	T 20	36.8	6 months	4 x	17-27	60% of Max Heart Rate	Jog	126	121	?	?
	C 13	34.8	6 months					119	118	?	?
Kukkonen et al 1982	T 17	39	4 months	3 x	50	40-66% Heart Rate Reserve	Walk/Jog/ Cycle/Ski	133	127	86	80*
	C 17	39	4 months					129	124	86	75*

* P <0.05 ? = Not Given T = Training Group C = Control Group

TABLE 2.

LONGITUDINAL CONTROLLED STUDIES IN NORMOTENSIVES -

EFFECTS ON AEROBIC FITNESS AND BODY WEIGHT.

AUTHORS	VO_2 MAX (ml kg^{-1} min^{-1}) TEST 1	TEST 2	PERCENTAGE CHANGE	BODY WEIGHT (kilograms) TEST 1	TEST 2
Mann et al 1969	2 minute increase in exercise time.		Exercise Time + 18%	0.85 kg. Decrease*	
DeVries et al 1970	33.9±7.6	$35.5\pm7.03^{+}$	+4.7% (estimated VO_2 Max)	76.1± 9.9	75.2±9.3*
Pollock et al 1971	29.9	38.9^{+}	+30%	77.6±5.4	76.3±5.6*
Bonnano et al 1974	33.4±5.1	35.5±5.1*	+6.3%	N.S.	
Gettman et al 1976	44.9±5.5	50.7±5.3	+12.9%	N.S.	
Millesis et al 1976	41.5±5.2	$48.2\pm 5.5^{+}$	+16.1%	80.6±12.5	78.9±11.6+
Wolfe et al 1979	42.4±2.4	$50.0\pm2.5^{*}$	+18% (estimated VO_2Max)	N.S.	
Kukkonen et al 1982	42.0±1	46 ± 2^{0}	+9.5% (estimated VO^2Max)	N.S.	

*P <0.05 +P <0.01 ^{o}P <0.001

N.S. = Not Significant.

comparison can be made.

2.2.6(a) Characteristics of Training Programmes in Normotensive Subjects

In this review a representative number of controlled studies with SBP under 140 mmHg and DBP under 90 mmHg has been selected (see Tables 1 and 2). The number of subjects has varied from 12 to 144 and the age has ranged from 20 to 70 years. Most studies have had a frequency of training sessions of three times per week (range 3-5) and the duration of training sessions has varied between 17- 60 minutes. The intensity of training has varied greatly. A variety of testing methodology was used to determine fitness improvement. All exercise groups demonstrated a significant increase in fitness while all control group fitness levels remained constant. Some studies have measured Vo2 Max directly (Pollock et al; 1971; Gettman et al; 1976), others (Wolfe et al; 1979; Kukkonen et al; 1982) have estimated Vo2 Max or employed a maximal treadmill test (Mann et al; 1969) to evaluate fitness changes.

2.2.6(b) Body Weight and Blood Pressure Changes in Normotensive Subjects

The body weight of subjects showed a varied response. Four out of the eight studies selected had a significant fall in body weight (Mann et al; 1969; De

Vries et al; 1970; Pollock et al; 1971; Millesis et al; 1976). In five studies (Mann et al; 1969; Bonnanno et al; 1974, De Vries et al; 1970, Pollock et al; 1971; and Kukkonnen et al; 1982; a modest but significant fall in diastolic blood pressure between 2-6 mmHg was reported, but in two of these studies (Bonanno and Kukkonen) the control groups also demonstrated a significant decrease in DBP. Systolic blood pressure fell significantly in the studies of De Vries and Mann while no control group on any of any of the eight studies quoted showed a significant fall. Table 2 provides information on fitness and body weight changes.

2.2.7 Aerobic Training and Hypertensive Subjects

This discussion relates to studies published between 1967 and 1985. Tables 3 and 4 give a representative sample of the research in this area and include information on the mode of training, mode of assessment of cardiorespiratory function, and the effect of the training on blood pressure.

2.2.7(a) Characteristics of Training Programme in Hypertensive Subjects

Physical fitness/high blood pressure studies have been characterised by the use of large muscle groups as the main mode of exercise. Most studies have had a

TABLE 3.

EFFECTS OF AEROBIC TRAINING ON RESTING BLOOD PRESSURE IN HYPERTENSIVE SUBJECTS.

AUTHORS	CHARACTERISTICS OF SUBJECTS NUMBER	AGE	TRAINING PROGRAMME LENGTH	FREQ. (DAYS)	DURATION (MINS)	INTENSITY	MODE	BLOOD PRESSURE (mm. Hg) SYSTOLIC PRE	SYSTOLIC POST	DIASTOLIC PRE	DIASTOLIC POST
Johnson et al 1967	T 4 C -	?	10 weeks	3 x	35	160-180 Beats/Min.	Run	188	195	103	105
Boyer et al 1970	T 23 C -	49	6 months	2 x	30-35	70% Heart Rate Reserve	Walk/ Jog	159	146*	105	93*
Hanson et al 1970	T 8 C -	30-54	7 months	3 x	60	?	Run/Squash/ Basketball	150	134	86	75*
Bonnano et al 1974	T 12 C 15	41 43	12 weeks	3 x	30-35	70-85% of Max Heart Rate	Walk/ Jog	148 150	135* 147	97 101	83+ 90+
De Ressl et al 1977	T 10 C -	38-53	4 weeks	5 x	24	70% VO_2 Max	Cycle	182	176	99	98
De Plaen et al 1980	T 6 C 4	44 47	3 months	3 x	60	60-70% VO_2 Max	Walk/Jog Cycle	169 158	168 154	108 113	114 107
Roman et al 1981	T 30 C -	30-69 Female	7 months	3 x	30	70-85% of Max Heart Rate	Walk/ Jog	182	161^{o}	114	97^{o}
Kukkonen et al 1982	T 13 C 12	42 42	4 months	3 x	50	40-66% Heart Rate Reserve	Cycle/Jog X Country Ski	145 140	136* 140	99 97	88^{o} 88+
Hagberg et al 1983	T 9 C ?	16	6 months	3 x	30	60-65% VO_2 Max	Run	143	133* No Change	94	78
Kiyonga et al 1985	T 9 C -	46 (Male/ Female)	20 weeks	3 x	60	50% VO_2 Max	Cycle	157	136^{o}	104	90^{o}
Duncan et al 1985	T 44 C 12	30 30	16 weeks	3 x	60	70-80% of Max Heart Rate	Walk/ Jog	146 145	134* 139	94 93	87^{o} 96

* P<0.05 + P<0.01 ^{o}P<0.01 ? = Not Given T = Training Group C = Control Group All Subjects Male Unless Stated Otherwise.

TABLE 4

LONGITUDINAL STUDIES IN HYPERTENSIVE SUBJECTS EFFECTS ON AEROBIC FITNESS AND BODY WEIGHT.

AUTHOR	VO_2 MAX (ml kg^{-1} min^{-1}) TEST 1	TEST 2	% CHANGE	BODY WEIGHT (kilograms) TEST 1	TEST 2
Johnson et al 1967	Apparent increase in physical work capacity (No statistics given)		-	N. S.	
Boyer et al 1970	No Exercise Test		-	?	?
Hanson et al 1970)	Increase in physical work capacity 135 watts to 167.5 watts. (No statistics given)		-	?	?
Bonnano et al 1974	33.4	35.5°	+ 6%	N. S.	
DeRessl et al 1977	PWC 130 rose from 106.6 to 127.5 watts	*	+20%	82.8	79.8*
DePlaen et al 1979	31.8	35.9+	+13%	N. S.	
Roman et al 1981	0.89 (litres min_{-1})	1.53°	+72%	?	?
Kukkonen et al 1982	41 Estimated	45 *	+10%	78.2	77.0+
Hagberg et al 1983	39.3	43.0+	+ 9%	N. S.	
Kiyonga et al 1985	Decrease in sub maximal heart rate 123 to 108 beats/minute.	+	-	65.6	63.6+
Duncan et al 1985	39.7	44.5°	+12%	N. S.	

* P <0.05 + P <0.01 O P <0.001
? = Not Given.
NS = Not Significant

frequency of training sessions of 3 times per week (range 2-5). In some cases the quantification of intensity has received scant attention and it is not clear how long the participants operated at the prescribed intensity as the subjects were also involved in warm up exercises and games. The duration of sessions ranged from 24-60 minutes and the length of training has varied from 1 month to 27 months.

In most studies in the 1970's the mean age of the subjects has been around 40. Almost all of the subjects have been male. The number of subjects has ranged from 4 to 56. Clearly the conclusions from small groups are limited as interpretation from these studies could be distorted by the influence of one subject. Various groups have included essential hypertensives and normotensives in their groups. It is difficult to compare subjects with a wide range of initial blood pressure levels as blood pressure of 188/103 mmHg may have a different pathology to a group with a blood pressure of 150/86 mmHg.

In endurance type programmes, it is to be expected that the exercise prescription would elicit a training effect. If previously sedentary subjects show no improvement in Vo2 Max, it is deemed that insufficient adaption has occurred and the training programme has not promoted a training effect. Some studies have provided flimsy evidence that the training programme

has produced an improvement in the aerobic system (Boyer et al; 1970; Johnson et al; 1967). Some studies have demonstrated a dramatic increase in Vo2 Max (Roman et al; (1981) while others have produced only a more modest improvement (Kukkonen et al; 1982). Measurement has varied from sub maximal heart rates, a measure of physical work capacity or prediction of Vo2 Max to evaluate the effectiveness of the training programme. As the mode of assessment of cardiorespiratory function has varied, it is difficult to compare several of the studies.

It is difficult to identify a training variable which appears to influence high blood pressure over any other but reviews by some authors give some indication of the more important factors. Fagard, (1985) used a multiple regression analysis to establish the most important determinant of blood pressure response to training. He found that the initial level of pressure and the magnitude of change in physical work capacity to be the most important factors. Seals et al; (1984a) in their review concede that a clear trend cannot be established between the magnitude of increase in Vo2 Max and fall in blood pressure. They postulate that a substantial increase in Vo2 Max may be necessary to promote a considerable reduction in blood pressure. They add however, that marked cardiovascular adaptions can result at rest after a training programme with no alteration in Vo2 Max.

Some studies have reported a decrease in blood pressure but they have not included nontrained hypertensive controls. These results must be interpretated with caution as some studies which have shown a decrease in blood pressure in the exercising group have found a significant decrease in inactive controls. Systolic and/or diastolic blood pressure fell significantly in the studies of Bonnano et al; (1974); Hagberg et al; (1983); Kukkonen et al; (1982); and Duncan et al; (1985) but in the Bonnano and Kukkonen studies a significant decrease was found in some of the control values. Conclusions about the the effect of training on hypertensives are often unclear as several interelated factors such as decreases in total body weight make clear cut decisions difficult.

There are other methodological differences which present problems. Some studies give no information on how many readings were taken or when they were taken. Others have taken several observations over a few days while some have preceeded the exercise programme with a screening period which has demonstrated a reduction in blood pressure before training has begun.

2.2.7(b) Body Weight Changes and Hypertensive Subjects

Some evidence indicates that resting blood pressure can be reduced through training which is accompanied by

weight loss (Seals et al; 1984a). In the review of Kenny et al; (1984) the authors conclude that weight loss undoubtedly facilitates a decrease in blood pressure in the obese. However, Fagard et al; (1985) in their review of the literature conclude that physical activity does influence blood pressure directly. They do concede, however, that significant alterations in blood pressure have taken place without a significant decrease in body weight or vice versa.

SECTION THREE

LIPIDS

2.3.1 Lipids

Hyperlipidemia is the name given to the clinical condition characterised by increased levels of fat (lipid) in the blood. Cholesterol and triglycerides (TRIG) are the fats most commonly linked with CHD. Cholesterol, a derived fat, contains no fatty acids but has some of the physical and chemical characteristics of fat. Cholesterol is found in all cells. The liver is the major organ which synthesises cholesterol but some tissues including the artery walls also synthesise cholesterol. Cholesterol can also be ingested in food. Other factors recognised as influencing the plasma level of total cholesterol are sex, hypertension, obesity and a family history of heart disease. Lipids are insoluble in blood plasma and must be combined with a "carrier" to transport them - lipoproteins.

Lipoproteins are clasified into major categories depending on their densities. They are Chylomicrons, very low density lipoprotein (VLDL), Low density lipoprotein (LDL), and high density lipoprotein (HDL). LDL accounts for 75% of the total serum cholesterol and is considered to be more closely linked to CHD than

total cholesterol. This lipoprotein is thought to be important in carrying cholesterol to the peripheral cells. Moreover, it is suggested that high levels of LDL are responsible for arterial wall damage which leads to the development of artherosclerotic plaque. Conversely, HDL carries cholesterol away from the cells to the liver. It is suggested that HDL may inhibit the accumulation of cholesterol in the cells by restricting the uptake of LDL by the cells. This is the "reverse transport" role of HDL (Lewis, 1983).

Triglyceride, a simple fat is made up of 3 fatty acids and a molecule of glycerol. More than 95% of the body fat is in the form of triglyceride.

2.3.2 .Lipid Levels and CHD Risk

While it is now accepted that elevated total cholesterol increases CHD risk, there is consensus that it is the manner in which cholesterol is transported in the blood which has a greater bearing on the development of CHD than the total blood cholesterol concentrations. Epidemiological studies have shown that high levels of total cholesterol and LDL cholesterol are associated with an increased CHD risk (Consensus Conference, 1985). On the other hand, high levels of HDL appear to offer some degree of protection from CHD (Miller et al; 1979). In their review, Goldberg et al; (1985) place great emphasis on HDL as a

predictor of CHD. They state that the CHD predictor value of HDL cholesterol has been estimated to be four times that of LDL and eight times that of total cholesterol. A weaker association has been found between LDL and CHD, but Lewis, (1983) in his review indicated that the predictive power of HDL over LDL had not been confirmed in all studies. A low level of HDL was associated with an acceleration of the development of CHD but it was reported that an increase in HDL decreases CHD risk, no matter the level of LDL (Miller, 1977). However, as yet no long term studies have shown that an increase in HDL and a decrease in LDL will lead to a lowering of CHD mortality rates.

The population level of total cholesterol for the British population averages around 6.0 mmol/litre. In the Townhead district of Glasgow 47% of the 1896 males screened had a total cholesterol greater than 5.5 mmol/litre (Greater Glasgow Health Board, 1988). The recommendations of the European Atherosclerosis Society, (EAS) (1987) highlight the need to lower total cholesterol levels. The EAS study group considered that total cholesterol levels of 5.2-6.5 mmol/litre warranted dietary advice and correction with consideration also given to other CHD risk factors. This value has been influenced by the results of the Multiple Risk Factor Intervention Trial, (MRFIT) (1982). Although this study provided no threshold level of total cholesterol, it demonstrated an increase

in mortality as the level of total cholesterol rose. In the MRFIT study subjects in the lowest quartile, who had a total cholesterol level of less than 4.7mmol/litre showed the lowest CHD mortality. The EAS study group considered it a realistic goal to lower total cholesterol to 4.65 mmol/litre for people under 30 years and 5.2 mmol/litre for those over 30 years. They suggested that individuals in the category 6.5-7.8 mmol/litre should be considered for drug therapy if a lipid lowering diet was not successful.

While total cholesterol has been shown to be a predictor of CHD, the ratio of total cholesterol/HDL might be more useful in determining CHD risk. Moreover, there is agreement that the total cholesterol/HDL ratio is of great importance in the development of CHD. Brooks and Fahey, (1984) suggest a ratio of 4 as an acceptable level. A total cholesterol value which remains unchanged may mask any alterations in the sub-divisions within the the total figure. Thus an increase in HDL with no movement in total cholesterol levels would favourably adjust the total cholesterol/HDL ratio.

Several studies have demonstrated that a lowering of total cholesterol levels has resulted in a decrease in CHD mortality rates. For example, the Lipid Research Clinics Coronary Prevention Trial Results, (1984) has demonstrated that drug induced decreases in total

cholesterol and LDL has resulted in a decrease in CHD deaths or non fatal myocardial infarction.

It has not been clearly established that serum concentrations triglycerides are directly linked with the development of CHD (Miller et al; 1977). Although it has been suggested that triglycerides may be associated with CHD independent of cholesterol levels (The Stockholm Prospective Survey, Carlson et al; 1972), some studies have only shown triglycerides to be linked with CHD mortality through its association with total cholesterol levels. However, a Scandinavian study has reported a direct link between triglycerides and CHD. Pelkonen et al; (1977) followed 1648 middle aged men for 7 years. Using multivariate analysis they showed that total cholesterol and triglyceride concentrations were independently associated with CHD mortality rates. Their results suggested that the relationship between triglyceride and CHD death was not linear. Moreover, the authors concluded that triglyceride levels greater than 1.7 mmol/ litre increased the risk of cardiovascular death. Conversely, Carlson et al; (1972) found that CHD increased linearly with higher concentrations of triglycerides and cholesterol.

2.3.3 Measurement of Lipids

Analysis of lipids is normally carried out in hospital

laboratories using automatic analysers. Some research carried out in a number of American laboratories has shown inconsistent results between different hospitals (Goldberg et al; 1987). Concern over validity and reproducability of lipid analysis is widespread as the papers from around the world indicate (Jacobs et al; 1982; Gosland, 1985). Godsland, (1985) concluded that strict control of test conditions produced no decrease in intra-individual variation which he found to be high. The mean co-efficients of variation were 20.9%, 9.2% and 9.8% for triglycerides, total cholesterol and HDL respectively. In recognition of the potential differences between institutions biochemistry laboratories in Britain send samples regularly to a central institution for comparison (See Appendix C).

2.3.4 Factors Influencing Lipids Levels

Lipid levels can be influenced by several factors including seasonal variation, dietary manipulation, alcohol, smoking, medication and genetic factors (Sacks et al, 1981; Eichner, 1985; Criqui et al; 1980; Goldberg et al; 1987; Hartung, 1984; Arntzenius et al; 1985; Councilon Scientific Affairs, 1983). Thus, any study which attempts to examine the effects of exercise on lipid levels must attempt to standardise for all extraneous influences.

2.3.5 Fitness and Lipid Levels

Cross sectional studies have demonstrated that more active individuals may have a more favourable lipid profle compared to sedentary subjects (Dufaux et al; 1982). Caution, however, in the interpretation of some of these studies may be necessary. Athletes and more active individuals usually differ from inactive people in several ways. The active are normally less fat, and alcohol and tobacco use is different and diets tend to be dissimilar. Some individuals may be predisposed to exercise as a result of their genetic make-up. Further difficulties in the interpretation of cross sectional studies may arise when an attempt is made to find group differences when the range of lipids in the whole population is small or where low sample numbers limit meaningful analysis.

2.3.5(a) Fitness - Low Density Lipoprotein and Total Cholesterol

There is no conclusive evidence supporting a direct relationship between regular physical activity and the concentrations of LDL or total cholesterol. There are some studies which have failed to demonstrate a significant difference in total cholesterol between endurance athletes and matched sedentary individuals Adner et al; 1980; Hurter et al; 1972). Other studies

which have compared middle distance runners with sedentary controls have found lowered cholesterol levels in the more active groups. The sedentary subjects however, were more obese and their diet did differ considerably from the exercising group (Martin et al; 1977; Hartung et al; 1980).

2.3.5(b) Fitness and High Density Lipoprotein

Cross sectional studies have in the main shown very active individuals to have higher levels of HDL. Adner et al; (1980) demonstrated HDL levels 20% higher in runners who covered 500 miles per year compared to sedentary controls. Furthermore, Deshaies et al; (1982) demonstrated that Olympic athletes had HDL levels 20% higher than inactive North Americans. Additional evidence is found in the study of Hartung et al; (1980) which revealed that joggers (3 miles per week) had HDL levels 34% greater than sedentary controls while marathoners had HDL levels 13% higher than the joggers.

2.3.5(c) Fitness and Triglycerides

Individuals involved in vigorous activity have shown low serum triglyceride concentrations e.g. active middle aged men, top class marathoners and female distance runners (Martin et al; 1977; Wood et al; 1977). Endurance activities appear to promote some

lowering effect on triglyceride levels. Haskell, (1984) speculated that some of this reduction may be in part linked to the low levels of body fat as well as to increased lipoprotein lipase (LPL) activity associated with endurance athletes. The age related increase in triglyceride levels appears to be prevented by physical activity. Triglyceride levels were found to be around 0.9 mmol/litre for endurance runners compared with 1.7 mmol/litre in sedentary middle aged men (Martin et al (1977). Although some cross sectional studies have indicated that physical activity in work and/or leisure may appear to be directly related to lower triglyceride levels, some of the evidence is inconclusive (Montoye et al; 1978). For example, Montoye et al; (1978) removed the effects of age, weight, sum of skin folds and found that total cholesterol and triglyceride were unrelated to Vo2 Max.

2.3.6 Aerobic Training and Lipid Changes

Many studies which have investigated the effect of exercise on lipids have produced conflicting findings. These inconsistencies may be explained by the variety of the training stimulus used in the different studies, the lack of dietary analysis as well as the confounding factor of changes in body composition during training. Tables 5 and 6 give details of a cross section of studies in this area. Reference to studies not listed in these tables will also be made. The training

prescription has in almost all cases produced a significant increase in fitness levels, but the magnitude of change in fitness has been over a wide range. Initial lipid levels have also had a bearing on the changes produced by aerobic programmes.

Vu Tran et al; (1983) conducted a meta analysis of 66 training studies which measured blood lipids and lipoproteins. A total of 2086 experimental and 839 control subjects were involved in the study. Overall, total cholesterol fell significantly by 0.26 mmol/litre, triglycerides by 0.18 mmmol/litre, and LDL by 0.13 mmol/litre. HDL showed a non significant increase of 0.03 mmol/litre. The total cholesterol/HDL ratio fell significantly by 0.48. Higher initial levels of total cholesterol and LDL resulted in a greater decrease in these variables following training. Those subjects with lower initial HDL values produced greater increases after training. Vu Tran et al; (1983) emphasised that the weight reduction which had taken place during the training may have had a more favourable effect on the lipid profile. They concluded that exercise appeared to promote beneficial changes in lipids. However, they expressed caution in the interpretation of these findings between training and lipids and lipoproteins. They stressed that the interaction of initial lipid levels, age, length of training, Vo2 Max, body weight and percentage body fat together with the effects of exercise made

TABLE 5.

EXERCISE STUDIES AND CHANGES IN LIPID PROFILES -
CHANGES IN FITNESS LEVELS, PERCENTAGE BODY FAT AND BODY WEIGHT.

STUDY	TYPE OF TEST	WEEKS OF TRAINING	TEST 1	TEST 2	P VALUE	% FAT TEST 1	% FAT TEST 2	P VALUE	WEIGHT	P VALUE
Streja et al 1979	Maximal Treadmill Test	13	8 mins 40 secs.	9 mins 33 secs.	<0.005	No Change			No Change	
Huttunen et al 1979	Bicycle	16	3.36 Est. VO_2 Max	3.69 L/min.$^{-1}$	<0.01	Not Given			-0.91 Kg.	<0.01
Shephard et al 1980	Step	26	40.5 Est. VO_2 Max	42.5 ml kg^{-1}min^{-1}	<0.01	No Change			-2.3 Kg.	<0.01
Peltonen et al 1981	Bicycle PWC/150	15	220 watts	227 watts	<0.001	Not Given			No Change	
Allison et al 1981	VO_2 Max Treadmill	8	50.0* 51.8+	52.7 ml kg^{-1}min^{-1} 56.4 ml kg^{-1}min^{-1}	<0.05 <0.05	No Change 11.6	12.5	<0.05	No Change No Change	
Hartung et al 1981	Treadmill	12	31.8 Est. VO_2 Max	37.5 ml kg^{-1}min^{-1}	<0.01	20.3	17.8	<0.01	No Change	
Brownell et al 1982	VO_2 Max Bicycle	10	31.3	34.5 ml kg^{-1}min^{-1}	<0.05	Not Given			-1.0 Kg.	<0.001
La Rosa et al 1982	Treadmill	52	8.4 mets	8.7 mets	<0.001	52.0 Sum of skinfolds	50.2	<0.05	No Change	
Pauly et al 1982	Bicycle	14	38.4 Est. VO_2 Max	43.2 ml kg^{-1}min^{-1}	<0.05	No Change			No Change	
Linder et al 1983	Bicycle	8	16.2	18.7 kpm min^{-1} kg^{-1}	<0.05	Not Given			-3.0 Kg.	<0.05
Gaesser et al 1984	VO_2 Max Bicycle	18	H. 43.3 L. 37.7	50.1 ml kg^{-1}min^{-1} 42.5 ml kg^{-1}min^{-1}	<0.05 <0.05	18.0 19.0	15.9 17.2	<0.05 <0.05	No Change	
Savage et al 1986	VO_2 Max Treadmill	10	H. 43.2 L. 37.4	46.6 ml kg^{-1}min^{-1} 38.4 ml kg^{-1}min^{-1}	<0.05 N.S.	104.3 126.5 Sum of skinfolds	97.0 115.8	<0.01 <0.01	No Change	

+ 45 Minute Group * 30 Minute Group NS = No Significant Change H = High Intensity Group L = Low Intensity Group

TABLE 6.

EXERCISE STUDIES AND CHANGES IN LIPID PROFILE.

STUDY	DESCRIPTION	TOTAL CHOLESTEROL mmol/litre Test 1	Test 2	HIGH DENSITY LIPOPROTEIN mmol/litre Test 1	Test 2	LOW DENSITY LIPOPROTEIN mmol/litre Test 1	Test 2	TRIGLYCERIDES mmol/litre Test 1	Test 2
Streja et al 1979	32 males (35-68 years) 13 weeks of walk/jog 3 x per week. 70-85% MHR for 20-30 mins per session. NO CONTROL GROUP.	5.94	6.22	0.93	1.02	3.87	4.13	2.50	1.81
		(<0.05)		(<0.01)		(NS)		(NS)	
Huttunen et al 1979	50 males (40-45 years) 2 months jog, cycle 3 x per week at 40% HRR two months every second day 66% HRR for 30 mins per session. CONTROL GROUP - NO CHANGE	6.8	6.13	1.27	1.41	4.8	4.22	1.54	1.27
		(NS)		(<0.01)		(NS)		(<.001)	
Shephard et al 1980	21 males (mean age 34 years) 6 months of aerobic activity 2 x per week for 15-17 mins per session. CONTROL GROUP - NO CHANGE.	5.35	5.13	1.40	1.39	3.34	3.07	1.45	1.60
		(<0.05)		(NS)		(<0.05)		(NS)	
Peltonen et al 1981	20 males (31-49 years) 15 weeks aerobic activity 3 x per week at heart rate 140-160 b/min for 30-60 mins per session. CONTROL GROUP - NO CHANGE	5.83	5.61	1.20	1.28	4.13	3.85	1.19	1.17
		(NS)		(<0.01)		(<0.05)		(NS)	
Allison et al 1981	25 men (age 17-26 years) 8 weeks 3 x per week of fitness classes at 85% of MHR for 30 mins or 45 mins.								
	30 min =	4.11	4.22	1.48	1.30	2.21	2.45	0.98	1.03
	45 min =	4.50	4.56	1.55	1.39	2.48	2.68	1.02	1.06
	CONTROL GROUP - NO CHANGE	(NS)		(<0.05)		(NS)		(NS)	
Hartung et al 1981	18 male coronary patients, (32-67 years) 3 months of walk, jog, cycle 3 x per week of 70% VO_2 Max for 20-40 mins. NO CONTROL GROUP	5.42	5.29	1.06	1.22	3.55	3.35	1.79	1.65
		(NS)		(<0.01)		(NS)		(NS)	
Brownell et al 1982	24 males (mean age 42 years) 10 weeks 70% MHR in Y.M.C.A. program for 15-20 mins per session 3 x per week. NO CONTROL GROUP.	5.35	5.02	1.10	1.12	3.43	3.21	2.0	1.61
		(<0.04)		(NS)		(NS)		(<0.02)	

TABLE 6. (Contd.)

STUDY	DESCRIPTION	TOTAL CHOLESTEROL mmol/litre Test 1	Test 2	HIGH DENSITY LIPOPROTEIN mmol/litre Test 1	Test 2	LOW DENSITY LIPOPROTEIN mmol/litre Test 1	Test 2	TRIGLYCERIDES mmol/litre Test 1	Test 2
La Rosa et al 1982	110 post coronary males (30-64 years) 1 year aerobic activity 3 x per week at 70-85% MHR for 30-40 mins per session. CONTROL GROUP - NO CHANGE.	5.75	5.81	1.14	1.17	3.87	3.98	1.61	1.56
		(NS)		(NS)		(NS)		(NS)	
Pauly et al 1982	73 male and female (mean age 36) 14 weeks of running/cycling <2 - <3 times per week at 65-85% HRR for 20 mins per session. NO CONTROL GROUP.	5.40	5.00	1.71	1.68			1.18	0.98
		(<0.05)		(NS)		-		(<0.05)	
Linder et al 1983	51 males (11-17 years) 8 weeks of walk/jog 4 x per week at 80% of MHR for 30 mins per session. CONTROL GROUP - NO CHANGE.	3.82	3.80	1.07	0.97	2.43	2.50	0.95	0.95
		(NS)		(NS)		(NS)		(NS)	
Gaesser et al 1984	16 males (range of 20-30 years) 18 weeks of cycling 3 x per week - 1 group 80-85% of VO_2 Max for 25 mins. 1 group 45% of VO_2 Max for 50 mins per session. NO CONTROL GROUP.	H. 4.73	4.70	1.09	1.16	3.3	3.1	0.66	0.94
		L. 4.70	4.66	1.16	1.23	3.1	2.99	0.94	0.97
		(NS)		(NS)		(NS)		(NS)	
Savage et al 1986	30 males (age 37 years) 10 weeks of walk/jog 3 x per week 40% or 75% of VO_2 Max for 2.8 to 4.8 kilometres per session. CONTROL GROUP - NO CHANGE	H. 5.45	5.26	1.61	1.18	3.54	3.45	1.08	1.12
		L. 4.94	5.17	1.61	1.40	2.81	2.99	1.34	1.33
		(NS)		(<0.05 for H group)		(NS)		(NS)	

( ) = P Value (NS) = Not Significant MHR = Maximum Heart Rate HRR = Heart Rate Reserve

H = High Intensity Group L = Low Intensity Group

interpretation of these findings somewhat difficult.

2.3.7 Lipids and Training Stimulus

Athough several longitudinal studies have in general supported the belief that aerobic exercise will promote a favourable lipid profile, it should be emphasised that a limited number of studies have included controls in the research design. Haskell, (1987) in his review of aerobic training and total cholesterol changes stresses that differences in exercise intensity and duration of training sessions or the length of the training programme do not offer any explanation for the great variation in the results in these studies (see Table 6).

Several studies have used a similar training programme of 30 minutes duration three times per week and produced discordant results. Altekruse et al; (1979) and Lopes et al; (1974) showed that middle aged men who induldged in a short term training programms could reduce their total cholesterol levels. However, other workers have been unable to demonstrate similar decreases in total cholesterol in middle aged men (La Rosa et al; 1982; Leon et al; 1979). A similar confusing situation exists with LDL where aerobic training has not lowered LDL after training (Allison et al; 1981; Streja et al; 1979). However, decreases in LDL have been shown by Peltonen et al; (1981) and

Shephard et al; (1980).

The effect of training on HDL has resulted in increases in HDL (Peltonen et al; 1981; Streja et al; 1979) while other studies have found no change (Brownell et al; 1982; La Rosa et al; 1982). A small number of studies has demonstrated a reduction in HDL (Allison et al; 1981; Savage et al; 1986). The variety of training stimulus, the lack of control of the diet and the varying length of the studies may all have contributed to the wide ranging findings.

HDL is inversely related to body weight. Many aerobic exercise programmes have reported a fall in body weight. However, there appears to be some confusion with regard to body weight and HDL changes related to training. Favourable changes in HDL were shown to be caused either through aerobic training or fat loss through diet (Wood et al; 1988). However, the exercise group HDL improvement was not independent of a loss in body fat. The findings of Huttunen et al; (1979) and Peltonen et al; (1981) do not support the contention that a decrease in body weight is essential for improvement in HDL. Indeed, Huttunen found that the greatest rise in HDL came from those who had least weight loss. Conversely, Brownell et al; (1982) showed a significant decrease in body weight of 1.0 kg, but failed to increase HDL levels significantly. The literature in this issue is still unclear.

Increases in fitness levels (measured by Vo2 Max and treadmill time) as a result of endurance training failed to demonstrate reductions in triglyceride levels (Gaesser et al; 1984; Peltonen et al; 1981). It should be stressed that these subjects had triglyceride levels within normal range .i.e. 0.66 to 1.19 mmol/litre. There is some evidence to show that training will lower triglyceride levels in subjects with relatively high baseline levels as in the study by Brownell et al; (1982) where the pre-training triglyceride level was 2.0 mmol/litre. Conversely, there are some other studies where training has failed to lower triglyceride levels greater than 2.0 mmol/litre (Streja et al; 1979). It may be that subjects with elevated triglyceride levels could benefit from endurance training.

SECTION FOUR

BODY COMPOSITION

2.4.1 Body Composition

The subject of weight, diet and exercise has become increasingly popular as it is now recognised that there is an association between obesity and a worsening of CHD risk factors.

Height and weight tables are still used extensively to determine the extent of "overweightness" based on age and frame size. It should be noted that there is no scientific basis for frame sizes and there is no recognised preocedure whereby frame sizes can be categorised (Garrow, 1979). It is possible for an individual to be overweight based on height weight tables but have very small amounts of fat. American footballers are often cited as examples of individuals who are overweight according to height-weight tables but have below average body fat (Mc Ardle et al; 1986). It is clearly more desirable to determine the body composition of the individual.

The human body consists of many component parts. However, the two component parts most often used with regard to body composition are the fat free compartment and the fat compartment. Sometimes the fat free

compartment is referred to as the lean body mass.

2.4.1(a) Percentage Body Fat

There is no consensus as to what percentage body fat or range of values is to be considered obese. Studies of groups in the western world indicate that the population is becoming more obese and the incidence of obesity rise as the population ages. The increase in obesity with age is seen as a cultural phenomenon and is considered to be detrimental to health. Obesity is defined by McArdle and Katch, (1986) "as excessive enlargement of the body's total quantity of fat." The criteria suggested for a "normal range" is to use one standard deviation from the mean as the normal range. In North America, the threshold for obesity would be 20% body fat in young men and in older men obesity would be defined as above 30%. The authors conclude however, that "average" population values should not be used as a reference standard and should not be allowed to dictate that the older the individual the fatter he will become. There is agreement that the essential amount of fat is around 3-4% (Durnin, 1984).

2.4.2 Obesity as a CHD Risk Factor

Is "overweight" associated with CHD or a cause of CHD? Obesity could be responsible for inactivity which could in turn result in CHD. Although obesity has received

great noteriety as a CHD risk factor, until recently research has indicated a poor relationship between obesity and CHD. Obesity is interelated with other risk factors e.g. hypertension, hypercholesteremia and diabetes mellitus. Obesity is certainly not considered to be a primary CHD risk factor but the following evidence clearly indicates that there is a link between obesity and CHD.

Lew et al; (1979) described the mortality rates in relation to body weight of 750,000 men and women in a United States population. The authors sub-divided their enormous sample into seven weight categories. They found that mortality from CHD was greatly increased in the higher weight categories. CHD mortality was 55% higher among those 30-40% heavier than average and around 100% higher among those more than 40% overweight. Furthermore, post mortem studies have revealed a high correlation between obesity per se and arthersclerosis (Wilkins et al; 1959). Further convincing evidence of the link between obesity and CHD is provided by Hubert et al; (1983) who examined the relationship between the degree of obesity and the incidence of CHD disease in 5209 males and females. Desirable weight was derived from the 1959 Metropolitan Life Insurance Companies by taking the midpoint of the weight range for the medium build at a particular height. Criteria were established for CHD diagnoses including angina, myocardial infarction and sudden

death. During the 26 year follow-up 870 men and 688 women were found to have some form of CHD. The authors concluded that an increase in weight after young adulthood is associated with an increased risk of CHD. Obese individuals often have hypertension and elevated blood lipid levels (Lancet, 1985; Stamler, 1978; Angel, 1978). Moreover, various studies have shown that weight loss is associated with a decrease in blood pressure and an improvement in the lipid profile (Pacy, 1986; Brownell et al, 1981; Andrews, 1982). A review in the Lancet, (1985) highlighted the fact that reductions in hypertension achieved through weight loss has much to commend it. Clearly, the possible non pharmacological treatment of hypertension has great appeal as it is cheap and non pharmacological treatments avoid the associated side effect problems.

While it must be conceded that obesity is not a primary risk factor, obesity is a contributory factor in CHD and body fat reduction will contribute to a decrease in CHD risk.

2.4.3 Energy Balance

Fat levels can be altered by "unbalancing" the energy balance equation. There are three ways to "unbalance" the energy balance equation:

1) Reduce caloric intake below daily requirements.

2) Maintain regular food intake and increase energy expenditure through exercise.
3) Decrease food intake and increase energy expenditure through exercise.

Kilocalorie abbreviated kcal represents the amount of heat necessary to increase the temperature of one kilogram of water by one degree centigrade from 14 to 15 degrees centigrade. The energy equivalent of losing one kilogram of fat is 7000 kcal. If the individual expends 7000 kcal more than is consumed, this is equivalent to one kilogram of fat loss. Conversely, if the individual consumes more than 10,000 kcal of fat than is expended, an extra kilogram of fat will be added. The apparent discrepancy betwee 10,000 and 7,000 kcal is explained by the mechanical and chemical inefficiency of depositing fat (Durnin, 1985).

2.4.4 Methodology of Estimating Energy Expenditure

Energy expenditure is normally calculated directly or indirectly in the laboratory. Direct assessment is made using a whole body calorimeter. As this method involves a large non-portable chamber, other indirect methods have been developed. Measurement of expired air and evaluation of the concentrations of oxygen and carbon dioxide enables an estimate of energy expenditure to be made. While this method has been occasionally employed outdoors, it involves

sophisticated equipment and is logistically very difficult to carry out.

Several researchers (Margaria et al; 1963; Shephard et al; 1969b) have measured the calorie cost of walking and running using measurement of expired oxygen and carbon dioxide. However, measurement or estimation of the energy cost of individuals who are participating in an indoor fitness session which involves different types of activity is logistically very difficult to undertake. Measurement of energy expenditure indoors would involve the use of Douglas bags or an "on line" automatic analysing system. Clearly, this operation would involve a great deal of time and require sophisticated equipment.

It may be possible to gain a very rough estimate of energy expenditure by measuring heart rate during the exercise period and translating this heart rate into a percentage of Vo2 Max. If the Vo2 Max were known, it would be possible to estimate the energy expenditure by calculating the percentage of Vo2 Max at which the subjects were exercising. Mc Ardle et al; (1986) give guidance on the eqivalent percentage maximum heart rate and percentage Vo2 Max. It should be noted that Durnin, (1985) is sceptical of the use of heart rates to predict energy expenditure as he believes that heart rates are influenced by factors other than the tissues need for oxygen. Thus, heart rates are contaminated by

other factors and distort any estimate of energy expenditure. However, such extraneous influences are less likely to play a role in modifying the exercise heart rate as opposed to resting heart rate.

2.4.5 Post Exercise Increases in Energy Expenditure

Exercise increases energy expenditure during a 30 minute period at 60%-75% HRR. The energy expenditure will become fairly stable after the initial period of adjustment. It is claimed by some that exercise has a long lasting effect on the level of energy metabolism. Indeed, it is suggested that the basal metabolic rate (BMR) remains elevated for some hours after exercise. Clearly, if this assumption were true, the increase in BMR would result in a marked increase in energy expenditure. The acute effects of aerobic training on BMR have been investigated by Durnin, (1985). He concluded that the increase in BMR after training has an insignificant effect on energy expenditure (Durnin, 1985). However, it should be noted that some studies have demonstrated an increase in BMR following high intensity long duration exercise. A review in the Lancet, (1988) highlights that there are discrepancies in the findings of studies in this area. It may well be that the magnitude of error in the measurment of the BMR masks any trend in the studies that have been carried out.

A further consideration in the calculation of energy expenditure is the resting metabolic rate. Subtraction of the resting metabolic rate (RMR) from the total energy cost of the activity is necessary to calculate the net energy cost of the exercise session. Durnin et al; (1967) constructed tables which detail the resting rate of energy expenditure. These tables have been derived partly from the literature and partly from unpublished observations from the authors. The RMR is dependant on body weight and body composition and the RMR ranges from 0.78 kcal/min in 45kg females to 1.39 kcal/min in lean 80kg males.

2.4.6 Energy Intake

Accurate measurement of energy intake requires meticulous methodology in the weighing and itemising of all food eaten. Thereafter, it is necesarry to analyse the food recordings and calculate the energy in the constituent parts of the food using formula which take into consideration the energy contained in fat, carbohydrate, protein, and alcohol (Paul et al; 1978). This procedure can be done using computer programmes. There are packages which enable subjects to record their diet using a code system (Balance your Diet, Cambridge University Press). These recordings are subsequently analysed using a computer programme which provides information on total energy intake, carbohydrates, fat and protein. Any type of energy

intake measurement is difficult to undertake. It is unlikely that subjects will give good compliance over extended periods of time as the recording of every item of food is extremely laborious. Therefore, it is better to ask subjects to provide detailed information over short periods of time and hopefully gain an accurate picture. As diet may differ considerably over a week-end, it is better to include a week-end in the data collection period.

2.4.7 Measurement of Body Fat

Body fat can be measured directly or indirectly. Direct measurement involves the chemical analysis of human cadavers while indirect techniques include hydrostatic weighing, skinfold and circumference measurement. Further information on the measurement of body fat is given in Mc Ardle et al; (1986).

2.4.7(a) Hydrostatic Weighing

Hydrostatic weighing involves weighing the individual in water and in the air. As the density of fat and fat free mass differ it is possible to estimate the body composition. This method has been established as the "gold standard" for body composition measurement. It has some limitations. The subject must be fully immersed in water and "non water" subjects may be apprehensive about this situation and refuse to take

part. The apparatus involved is costly and difficult to transport.

2.4.7(b) Skinfold Measurement

The limitations of cost, technical complexity and time involving processes of some methods of assessing body fat has led to the development of skinfold techniques. In some cases skinfold alone or skinfold in conjunction with anthropometric variables have been used to determine body fat. From skinfold measurement, equations have been formulated to predict body density. Body density in itself is not a precise estimator of body composition. However, from the body density prediction, equations have been produced to predict percentage body fat (Mc Ardle et al; 1986).

Although skinfold measurement is less complex and less time consuming than other forms of body composition assessment, it is subject to error. As the body ages, there is a greater deposition of fat internally rather than immediately under the skin Durnin, (1984). Predictive equations are population specific. Some equations for estimating body fat could be called descriptive rather than predictive equations. Moreover, various researchers have used different anthropometric sites. Thus, it is essential to determine the actual site. Lack of standardisation of skinfold technique can lead to a further source of error.

2.4.8 Exercise and Fat Loss

A considerable number of investigators have examined the effects of exercise on body composition. Wilmore, (1983) in his review of 55 studies on aerobic exercise and body composition considers that changes in body composition have been minimal. He highlights the fact that much greater alteration in body composition would be expected from animal studies. Wilmore's evidence is that a mean decrease in body fat of only 1.6% took place in 55 human training programmes of durations between 6-104 weeks. He concludes by stressing that much tighter control of energy intake and energy expenditure must be made to clarify some of the discrepancies in this area.

Various studies suggest that there is a threshold for fat loss. The individual must expend a minimum number of calories in a session or in a week to alter body composition. Some research by Pollock et al; (1984) indicates that 3 or 4 sessions per week are more effective than 2. In one study the energy expenditure was similar in 2 groups, one of which trained 2 times per week for 45 minutes per session and the other 3 times at 30 minutes. The latter group was the only one to show a fall in body fat. These results are quite surprising considering the fact that the energy expenditure of the 2 programmes was the same. These findings have influenced researchers in the field to

recommend that training should take place three times per week and have a minimum energy expenditure of 900 kcal per week (Pollock, 1972; Pollock et al; 1975; Pollock et al; 1978; Pollock et al; 1979).

A number of studies which have examined the effect of aerobic exercise on body composition in middle aged men are listed in Table 7. Direct comparison is often difficult because of a lack of dietary control and the problems involved in estimating energy expenditure.

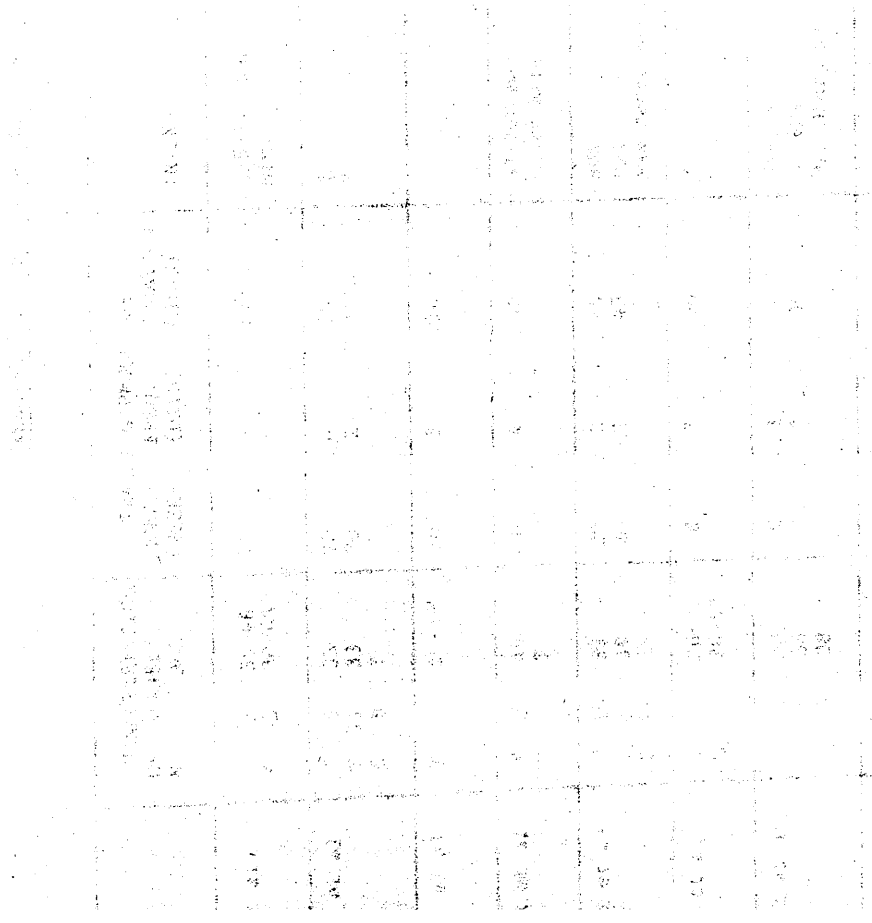

TABLE 7.

EXERCISE STUDIES/BODY WEIGHT/BODY FAT CHANGES.

AUTHORS	CHARACTERISTICS OF SUBJECTS N	AGE	TRAINING PROGRAMME LENGTH (WEEKS)	FREQ. (DAYS)	DURATION (MINS)	INTENSITY	MODE	WEIGHT (KILOGRAM) TEST 1	TEST 2	PERCENTAGE BODY FAT TEST 1	TEST 2
Oscai et al 1968	T 5	35-46	16	3	15-30	155-170 beats/ min.	Run	88.1	83.6 (<0.05)	23.6	20.0 (<0.05) skinfold
	C 5	30-45						87.9	87.0 (NS)	23.6	23.7 (NS)
Pollock et al 1969	T 8	33	20	4	30	?	Walk/Jog	79.7	76.8 (<0.05)	19.6	18.6 (<0.05)
	T 11	33	20	2	30	?	Walk/Jog	80.2	80.3 (NS)	18.0	18.9 (NS)
	C 8	?						73.8	74.1 (NS)	20.3	21.2 (<0.05)
Wilmore et al 1970	T 55	17-59	10	3	14	?	Jog	79.6	78.6 (<0.05)	18.9	17.8 (<0.05)
Pollock et al 1971	T 16	49	20	4	40	63-76% Max Heart Rate	Walk	77.6	76.3 (<0.05)	22.0	20.9 (<0.05)
	C 8	?						82.2	82.2 (NS)	22.3	22.8 (<0.05)
Pollock et al 1972	T 10	39	20	2	45	80%	Walk/Jog	81.3	80.4 (NS)	23.3	22.9 (NS)
	T 12	39	20	2	45	92%	Walk/Jog	79.4	78.7 (NS)	22.9	22.1 (<0.05)
	C 12	39				Max Heart Rate		82.6	82.0 (NS)	23.1	22.8 (NS)
Misner et al 1974	T 8	26-57	8	3	30	?	Jog	84.7	83.9 (NS)	27.9	25.5 (<0.05)
	C 6	26-57						79.0	78.8 (NS)	19.4	19.4 (NS)
Pollock et al 1975	T 9	38	20	3	30	87-90%	Walk	85.2	83.9 (NS)	22.4	19.4 (<0.05)
	T 9	38	20	3	30	87-90%	Run	84.7	83.4 (<0.05)	21.7	20.4 (<0.05)
	C 7	38				Max Heart Rate		81.0	81.0 (NS)	21.2	21.1 (NS)
Van Handel et al 1976	T 6	35-56	10	3-4	20-30	75-85% of VO_2 Max	Walk/Jog	84.9	85.2 (NS)	21.3	24.0 (NS)
Millesis et al 1976	T 17	20-35	20	3	30	85-90% Max Heart Rate	Walk/Jog	80.6	78.9 (<0.05)	14.2	13.6 (<0.01)
	C 16	20-35						72.1	73.2 (NS)	12.5	13.0 (NS)

T = Training Group NS = Not Significant C = Control Group ? = Not Given

Body fat measured by densitometry unless stated otherwise.

SECTION FIVE

FLEXIBILITY

2.5.1 Flexibility

Flexibility is defined as the range of possible movement in a joint or a series of joints. Two types of flexibility were identified by Fleishman, (1964), extent flexibility and dynamic flexibility. The former is where the emphasis is on the ability to move or stretch the body as far as possible in one movement (eg., yoga) and the latter is the ability to make repeated flexing and stretching movements of a ballistic nature.

Research has shown that flexibility tends to be specific to a particular joint (Alter, 1988). The most important factors in determining flexibility are muscles and ligaments while tendons are considered less important (Corbin and Noble, 1980). The lengths of muscles and ligaments determine to a great extent the amount of movement possible at each joint. For example, the extensibility of the knee joint is limited by the hamstrings. The main sources of resistance at the normal extremes of joint motion are big joint capsules, tendons and muscles. However, it should be noted that in some joints it is bone contact which is the limiting factor as in the olecranon process in the

elbow joint. Research indicates that resistance to stretch when a muscle is relaxed, is not from the myofibrillar elements but is linked to the connective tissue sheathing around and within the muscle (Johns and Wright; 1962). Connective tissue consists of collagen fibres which are embedded in a protein polysaccharide matrix. Connective tissue has an elastic and viscous (plastic) element. When connective tissue is stretched, it behaves as though it has both viscous and elastic elements connected in series. As connective tissue is stretched, elongation takes place in the elastic and viscous elements. However, when the stress is removed, it is only the plastic deformation which remains. Thus, flexibility exercises should aim to produce plastic deformation which will promote a permanent increase in range of movement.

It is believed that inactivity causes muscles and connective tissue to lose their normal extensibility and thus flexibility is reduced (Jensen and Fisher, 1972). Moreover, inactivity may also result in an accumulation of fat which may further restrict flexibility. Advancing age is associated with stiffness. However, in his review, Hockey, (1973) refers to research when he states that children become less flexible to around the age of 12. Thereafter, flexibility increases into young adulthood after which flexibility decreases with age. Tissue changes account for some decrease in flexibility and diminishing

activity is believed to be another contributory factor. It is suggested that the more active the person is, the greater the flexibility. However, the activity must be performed through the full range of movement of each specific joint. If there is a limited range of movement during the activity (e.g. endurance runners who do not perform flexibility exercise) a reduced range of movement may result (Round table, National Sports and Coaching Association, 1984). Furthermore, flexibility is temperature related and is influenced by local warming and cooling. Local warming will enhance flexibility (Beaulieu, 1980).

2.5.2 Flexibility and Health

Some of the evidence that good flexibility is beneficial to health is sometimes anecdotal or theoretical. However, research highlights the importance of flexibility in relation to health. Low back pain is a prevalent medical complaint in the western world which results in great expense and discomfort (Nachemson, 1988). There is often no concensus on the cause of back pain. Nachemson, (1988) referred to a study at the Boeing Works in Seattle which found that an increase in flexibility had no influence on low back pain whereas Corbin et al; (1980) stress that much low back pain is the result of weak in flexible muscles. Furthermore, Kraus et al; (1961) highlighted the fact that 80% of those who suffered

lower back pain in their subjects had some form of muscular deficiency. The assessment of low back pain sufferers included a test battery which identified muscle weakness or stiffness. In only 20% of the low back pain sufferers could a well defined diagnosis be made. Two hundred and thirty three cases were followed for 8 years. It was found that increases in muscle strength and flexibility paralleled improvement in low back pain. The authors concluded that "muscle insufficency" may cause backache and that they have demonstrated that appropriate exercise can improve muscle strength and flexibility and lower the incidence of back pain. Nachomson, (1988) indicated that in the majority of his patients the cause of low back pain was unknown. However, he agreed that physical activity could promote healing in low back pain.

Some authorities in this area (Pollock et al; 1984; Corbin et al; 1980) consider that flexibility and strength in the abdominal, back and hamstring muscles to be important in the prevention and rehabilitation of low back pain. Thus, there is evidence that connective tissue stiffness which restricts joint movement may promote low back pain. Moreover, weakness in the muscles of the trunk have also been linked with low back pain. On the other hand, excessive flexibility may produce an unstable joint and poor stability may result in injury.

2.5.3 Measurement of Flexibility

As flexibility is specific to each joint, it is not possible to measure the flexibility of one joint using one test and relate it to other joints (Pollock et al, 1978). Various types of apparatus have been devised to measure flexibility. Reilly, (1981) outlined the various instruments and how they are used, eg., goniometer, flexometer, electrogonometer and photography. Other forms of tests which do not require the use of a goniometer have been developed, some of which involve the use of fairly simple apparatus. Hockey, (1973); Campbell and Tucker, (1967) give examples of these tests, for example Sit and Reach Test. The Sit and Reach Test is a measure of flexibility in the lower back and hamstring muscles (Wells and Dillon, 1952).

2.5.4 Development of Flexibility

Although there is general agreement among researchers that flexibility can be improved, there is no consensus as to how increased flexibility can be best achieved (Corbin and Noble, 1980). Hockey, (1973) considers that it is necessary to move the body parts through complete ranges of motion several times per day in order to maintain adequate flexibility. Furthermore, Corbin and Noble, (1980) refer to research when they state that whatever technique is used, the muscular

connective tissue must be stretched beyond the normal length to be effective. A limited number of studies in the area suggest that both ballistic(bobbing movements) and slow tension (static stretching) exercises increase flexibility but ballistic movements are more likely to cause injury (De Vries 1962; Etnyre et al; (1987). While most sports are of a ballistic nature, it may be appropriate for athletes to include ballistic stretching in their flexibility programmes. However, static stretching is recommended for health related flexibility as the static stretch is less inclined to evoke the stretch reflex which will in fact limit the development of flexibility. The stretch reflex is a protective compensating mechanism which allows the muscle to adapt without relying on higher centres for control.

Proprioceptive neuromuscular facilitation (PNF) is a stretching technique which involves the individual in sequentially contracting the muscle to be stretched followed by stretching of the desired muscle. Corbin et al; (1980) in their review indicated that PNF was as least as effective as static and ballistic stretches in the development of flexibility. However, they concede that comparison of the various studies is difficult as different studies have used a wide variety of PNF procedures. Several authors suggest that in a static programme that a stretched position should be held in one position for around 30 seconds and this period of

time should be increased to around one minute as the training programme progresses in the subsequent weeks. It is recommended that ballistic exercises are performed for between 30 - 60 seconds (De Vries, 1962; Hardy et al; 1986). Although comparisons between static, ballistic and PNF suggest that PNF procedures produce the most favourable results, Etnyre et al; (1987) highlight the fact that differences in methodology and research design make comparison difficult. Indeed, Etnyre et al; (1987) concluded from their review that the question of which stretching method is most effective has not been answered.

2.5.5 Training Studies and Flexibility

De Vries, (1962) referred to his experience in the area when he stated that he has found that the lower back and hamstring muscles were the most resistant of all joint complexes to improvement. Furthermore, he stressed that extra emphasis should be placed on these areas as the flexibility of the hamstrings and the lower back were very resistant to improvement. However, Fox, (1979) stated that significant gains in flexibility are to be expected in 5 weeks if stretching activities are performed twice per week.

The question of how to improve flexibility has apparently received limited emphasis where research is concerned, and although the Sit and Reach Test is

mentioned as a flexibility test in several textbooks, it appears that this test has not been used very often to evaluate the effectiveness of training programmes. Several studies have used the Sit and Reach test to evaluate the effectiveness of training programmes. Moreover, different scales have been used and the obscure reporting of data in others have caused confusion. In an attempt to standardise comparison, the values from other studies have been changed to centimetres and 30 has been taken as the point which coincides with the plane of the bottom of the feet.

Comparison between studies is made difficult because different amounts of time have been spent on flexibility and various types of exercise have been included in the programme. Furthermore, the duration of the studies has varied and differing populations have been investigated. Details of some of the training programmes are sketchy. Indeed, the fact that some of these training programmes may have greater priorities in other fitness components may explain the paucity of studies and detail in this area.

Flexibility as judged by the Sit and Reach Test in the Marcinik et al; (1985) study did not improve after three different types of training programmes - aerobic/calisthenic and aerobic/circuit training at 40% and 60 % of a one rep maximum. The authors conceded that none of their programmes stressed flexibility

development. Therefore, it is not surprising that no significant changes in flexibility took place. This finding prompted the authors to examine their physical conditioning programmes.

Another study which failed to improve flexibility in 16 males was that of Wilmore et al; (1978). The subjects performed circuit weight training for 10 weeks, three times per week. Each session lasted 22.5 minutes and was performed on a Universal Gym. Exercises were performed at 40%-55% of the subjects maximum strength for 30 seconds at each station followed by 15 seconds rest. The initial value on the sit and Reach test was 30.5 centimeters which showed a non significant increase to 33.8 centimeters.

In the study by Van Gool et al; (1981) 16 males (mean age =41 years) exercised for one hour by performing circuit training which included one lower back and hamstring exercise. The Sit and Reach Test was used to evaluate changes in flexibility. No information on values is available from the paper but the authors concluded that there was a slight but significant increase in flexibility.

In their comparison of static, dynamic and PNF techniques, Lucas et al; (1984) used the Sit And Reach Test to assess the effectiveness of their training programmes. All three groups showed a significant

increase in flexibility - static (27 to 30) ballistic (28 to 31) and PNF (27 to 30) centimeters. All three groups performed their flexibility sessions three times per week for seven weeks. The 63 female college students performed aerobic dance as well as their flexibility exercises.

2.5.6 Norm Tables

Pollock et al; (1984) provide norm tables for flexibility as measured by the Sit and Reach Test. These tables are derived from an American population and offer some form of comparison with other groups (see Table 8).

TABLE 8

FLEXIBILITY NORMS

Sit and Reach Test (centimetres)

Excellent	>50
Good	42-48
Average	30-40
Fair	28-25
Poor	<23

Thirty is equal to level with the plane of the foot. The values have been changed from inches to centimetres.

Pollock et al. 1984.

SECTION SIX

LOCAL MUSCULAR ENDURANCE

2.6.1 Local Muscular Endurance

Local muscular endurance (LME) refers to the ability of a single muscle group to carry on working in the form of either a sustained contraction (isometric) or repeated contractions (isotonic). Many factors are involved in local muscular endurance. Indeed, local muscular endurance is a complex physiological and psychological phenomenon. Factors influencing LME include strength, anaerobic and aerobic enzymes, buffering mechanisms and muscle blood flow (Sharp, 1978).

Strength is not directly correlated with local muscular endurance. Sharp, (1980a) pointed out that muscles function at their best at one third of the maximum force that they can exert where local muscular endurance is concerned. Thus, in certain circumstances, increases in muscular endurance may be gained by increasing the local muscular endurance of the muscles or the strength of the muscles, or both. The concentration of myoglobin in skeletal muscle is important. Before the oxygen transport system can "meet the demand for oxygen", the oxygen bound to the

myoglobin is consumed. This small amount of oxygen delays the build-up of lactic acid in the muscles and the blood. The greater the myoglobin stores, the longer it will be before the muscles are "inhibited" by lactic acid. Increases in myoglobin will help increase the consumption of oxygen as myoglobin's main function is aiding diffusion of oxygen from the cell membrane to the mitochondria, where oxygen is consumed (Fox, 1979). The oxygen utilising enzymes which are found in the mitochondria provide adenosinetriphosphate (ATP). Thus, mitochondria provide energy for muscle contraction. A muscle which has a great many mitochondria will be able to continue contraction/relaxation cycles for a long period of time provided the supply of nutrient remains adequate. The local blood vessels must supply sufficient blood and therefore oxygen to the working muscles as well as carrying away waste products. Thus, a well developed capillary network can enhance local muscular endurance (Astrand and Rodahl, 1986).

When the oxygen system is not available, ATP can be reformed from the energy which arises from the anaerobic breakdown of glucose. The glucose is stored in muscle in the form of glycogen (Mathews and Fox, 1976). As glucose is broken down and energy is produced, lactic acid builds up in the muscle and blood stream and this accumulation of lactic acid can cause

fatigue. Fatigue during sustained high intensity anaerobic activity is associated with an increase in hydrogen ions and a lower pH. Hydrogen ions inhibit glycolysis by reducing phosphofructokinase (PFK) activity. (PFK is the rate limiting enzyme in glycolosis). Furthermore, Astrand and Rodahl, (1986) discuss the possibility that hydrogen ions alter the binding of calcium ions to muscle regulatory proteins and therefore reduce the contractile force. Moreover, Astrand and Rodahl, (1986) hypothesise that a decrease in pH could reduce the myofibrillar ATPase activity which would decrease the rate of ATP turnover and cross bridge cycling.

The chain of events leading from the brain to cross bridge interaction comprises of several component parts. Fatigue in this chain may be the result of impairment in one or more of the links in the chain (Gibson et al; 1985). Several authors consider that there is fatigue at the neuromuscular junction, but there is dispute as to whether fatigue in this area is caused by intense exertion of short duration or whether fatigue results from work of long duration (Stephens and Taylor, 1972; Sharkey, 1975). However, Kugelberg et al; (1979) suggest that failure in the motor endplate transmission is unlikely as endplate transmission becomes limited at firing rates around 100 hz while human motor nerves in an endurance situation only fire around 20hz. Decline in the EMG has been

been found not to parallel force decrease in repetitive exercise (Spurway, 1987). Thus, electrical failure is unlikely to be the source of fatigue.

2.6.2 Local Muscular Endurance and Health

The implications of LME and health have been discussed in the chapter on flexibility. Several authors consider that weak abdominals and/or lack of flexibility in the lower back or hamstrings can be a contributory factor in low back pain (Patton et al; 1986; Pollock et al; 1984). As has been expressed earlier, low back pain is a serious, widespread health problem which, in some circumstances could be alleviated by an appropriate exercise programme.

2.6.3 Measurement of Local Muscular Endurance

As muscular endurance is specific to each of the muscle groups, separate tests of each muscle group are necessary to evaluate local muscular endurance in a subject (Hockey, 1973).

There are various tests to measure local muscular endurance, pull-ups, sit-ups, push-ups. However, Pollock et al; (1978) pointed out that some of these tests penalise subjects who have a heavy body weight. In order to overcome this problem in LME testing, they suggested that it is possible to use a fixed percentage

of the individual's body weight as the resistance, and the individual performs as many repetitions as possible. They indicated that the debate continues as to what the actual percentage of the subject's body weight should be for the muscle group being tested or a fixed percentage of the indivdual's absolute strentgh as demonstrated in one repetition.

It is difficult to find apparatus to measure LME. Sharp, (1980a) states that it is necessary to establish a "defined movement" in a certain time. Thus, the movement must be standardised and the pace of the movement must be regulated by a metronome. If no metronome is used, the subjects will have the opportunity to vary the speed of movement and there could be great differences in the speed of movement between different subjects. However, with unknown ability, in a very mixed population, a standard cadence can make the test awkward for subjects. Difficulty in the maintainance of the cadence or problems in selecting a cadence which is appropriate for all can produce difficulties for the tester and the subjects. Thus, in some instances sit-ups have been established for a given time period e.g. how many sit-ups can a person perform in one minute? This type of test is advocated by Pollock et al; (1984) as LME in the abdominal area is considered to be important in a health related fitness programme.

2.6.4 Development of Local Muscular Endurance

In training for LME it is important to examine the energy systems involved as well as the movement, the speed, and the number of repetitions and the forces involved. Energy is released when ATP is broken down. There are three main systems through which this energy becomes available for muscular contraction. If great forces are applied, it is likely that the muscles will fatigue quickly. In athletic performance, movements which last for 10 to 15 seconds will probably have large forces or slightly smaller forces will be applied more quickly. Thus, the ATP,creatine phosphate (CP) system should be trained. Astrand and Rodahl, (1986) suggest the following method to train the ATP, CP system. They state that the stores of ATP,and CP should be greatly reduced within 15 seconds of maximum effort. Rest periods should be sufficiently long to allow the ATP, CP system to recover. If little rest is given, more emphasis will be placed on the lactic acid system and the oxygen system (Campbell, (1980). Astrand and Rodahl,(1986) suggest a 3 minute rest between efforts.

In near maximal or maximal movements of around 60 seconds, the lactic acid system is the predominant source of energy, therefore it is this energy source which should be trained. Astrand and Rodahl, (1986) state that there should be a maximum effort of one

minute duration followed by 5 minutes rest. A rest of 5 minutes enables the athlete to maintain a high quality of performance. Campbell, (1980) considers that the work bout should be between 15 to 45 seconds and states that the term "quality" refers to the capability of the athlete to reproduce as closely as possible his previous best performance and she considers that this type of training allows the athlete to work at high intensity as a 5 minute active rest period allows the lactic acid levels to drop. Lamb, (1978) states that the training should be very intense so that the enzymes involved in anaerobic energy production are taxed. This sequence should be repeated four to five times.

If it is necessary for the movements to continue for periods longer than one minute, greater and greater emphasis is put on the aerobic energy supply. Thus, the training should aim to affect the mitochondria. Sharp, (1980b) suggests training where the subject works for 30 seconds, rest 30 seconds, for a period of 10-15 minutes. This type of training would have an effect on the mitochondria, myoglobin and capillaries in the muscle. Thus, it is necessary to establish which energy system predominates in a particular activity. However, in many instances certain activities may require energy from two or three energy sources.

Few studies have employed sit-ups to evaluate the effectiveness of a training programme on abdominal endurance. Furthermore, the quantification of the amount of work placed on the abdominals is difficult as the description of the training programmes have not been reported in great detail.

A two minute sit-up test was used by Capen, (1950) to monitor changes in LME in a weight training group and a "conditioning group". The conditioning group followed a varied training programme which consisted of running, conditioning gymnastics and hand combats. The training programme lasted 11 weeks. The conditioning group showed a highly significant increase in sit-ups from 29 to 47 repetitions. Marcinik et al; (1985) examined the effect of three different programmes (aerobic, calisthenics and aerobic circuit training at two different work intensities) on various fitness parameters. During the 10 week study the males performed three sessions per week as well as participating in three 40 minute runs per week. Bent knee sit-ups for 90 seconds were used to measure muscular endurance in the trunk. All three groups had virtually the same adjusted mean score of 42 repetitions at test 1. All three groups showed a significant increase from the initial mean value. The increase of three repetitions represents a percentage

increase of 8% for each of the three groups.

2.6.6 Norm Tables

The Canadian Standardised Test of Fitness, (1986) has produced norm tables which give guidance for the general population. Similarly, Pollock et al; (1984) have published norm tables provided by the Health Improvement Program, National Athletic Health Institute in the United States. These tables give norms for sit-ups performed for one minute (see Table 9).

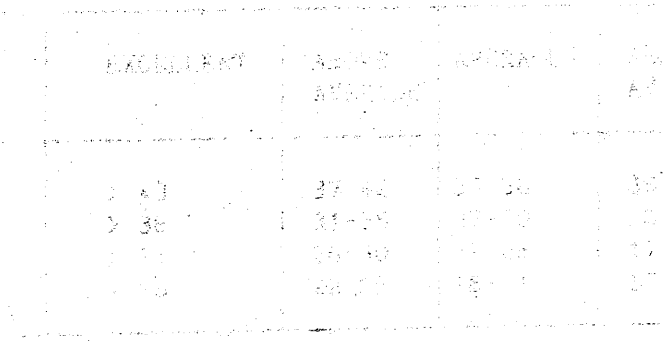

TABLE 9.

SIT-UP NORMS

Muscular Endurance Test Standards Repetitions
(Sit-Ups in 60 seconds) for males.

Pollock et al; 1984.

AGE	EXCELLENT	GOOD	AVERAGE	FAIR	POOR
20-29	> 48	43-47	37-42	33-36	0-32
30-30	> 46	35-39	29-34	25-28	0-24
40-49	> 35	30-34	24-29	20-23	0-19
50-59	> 25	25-29	19-24	15-18	0-14

Canadian Standardised Test of Fitness 1986.

AGE	EXCELLENT	ABOVE AVERAGE	AVERAGE	BELOW AVERAGE	POOR
20-29	> 43	37-42	33-36	39-32	<28
30-39	> 36	31-35	27-30	22-26	<21
40-49	> 31	26-30	22-25	17-21	<16
50-59	> 26	22-25	18-21	13-17	<12

CHAPTER 3

SECTION ONE

METHOD

3.1 Subjects, Design, Measurements, and Equipment

3.1.1 Subjects

All sections of the University community, students, academic staff and technical staff participate in training programmes offered by the Department of Physical Education and Sports Science. Therefore, it was considered appropriate that volunteers for this study should be taken from each sub group within the University. Thus, it was accepted that a wide age range would be involved. In an attempt to ascertain if any seasonal variation or testing effect influenced the results, a control group consisting of volunteers from the University community was included in the study. Control subjects were matched for age and weight as much as possible with the exercisers.

3.1.2 Study Design

An examination of the possible changes in the variables which were to be measured was made in an attempt to

determine the numbers necessary for an exercise group and a control group. Previous work (Grant et al; 1988) has shown that a 10 week aerobic training programme produced a significant fall in body fat and a significant increase in aerobic fitness with 20 exercisers. There were no significant changes in the control group which numbered 10. The great variability in the blood pressure changes resulted in a non significant decrease in blood pressure. While it is clearly desirable to have as large a study population as possible, the resources of the present study were limited. It was felt that 25 exercisers and 20 controls would allow for meaningful analysis to be performed. Advertisements were placed throughout the University asking for volunteers to undertake a 10 week training programme. Volunteers were assigned to the exercise and control groups. All subjects had to be sedentary and male. It has been shown that subjects who have a previous exercise background do not display the same magnitude of physiological change to training compared to previously sedentary individuals (Pollock, 1973). Therefore, it was essential that only sedentary individuals were recruited. Subjects were given an activity questionaire to ensure that only previously inactive subjects entered the study. The questionaire is a modified version of the population questionaire designed by the University of Toronto Department of Physical Education and Recreation. The physical activity questionaire which included a medical and

smoking history and alcohol consumption was given to each subject. Prospective subjects who were found to have recently undertaken strenuous exercise were excluded from the study. Exercisers and controls were asked to maintain their present lifestyle and eating habits, tobacco usage and alcohol consumption throughout the study. All subjects were given an outline of the study so that they knew what was involved and were asked to sign a consent form (Appendix A).

The subjects in the exercise group agreed not to take part in any other "significant" exercise during the 10 week period of the project. The "controls" agreed that they would not embark on any extra form of exercise during the 10 week period.

3.1.3 Pilot Study

Testing was conducted in the testing room of the Physical Education and Sports Science Department. A pilot study was carried out to assess the suitability of the proposed test battery. The results from the pilot study showed that the tests were reliable (See Appendix B for details of the pilot study).

3.1.4 Equiupment

The main factor in the choice of tests was the

availability of appropriate apparatus. A list of the equipment used in the study is given in Appendix D.

3.1.5 Order of Testing

All subjects reported to the testing area in shorts, vest, socks and gym shoes. All subjects were tested individually. The name, age and date of birth were recorded. The order of subsequent testing was:

1. Blood pressure and resting heart rate
2. Weight
3. Height
4. Estimated body fat
5. Estimated oxygen uptake
6. Sit ups
7. Sit and Reach Test
8. Blood cholesterol (on a separate day)

An attempt was made to control as many extraneous factors as possible. It was not possible to control humidity in the testing area but the room temperature was maintained between 18 and 22 degrees centigrade which is recommended by the American College of Sports Medicine, 1986 (A.C.S.M; 1986). Sub-maximal variables can be affected by circadian rhythms. Reilly et al; (1984) have shown that sub-maximum Vo2, and heart rate can vary with the time of testing. Accordingly, sub-

jects were tested within 2 hours of the previous test.

SECTION TWO

METHODOLOGY EMPLOYED

3.2.1 Subject Preparation

Tests must be highly reproducable if they are to be of value. Elimination of variations in the condition of the subject will help increase the reproducability of the tests. In accordance with the recommendations of the ACSM; (1986) subjects were asked to refrain from caffeine and tobacco for 3 hours before the test. They were asked to avoid alcohol for the 24 hour period before testing. Each subject was asked to verify that he had complied with the above requests.

3.2.2 Blood Pressure

Blood pressure measurements were taken using a Copal Digital sphygmomanometer (see Appendix D). The subjects sat in a chair for 5 minutes. Thereafter, measurements were taken at 5, 7, and 9 minutes. The mean of the three readings was recorded (American Heart Association, 1980). During all measurements the sphygmomanometer cuff was placed level with the subject's heart and the cuff was wrapped around the

subjects' upper arm. All measurements were made on the left arm. The brachial artery was located by palpating the arm approximately 2 inches above the olecranon process. The cuff was placed evenly over the arm with the microphone mark to the inside of the arm. The microphone mark was adjusted so that the microphone mark lay over the pulse point. The elbow of the subject rested on a table so that the microphone mark was at the same height as the heart. The arm was relaxed with the palm bent gently inward. The cuff was inflated by pressing the pressurisation button. The blood pressure reading was given by a digital display. The cuff was completely deflated before further measurements were taken. Sitting heart rates were monitored during blood pressure measurement by the Copal monitor. The mean of the three heart rates was recorded.

3.2.3 Body Composition

The subject's weight was taken on scales and height measured. Body composition was estimated using the method described by Durnin and Womersley, (1974). Skinfold calipers (see Appendix D) were used to measure skinfold on the triceps, biceps, subscapularis and supra-iliac sites. The Durnin and Womersley equations particularly commended themselves because the subjects from whom they were derived were selected from a Glasgow population. This factor would help reduce

3.2.3(a) Body Composition - Method and Equipment

All measurements were taken on the right side of the body. A Holtain caliper was used (See Appendix D). The sum of the 4 measurements was applied to the Durnin and Womersley, (1974) formula to predict percentage body fat. A reliability study on skinfold measurement was conducted and the results are in Appendix B. Skinfold measurements were taken at the sites outlined below. The thumb and forefinger were used to lift the skinfold before the caliper jaws "caught" the fold approximately 1 centimetre below the forefinger and the thumb. Two seconds elapsed before a reading was taken to the nearest 0.2 centimetre. Three measurements were taken on each site and the average of the three scores was recorded. The subject stood with equal weight on each foot throughout the test. The triceps measurement was taken midway between the acromion and olecranon processes with the arm hanging loosely at the subject's side. The biceps measurement was made with the arm supinated and the site was in the middle of the muscle belly. At the subscapular site, the skinfold was picked up below the inferior angle of the scapula, at an angle of 45 degrees to the vertical. The fingers touched the bone to standardise the location of the site. At the suprailiac site the skinfold was grasped just above the iliac crest on the mid axillary line.

The skinfold was vertical except on a few occasions where the subjects were particularly fat in this area.

3.2.4 Aerobic Test Protocol

Equipment for a direct measurement of Vo2 Max was not available. Therefore, an indirect method had to be chosen. As a Monark bicycle ergometer and heart rate recording equipment (See Appendix D) were available, it was decided to use an Astrand Ryhming nomogram to estimate Vo2 Max. A bicycle ergometer would enable a comparison to be made between standardised sub-maximal work loads used in other studies as well as allowing a prediction of Vo2 Max to be made.

3.2.4(a) Aerobic Test - Method and Equipment

The saddle height was adjusted so that when the pedal was 'down' the subject's leg was almost fully extended. The electrodes were attached and it was established that there was a stable heart rate reading. A short introductory period was given to familiarise the subjects with the equipment. A common work load of 60 watts was given for 3 minutes for the following reasons.

1) This period provided a warm-up.
2) The experimenter was able to check that the subject could maintain the cadence. Cadence was

maintained/monitored by a digital display on the bicycle and by an audible metronome produced by a taperecorder. Thereafter, the work load was increased depending on the response to work load during the warm-up. An attempt was made to elicit heart rates between 75%-85% of age predicted maximum heart rate.

After 3 minutes the heart rates from the "warm -up " work load would provide guidance for the second work load which would in turn provide information for the selection of the third and final work load. In some instances the second work load produced an appropriately high heart rate response so that the duration of the second workload was extended from 3 minutes to 5 minutes and the test was terminated. Subjects who only completed two work loads were asked to return on another day to have an other bicycle test with three work loads. The third work load was maintained for 5 minutes. It was recognised that marked improvements in fitness would result in a decrease in sub-maximal heart rate and that the final workload used in test 1 may not elicit an appropriately high heart rate. It was decided to replicate the protocol used in test 1 and if necessary to conduct a fourth work load of 5 minutes duration after a 5 minute recovery period which included a 3 minute "cool down " at 50 watts in an attempt to accelerate the removal of lactate (Brooks et al; 1984). The results from the

pilot study showed that heart rates had dropped to 60% of maximum after the 5 minute recovery period. Thus, it was felt that this recovery period was sufficient and the subsequent fourth work load would not be influenced by fatigue from an earlier work load.

The steady state heart rate for the first two work loads was determined by calculating the mean heart rate for 2:00 2:30 and 3:00 minutes. The third and fourth work load steady state heart rates were calculated by taking the mean of the heart rates recorded at 4:00, 4:30 and 5:00 minutes. The steady state heart rate at the fourth work load was used for most of the exercise group at test 2 to predict Vo2 Max. The heart rate value was "fed in " to a calculator to predict Vo2 Max using the method outlined by Astrand et al; (1954).

3.2.5 Sit-ups

Fitness sessions at the University involve exercises to promote local muscular endurance and flexibility in an attempt to reduce the possibility of back pain /injury. Therefore, it was necessary to devise a means of measuring the effects on these variables. A modified version of the sit-up test outlined by the Canadian Standardised Test of Fitness, (1986) was used. The subject lay on his back and the knees were bent and the thumb and forefinger grasped the ear lobes. The elbows were outstretched directly in front of the head. The

degree of knee flexion was assessed by placing the subject's foot as close to the buttock as possible and marking the extremity of the big toe. The heel was then placed at the "big toe" mark. The subject was asked to "sit-up" and touch his knees with his elbows and return the shoulders to the mat. Practice was given until the subject could perform the proper movement. The subject was asked to complete as many sit-ups in one minute as possible. The experimenter held the subject's ankles and counted the score as the subject proceeded. The total number of sit-ups in one minute was recorded.

3.2.6 Flexibility

The only apparatus available to measure flexibility was the Sit and Reach apparatus (See Appendix D). However, it was felt that this test would provide a measure of flexibilty in the back of the legs (hamstrings) and the lower back (Wells and Dillon, 1952). The subject performed the test in bare feet. The subject sat with legs extended, big toes touching, and the hands slid as far forward as possible on a scale which is marked in centimetres on top of the apparatus with the 30 centimetre line directly over the feet, which are placed flat against a cross board. The score was determined by the distance reached by the fingers. Thus, a subject who reaches directly above the feet is given a score of 30, while the subject who reaches 10

centimeters behind the feet is given a score of 20. Each subject was given three practice trials so that the subjects could become familiar with the apparatus and the procedure of the test, and three trials, the best of which was recorded.

3.2.7 Plasma Lipids

It was decided that total cholesterol, HDL and triglycerides should be measured. LDL was not measured as doing so invloves more lengthy procedures. All subjects were asked to report 12 hours fasted to provide a blood sample so that an assessment of total cholesterol HDL and tryglycerides could be made. Sampling took place before training commenced and 4 days after the final training session in the study. The subject sat for five minutes before ten ml of blood was drawn from the anti cubital vein by qualified personnel. Some of the test 1 blood sample was stored at the appropriate temperature and analysis was carried out 10 weeks later using the same reagent as test 2. Analysis was also carried out on the test 1 samples immediately after test 1.

All analysis was carried out by senior biochemists at the Western Infirmary, Glasgow. Serum total cholesterol and triglyceride concentrations were measured on a Cobas-Bio centrifugal analyser, Roche Diagnostics, (see Appendix D) employing enzymatic methods - that is, with cholesterol oxidase and aminoantipyrine for cholesterol and glycerol kinase and pyruvate for triglyceride - Merckotest reagents being used in each case (see Appendix D). High density lipoprotein cholesterol was separated by using Biomerieux precipitating reagents and the cholesterol concentration estimated as above. Appendix C gives the results of the precision checks carried out at the Biochemistry Department, Western Infirmary, Glasgow.

3.2.8 Training

Each subject was given a Department of Physical Education and Recreation booklet, "An Introduction to Fitness Programmes at Glasgow University", to provide information on fitness and training as well as a timetable outlining the times when Tune-ups took place throughout the week. A 60% and 75% HRR was calculated using the resting heart rate and predicted maximum heart rate for each subject to ensure that every subject was given a training heart rate which was above the minimum threshold for the improvement of Vo2 Max. A ceiling of 75% HRR was established to prevent subjects training at high intensity this has been shown

to produce more injuries and can be psychologically very demanding (Pollock et al; 1984). Training was conducted three times per week in groups in the organised "Tune-up sessions" which consisted of 20 minutes of aerobic activity, 5 minutes of local muscular endurance and 5 minutes of flexibility exercises. (Appendix F gives examples of the Tune-ups used in this study). All group sessions were supervised by the physical education staff of the Department of Physical Education and Sports Science. A record of physical activity was kept by both the exercise and control groups in a training diary. Training heart rates were counted by monitoring the radial pulse for 15 seconds during or at the conclusion of the aerobic training period. The count was started as soon as the pulse could be found. The heart rate for the 15 second period was multiplied by 4 to give a figure in beats per minute. Heart rate recorders (Sport Tester PE 3000) with a memory mode (see Appendix D) were used to monitor heart rates throughout the training session. All subjects made use of the heart rate monitors at some time during the 10 week period. Appendix E gives an example of the training diary which each subject had to complete each week.

3.2.9 Tobacco Usage and Alcohol Consumption

Subjects recorded tobacco usage and alcohol consumption in the diaries provided.

3.2.10 Diet

An assessment of diet was undertaken for a five day period in the first 2 weeks of the study and the last 2 weeks of the study. Each 5 day period included a weekend and started on a Friday and finished on a Tuesday evening. During these periods all subjects were asked to record all the food and drink consumed within the 5 day period. The subjects recorded all dietary information in a diary published by Balance Your Diet, Cambridge University Press. The diaries of the food intakes were coded using code numbers from the Balance Your Diet, Cambridge University Press Handbook. This programme was limited to the foods in the handbook and in some instances no recording could be made. Appropriate analysis was carried out using the Balance your Diet, Cambridge University Press Handbook and Computer Programme to determine if there had been any significant change in energy intake or the composition of the food. The results of test 1 and test 2 were brought together and inserted into the mainframe computer at Glasgow University.

CHAPTER FOUR

RESULTS

4.1.1. Statistical Treatment of Data

All mean scores and statistical tests were carried out by the computer at Glasgow University.

The analysis investigated which of the variables showed a significant average difference between Test 1 and Test 2 for the 21 exercisers and 22 controls separately and between each group.

Paired t-tests were used to assess if a particular group (exercise or control) had shown an average improvement from Test 1 to Test 2.

Two sample t-tests were used to determine if the exercisers demonstrated a greater average improvement than the controls.

The 95% Confidence Intervals indicate the range of average improvement.

This analysis answered the following:-

1) Does the exercise group show an average improvement from Test 1 to Test 2?

2) Does the control group show an average improvement from Test 1 to Test 2?

3) Does the exercise group show a greater average improvement than the control group?

TABLE 10

AGE (YEARS)

Mean age and standard deviation of exercise and control groups.

	MEAN	S. D.
Exercise Group	36. 95	10. 24 (Range 21-58)
Control Group	38. 64	7. 85 (Range 17-54)

TABLE 11.

ADHERENCE TO STUDY.

	NUMBER	MEAN NUMBER OF TUNE-UP SESSIONS ATTENDED	S.D.	RANGE
Exercisers	21	26.67	± 2.93	21-31

Five subjects failed to complete a minimum of 20 Tune-ups and were eliminated from the analysis.

TABLE 12

WEIGHT (Kilogrammes)

Mean body weight, median, standard deviation, for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	77.60	73.70	11.42
	Test 2	76.66	72.40	10.29
Control Group (n=22)	Test 1	74.95	73.95	8.90
	Test 2	74.90	74.35	8.76

TABLE 13

WEIGHT (Kilogrammes)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.94	2.26	1.91	0.07	(+0.09, -1.98)
Control Group	-0.05	0.784	0.30	0.77	(+0.30, -0.40)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.94	1.72	0.09	(+0.18, -1.97)
Control Group	-0.05			

The exercise group showed a fairly large non significant average decrease in body weight. There was a very small non significant average decrease in the body weight of the control group.

TABLE 14

RESTING HEART RATE (beats/minute)

Mean resting heart rate, median and standard deviation for the exercise group and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	74.71	74.00	8.47
	Test 2	63.10	63.00	7.02
Control Group (n=22)	Test 1	71.14	71.00	7.85
	Test 2	67.09	66.00	9.26

TABLE 15

RESTING HEART RATE (beats/minute)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-11.62	8.56	6.22	0.0000	(-7.72, -15.52)
Control Group	-4.05	8.32	2.28	0.033	(-0.35, - 7.74)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-11.62	2.94	0.0054	(-2.4 , -12.8)
Control Group	- 4.05			

The exercise group and the control group demonstrated a significant average decrease in resting heart rate as shown by a paired t-test. A two sample t-test and confidence intervals showed that the resting heart rate showed a greater average decrease for the exercise group over the control group from Test 1 to Test 2.

TABLE 16

SYSTOLIC BLOOD PRESSURE (mmHg)

Mean systolic blood pressure, median, standard deviation, for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	129.76	128.00	14.68
	Test 2	131.14	127.00	14.20
Control Group (n=22)	Test 1	125.91	124.00	11.24
	Test 2	126.23	126.00	10.24

TABLE 17

SYSTOLIC BLOOD PRESSURE (mm. Hg)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	1.38	8.87	-9.70	0.49	(+5.52, -2.76)
Control Group	0.32	9.38	-0.16	0.88	(+4.48, -3.84)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference		t	P Value	95% Confidence Interval
Exercise Group	1.38	0.3	0.30	-0.71	(+6.8, -4.6)
Control Group	0.32				

The systolic blood pressure in the exercise and control group showed a very slight non-significant average increase from Test 1 to Test 2.

TABLE 18

DIASTOLIC BLOOD PRESSURE (mm. Hg)

Mean diastolic blood pressure, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S. D.
Exercise Group (n=21)	Test 1	85.29	84.00	8.10
	Test 2	85.33	85.00	11.74
Control Group (n=22)	Test 1	80.95	80.00	8.28
	Test 2	82.14	82.00	7.82

TABLE 19

DIASTOLIC BLOOD PRESSURE (mm. Hg)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	0.05	8.36	-0.03	0.98	(+3.95, -3.85)
Control Group	1.18	4.17	-1.30	0.21	(+3.08, -0.71)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	0.05	-0.55	0.59	(+3.1, -5.4)
Control Group	1.18			

The diastolic blood pressure in the exercise and control group showed a very slight and non significant average increase from Test 1 to Test 2.

TABLE 20

BICEP SKINFOLD (millimetres)

Mean bicep skinfold, median, standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	5.69	5.80	1.67
	Test 2	5.47	5.50	1.51
Control Group (n=22)	Test 1	5.47	5.30	1.65
	Test 2	5.41	5.25	1.65

TABLE 21

BICEP SKINFOLD (millimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.20	0.69	1.31	0.20	(+0.12, -0.52)
Control Group	-0.06	0.55	0.49	0.63	(+0.19, -0.31)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.20	0.72	0.47	(+0.25, -0.53)
Control Group	-0.06			

The bicep skinfold in the exercise and control group showed a very slight but non significant average decrease.

TABLE 22

TRICEP SKINFOLD (millimetres)

The mean tricep skinfold, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	12.35	11.10	3.98
	Test 2	12.12	11.10	3.94
Control Group (n=22)	Test 1	12.12	12.50	4.15
	Test 2	11.48	11.40	3.47

TABLE 23

TRICEP SKINFOLD (millimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.23	1.31	0.78	0.44	(+0.39, -0.84)
Control Group	-0.64	1.59	1.85	0.08	(+0.08, -1.36)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.23	0.91	0.37	(+1.33, -0.51)
Control Group	-0.64			

There were no significant changes in the tricep skinfold of the exercise or control group from Test 1 to Test 2.

TABLE 24

SUBSCAPULAR SKINFOLD (millimetres)

Mean subscapular skinfold, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	13.81	14.00	3.00
	Test 2	13.33	13.10	3.20
Control Group (n=22)	Test 1	14.16	13.40	4.49
	Test 2	13.74	13.40	4.56

TABLE 25

SUBSCAPULAR SKINFOLD (millimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.49	1.34	1.63	0.12	(+0.14, -1.11)
Control Group	-0.42	0.93	2.11	0.047	(-0.01, -0.83)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.49	0.19	0.85	(+0.66, -0.80)
Control Group	-0.42			

The control group showed a significant average decrease in the subscapular skinfold from Test 1 to Test 2 as shown by a paired t-test. There was no significant difference in the exercise group subscapular skinfold from Test 1 to Test 2. A two sample t-test showed that there were no significant differences in the changes in the exercise group and control group from Test 1 to Test 2.

TABLE 26

SUPRA-ILIAC SKINFOLD (millimetres)

Mean supra-iliac skinfold, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	19.42	18.20	8.94
	Test 2	18.60	16.80	8.16
Control Group (n=22)	Test 1	17.65	17.55	6.26
	Test 2	17.45	17.85	7.26

TABLE 27

SUPRAILIAC SKINFOLD (millimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.83	3.74	0.99	0.33	(+0.92, -2.57)
Control Group	-0.21	2.55	0.37	0.72	(+0.95, -1.36)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.83	0.62	0.54	(+1.42, -2.66)
Control Group	-0.21			

There were no significant changes in the suprailiac skinfold of the exercise or control group from Test 1 to Test 2.

TABLE 28

TOTAL SKINFOLD (millimetres)

Mean total skinfold, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	51.37	50.20	15.93
	Test 2	49.52	46.30	15.46
Control Group (n=22)	Test 1	49.44	48.45	15.21
	Test 2	48.50	47.45	15.85

TABLE 29

TOTAL SKINFOLD (millimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-1.85	5.32	1.55	0.14	(+0.64, -4.33)
Control Group	-0.94	2.76	1.55	0.14	(+0.32, -2.19)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-1.85	0.68	0.50	(+1.80, -3.64)
Control Group	-0.94			

The total skinfold in the exercise and control group showed a non-significant average decrease from Test 1 to Test 2.

TABLE 30

PERCENTAGE BODY FAT

Mean percentage body fat, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group	Test 1	22.17	22.00	5.02
	Test 2	21.57	22.00	4.97
Control Group	Test 1	22.40	22.60	5.18
	Test 2	22.21	22.55	5.35

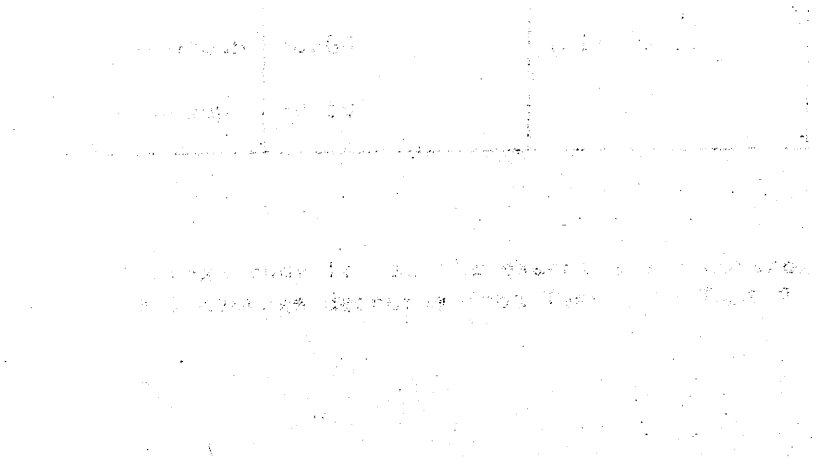

TABLE 31

PERCENTAGE BODY FAT

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	0.60	1.44	1.91	0.07	(+0.06, -1.26)
Control Group	0.19	0.70	1.27	0.22	(+0.12, -0.50)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.60	1.17	0.25	(+0.31, -1.12)
Control Group	-0.19			

The percentage body fat in the exercise and control group showed a non significant average decrease from Test 1 to Test 2.

TABLE 32

STEADY STATE HEART RATE BETWEEN TWO AND THREE MINUTES IN THE FIRST WORK LOAD (beats/minute).

Mean heart rate, median and standard deviation for the exercise and control group at Test 1 and Test 2.

	MEAN	MEDIAN	S.D.
Exercise Group (n=21)	104.90 93.05	102.00 93.00	12.34 10.35
Control Group (n=22)	104.95 101.15	105.00 100.50	12.23 10.16

TABLE 33

STEADY STATE HEART RATE BETWEEN TWO AND THREE MINUTES IN THE FIRST WORK LOAD. (beats/minute).

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-11.86	7.39	7.35	0.0000	(-8.49, -15.22)
Control Group	- 3.90	10.06	1.73	0. 099	(+0.81, - 8.61)

Two Sample t-test of Differences and 95% Confidence Interval of Average Improvement (Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-11.86	2.87	0.0069	(-2.3, -13.6)
Control Group	- 3.90			

The exercise group demonstrated a significant average decrease in heart rate from Test 1 to Test 2 as shown by a paired t-test. There was no significant decrease in the control group heart rate from Test 1 to Test 2. A two sample t-test and confidence intervals demonstrated that the heart rate showed a greater average decrease for the exercise group over the control group from Test 1 to Test 2.

TABLE 34

STEADY STATE HEART RATE BETWEEN TWO AND THREE MINUTES IN THE SECOND WORK LOAD (beats/minute).

Mean heart rate, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	124.47	123.00	9.76
	Test 2	112.65	112.00	7.29
Control Group (n=16)	Test 1	124.00	122.50	8.57
	Test 2	117.93	119.00	8.40

TABLE 35

STEADY STATE HEART RATE BETWEEN TWO AND THREE MINUTES IN THE SECOND WORK LOAD (beats/minute).

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-11.82	7.45	6.35	0.0000	(-7.87, -15.77)
Control Group	- 6.07	5.61	3.90	0.0018	(-2.71, - 9.43)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-11.82	2.37	0.025	(-0.8, -10.7)
Control Group	- 6.07			

The exercise and control groups demonstrated a significant average decrease in heart rate from Test 1 to Test 2 as shown by a paired t-test. A two sample t-test and confidence intervals demonstrated that the heart rate showed a greater average decrease for the exercise group over the control group from Test 1 to Test 2.

TABLE 36

STEADY STATE HEART RATE BETWEEN FOUR AND FIVE MINUTES IN THE FINAL WORK LOAD (Beats/minute)

Mean heart rate, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	148.48	149.00	10.22
	Test 2	131.76	131.00	8.02
Control Group (n=20)	Test 1	147.81	148.00	9.08
	Test 2	143.10	144.00	11.48

TABLE 37

STEADY STATE HEART RATE BETWEEN FOUR AND FIVE MINUTES IN THE FINAL WORK LOAD (beats/minute).

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-16.71	7.02	10.91	0.0000	(-13.52, -19.91)
Control Group	- 4.70	6.16	3.41	0.0029	(- 1.82, - 7.58)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-16.71	5.83	0.0000	(-7.8, -16.2)
Control Group	- 4.70			

The exercise and control groups demonstrated a significant average decrease in heart rate from Test 1 to Test 2 as shown by a pairfed t-test. A two sample t-test and confidence intervals demonstrated that the heart rate showed a greater average decrease for the exercise group over the control group from Test 1 to Test 2.

TABLE 38

MEAN INCREASE IN HEART RATE (beats/minute) FROM FOUR TO FIVE MINUTES DURING THIRD WORK LOAD.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	2.29	1.00	2.05
	Test 2	1.19	1.00	1.47
Control Group (n=20)	Test 1	1.90	2.00	1.67
	Test 2	2.05	2.00	1.83

TABLE 39

ESTIMATED VO_2 MAX LITRES MINUTE^{-1}

Mean estimated VO_2 Max, median, and standard deviation at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	2.55	2.46	0.52
	Test 2	3.02	2.95	0.54
Control Group (n=20)	Test 1	2.45	2.40	0.46
	Test 2	2.52	2.54	0.47

TABLE 40

ESTIMATED VO_2 MAX LITRES MINUTE^{-1}

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+0.47	0.20	-9.64	0.0000	(+0.38, +0.56)
Control Group	+0.13	0.20	-3.68	0.0016	(+0.03, +0.23)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+0.47	5.38	0.0000	(+0.22, +0.48)
Control Group	+0.13			

The exercise group and the control group demonstrated a significant average increase as shown by a paried t-test. A two sample t-test and confidence intervals demonstrated that the estimated VO_2 Max showed a greater average increase for the exercise group over the control group from Test 1 to Test 2.

TABLE 41

ESTIMATED VO_2 MAX ($ml.kg^{-1}.min^{-1}$)

Mean estimated VO_2 Max, median and standard deviation at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	32.97	33.30	5.70
	Test 2	39.55	39.40	6.62
Control Group (n=20)	Test 1	32.12	31.60	5.90
	Test 2	33.62	32.40	6.12

TABLE 42

ESTIMATED VO_2 MAX ($ml.kg^{-1}min^{-1}$)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+6.58	2.68	-9.29	0.0000	(+5.36, +7.80)
Control Group	+1.65	2.66	-4.06	0.0007	(+0.37, +2.93)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+6.58	5.84	0.0000	(+3.22, +6.64)
Control Group	+1.65			

The exercise group and the control group demonstrated a significant average increase as shown by a paired t-test. A two sample t-test and confidence intervals demonstrated that the estimated VO_2 Max showed a greater average increase for the exercise group over the control group from Test 1 to Test 2.

TABLE 43

FLEXIBILITY (Centimetres)

Mean flexibility, median and standard deviation for the exercise and control groups at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=20)	Test 1	28.00	31.00	9.07
	Test 2	34.10	36.00	7.95
Control Group (n=20)	Test 1	28.20	28.50	6.84
	Test 2	28.45	29.50	6.79

TABLE 44

FLEXIBILITY (Centimetres)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+5.35	2.30	-7.99	0.0000	(+3.95, +6.75)
Control Group	+0.25	0.58	-0.43	0.67	(+1.47, -0.97)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+5.35	5.74	0.0000	(+3.30, +6.90)
Control Group	+0.25			

The exercise group demonstrated a significant average increase in flexibility from Test 1 to Test 2 as shown by a paired t-test. There was no significant increase in the control group flexibility score from Test 1 to Test 2. A two sample t-test and confidence intervals demonstrated that the flexibility score showed a greater average increase for the exercise group over the control group from Test 1 to Test 2.

TABLE 45

SIT-UPS (REPETITIONS)

Mean sit-ups, median and standard deviation at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=21)	Test 1	27.57	28.00	6.29
	Test 2	34.43	34.00	4.92
Control Group (n=21)	Test 1	28.05	27.00	7.35
	Test 2	29.86	30.00	8.37

TABLE 46

SIT-UPS (Repetitions)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+6.86	3.64	-8.64	0.0000	(+5.20, +8.51)
Control Group	+1.81	2.36	-3.52	0.0022	(+0.74, +2.88)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+6.86	5.34	0.0000	(+3.12, +6.97)
Control Group	+1.81			

The exercise group and control group demonstrated a significant average increase in sit ups as shown by a paired t-test. A two sample t-test and confidence intervals demonstrated that the sit-up score showed a greater average increase for the exercise group over the control group from Test 1 to Test 2.

TABLE 47

TOTAL CHOLESTEROL (mmol/litre)

Mean total cholesterol, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=19)	Test 1	6.50	7.00	1.22
	Test 2	5.60	5.40	1.09
Control Group (n=20)	Test 1	6.21	6.25	1.20
	Test 2	5.47	5.50	0.84

TABLE 48

TOTAL CHOLESTEROL (mmol/litre)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-0.90	0.76	5.20	0.0000	(-0.539, -1.271)
Control Group	-0.74	0.59	5.53	0.0000	(-0.457, -1.013)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.91	0.78	0.44	(+0.27, -0.62)
Control Group	-0.74			

The exercise group and the control group demonstrated a significant average decrease in total cholesterol as shown by a paired t-test. A two sample t-test and confidence intervals showed that there was no significant difference in the changes from Test 1 to Test 2 between the exercise and control groups.

TABLE 49

HIGH DENSITY LIPOPROTEIN (mmol/litre)

Mean high density lipoprotein, median and standard deviation at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=19)	Test 1	1.12	1.05	0.22
	Test 2	1.29	1.25	0.24
Control Group (n=19)	Test 1	1.16	1.20	0.20
	Test 2	1.26	1.25	0.27

TABLE 50

HIGH DENSITY LIPOPROTEIN

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+0.17	0.18	-4.23	0.0005	(+0.86, +0.26)
Control Group	+0.13	0.16	-3.56	0.0022	(+0.05, +0.21)

Two Sample t-test of Differences and 95% Confidence Interval of Average Improvement (Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+0.17	-0.73	0.47	(+0.15, -0.07)
Control Group	+0.13			

The exercise group and the control group demonstrated a significant average increase in high density lipoprotein as shown by a paired t-test. A two sample t-test and confidence intervals showed that there was no significant difference in the changes from Test 1 to Test 2 between the exercise and control groups.

TABLE 51

TOTAL CHOLESTEROL/HIGH DENSITY LIPOPROTEIN RATIO

Mean cholesterol intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=19)	Test 1	6.00	6.20	1.54
	Test 2	4.51	4.25	1.40
Control Group (n=19)	Test 1	5.58	5.85	1.89
	Test 2	4.57	4.35	1.43

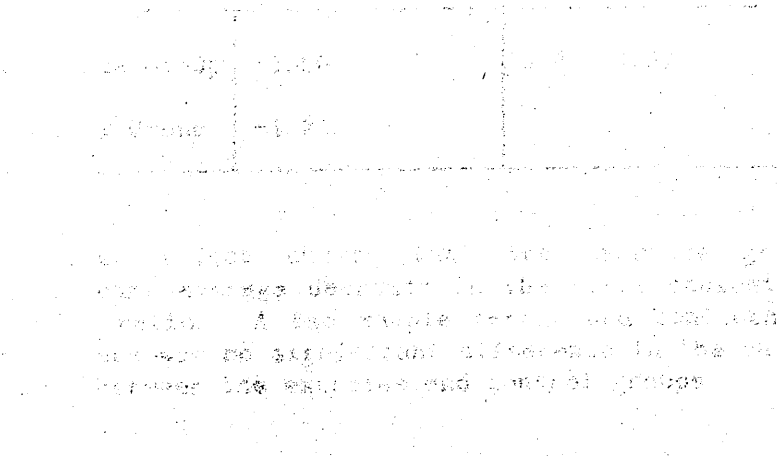

TABLE 52

TOTAL CHOLESTEROL - HIGH DENSITY LIPOPROTEIN RATIO.

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	-1.50	0.95	3.14	0.01	(0.53, 2.47)
Control Group	-1.21	1.01	1.89	0.07	(-0.07, 2.09)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-1.50	0.91	0.37	(-0.36, 0.94)
Control Group	-1.21			

A paired t-test showed that the exercise group demonstrated a significant average decrease in the total cholesterol - high density protein ratio. A two sample t-test and confidence intervals showed that there was no significant difference in the changes from Test 1 to Test 2 between the exercise and control groups.

TABLE 53

PLASMA TRIGLYCERIDES (mmol/litre)

Mean plasma triglycerides, median and standard deviation for the exercise group and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=19)	Test 1	1.38	1.40	0.53
	Test 2	1.36	1.20	0.54
Control Group (n=20)	Test 1	1.34	1.25	0.52
	Test 2	1.42	1.20	0.70

TABLE 54

PLASMA TRIGLYCERIDES (mmol/litre)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+0.02	0.54	0.17	0.87	(+0.24, -0.28)
Control Group	+0.09	0.50	0.76	0.46	(+0.31, -0.15)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	-0.02	0.63	0.53	(+0.23, -0.45)
Control Group	+0.09			

There were no significant differences for the exercise or control group from Test 1 to Test 2.

TABLE 55

CURRENT SMOKING HABITS OF EXERCISE GROUP AND CONTROL GROUP AT TEST 1 AND TEST 2.

	Smokers	Non Smokers
Exercise Group (n=21)	2 (9.5%)	19 (90.5%)
Control Group (n=22)	6 (27.3%)	16 (72.7%)

TABLE 56

FIVE DAY ENERGY INTAKE (KCALS)

Mean energy intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	10550	10389	2509
	Test 2	11821	12289	3496
Control Group (n=8)	Test 1	11074	11196	1755
	Test 2	10970	11647	1677

TABLE 57

FIVE DAY ENERGY INTAKE (Kcals)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+1271	2516	-1.18	0.25	(-920, +3463)
Control Group	- 103	2139	0.11	0.20	(-2971, +2071)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+1271	1.33	0.20	(-761, +3510)
Control Group	- 103			

There was no significant change in the energy intake of the exercise or control group from Test 1 to Test 2.

TABLE 58

FIVE DAY ALCOHOL INTAKE (KCALS)

Mean alcohol intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	3633	2205	3896
	Test 2	4081	3845	3468
Control Group (n=8)	Test 1	2365	1191	3155
	Test 2	2598	2097	2687

TABLE 59

FIVE DAY ALCOHOL INTAKE (Kcals)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+449	2683	-0.34	0.73	(-2208, +3106)
Control Group	+232	1624	-0.15	0.88	(-3127, +3592)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+449	-0.21	0.84	(-1933, +4331)
Control Group	+232			

There was no significant change in the alcohol intake of the exercise or the control group from Test 1 to Test 2.

TABLE 60

FIVE DAY FAT INTAKE (GRAMS)

Mean fat intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	385.2	405.0	142.9
	Test 2	439.1	449.7	204.4
Control Group (n=8)	Test 1	417.3	468.0	129.5
	Test 2	383.6	389.5	111.5

TABLE 61

FIVE DAY FAT INTAKE (Grams)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+53.9	136.6	-0.87	0.39	(-73, +181)
Control Group	-33.7	112.6	-0.52	0.61	(-172, +105)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+53.9	-1.58	0.13	(-27, +209)
Control Group	-33.7			

There was no significant change in the fat intake of the exercise or the control group from Test 1 to Test 2.

TABLE 62

FIVE DAY CARBOHYDRATE INTAKE (GRAMS)

Mean carbohydrate intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	1325.4	1257.3	353.3
	Test 2	1471.4	1429.3	405.5
Control Group (n=8)	Test 1	1342.8	1235.2	283.3
	Test 2	1479.8	1520.2	342.9

TABLE 63

FIVE DAY CARBOHYDRATE INTAKE (Grams)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+145.7	302.0	-1.08	0.29	(-128, +419)
Control Group	+136.9	387.1	-0.81	0.43	(-224, +489)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+145.7	0.34	0.73	(-284, +302)
Control Group	+136.9			

There was no significant change in the carbohydrate intake of the exercise or the control group from Test 1 to Test 2.

TABLE 64

FIVE DAY PROTEIN INTAKE (GRAMS)

Mean protein intake, median and standard deviation for the exercise and control group at Test 1 and Test 2.

		MEAN	MEDIAN	S.D.
Exercise Group (n=17)	Test 1	368.8	335.2	77.5
	Test 2	372.7	365.5	137.5
Control Group (n=8)	Test 1	378.9	408.5	50.4
	Test 2	361.4	356.1	26.0

TABLE 65

FIVE DAY PROTEIN INTAKE (Grams)

Statistical Analysis

Paired t-test and 95% Confidence Interval

	Difference Mean	S.D.	t	P Value	95% Confidence Interval
Exercise Group	+ 3.9	135.7	-0.1	0.90	(-76, + 84)
Control Group	-17.5	-17.5	-0.82	0.82	(-64, + 28)

Two Sample t-test of Differences and
95% Confidence Interval of Average Improvement
(Exercise Over Controls).

	Mean Difference	t	P Value	95% Confidence Interval
Exercise Group	+ 3.9	0.43	0.67	(-82, +125)
Control Group	-17.5			

There was no significant change in the protein intake of the exercise or the control group from Test 1 to Test 2.

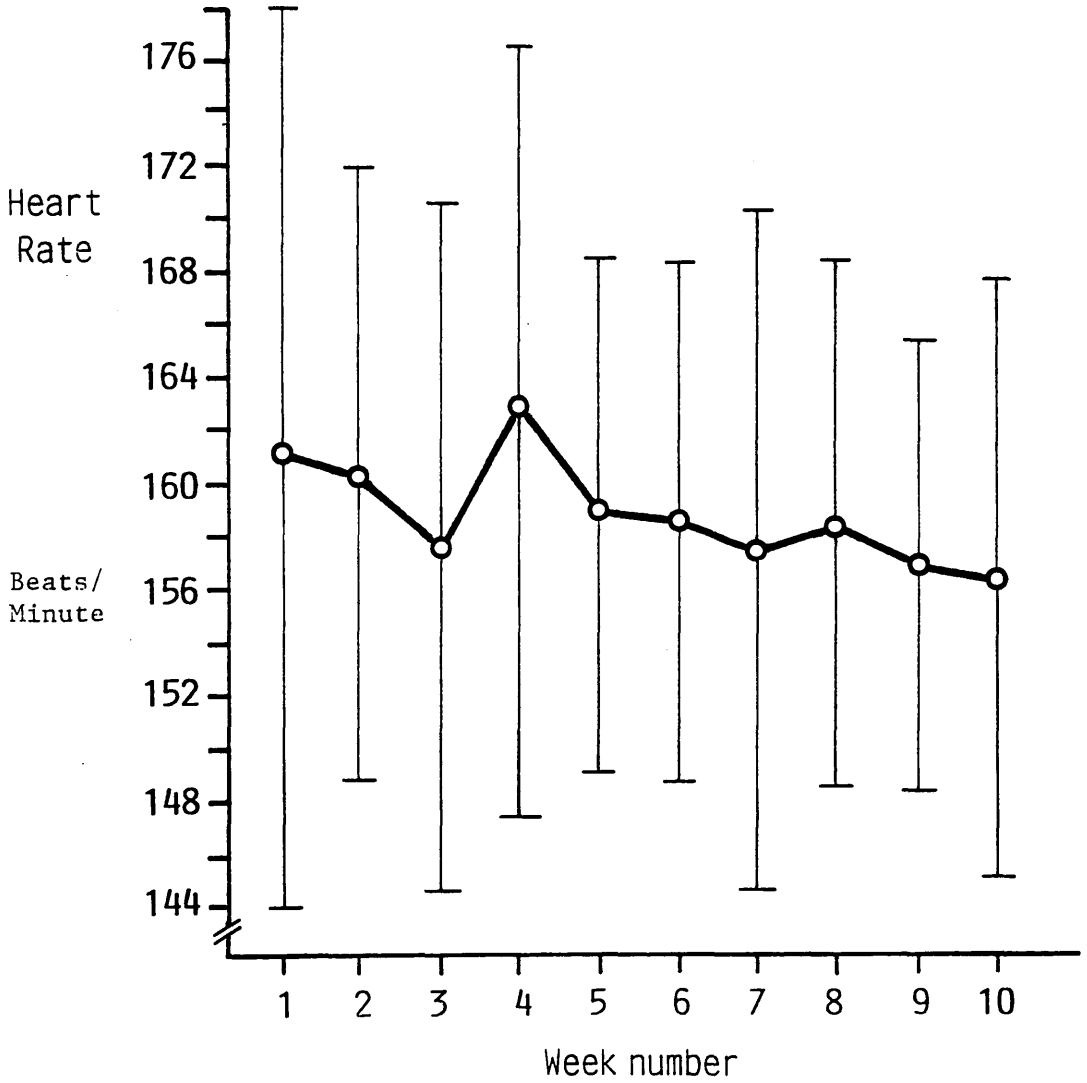

FIGURE 1.

Mean Training Heart Rate and Standard Deviation for the Exercise Group

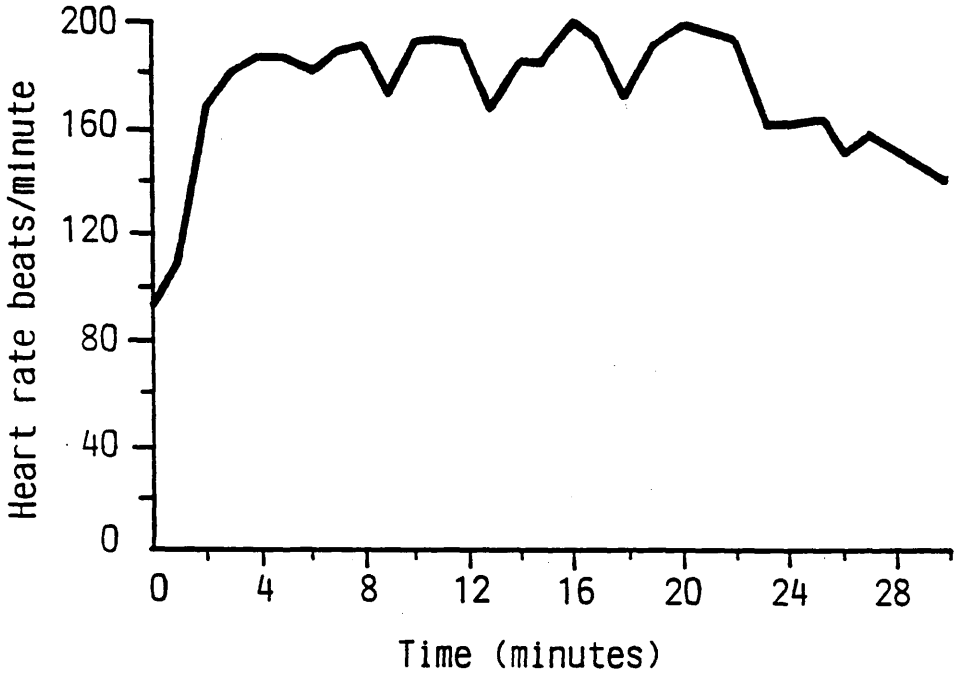

FIGURE 2.

Heart rate response of a 23 year old to a tune-up

CHAPTER FIVE

DISCUSSION

SECTION ONE

AEROBIC POWER

5.1.1 Training Effect

In order to achieve a training effect, i.e., an improvement in the area for which one is training, it is necessary to overload the particular system involved. Figure 1 shows that the mean training heart rate for the exercise group was around 88% of predicted maximum heart rate. Thus, it is not surprising that the exercise group improved their aerobic efficiency as they trained three times per week for 30 minutes at around 80% Vo2 Max. This level of training is considered by ACSM; (1978) to be sufficient to improve aerobic efficiency. Aerobic training results in adaptations in several mechanisms. Therefore, it is not surprising that the exercise group demonstrated some typical characteristics of improved aerobic efficiency.

5.1.2 Resting Heart Rate Bradycardia

The exercise group showed a significant decrease in resting heart rate compared to the control group. The 95% confidence interval showed that the average improvement in the exercise group was between 2.4 and 12.8 beats/minute greater than the control group. The mean decrease of the exercise group of 12 beats/minute compares favourably with other studies. For example, the 30 minute, 20 week walk/run programme of Millesis et al; (1976) at 85% to 90% of maximum heart rate showed a fall from 68 to 60 beats/minute in resting heart rate while the 2 day per week jogging programme of Pollock et al; (1972) at two different intensities at 92% and 80% of maximum heart rate produced a non significant fall in the 90% group from 65 to 61 beats/minute and a significant decrease in the 80% group from 68 to 63 beats/minute in resting heart rate.

It should be noted that a paired t-test showed that the control group resting heart rate fell significantly from 71 to 67 beats/minute from test 1 to test 2. Familiarity, less apprehension as well as a slight improvement in aerobic fitness could be the explanation for this significant decrease.

5.1.3 Physiological Mechanisms of Resting Bradycardia

The physiological mechanisms which underlie a resting bradycardia have yet to be clearly established. In his review, Fox, (1980) indicates that the bradycardia results mainly from an increase in parasympathetic activity. However, Scheur et al; (1977) suggest that a simultaneous decrease in sympathetic tone cannot be ruled out. Furthermore, an increase in blood volume has been shown to parallel a reduction in resting heart rate (Clausen, 1977). Moreover, Astrand and Rodahl (1986), postulate that a resting bradycardia is a consequence of a primary increase in stroke volume. The concomitant increase in stroke volume is associated with several factors which will be discussed under sub maximal bradycardia.

5.1.4 Sub Maximal Heart Rate

It is important to note that the small heart rate increases betweeen 4 and 5 minutes (2.3, 1.2 beats/min in the exercise group and 1.9 and 2.1 beats/min in the control group) in the third work load of test 1 test 2 verifies that the subjects had in fact attained a steady state. The mean significant decreases of the exercise group at the first, second and third workload were 12, 12 and 17 beats/min respectively. Comparison with other training programmes shows that the decrease

in sub-maximal heart rate is similar to these studies. However, these studies did not include control groups. In the study of Van Handel et al; (1976) decreases in heart rate of 18 beats/min were seen from the initial level of 128 beats/min. Saltin et al (1969) reported a fall in sub maximal heart rate of between 10 to 16 beats/min where the range of heart rate was between 120 to 160 beats/min. As in this study, the greatest absolute difference in heart rate from test 1 to test 2 was seen at the highest work load. The Saltin exercise programme was similar in respect to frequency, duration and intensity to this study. Clausen, (1977) indicates that the magnitude of sub maximal bradycardia is related to the duration, intensity, initial fitness level, and length of the study. The decrease in heart rate for the control group in the final work load, as shown by 95% confidence intervals, was between 1.8 and 7.6 beats/min. The 95% confidence intervals show that the magnitude of decrease was much greater for the exercise group than the control group. In the final work load the exercise group showed an average decrease over the controls of between 7.8 and 16.2 beats/min as shown by 95 % confidence intervals.

The fact that the control group demonstrated a significant fall in heart rate in the second and third work loads points to the fact that other variables unrelated to the training programme influenced the heart rates. However, it should be noted that the

activity levels of the control group were much higher after test 1. Test 1 was carried out in the first week of the New Year. Most subjects reported that they had been extremely indolent over the holiday period whereas several became much more active after the vacation. Activity diaries showed that several control subjects walked to and from work. For some, the total weekly walking time was 3 to 4 hours per week. Thus, the higher activity levels could be one reason for a sub maximal bradycardia. Walking studies have reported significant gains in aerobic fitness (Hudson et al; 1988). Furthermore the walking can elevate heart rates to appropriate training intensities in a wide ranging population (Pocari et al; 1987). Clearly this mode of regular exercise could have influenced the aerobic fitness levels of some control subjects.

5.1.5. Factors Influencing Sub Maximal Heart Rate

Other factors could have affected the sub maximal heart rate. Sub-maximal variables are prone to extraneous influences such as environmental factors, anxiety, lack of familiarity and circadian rhythms. These factors could explain the sub-maximal bradycardia in the control group. Marked variations in laboratory temperature could influence the results (Rowell, 1974). The temperature in the testing room was maintained between 18 and 20 degrees centigrade. Therefore, fluctuation in temperature cannot be the explanation

for the reduction in heart rate. Perhaps the subjects had become more familiar with the experimenter and the procedures. This contention is supported by Shephard, (1969a) who indicates that greater familiarity is likely to lead to a reduction in sub maximal heart rate. Repetition of a mode of exercise results in an increased efficiency. Shephard describes habituation as a form of negative conditioning which leads to a diminished anxiety in an experimental situation.

Another contributory factor to a decrease in sub maximal heart rate is the mechanical efficiency of the subject. Some of the decrease in sub maximal heart rate could be attributed to a fall in sub maximal Vo2. Ekblom et al; (1968) showed a fall in sub maximal cost after training whereas Saltin et al; (1969) reported no change. It could be that the subjects were more efficient at test 2 and less cumbersome movements resulted in a lower oxygen cost and heart rate. Thus, the control decrease on sub maximal heart rate could be explained by a learning effect, reduced anxiety, a reduction in the sub maximal oxygen cost or improved mechanical efficiency.

However, 95% confidence intervals show that the magnitude of change of the exercise group to be much greater than that of the controls. Therefore, the exercise group sub maximal bradycardia could be partly due to the possible explanations of the control

bradycardia and the effects of the training programme. However, the large decrease in the exercise group heart rate can be attributed to training.

The sub maximal bradycardia found in this study is a typical response to aerobic training and is considered to be an acceptable measure of an adequate training stimulus (Seals et al; 1984b).

5.1.6. Mechanisms of the Reduction in Sub-Maximal Heart Rate

The mechanisms associated with a bradycardia are complex and are as a result of possible changes in several variables. Both central and peripheral mechanisms can be implicated in submaximal bradycardia. Several studies have reported a reduction in submaximal CO after training (Andrew et al; 1966; Ekblom et al; 1968). However, Ehansi et al; (1987) and Saltin et al; (1969) found no significant change. All of these studies have demonstrated an increase in sub-maximal SV.

There is agreement that aerobic training results in an increased SV and bradycardia in previously sedentary subjects. The SV is influenced by the preload, (load on heart prior to contraction) the after load (load on the heart after contraction), and the contractility of the heart.

Assuming CO is relatively constant, a decrease in heart rate and a larger SV are interrelated. A lower heart rate results in an increased filling time for the heart. The increased filling time will result in an increased diastolic volume which will enhance the contractile potential of the cardiac muscle and hence increase the volume of blood ejected according to the Frank Starling mechanism. Blomquist et al; (1983) advocate that the increased end diastolic volume is the most important factor involved with the increase in SV. In their review, they highlight the fact that most human studies have been unable to demonstrate enhanced contractility of the heart after training. Increases in blood volume could increase the pre-load by increasing the venous return. Saltin et al; (1969) showed a 5% increase in blood volume in middle aged men after 10 weeks training. The question of whether the fall in heart rate follows an increase in SV is under debate. Clausen, (1977) quotes training studies in coronary heart patients whose heart rate fell as well as a decrease in SV. Moreover, short tern endurance training studies have shown that there is an increase in left ventricular end-diastolic diameter at rest (Park et al; 1985). An exercise induced increase in chamber size has been shown to develop within several weeks (Saltin et al; 1969). Debate continues as to whether the increase in stroke volume can also be attributed to an increased vascularity in the

myocardium as well as an increase in mitochondiral mass (Mc Ardle et al; 1986).

Starling's law is not the sole determinant of SV. A reduction in the after load i.e. a decrease in the peripheral vascular resistance would increase the SV. A decrease in exercise mean blood pressure would reduce the after load which would result in a reduction in the mean arterial pressure and increase the stroke volume. Reductions in exercise systolic pressure have been reported by Fagard, (1981) in his review. A decrease in exercise SBP would lower mean BP, reduce TPR and decrease the after load. Astrand and Rodahl, (1986) suggest that a reduction in sympathetic drive and a development of the capillary network will contribute to a decrease in total peripheral resistance. Some studies suggest that an increased parasympathetic activity is partly responsible for exercise bradycardia but some evidence indicates that there may be a simultaneous decrease in sympathetic activity (Fox; 1980; Winder et al; 1978; Ekblom et al 1973). Furthermore, Fox et al (1980) discussed the possibility that an increased SV at approximately the same cardiac output may mean that sympathetic stimulation is not necessary. In addition, a decrease in the intrinsic rate of sinoatrial node activity is another explanation for the bradycardia (Mc Ardle et al; 1986).

5.1.7. Peripheral Adaptations

The fact that experiments have shown a sub maximal bradycardia only in the exercised trained limb demonstrates that local adaptions within the trained muscle must have some importance (Saltin, 1976). With no fall in sub maximal oxygen consumption Clausen, (1977) suggests that this may be due to, less blood being supplied to the active muscles and a concomitant increase to the non "working" areas after training. Holloszy, (1973) considers that the more efficient muscles' ability to extract oxygen is the explanation for a decrease in blood flow to the working muscles and this fact could explain a fall in CO as the muscles are more efficient in extracting oxygen.

It is assumed that the blood flow to the trained muscles is decreased (Clausen; 1977). Although there is very little scientific evidence to back up this statement, it is based on the fact that most studies have found no decrease in CO but a concomitant increase in blood flow to non exercising tissues. Therefore, it is deduced that blood flow to the working muscles is reduced. However, it should be noted that Saltin et al: (1976) failed to demostrate any difference in blood flow between trained and untrained legs of 4 subjects. These subjects trained one leg and measurement of blood flow and A-Vo2 Diff was made after training. While

there was no significant difference in blood flow after training between the trained and untrained. The A-Vo2 Diff in the trained leg was greater. Gollnick et al; (1984) in their review, consider that blood flow to contracting muscle during sub maximal work is similar when power production by the muscle is constant.

It is not possible to speculate on any skeletal adaptions in this study as no investigations in this area were carried out. However, the literature suggests that there are several adaptations which take place in the muscle which would be expected after a period of aerobic training (Astrand and Rodahl; 1986).

Biochemical and cellular changes within the muscle promote an increase oxygen extraction. An increase in the oxidative capacity of skeletal muscle may result from several adaptive responses. These changes include an increase in capillary density, mitochondrial volume and an increase in the enzymes involved in aerobic metabolism (Astrand and Rodahl, 1986). Training of the endurance type results in an increase in the size of the muscles, but there is also a greater number of capillaries for each muscle fibre (Ingjer, 1979). Increases in capillary density may in fact lessen the distance between the cell interior and the blood. An increase in the capillary bed has the advantage that gas, substrate and metabolite exchange will be improved. Thus, the supply of oxygen to the working

muscles is enhanced.

While an increase in myoglobin takes place, it has yet to be proved that there is enhanced diffusion of oxygen from the cell membrance to the mitochondria (Mathews et al; 1976). With training, there is an increase in the number and size of mitochondria and an increase in the concentration and the activity of the oxidative enzymes in the mitochondria in the muscles (Gollnick, 1969; Barnard et al; 1970; Saltin et al; 1976). A greater utilisation of free fatty acids at a standardised sub maximal work load is another feature of aerobic training adaptions (Gollnick et al; 1984).

Within the limitations of this study it is impossible to establish the cause or causes of the post training bradycardia. However, an increase in blood volume, an increase in parasympathetic tone, a larger stroke volume as well as peripheral adaptations are all possible reasons for sub maximal bradycardia. Both central and peripheral mechanisms are implicated in sub maximal bradycardia.

5.1.8. Health Implications

The reduction in sub maximal heart rate has implications for health. A lowering of heart rate at a given absolute work load will increase the efficiency of the heart. Blood pressure is often lower or the

same at a standard work load after a training programme. An estimate of the myocardial Vo2 is the Rate Pressure Product (heart rate x systolic blood pressure). Therefore, the myocardial oxygen consumption will be lower at a given load and perfusion of the myocardium greater as the diastolic phase is longer at a given workload after training. With a decreased sub maximum heart rate, the heart is capable of a greater maximum performance. Thus, after appropriate training the individual can accomplish sub maximal tasks more easily and can work harder and longer. A reduced myocardial oxygen cost at a given absolute work load alse reduces the possibility of ischaemia. Training has the effect of increasing the heart's capability to work and the lower sub maximal heart rate reduces circulatory strain at sub maximal levels (Clausen, 1977).

5.1.9. Predicted Vo2 Max

Bearing in mind that the results of the Vo2 Max were gained from a prediction, the validity of the values may be questioned. However, a value of 33 ml/kg/min is close to other studies which have examined similar age groups (see Table 66). The initial level of Vo2 Max was typical of sedentary middle aged males. Thus, it would be expected that this value would be increased with training. The magnitude of increase of 20 per cent in Vo2 Max in this study is in agreement with the

literature. Astrand et al; (1986) confirm that 3 sessions of 30 minutes duration for 2-3 months at an appropriate intensity will result in an increase in aerobic power in the order of 10-20%.

Prediction of Vo2 Max allows for comparison with other studies. The training heart rate of 88% of maximum heart rate in this study is a similar intensity to that of Bonnano et al; (1974) and Pollock et al: (1975). As Table 66 shows this 10 week study has produced a percentage increase which compares favourably with other studies with a similar training prescription and initial values. A great variety of response to training has been reported and there is a suggestion that the trainability of middle aged men is less than in younger men (Pollock, 1973). However, the 18.1% increase of the Saltin study was considered to be as a consequence of the higher intensity interval training in that study. The large increase in this study and in the Saltin study may reflect the statement of Wenger and Bell, (1986) who consider that the greater the challenge to the oxygen transport system the greater the gains in aerobic fitness.

5.1.10 Mechanism of Increase in Vo2 Max

An increase in Vo2 Max is associated with an increase in cardiac output and A-Vo2 Diff (Ekblom et al; 1968).

TABLE 66.

EXERCISE STUDIES - AEROBIC CHANGES IN MIDDLE AGED MEN.

STUDY	CHARACTERISTICS OF SUBJECTS N	AGE	TRAINING PROGRAMME LENGTH WEEKS	FREQUENCY DAYS	DURATION (MIN)	INTENSITY	MODE	ml kg^{-1} min^{-1} TEST 1	TEST 2	% INCREASE
This Study	21	37	10	3	30	80% VO_2Max	Walk Run Skip	33.0	39.6*	20%
Ribisl et al 1969	15	40	20	3	35	?	Run	40.1	45.5*	14%
Saltin et al 1969	42	41	8-10	2+	45	Interval Training	Run	37.5	44.3*	18%
Pollock et al 1971	16	49	20	4	40	63-76% Max Heart Rate	Walk	29.9	38.9*	30%
Pollock et al 1972	12	39	20	2	45	92% Max Heart Rate	Run	36.0	40.1*	11%
Bonnano et al 1974	20	41	12	3	30-35	70-85% Max Heart Rate	Run	33.4	35.5*	6%
Pollock et al (1975)	9	38	20	3	30	90% Max Heart Rate	Run	36.8	41.1*	12%
Van Handel et al 1976	6	35-56	10	3-4	20-30	75-85% VO_2 Max	Jog	34.0	40.7*	20%
Brownell et al 1982	24	42	10	3	15-20	70% Max Heart Rate	Run	31.3	34.5*	10%
Duncan et al 1985	44	30	16	3	60	70-80% Heart Rate	Run	39.7	44.5*	12%

* $P<0.05$ or less Max = Maximum

It should be noted however, that Hartley et al; (1969) in their study of middle aged men suggest that a less pronounced increase in Vo2 Max after aerobic training may be as a result of the older subjects inability to improve maximal A-Vo2 Diff. As maximum heart rate is the same or shows a decrease after training, the increase in cardiac output is due to an increase in stroke volume. The increase in stroke volume may be partly due to an increase in blood volume and hence a greater venous return as well as a greater ventricular volume. However, Hammond et al; (1985) state that it is not clear whether the predictable increase in CO with training is the result of reduced outflow impedance, increased chamber size, or enhanced contractility. Moreover, there is speculation that there is an increase in left ventricular diastolic compliance and perhaps greater systolic contractility Park et al; (1985). The skeletal muscle adaptions which enhance A-Vo2 Diff have been discussed earlier. There is no consensus as to whether maximum muscular blood flow is increased (Lamb, 1984). However, Clausen, (1977) and Shephard, (1982) in their reviews concluded that there is an increase in blood flow. An increased capillary network results in the maintanance of a near optimal transit time for blood flow (Astrand and Rodahl; 1986). Saltin, (1985) concluded that the primary importance of a larger capillary bed as a result of endurance training is not to accommodate blood flow but to maintain or increase the mean transit

time of the red cells passing through the capillaries. An increased diffusion time and shorter diffusion distance will enhance oxygen transfer (Vander et al; 1975).

5.1.11 Summary

This study verifies that a training programme of 3 times per week for 30 minutes duration at an intensity of 80% of Vo2 Max will result in an enhanced aerobic efficiency.

Blood Pressure

5.2.1 Baseline Levels

The mean blood pressure value for the exercise group was 130/85 mmHg. Therefore, the exercise group was normotensive as was the control group which had initial values of 126/81 mmHg. These initial values are low compared with one study conducted in the Glasgow area. Grant et al; (1988) found a group of 42 year old males to have initial levels of 139/92 mmHg whereas the the study of Findlay et al; (1987) reported a similar baseline level of 133/87 mmHg to this study. A comparison with a recent Greater Glasgow Health Board Baseline Study, (1988) of almost 6000 male and female subjects with ages between 20 and 50 years shows that the exercisers and controls were outwith the 21% of subjects with a DBP of 90mmHg or over. Similarly, around 21% of those subjects measured in the Glasgow Health Board study had a SBP of 140 mmHg or over. Clearly the subjects in this study can be categorised as normotensive.

5.2.2 Comparison with Other Studies

The small non significant increase in systolic blood

pressure in the exercise group was mirrored by a similar small non significant increase in control systolic blood pressure. Similarly, there was no signifiacnt change in the diastolic blood pressure of either group. Comparison with the other studies which have had similar initial blood pressure levels reveals that a dramatic fall in blood pressure is unlikely as it is not possible to reduce blood pressure in the normal range by a large amount. While some studies have demonstrated a small but significant decrease in blood pressure, (Mann et al; 1969; De Vries et al; 1970; Pollock et al; 1971), other studies with similar initial readings have failed to demonstrate a significant reduction in blood pressure. For example, Millesis et al; (1976) reported almost identical baseline values to this study but did not find any significant decrease in blood pressure after 20 weeks of walking and running. It is difficult to compare some other studies as non exercising controls were not included in the research design. Thus, the decrease in similar baseline readings of Findlay et al; (1987) must be treated with caution. After 30 weeks of marathon training blood pressure fell from 133/87 mmHg to 114/73 mmHg. Indeed, the authors concede that the fall in blood pressure may be due to a familiarisation effect.

5.2.3 Training Stimulus

In all the studies in Table 2 all the exercise groups

demonstrated an improvement in fitness. This characteristic was replicated in this study. Therefore, the fact that there was no fall in blood pressure of the exercise group cannot be attributed to the fact that the training programme was an insufficient stimulus for the improvement in fitness. The decrease in sub maximal heart rate and increase in estimated Vo2 Max are testimony to an improvement in aerobic efficiency. It was to be expected that the training programme would elicit a training effect. Therefore, it is impossible to interpret changes in blood pressure and fitness levels. Furthermore, it is not essential to change Vo2 Max to elicit changes in cardiovascular factors at rest (Seals et al; 1984b).

5.2.4 Body Weight

The fact that some studies which have shown a fall in body weight and a concomitant decrease in blood pressure has led some to suggest that a decrease in body weight is interelated with a fall in blood pressure. Although weight loss after training is associated with a decrease in blood pressure, the evidence is not conclusive. Thus, the non significant weight loss in this study cannot be considered to be a major reason for no change in blood pressure. Normotensives who have lost weight and decreased blood pressure after a period of exercise include the studies of Mann et al; (1969); De Vries et al; (1970) and

Pollock et al; (1971). It should be noted that in Findlays 30 week marathon training programme that weight loss was greatest in first 15 weeks but blood pressure continued to fall in the second 15 weeks despite a less marked decrease in the rate of weight loss. Findlay et al; (1987) hypothesise that the decrease in blood pressure associated with considerable weight loss may be due to a fall in the levels of circulating catecholamines. However, Fagard et al; (1985) in their review indicated that decreases in blood pressure can be expected after aerobic training even although no fall in body weight takes place.

5.2.4 Sympathetic Activity

The fact that the resting and sub maximal heart rates were lowered in the exercisers after training is suggestive of a reduction in sympathetic tone. A reduction in sympathetic acivity is associated with a lowering of blood pressure. However, sympathetic activity is low at rest and may not be greatly affected by endurance training. Fagard, (1985) stated that a decrease in autonomic activity may in fact be partly be responsible for a training induced bradycardia, but it cannot be ascertained to what extent it influences blood pressure. Despite the possible fall in sympathetic activity there was no reduction in blood pressure.

5.2.5 Summary

Although aerobic exercise has been recommended as a non pharmacological method of lowering blood pressure, this 10 week aerobic training pogramme has failed to achieve this aim. The lack of significant decrease in blood pressure in this study was to be anticipated as previous studies have shown that dramatic falls in blood pressure would not be expected in a group of subjects who were well within the range of normal blood pressure.

5.2.6 Further Research

The adverse side effects, high cost and the making of asymptomatic subjects "medical dependent" associated with pharmacological treatment of hypertension make alternative methods of treatment appealing. Aerobic training is cheap, often creates a feeling of well-being and is easily accessible to most of the population. Thus, it is important that further investigation takes place on the effects of aerobic training on high blood pressure. Clearly, an established hypertensive group needs to be found before meaningful study can begin. It is important to establish that exercise can influence hypertension but it would be of great value to determine the mechanisms responsible fo any decrease in blood pressure. Therefore future studies should incorporate measurement

of haemodynamic and humoral changes. Various studies have investigated the effect of training on the mechanisms involved in a reduction of blood pressure, in particular the haemodynamic and humoral response to training. These investigations have produced conflicting findings (Fagard et al; 1985; Kenney et al; 1984; Johnson et al; 1967; Hagberg et al; 1983). Obviously non exerising control groups with the same characteristics as the exercisers should be included in the research design of the study.

LIPIDS

5.3.1(a) Total Cholesterol - Baseline Levels

The total cholesterol level for the exercisers at test 1 was 6.50 mmol/litre and 6.21 mmol/litre for the control group. These values are fairly high and the European Artherosclerosis Society, (1987) would recommend that some action be taken to lower this figure to reduce CHD risk. Total cholesterol showed a dramatic decrease in both groups. However, the fact that the control group demonstrated a similar decrease to the exercise group means that there is some other underlying factor other than exercise which has infuenced total cholesterol levels.

5.3.1(b) Total Cholesterol - Comparison With Other Studies

This study agrees with the studies of Linder et al; 1983; La Rosa et al; 1982; Huttenen et al; 1979 and Peltonen et al; 1981 that aerobic training per se does not lower total cholesterol. However, Shephard et al; (1980) and Altekruse et al; (1973) have shown a significant decrease in total cholesterol. Both controlled and non controlled studies have failed to change total cholesterol levels. In some studies the

pre-training levels were low and considered to be not very amenable to change. Examples of low pre-training levels are those of Allison et al; (1981) at 4.11 mmol/litre and Linder et al; (1983) at 3.82 mmol/litre. However, Pauly et al; (1982) significantly lowered total cholesterol from 5.40 to 5.00 mmol/litre and other studies (e.g. Brownell et al; (1982) from 5.35 to 5.02 mmol/litre; and Altekruse et al; (1973) from 5.81 to 5.20 mmol/litre) have reported decreases in total cholesterol with initial levels below that of this study.

5.3.1(c) Total Cholesterol - Training Stimulus

No training characteristic can be identified which might influence total cholesterol (Haskell, 1987). Therefore, it is not possible to isolate any training variable in this study as the training stimulus in this study is similar to many other studies. Moreover, this study demonstrated an increase in fitness, as in many other studies. The results of this study do support the caution expressed by Goldberg et al; (1987) who cast doubt on the statement that aerobic training by itself, is an independent modifier of total cholesterol.

5.3.1(d) Total Cholesterol - Body Weight

There is conflict regarding the effect of body weight

changes on total cholesterol levels after training. Altekruse et al; (1973) suggest that a drop in body weight as a result of an exercise programme will lead to a decrease in total cholesterol. A non significant fall in body weight and body fat took place in this study and no decrease in total cholesterol was found. The 10 week study of Savage et al; (1986) did not effect body weight but produced a significant decrease in the sum of skinfolds. Despite this decrease in body fat, no fall in total cholesterol was reported. In the study of Hartung et al; (1981) body weight remained the same, body fat fell by 2.5 per cent but no change was found in total cholesterol. Conversely, the middle aged males in the Brownell et al; (1982) study showed a concomitant decrease in body weight and total cholesterol. However, Vu Tran et al; (1983) in their meta analysis showed that subjects with the greatest fall in body weight displayed the greatest decrease in total cholesterol. Therefore, it may not be surprising that the non significant fall in body weight in this study resulted in a non significant decrease in total cholesterol.

5.3.2(a) High Density Lipoprotein - Baseline Levels

The initial HDL level in the exercise group was 1.12 mmol/litre and the control group was 1.16 mmol/litre. These values are in the mid range. Therefore, it would be expected that they were amenable to improvement.

The exercise group and the control group showed a significant increase in HDL as shown by a paired t-test. However, there was no significant change between the exercise group and the control group in this study. Obviously other factors have influenced HDL levels even although the fitness level of the exercise group improved markedly compared with the control group.

5.3.2(b) High Density Lipoprotein - Comparison with Other Studies

This study has not produced a similar result to the controlled studies of Huttenen et al; (1979) and Peltonen et al; (1981) whose 16 and 15 week studies respectively, produced significant increases in HDL despite higher baseline levels. However, the findings of this study are in agreement with the results of Brownell et al; 1982; Linder et al; 1983; and Gaesser et al; 1984; who showed no significant change in HDL with similar baseline levels to this study.

5.3.2(c) High Density Lipoprotein - Effect of Training Intensity

While most studies have improved fitness levels, Shephard et al; (1982) speculated that the significant but small increase in estimated Vo2 Max in their study was a reflection on the low intensity traning levels of the subjects. They considered the low intensity

training programme to be responsible for the lack of change in HDL. The significant and fairly large improvements in aerobic fitness found in this study shows that the training programme was effective. With training heart rates of 160 beats/min (80% Vo2 Max) during the 10 weeks of this study it was to be expected that the training intensity was high enough to produce a training effect. However, Peltonen et al; (1981) (140-160 beats/min) and Huttunen et al; (1979) (66% HRR) have produced a significant increase in HDL with similar levels of intensity to this study. Similar training heart rates have failed to elevate HDL levels (Pauly et al; 1982). Furthermore, Gaesser et al; (1984) failed to demonstrate any significant increase in HDL in a low (45% Vo2 Max)or high intensity (85% Vo2 Max) despite an increase in Vo2 Max in both groups. A similar pattern is evident when training sessions of tbe same duration and frequency are compared. There are examples of HDL levels rising, Streja et al; (1979), remaining the same Brownell et al; (1982), and lowering Allison et al; (1981).

5.3.2(d) High Density Lipoprotein - Length of Training Programme

Some researchers have suggested that improvements in fitness levels are insufficient in themselves to alter HDL levels. The study of Williams et al; (1982) did not demonstrate changes in HDL until the subjects had

maintained 10 miles per week of running for at least 9 months. Williams et al; (1982) also reported that despite an earlier increase in fitness and a decrease in body weight the changes in HDL did not manifest themselves till later. They suggested that this training threshold might be "turned on" by some physiological process. The authors postulate that an increase in lipoprotein lipase (LPL) could be responsible. Moreover, the authors consider that these findings have implications for health. They concluded that a prolonged period of training was necessary for changes in HDL to be seen. Williams et al; (1982) emphasise that changes in fitness, percentage fat and lipids were not simultaneous. Improvements in percentage fat and fitness were recorded before lipids improved. Similarly, Kavanagh, (1987) has also found a threshold for HDL increases. His post myocardial infarction patients had to cover 12 miles per week, regardless of pace, before increases in HDL took place. Thus, the length of the training programme or the duration of each training session may be responsible for the increase in HDL. Kavanagh, (1987) stressed that HDL levels dropped when training distance dipped below 12 miles per week. However, it must be conceded that several studies of short duration have disputed the notion that relatively long term training programmes are necessary to favourably influence HDL. Farrell et al; (1980) agree that alterations in HDL may lag behind changes in aerobic fitness. Although Vo2

Max had increased after 4 weeks, there was no change in HDL. However, after 8 weeks of 3-4 times per week of running at 70% of Vo2 Max HDL rose from 1.32 to 1.48 mmol/litre. The subjects in Streja's study (1979) only covered 8.5 miles per week in their 13 week programme and increased HDL levels from 0.93 to 1.02 mmol/litre. However, other short duration studies have failed to demonstrate any increase in HDL (Brownell et al; 1982; Linder et al; 1983; and Savage et al; 1986). Conversely, studies of 15 and 16 weeks duration have shown elevations in levels of HDL (Huttunen et al; 1979; Peltonen et al; 1981).

5.3.2(e) High Density Lipoprotein - Body Weight

No clear trend can be established between HDL changes and body weight decreases. Tables 5 and 6 show that there are instances of an increase in HDL with no fall in body weight (Streja et al ;1979; Peltonen et al; 1981;). Thus, the non significant fall in body weight in this study cannot be given as the reason for the non significant increase in HDL.

5.3.2(f) High Density Lipoprotein Subfractions

Subclasses of HDL, HDL2 and HDL3, may provide more information on CHD risk. Higher levels of the HDL2 fraction is thought to offer more protection from CHD (Lewis, 1983). Moreover, some investigation has shown

that it is HDL2 which changes with training (Ballantyne et al; 1982). These sub fractions were not measured in this study, therefore it could be that changes took place in these sub fractions and the beneficial effects of the exercise programme were not revealed.

5.3.2(g) High Density Lipoprotein - Triglycerides

It is unlikely that any possible increase in HDL in this study did not happen because the training programme failed to influence the triglyceride levels which were below the EAS; (1987) threshold for intervention. Selection of subjects with triglyceride levels which are unlikely to change may preclude an increase in HDL levels. However, Huttunen et al; (1979) has shown that an increase in HDL with a fall in triglyceride levels from 1.54 to 1.27 mmol/litre. Conversely, Leon et al; (1979) have shown an increase in HDL despite no significant fall in baseline triglyceride levels of 1.86 mmol/litre whereas Brownell et al; (1982) lowered triglycerides and total cholesterol levels after similar training to this study but the HDL levels stayed unchanged.

Two enzymes are implicated in the regulation of HDL and triglycerides. These are lipoprotein lipase (LPL) and hepatic lipase (HL). It remains to be established whether enzymatic changes are a function of exercise alone or whether weight loss and exercise are

interrelated (Nikkila et al; 1978; Dufaux et al; 1982; Peltonen et al; 1981; Sutherland et al; 1980;).

It is thought that higher HDL levels in long distance runners are partly as a result of an increased LPL activity and a concomitant increased turnover of triglyceride rich lipoproteins. A response of endurance training is the increase in muscle and adipose tissue LPL. Dufaux et al; (1982) concluded that there is a close relationship between the metabolism of triglyceride rich lipoproteins and HDL. It is suggested that increases in HDL are caused by an increased activity of LPL.

The explanation given by Astrand and Rodahl (1986) is that an increase in HDL is caused by a more extensive capillary bed which results in an increase in LPL activity. The increase in skeletal muscle capillary density may be responsible for higher LPL levels as the capillary endothelium is increased. Saltin et al; (1969) indicate that middle aged sedentary males may need longer to adapt to aerobic training. Thus, it may well be that the lack of improvement in A-Vo2 difference is indicative of a lack of adaptation found in some studies of middle aged men and may explain the lack of increase in HDL levels in some studies which have used middle aged men as subjects. Therefore, the short length of this study may be an explanation for the non significant increase in HDL.

5.3.2(h) Total Cholesterol/High Density Lipoprotein Ratios

The change in the total cholesterol/HDL ratio for the exercise group was not significantly different from the control group.

5.3.2(i) Changes in Total Cholesterol and High Density Lipoprotein

It has been suggested that movement in total cholesterol is predictive of HDL changes. Haskell, (1984) indicated that when total cholesterol is lowered for any reason absolute concentrations tend to fall. However, Lipson et al; (1980) reported a fall in total cholesterol from 4.03 to 3.62 mmol/litre over a six week period and no change in HDL. It may be that it is necessary for an exercise programme to promote a significant increase in total cholesterol and a decrease in triglycerides or both, if it is to elevate HDL (Allison et al; 1981). The study of Streja et al; (1979) is an example where HDL increased significantly and total cholesterol also rose. However, Peltonen et al; (1981) have shown HDL increases without an increase in total cholesterol. Thus, changes in total cholesterol do not seem to be predictive of movement in HDL levels and cannot provide an explanation for the results in this study.

5.3.3(a) Triglycerides - Baseline Levels

Both the exercise and control groups had initial levels well within the acceptable levels outlined by the European Artherosclerosis Society, (1987). The exercise group triglyceride levels showed a small but non significant fall from 1.38 to 1.36 mmol/litre while the control group triglyceride level demonstrated a non significant increase from 1.34 to 1.42 mmol/litre.

5.3.3(b) Triglycerides - Comparison With Other Studies

The findings in this study are in accordance with the non controlled study of Streja et al; (1979) and the controlled studies of Linder et al; (1983) and Peltonen et al; (1981). However, similar types of training programmes in non controlled studies (Brownell et al; 1982; Pauly et al; (1982) have produced decreases in triglycerides as well as the controlled study of Huttenen et al; (1979). The initial level of the Pauly study was 1.18 mmol/litre which could be considered to be rather low to be influenced by training whereas the baseline level of 2.0 mmol/litre of the Brownell study was more amenable to change (Haskell, 1984). The initial levels of 1.38 mmol/litre (exercise group) and 1.34 mmol/litre (control group) in this study are similar to those of Huttunen et al; (1979) of 1.54 mmol/litre and La Rosa et al; (1982) of 1.61 mmol/litre. The former showed a decrease over 4 months

whereas the one year study of La Rosa resulted in no change. Haskell, (1984) suggests that levels under 1.35 mmol/litre are unlikely to change with training. Huttunen et al; (1979) agree that training will only influence triglyceride levels when they are already elevated. However, Peltonen et al; (1981) found that their controls had a slight increase in triglyceride levels whereas the runners' values remained constant and the authors suggest that running may have produced some benefit. It should be noted that the baseline levels in the Peltonen study was 1.19 mmol/litre. Thus, it is not surprising that there was no change in triglyceride levels in this study as the literature indicates that these baseline levels are unlikely to change with aerobic training.

5.3.3(c) Triglycerides - Training Stimulus

No training characteristic can be identified which can be associated with a decrease in triglycerides (Haskell, 1984). An examination of Table 6 shows that similar training programmes have produed a variety of response. Similar training prescriptions to this study have resulted in a fall in triglycerides (Pauly et al; 1982) or no change (Gaesser et al; 1984). Neither the low or high intensity groups of Gaesser had an effect on triglyceride levels. It must be conceded that the very low pre-training levels would not be likely to change.

Although the Huttunen et al; (1979) study lasted 16 weeks, the authors reported that the triglyceride levels fell after 8 weeks. It seems that the short duration of this study could not be the reason for no change in triglyceride levels.

5.3.3(d) Triglycerides - Body Weight

The non significant change in body weight is unlikely to be the reason for the lack of change in triglycerides. While some studies have demonstrated a fall in body weight and triglycerides (Brownell et al; (1982), the study of Huttenen et al; (1979) disputes the notion that a decrease in body weight is essential if triglyceride levels are to fall. While Huttunen reported a small non significant fall in body weight, they indicate that triglyceride changes cannot be attributed soley to a weight decrease. They state that a fall in triglycerides was not related to a decrease in body weight. The authors emphasise that a highly significant decrease in triglycerides took place in those who maintained the same body weight throughout the study. Moreover, the massive weight loss of 5.7 kg in the 16 week study of Leon et al; (1979) failed to lower the pre- training triglyceride level of 1.86 mmol/litre. Thus, it may be difficult to attribute the lack of movement in triglyceride levels to a non significant decrease in body weight in this study.

5.3.4. Diet

An attempt was made to monitor diet as closely as possible. This study showed no changes in total calorie intake, or the amount of protein, carbohydrate and fat consumed for the exercise or the control group. However, it must be conceded that there are limitations to the method of dietary analysis used in this study. It was only possible to sample the diet for 5 days in the early part of the study and for 5 days near the end of the study. Although subjects were asked to maintain the same diet throughout this study, there could have been dietary changes which this dietary evaluation did not detect. The subjects may have become more health conscious in the middle of the study but reverted to their former diet near the end of the study. Moreover, this study began immediately after the Christmas holidays. Therefore, it is anticipated that the subjects consumed an "unusual" diet during the holiday period. Clearly it is not possible to sustantiate this hypothesis but enormous dietary variation before test 1 could have influenced the initial lipid scores on test 1. However, the dietary results are very similar to a previous study (Grant et al; 1988) where the subjects weighed all food for the same time periods to this study.

There is a suggestion that dietary analysis may not be sensitive enough to detect elements which have a

bearing on lipids. Possible limitations in dietary analysis are shown by Thomson et al; (1984) who followed 10 distance runners for 21 days during which time the runners ran 16 kilometres and consumed a diet produced by a university kitchen. The HDL fell from 1.81 to 1.63 mmol/litre. The authors postulate that elements within the diet which were unable to be defined had influenced the HDL levels. In the Thompson study it has been suggested that the increase in energy cost of a training programme has resulted in dietary changes. Part of the increase in energy intake was to compensate for the increased energy cost of exercise. A portion of the greater energy intake came from saturated fat which may have made a contribution to the decrease in HDL levels. This explanation may be plausible in this study as no assessment of polyunsaturated or unsaturated fat was made. In the Allison et al; (1981) study an increase in dietary fat was reported. The authors suggested that an increase in saturated fat would lead to greater levels of LDL with no change in total cholesterol and a reduction in HDL. Furthermore, Hartung, (1984) has shown that dietary fat restriction allied to very high levels of exercise did not prevent HDL levels from declining significantly. However, in this study fat intake remained constant at the two sampling periods.

5.3.5 Alcohol

No change in alcohol consumption was found in the dietary survey. Therefore, changes in alcohol consumption could not be a reason for any movement in HDL levels. It should be noted that Eichner, (1985) indicated in his review, that alcohol may increase HDL 3 but he stressed that it is HDL 2 which is associated with protection from heart disease.

5.3.6 Smoking

Two exercisers smoked an average of 7.5 cigarettes per day and this level was maintained throughout the study while six controls smoked an average of 24 cigarettes per day. The smokers in both groups did not alter their smoking habits. Thus, changes in smoking habits did not influence total cholesterol or HDL levels. The small percentage of smokers (see table 55) in both groups shows that this sample is atypical of the Glasgow population. Forty per cent of the male population of Glasgow smokes (Greater Glasgow Health Board, 1988).

5.3.7 Sampling

The fact that both the control and the exercise groups showed "improvement" in total cholesterol and HDL

suggests that other factors other than exercise have influenced the results. Seasonal variation in total cholesterol levels was found by Gordon et al; (1987) who demonstrated that total cholesterol levels peaked in December and were lowest in June. However, dietary and weight changes could only explain one third of the seasonal variation which was only 0.20 mmol/litre. The magnitude of this seasonal variation is much smaller than the "improvement" seen in this study

The fact that both groups demonstrated a dramatic change in total cholesterol and HDL is suggestive of methodological error. However, as Appendix C shows, precision checks carried out at the Western Infirmary demonstrated that this explanation is unlikely. Jacobs et al; (1983) have shown that there is day to day variability in total cholesterol measurement but it is reasonable to assume that this variability would "even itself out".

Acute changes which are associated with training are found in triglycerides, LDL and HDL (Haskell, 1984). It is unlikely that the time of blood sample had any bearing on lipids in this study as sampling took place 3-4 days after cessation of training at the same time of day. While it has been shown that postural changes can effect total cholesterol levels, (Katan et al; (1987), this explanation is not plausible as all subjects gave samples in a seated position.

Furthermore, all subjects gave blood samples after a 12 hour fast and the blood samples were analysed using the same analyser. Moreover, the Biochemistry Department at the Western Infirmary participates in external control trials, the results of which have been favourable. In addition, the test 1 and test 2 samples were analysed using the same reagent and the test 1 samples produced the same results when analysed 10 weeks apart using different reagents.

5.3.8 Summary

This 10 week aerobic training programme did not improve the lipid profile of 21 middle aged men. Although the exercise group demonstrated a substantial decrease in total cholesterol and increase in HDL, this fall was not significantly greater than that observed in the control group. Clearly, there is some underlying factor which has influenced total cholesterol.

The time of year may have had a considerable influence on lipid levels and further research should take possible seasonal variations into account. These results highlight the fact that a control group is essential when the effect of aerobic training on lipids is evaluated. In the absence of a control group the changes in exercise group total cholesterol and HDL levels would have been statistically significant and attributed to aerobic exercise. Thus, the findings of

the many non controlled studies must be questioned. With fairly low initial levels of triglycerides, it was to be expected that no change would take place in triglyceride levels.

Reasons for the lack of increase in HDL include the fact that HDL changes may not occur until a certain weekly training time is maintained for a given duration. HDL levels are related to triglycerides and a downward movement in triglycerides may be necessary before HDL levels will rise. Possible unmeasured changes in the HDL subfractions may mask potential benefits

Despite improvements in fitness levels, this study suggests that a threshold for fitness changes does not correspond with movement in lipid levels. Furthermore, no training characteristic can be identified which may have influenced the results. In addition, an attempt was made to monitor as many extraneous factors as possible. It appears that diet, smoking and alcohol did not have a bearing on lipid levels.

5.3.9 Future Research

While this study has failed to provide positive evidence that aerobic training per se will favourably affect lipid levels, there is evidence that physically active people have a lipid profile which is associated

with a lower CHD risk. Thus, further study should investigate the concept of a training threshold for lipid changes but studies must take into account the pre-training levels as the magnitude of change appears to be dependant on baseline values. The findings of this study suggest that aerobic exercise cannot be recommended for a beneficial alteration in lipid profile. Further studies which include a longer training duration and an alteration to diet may yield more positive results than this present study. However, the investigation of the effects of aerobic training on lipid levels is made difficult by the many confounding factors. Clearly, there is a need to monitor, and if necessary regulate the factors which have a bearing on lipid levels.

BODY COMPOSITION

5.4.1 Baseline Levels

Comparison with other groups of a similar age reveals that the initial level of body fat was not particlarly high. Taylor, (1988) reported an initial body fat of 24.1% in a West of Scotland population of a similar age group while Grant et al; (1988) found an initial level of 26.4% in sedentary middle aged men. Although there is no threshold between percentage body fat and CHD risk, the initial figures of 22% body fat for the exercise and control groups in this study indicate that the subjects were not very obese but they were above the threshold limit of 20% body fat set by Mc Ardle et al; (1986) who stated that males over 20% body fat should be considered obese.

Body weight fell from 77.6 kg to 76.7 kg. The non significant decrease in body weight is equivalent to 1.16% body fat, presuming that all of the weight loss was fat. The non significant decrease in body fat of 0.6% as predicted from skinfold measurement is clearly not equivalent to the decrease in body weight. There are a number of reasons why this apparent discrepancy has taken place.

5.4.2 Experimental Error

The same experimenter carried out all the measurements but it may be that the experimenter was unskilled in the task of measuring skinfolds and consequently there was error in measurement. However, a pilot study carried out on a slightly leaner population demonstrated that the experimenter could reliably measure skinfolds (see Appendix B). Moreover, the control group body weight remained static between test 1 and test 2 while the skinfolds total showed a very small non significant decrease. Thus, any possible methodological error was not evident in the control group. Furthermore, the same skinfold caliper was used throughout the tests and and the caliper was calibrated before test 1 and test 2. Therefore, any potential problems with the apparatus can be discounted. Moreover, the experimenter has great experience with skinfold measurement and has taken part in several skinfold reliability studies as well as measuring skinfolds in a middle aged male population which underwent a 10 week running programme. In this running study, the significant 1.4 kg weight loss was mirrored by a similar significant loss in body fat (Grant et al;1988). Furthermore, Hyner et al; (1986) have shown that novices can measure skinfolds as accurately as experienced technicians.

A paired t-test showed that the subscapular skinfold measurement fell significantly in the control group. Despite the small mean decrease of 0.42 mm and the even smaller median decrease of 0.30 mm this fall was significant. It is suggested that the explanation may be that with so great a number of statistical tests undertaken in this study that this significant result occurred by chance. However, it would appear that any methodological or experimental error is not the likely cause of the lack of agreement between the weight loss and a decrease in body fat.

5.4.3 Influence of "Extreme" Subjects

Two subjects may have contributed to the lack of agreement between weight loss and fat loss. In test 1, one subject had a supra iliac score in excess of 50mm. Thus, it was not possible to accurately measure this site as the skinfold calipers would not measure beyond 50mm. Therefore, this score of 50 mm was a conservative estimate of the actual score on this site. This subject's body weight fell from 101.1 in test 1 to 97.5 kg in test 2 and percentage body fat fell from 30.0% to 27.3%. Clearly the difference in percentage body fat from test 1 to test 2 may have beeen greater if the supra iliac site had been measured accurately at test 1.

Greater discrepancy was found with another subject who

lost 8.1 kg between test 1 and test 2 but only a 2.1% decrease in body fat was recorded. When this vast discrepancy between percentage body fat loss and body weight loss was discovered, a subsequent measurement confirmed the readings of test 2. Clearly the test 1 reading could have been decidedly wayward but such a wide discrepancy from the true reading is unlikely. The supra iliac value on test 1 was 21.5 mm. While the supra iliac site can be particularly difficult to measure if this area has large amounts of fat, a value of 21.5 mm on test 1 suggests that this was not the case on this occasion. The subject reported that most of his fat at test 1 was around his abdomen and that he had lost approximately 6 inches around his waist. Thus, the decrease in weight from 96.1 kg to 88.0 kg was not mirrored by a similar fall in percentage body fat. The decrease in weight of this subject accounts for 41.1% of the total mean weight loss of the exercise group whereas the 2.1% fall in percentage body fat in this subject is clearly not representative of the 8.1 kg weight loss.

5.4.4 Internal v External Fat

Some recent cadaver studies (Clarys et al; 1987) indicate that the proportion of subcutaneous fat is greater than internal fat whereas some textbooks suggest that the proportion is 50/50. Nevertheless, it is possible that some internal fat was lost which was

not accounted for by skinfold measurement. However, in a 20 week training programme, Despres et al; (1985) found that training did not affect the proportion of subcutaneous to total fat. Therefore, it is unlikely that preferential internal fat loss over subcutaneous fat contributed to the imbalance between a decrease in body weight and body fat.

5.4.5 Choice of Sites

The four skinfold sites in the Durnin and Womersley, (1974) equations are restricted to the upper body. It may well be that the explanation of the lack of agreeement between body weight loss and percentage body fat loss could be that the four skinfold sites were not sensitive indicators of the total fat loss. It could be that other skinfold sites in the lower body need to be considered when the effects of training programmes on body fat are considered. The Durnin and Womersley, (1974) equations were calculated from moderately sedentary subjects. The population in this study was sedentary. However, recent work on female athletes by Sinning et al; (1984) suggested that other skinfold sites may in fact be more representative of total fat distribution. Substantiation of this hypothesis comes from Taylor, (1988) who assessed body composition in a previously sedentary middle aged men who underwent 30 weeks marathon training. He found that the Durnin and Womersley equations did not reflect body fat losses

compared with densitometry. The author concluded that lower body skinfold sites should be included when changes in body fat are to be assessed. It may be that the Jackson et al; (1978) equations which use 7 sites one of which is below the waist may have greater applicability to studies which examine the the effects of training on body composition. Recent work by Sinning et al; (1984) on female athletes found that the Durnin and Womersley equations over predicted body fat by 3.9% to 4.4% compared to densitometry.

A further reason for considering more skinfold sites is the wide variation in the distribution of fat in different individuals. Clarys et al; (1987), in their extensive research into skinfolds suggest that selection of skinfold sites should include all important storage levels and stress that sites in the legs should be included. These findings highlight the possiblility that the Durnin and Womersley,(1974) skinfold sites are not so sensitive to fat loss prompted by training as other skinfold methods.

5.4.6 Compressibility of the Sites

There is no evidence in the literature that there is a change in the compressibility of the skinfolds. However, training which results in fat loss may in fact reduce compressibility at the site as less fat under the skin will probably reduce skin tension.

5.4.7 Energy Balance

A decrease in body weight and percentage body fat may have been expected in this study as the energy intake of the exercisers remained the same and the energy expenditure increased during the 10 week training period. The magnitude of change in percentage body fat in this study is equivalent to a negative energy balance of 3259 kcals.

The probability of an increase in energy expenditure out with the training sessions was discounted. Brehm et al; (1986) has shown that the extra energy expenditure in the recovery period following steady state exercise only accounted for 3-17 kcal. These findings are in agreement with Durnin, (1985) whose scan of the literature confirms the belief that considerable energy expenditure can be gained after exercise can be misleading. It was not possible to measure energy expenditure during the Tune-ups. However, heart rate during the sessions showed that subjects worked at around 80% of Vo2 Max. Estimated Vo2 Max of 2.6 litres/minute was the value for the exercisers at test 1. Eighty per cent of 2.6 is 2.2 litres/minute which is equivalent to around 11 kcal/minute. The 80% of Vo2 Max was sustained for 20 minutes during the Tune-ups with a gradual decrease throughout the muscle conditioning and flexibility

sections. Figure 2 shows a representative graph of the heart rate during a Tune-up and illustrates that work levels fell in the last 10 minutes of the session. However, work rate did not fall below 50% of Vo2 Max. Thus, it is possible to estimate energy expenditure during the first 20 minutes i.e. 11 kcals/min and 6.5 kcals/min for the last 10 minutes. A deduction of 41.7 kcals per session for the RMR is necessary. The subjects performed on average 27 sessions. Therefore, 27 * (285 minus 41.7) (RMR) = 6569 kcals energy expenditure over the normal resting energy expenditure. This conservative estimate of increase in energy expenditure is equivalent to 0.94 kg of fat loss. This minimum theoretical fat loss has not been mirrored by the estimated fat loss as measured by skinfolds. It is worth remembering that the Durnin and Womersley, (1974) equations have a ± 5.0% error. This error is not systematic in one direction, therefore it is expected that any error would balance out.

5.4.8 Comparison with other Studies

The findings in this study are in agreement with Epstein et al; (1980) whose review has shown that fat loss does not show close agreement with net energy expenditure estimates. Taylor, (1988), in his re-analysis of the Epstein data showed that in almost all of the studies included in the Epstein review, that the theoretically calculated fat loss was greater than the

fat decrease actually measured. Epstein et al; (1988) postulate that the discrepancy between the increased energy expenditure and measured fat loss may be as a a result of two factors. According to Epstein and his colleagues, exercise may "stimulate" appetite or produce a decrease in non exercise activity. Therefore, these possibilities could provide some explanation for the lack of agreement between energy expenditure and fat loss in this study. However, in this study, there was no change in energy intake as measured by the dietary investigation.

The non significant decrease in body weight and body fat in this study is inconsistent with the findings of Wilmore et al; (1970) and Oscai et al; (1968). In both of these studies the subjects trained 3 times per week and the Wilmore study lasted 10 weeks whereas the study of Oscai had 16 weeks duration. The study of Oscai et al; (1968) showed a significant fall of 4.5 kg in body weight and a 3.6% decrease in body fat. The Wilmore et al; (1970) study reported a significant 1.08 kg fall in body weight and a significant 1.11% decrease in body fat. Wilmore et al; (1970) were unable to totally explain the the loss of 1.08 kg after their subjects ran 51.8 miles in 413 minutes. Although the Wilmore study had a much more modest total training time, 413 minutes compared to the 810 minutes covered in this study, it resulted in substantial changes in body composition. The Wilmore study did not measure energy

intake and the authors suggest an involuntary decrease in energy intake as the reason for the unexplained fat loss. The eight exercisers in the Misner et al; (1974) study performed three sessions per week for 30 minutes each session for eight weeks. A significant 2.3% decrease in body fat and a non significant fall in body weight of 0.8 kg was reported. However, the 10 week study of Van Handel et al; (1976) failed to lower either body weight or body fat with a similar training programme to this study. Van Handel et al; (1976) were unable to explain why there was a non significant increase in body fat.

5.4.9 Summary

The results of this study indicate that middle aged men who undertake regular Tune-ups three times per week and wish to decrease body fat cannot do so without alteration in diet.

5.4.10 Future Research

Studies which investigate the effects of aerobic training on body composition must attempt to measure energy intake and energy expenditure as closely as possible. The methodological problems of measuring energy intake and energy expenditure have imposed limitations on the study of exercise and weight loss. While it is possible to estimate energy expenditure

during running and cycling, it is extremely difficult to estimate energy expenditure in indoor training sessions. Moreover, the measurement of energy intake is fraught with difficulties. Furthermore, skinfold measurement of the appropriate sites which are sensitive to change or other measures of body composition may be necessary to improve the accuracy of assessing body composition changes. Clearly, it is desirable to find accurate but practical measures of energy intake, energy expenditure and body composition to clarify the effect of exercise on body composition.

SECTION FIVE

FLEXIBILITY

5.5.1 Pre and Post Training Levels

Both groups had virtually the same initial level of flexibility (28 centimetres). The exercise group showed a very significant six centimetre increase in flexibility whereas the control group score demonstrated a non significant increase of one centimetre. The large improvement in the exercise group is a clear indication that the exercise programme had a beneficial effect on flexibility. This increase is quite surprising considering that only 5 minutes were devoted to flexibility during each session and around 30% of this time was allocated to the lower back and hamstring regions. Thus, the statement of De Vries, (1962) that the lower back and hamstring muscles were very resistant to improvement has not been borne out by this study.

5.5.2 Comparison with other Studies

While there is no threshold for an acceptable level of flexibility as measured by the Sit and Reach Test, this increase has clearly resulted in a marked improvement in flexibility. Other studies which have used the Sit and Reach Test to evaluate changes in

flexibility have used a variety of training regimes, wide ranging age groups and male and female subjects. Therefore, comparison with other studies is difficult. However, the increase of 6 centimetres is the largest increase in flexibility that the author can find in the limited research in this area. While this study designated around 45 minutes to hamstring and lower back flexibility in the 10 week period, Lucas et al; (1984) devoted 105 minutes to the same areas. Clearly the increase in this study of 6 centimetres compares favourably with the 2 centimetre increase in the Lucas study. Unpublished work by Grant showed that six weeks of University "sweat sessions" which included three minutes of flexibility exercises failed to produce a significant increase in flexibility. In the "sweat sessions" less time was given to flexibility and no great emphasis was placed on static stretches. The initial level (as determined by the Sit and Reach Test) of the sweat session was 30 centimetres whereas the baseline level of the present study was 28 centimetres. Therefore, baseline levels cannot be the reason for the different response of the two studies.

5.5.3 Mechanisms of Improvement

No investigation of the possible mechanisms involved in an increase in flexibility was carried out in this study. However, research suggests that long duration stretching with elevated body temperatures are optimal

for the promotion of permanent plastic deformation (Alter,1988; La Ban, 1962). The stretching exercises in the Tune-up were carried out for a fairly long duration (around 20 seconds). Moreover, the stretching exercises were carried out at the end of an aerobic training programme. Therefore, it is to be expected that the body temperature of the subjects was fairly high. Sapega et al; (1981) highlight the fact that the plastic element shows most permanent deformation when muscles are stretched for a long duration and recommend that the body temperature is elevated. In addition, Sapega and his colleagues recommend that ballistic techniques should be avoided. In the Tune-ups, static stretching was emphasized. This methodology was employed to minimise the effect of the stretch reflex and enhance the improvement in flexibility. Sapega et al; (1981) indicate that the group 1a afferents which initiate the stretch reflex respond less intensely to static stretch than to dynamic stretching. Thus, it is not surprising that an improvement in flexibility took place.

5.5.4 Implications for Health

It is clear that the exercise group showed a dramatic increase in flexibility as measured by a Sit and Reach Test. This improvement may have a beneficial effect on posture and reduce the risk of low back pain.

5.5.5 Summary

This study has shown that a limited amount of flexibility exercises conducted three times per week can improve flexibility as measured by the Sit and Reach Test.

5.5.6 Future Research

Further study is needed to establish what is an acceptable level of flexibility in the various joint complexes for different populations. A linkage between performance in flexibility tests of the lower back and hamstring areas and lower back pain is desirable. It may also be important to establish the importance of flexibility programmes in primary and secondary prevention of lower back pain.

SECTION SIX

LOCAL MUSCULAR ENDURANCE

5.6.1 Pre and Post Training Values

The baseline scores of 28 repetitions for the exercise group and control group placed both groups as "fair" in Pollock's norm tables (see Table 9). The increase to 34 and 30 repetitions elevated both groups to the average category. However, the magnitude of increase in the exercise group was significantly greater. The 95% confidence interval of the differences between the exercise group and the control group demonstrates that the increase in the number of repetitions was due to the effect of 10 weeks of Tune-ups. The average increase in the exercise group over the control group was between 3.12 and 6.97 repetitions. The small but significant increase in repetitions of the control group may be explained by the fact that the subjects were more familiar with the test and were able to pace themselves better on test 2. It is difficult to undertake a one minute maximal test and perform optimally without prior experience of the task. Thus, the subjects may have overestimated their capability and gone too fast in test 1 whereas in test 2 the experience from test 1 prompted a less hurried approach at the start of the test.

5.6.2 Comparison with Other Studies

As with other studies, (Capen, 1950; Marcinik et al; 1985) training programmes which have included abdominal exercises in the exercise programme have increased local muscular endurance as measured by sit-ups. It is difficult to compare different training programmes as the training regimes have varied greatly. Moreover, the testing time durations have differed. However, all studies have demonstrated an increase in the sit-up score.

5.6.3 Mechanisms of Improvement

In all Tune-up sessions there are sit-ups and other abdominal exercises. Furthermore, the fact that the exercisers performed other movements such as running, hopping and running with knees up clearly could have an influence on local muscular endurance in the abdominal area. During the Tune-up sessions sit-ups were performed between 30 seconds and one minute at near maximal effort. Therefore, it is to be expected that the lactic acid system would be emphasised in the training. However, there is an aerobic contribution during this time period as well as during running and other activites. In addition, there is a contribution from the ATP and Cp system. Nevertheless, the one minute maximal test clearly places great emphasis on

the lactic acid system. Thus, it is not surprising that the near maximal or maximal efforts on sit-ups resulted in an improvement in the sit-up test.

During maximal exercise it has been found that there are higher levels of lactic acid in the muscles and the blood of trained subjects (Lamb, 1978). It is suggested that this increase in lactic acid is due to the fact that the trained subject can produce greater amounts of lactic acid. Training in young boys and adults has been shown to increase the activity and concentration of the glycolytic enzymes which has the result of speeding up the amount of glycogen broken down to lactic acid (Eriksson et al; 1973; Watson, 1983; Gollnick et al; 1973b)). Eriksson et al; (1973) and Gollnick et al; (1973b) have shown an increase in PFK of 117% and 83% respectively. The subjects in the Gollnick study worked between 75% and 90% of Vo2 Max while the Eriksson subjects participated in a mixture of aerobic and anaerobic activities. However, Houston et al; (1977) failed to demonstrate an increase in lactate dehydrogynase (LDH) activity after training which consisted of hill runs with a large anaerobic component. This form of training produced a significant increase in anaerobic performance as measured by a steep treadmill protocol. Houston et al; (1977) concluded that the absence of an increase in LDH does not exclude the fact that other glycolytic enzymes are the limiting factors in anaerobic performance.

Trained subjects have a greater tolerance for lactic acid and Lamb, (1978) states that this increased tolerance may in part be due to an increase in the motivation of the subject. Astrand and Rodahl, (1986) are in agreement when they concede that it is not certain whether this increased lactic acid tolerance is due to physiological or psychological influences.

Increases in ATP, CP have been shown after training (Eriksson et al; 1973). Karlson et al; (1972) have shown that ATP concentrations have increased after a three month running programme with an anaerobic contribution from "hill work". However, Watson, (1983) highlights the fact that other studies have demonstrated no change in ATP or CP concentrations after training. It should be noted that Gollnick et al; (1973a) concluded that an increase in ATP and CP is unlikely to alter anaerobic capacity.

As the training programme had an aerobic component, it is to be expected that training would produce an increase in the number of mitochondria and an increase in the level and concentration of oxidative enzymes (Ingjer, 1979; Barnard et al; 1970; Gollnick et al; 1973b). These changes would enhance the absorption of oxygen by the muscles and contribute to the increase in the scores of the subjects (Fox, 1979). Moreover, it has been shown that myoglobin levels can rise to

approximately three times that of the untrained (Sharp, 1980a). Thus, it may be that an increase in myoglobin levels provided more time before the abdominal muscles required energy from the lactic acid energy system. Furthermore, an increase in the number of capillaries in muscle with training would improve the blood supply and enhance LME because the increase in blood supply means that more oxygen can be supplied to the muscles (Hudlicka, 1982; Ingjer, 1979). Thus, there is more time for oxygen to diffuse into muscle and provide ATP. Furthermore, there is a greater capacity for waste products to be carried away.

5.6.4 Implications for Health

There is no threshold value which differentiates between an acceptable or unacceptable level of LME in the abdominal area. However, it is clear that the exercise group improved LME as measured by sit-ups. This improvement may have beneficial effects on posture and reduce the incidence of low back pain.

5.6.5 Summary

This study has shown that a 10 week Tune-up programme can improve LME as measured by sit-ups.

5.6.6 Future Research

It would be very appealing to find measures of local muscular endurance and flexibility which could be related to function and be some form of predictor of disability. Thus, potential problem cases could be identified and guidance on progress could be linked to performance in certain tests.

CHAPTER SIX

CONCLUSION

6.1.1 Conclusion

The following variables showed a greater average improvement in the exercise group over the control group from test 1 to test 2:

Resting heart rate
Steady state heart rate
Estimated Vo2 Max
Local muscular endurance (sit-ups)
Flexibility

There were no significant differences between the exercise group and the control group changes in the following variables:

Body weight
Percentage body fat
Blood pressure
Total cholesterol
Triglycerides
High density lipoprotein
Dietary intake
Alcohol consumption

A bradycardia during sub maximal exercise is an accepted indication of the effectiveness of a training programme. The reduction in heart rate reduces the myocardial oxygen cost at a given sub maximal heart rate and improves the efficiency of the heart. The magnitude of decrease in heart rate and increase in predicted Vo2 Max are testimony to the fact that the Tune-up programme was as effective as many other programmes in improving aerobic efficiency.

However, this improvement in aerobic fitness level was not mirrored by a favourable change in blood pressure or lipids. The baseline blood pressure levels were within normal limits and were unlikely to change whereas the almost parallel "improvement" of the exercise and control groups in some of the lipid values indicates that some underlying factor other than exercise influenced these values.

Despite an increase in energy expenditure and no increase in energy intake there was no significant decrease in percentage body fat. Perhaps a study of longer duration would have produced a significant decrease in percentage body fat and body weight.

The significant improvement in local muscular endurance and flexibility demonstrates the effectiveness of the training programme and these improvements may reduce the incidence of back pain in these individuals.

While this study has not shown an improvement in all variables, it has demonstrated an improvement in some health related variables. The use of exercise to promote health is appealing as exercise is cheap, has few side effects and often promotes a feeling of well-being. Further investigations into the efficacy of exercise are needed.

6.2.1 Limitations of Study

Subjects were not randomly selected.

Subjects were selected only from the University community.

The duration of the study lasted only 10 weeks.

The study was conducted immediately after Christmas and certain levels in some variables may not be representative of values throughout the year.

The dietary analysis was limited to two short periods of 5 days. Thus, the analysis may not have been representative of the dietary intake of the subjects.

The methodology of the dietary analysis may be questioned as the programme did not include all foods.

6.3.1 Recommendations for Future Studies

Subjects should be randomly selected.

The duration of the study should be longer.

It may be necessary to select a group with high blood pressure if the potential benefits of aerobic exercise are to be revealed.

The effect of dietary modification and aerobic exercise on body composition should be investigated.

The possibility of establishing a training threshold for improvement in the lipid profile is worthy of further study.

Energy expenditure and energy intake should be measured/estimated as accurately as possible.

Sensitive, but practical measures of body composition should be adopted.

The possibilty of including performance in sit-ups and the Sit and Reach Test measures in the assessment of low back pain has great appeal.

This type of study should be conducted on other sections of the community.

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APPENDIX A.

CONSENT FORM

DEPARTMENT OF PHYSICAL EDUCATION AND SPORTS SCIENCE

HEALTH RELATED FITNESS STUDY.

Informed Consent for Participation in Study and Exercise Tests.

EXERCISE TEST - (Exercisers and Controls).

The test involves 11 - 16 minutes of sub-maximal cycling with a progressive increase in workload. You will be able to terminate this test at any time if you so wish. Body fat, sit-up and flexibility tests will also be conducted. A fasting blood sample will be taken by qualified personnel.

Two tests will be conducted ten weeks apart.

TRAINING PROGRAMME - (Exercisers only).

The study will last for ten weeks during which time you will be asked to exercise in "Tune-ups" three times per week for 30 minutes in each session. Periodically during this time your heart rate will be monitored and you will be expected to complete a training diary regularly.

The tests which are conducted in this study do not constitute a medical examination. If you are in any doubt about your health or your suitability for this study you should consult your doctor.

I certify that to the best of my knowledge and belief, I have no physical or mental illness or weakness that would increase the risk to me of participation in this investigation.

I understand that I can withdraw from this study of my own volition.

SIGNATURE _____

DATE _____

APPENDIX B.

PILOT STUDY.

Objective of the Pilot Study.

B.1.1 Objective of Pilot Study.

A pilot study was undertaken to:

1) Determine the length of time the tests required.

2) Determine the suitability of the order of testing.

3) Determine the subjects' response to the test instructions and the test items.

4) Examine the problems which the experimenter may encounter with his verbal commands, and his operational system.

5) Test the apparatus.

6) Determine the reliability of the test items.

7) Evaluate the practicality of the intended test items.

B.2.1 Methods.

B.2.1.2. Subjects.
Nine subjects were involved in the pilot study. The subjects were aged between 22 and 58 years and were frequent users of the Department of Physical Education and Sports Science at Glasgow University. Thus, all of the group were recreationally active and could be categorised as being "above average" in fitness levels.

B.2.1.3 Equipment.
The equipment used was that described in Appendix D and the methodology employed was that described in the Methods Section of the main study.

B.2.1.4. Procedure.
Subjects came to the Department of Physical Education and Sports Science testing room on consecutive days (at the same time of the day) and underwent the test procedure outlined in the Methods Section of the main study.

B.3.1 Results of Pilot Study.

Tables B1 to B14 give the results of the tests used in the pilot study.

B.3.2 Two-Sided Sign Test.

For a 2-sided sign test one requires zero up to K* plus scores or zero up to K minus scores to achieve a significant 'test effect' at a 5% significance level. A 'test effect' is considered to be a consistent bias from one test to another (Daniel, 1978).

*K = (1 for 7 subjects)
(1 for 8 subjects)
(1 for 9 subjects)

B.4.1 Conclusions of the Pilot Study.

1) The length of the testing time was 30 minutes approximately, and it was felt that this time period was not sufficiently long to cause boredom, physical or mental fatigue or inhibit participation of the subjects in the main study.

2) The order of testing was considered to be suitable. The preceeding tests did not appear to produce significant fatigue in subsequent tests. The test instructions were adequate and no problems were encountered with the equipment.

3) A 2-sided sign test showed that there was no "test effect" from the pre-test to post-test (Daniel, 1978).

TABLE B1

SYSTOLIC BLOOD PRESSURE (mmHg)
Reliability Tests Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	119	113	-6
CG	110	111	+1
BS	114	117	+3
GMc	121	123	+2
AF	123	131	+8
GS	133	131	-2
JH	134	139	+5
BC	136	140	+4
MD	149	150	+1

Direction of Change 7 Plus, 2 Minus.

TABLE B2

DIASTOLIC BLOOD PRESSURE (mmHg)

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	52	56	+4
CG	72	72	0
BS	75	76	+1
GMc	75	74	-1
AF	81	85	+4
GS	67	64	-3
JH	82	82	0
BC	76	79	+3
MD	75	72	-3

Direction of Change 4 Plus, 4 Minus, 1 Zero.

TABLE B3

RESTING HEART RATE(beats/minute)

Reliability Tests Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	53	56	+3
CG	56	60	+4
BS	81	67	-14
GMc	73	54	-19
AF	48	55	+7
GS	57	59	+2
JH	59	59	0
BC	44	40	-4
MD	77	82	+5

Direction of Change 5 Plus, 3 Minus, 1 Zero.

TABLE B4

BICEP SKINFOLD (millimetres).

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	3.6	3.2	-0.4
CG	5.2	4.3	-0.9
BS	3.8	4.0	+0.2
GMc	3.5	3.6	+0.1
AF	5.0	5.5	+0.5
GS	2.8	2.4	-0.4
JH	4.8	4.8	0
BC	3.4	3.3	-0.1
MD	3.0	3.2	+0.2

Direction of Change 4 Plus, 4 Minus, 1 Zero.

TABLE B5

TRICEP SKINFOLD (millimetres).

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	8.4	8.0	-0.4
CC	8.2	9.2	+1.0
BS	11.5	11.1	-0.4
CMc	6.0	5.8	-0.2
AF	10.2	11.2	+1.0
GS	5.7	5.1	-0.4
JH	10.5	10.5	0
BC	8.5	8.5	0
MD	6.1	5.5	-0.6

Direction of Change 2 plus, 5 minus, 2 Zero.

TABLE B6

SUBSCAPULAR SKINFOLD (millimetres)

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	9.0	9.0	0
CG	12.0	11.8	-0.2
BS	10.0	10.2	+0.2
GMc	9.1	8.6	-0.5
AF	11.2	11.2	0
GS	8.5	9.0	+0.5
JH	9.8	10.0	+0.2
BC	9.5	10.0	+0.5
MD	8.0	8.2	+0.2

Direction of Change 5 Plus, 2 minus, 2 Zero.

TABLE B7

SUPRAILIAC SKINFOLD (millimetres)

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	6.1	5.5	-0.6
CG	13.5	14.6	+1.1
BS	12.0	10.0	-2.0
GMc	7.2	5.8	-1.4
AF	13.8	10.8	-3.0
GS	5.2	5.2	0
JH	16.5	16.8	+0.3
BC	5.8	5.1	-0.4
MD	5.7	8.0	+2.3

Direction of Change 3 Plus, 5 Minus, 1 Zero.

TABLE B8

PERCENTAGE BODY FAT.

Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	11.5	10.8	-0.7
CG	21.0	21.4	+0.4
BS	20.3	19.6	-0.7
BMc	15.4	14.3	-1.1
AF	19.2	19.0	-0.2
GS	13.1	12.9	-0.2
JH	16.8	16.9	+0.1
BC	16.9	16.7	-0.2
MD	9.4	10.5	+1.1

Direction of Change 3 Plus, 6 Minus

TABLE B9

STEADY STATE HEART RATE (beats/minute)
MEAN OF HEART RATE BETWEEN TWO AND THREE MINUTES IN THE FIRST WORK LOAD.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	86	82	-4
CG	75	77	+2
BS	97	88	-9
GMc	98	91	-7
AF	82	84	+2
GS	92	98	+6
JH	93	92	-1
BC	70	71	+1
MD	107	105	-2

Direction of Change 4 Plus, 5 Minus.

TABLE B10

STEADY STATE HEART RATE (beats/minute)
MEAN OF HEART RATE BETWEEN TWO AND THREE MINUTES IN THE SECOND WORK LOAD

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	115	117	+ 2
CG	109	107	- 2
BS	117	107	-10
GMc	-	-	-
AF	104	107	+ 3
GS	125	131	+ 6
JH	117	114	- 3
BC	-	-	-
MD	138	136	- 2

Direction of Change 3 Plus, 4 Minus.

TABLE B11

STEADY STATE HEART RATE (beats/minute)
MEAN OF HEART RATE BETWEEN FOUR AND FIVE MINUTES IN THE FINAL WORKLOAD

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	142	148	+6
CG	138	135	-3
BS	139	136	-3
GMc	142	141	-1
AF	141	142	+1
GS	156	159	+3
JH	147	143	-4
BC	132	136	+4
MD	167	164	-3

Direction of Change 4 Plus, 5 Minus.

TABLE B12

ESTIMATED VO_2 MAX ($ml.kg^{-1}\ min^{-1}$)

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	68.7	64.8	-3.9
CC	59.7	62.0	+2.3
BS	48.6	51.5	+2.9
GMc	39.8	40.8	+1.0
AF	55.5	54.4	-1.1
GS	49.1	47.9	-1.2
JH	59.3	61.6	+2.3
BC	45.3	42.8	-2.5

Direction of Change 5 Plus, 4 Minus.

TABLE B13

SIT AND REACH TEST (Centimetres)

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	34	36	+2
CG	02	05	+3
BS	35	35	0
GMc	28	28	0
AF	35	37	+2
GS	17	20	+3
JH	20	21	+1
BC	25	24	-1
MD	38	37	-1

Direction of Change 5 Plus, 2 Minus, 2 Zero.

TABLE B14

SIT-UPS (REPETITIONS)
Reliability Study Conducted in Pilot Study.

SUBJECT	TEST 1	TEST 2	SIGN TEST
JM	47	46	-1
CG	51	52	+1
BS	30	35	+5
GMc	38	37	-1
AF	49	51	+2
GS	40	43	+3
JH	39	42	+3
BC	33	32	-1
MD	43	40	-3

Direction of Change 5 Plus, 4 Minus.

APPENDIX C.

Total Cholesterol Precision Checks.

C.1.1 Results of Total Cholesterol Precision Checks.

The following results give details of the analytical precision checks for total cholesterol at the Western Infirmary, Glasgow.

Three patient plasmas with differing cholesterol concentrations - LOW = 4.9, MEDIUM = 7.6 and HIGH = 10.6 mmol/litre were assayed. Six replicates of each level were assayed 10 times over a period of 5 days. Quality control sera were also included in each run. (See Table C1). For the low level plasma, the within batch coefficient of variation (CV) ranged from 1.6 to 2.7% with the individual run means ranging from 4.84 to 5.09 mmol/litre, over the 10 run period (See Table C2). For the medium levels plasma, the corresponding figures were 1.4 to 4.6% for the CV, with the individual run means ranging from 7.83 to 8.06 mmol/litre (See Table C3). For the high plasma, the figures were 0.6 to 2.8% for the CV, with the individual run means ranging from 10.49 to 10.77 mmol/litre (See Table C4).

In addition, statistics were done on each individual run, which contained 6 replicates of each cholesterol level plus quality control material - i.e. 25 samples in each run, giving a range of cholesterol concentrations from 4.0 to 10.8 mmol/L. t-tests and correlation/regression were carried out for each run, there being no signfiicant difference between any one run and any/all of the others. The correlation coefficients obtained from each of the 10 runs were correlated against the others in turn, and were all greater than 0.995. The median cholesterol values for each of the 10 runs ranged from 7.92 to 8.09 mmol/L, with the difference between means ranging from 0.02 to 0.21 mmol/L.

Thus, it has been demonstrated that the reliability of total cholesterol measurement is extremely high.

TABLE C1.

PRECISION CHECK ON TOTAL CHOLESTEROL (mmol/litre)

ASSAY RUN	1	2	3	4	5	6	7	8	9	10
Low	4.96	4.87	5.06	5.12	4.99	4.95	4.88	5.14	5.10	5.19
Level	4.87	5.15	4.91	4.77	4.81	4.73	4.89	4.89	4.86	4.95
Plasma	5.03	5.06	5.11	5.04	4.99	5.20	5.06	5.09	5.05	5.25
4.9	5.09	5.00	4.95	4.88	5.02	5.23	4.75	5.03	4.99	5.15
	4.87	4.95	4.99	4.97	4.88	50.1	4.78	4.86	4.83	4.90
	4.99	5.07	5.06	4.95	4.95	5.20	4.70	5.00	4.96	5.08
Medium	7.90	7.83	7.98	7.78	7.95	7.87	7.87	8.10	8.04	8.02
Level	7.76	7.63	8.01	7.85	7.67	7.56	8.08	7.53	7.48	7.93
Plasma	8.08	7.94	7.57	7.83	7.95	8.06	7.90	7.97	7.97	8.09
7.6	7.92	7.71	7.66	7.96	7.75	7.77	7.49	8.08	8.03	8.09
	7.81	8.00	7.87	7.81	7.73	8.13	8.00	7.69	7.64	8.31
	7.82	7.88	7.91	8.10	8.67	8.14	8.06	8.13	8.08	7.03
High	10.66	10.86	10.69	10.70	10.52	10.80	10.53	11.14	11.06	11.02
Level	10.68	10.71	10.45	10.65	10.55	10.29	10.58	10.83	10.75	10.47
Plasma	10.86	10.55	10.63	10.41	10.66	10.75	10.60	10.75	10.68	10.83
10.6	10.75	10.62	10.51	10.30	10.53	10.51	10.32	10.70	10.63	10.43
	10.86	10.40	10.33	10.03	10.48	10.70	10.43	10.67	10.59	10.63
	10.68	10.50	10.34	10.66	10.49	10.74	10.33	10.53	10.46	10.13
	QUALITY CONTROL MATERIALS WITH ASSIGNED VALUES.									
	4.05	4.10	4.30	4.16	4.13	4.19	4.02	4.19	4.17	4.04
	10.52	10.30	10.64	10.43	10.63	10.85	10.20	10.48	10.41	10.39
	4.06	4.18	3.95	4.04	4.08	4.10	3.98	3.95	3.92	3.95
	10.47	10.12	10.66	10.50	10.78	10.64	10.62	11.03	10.95	10.53
	8.44	8.72	8.43	8.45	8.61	8.28	8.37	7.97	7.91	8.29

TABLE C2.

PRECISION CHECK ON TOTAL CHOLESTEROL (mmol/litre)

Low Level Plasma - 4.9 mmol/litre

ASSAY RUN	1	2	3	4	5	6	7	8	9	10
X	4.97	5.02	5.01	4.96	4.94	5.05	4.84	5.00	4.97	5.09
SD	0.09	0.10	0.08	0.12	0.08	0.19	0.13	0.11	0.10	0.14
CV%*	1.8	1.9	1.6	2.4	1.6	3.8	2.7	2.2	2.0	2.7
MIN	4.87	4.87	4.91	4.77	4.81	4.73	4.70	4.86	4.83	4.90
MAX	5.09	5.15	5.11	5.12	5.02	5.23	5.06	5.14	5.10	5.25

* Coefficient of Variation.

TABLE C3.

PRECISION CHECKS ON TOTAL CHOLESTEROL (mmol/litre)

Medium Level Plasma - 7.6 mmol/litre

ASSAY RUN	1	2	3	4	5	6	7	8	9	10
X	7.88	7.83	7.83	7.89	7.95	7.92	7.91	7.92	7.87	8.06
SD	0.11	0.14	0.18	0.12	0.37	0.23	0.22	0.23	0.25	0.14
CV%*	1.4	1.8	2.3	1.5	4.6	2.9	2.8	2.9	3.2	1.7
MIN	7.76	7.63	7.57	7.78	7.67	7.56	7.49	7.58	7.48	7.93
MAX	8.08	8.00	8.01	8.10	8.67	8.14	8.08	8.13	8.08	8.30

* Coefficient of Variation.

TABLE C4.

PRECISION CHECKS ON TOTAL CHOLESTEROL (mmol/litre)

High Level Plasma 10.6 mmol/litre

ASSAY RUN	1	2	3	4	5	6	7	8	9	10
X	10.75	10.68	10.49	10.53	10.54	10.63	10.55	10.77	10.70	10.59
SD	0.08	0.24	0.14	0.31	0.06	0.19	0.17	0.21	0.20	0.30
CV%*	0.7	2.2	1.3	2.9	0.6	1.8	1.6	1.9	1.9	2.8
MIN	10.66	10.40	10.33	10.03	10.48	10.29	10.33	10.53	10.46	10.17
MAX	10.86	11.12	10.69	10.96	10.66	10.80	10.82	11.14	11.06	11.02

* Coefficient of Variation.

APPENDIX D.

DETAILS OF EQUIPMENT USED IN THE STUDY

D 1.1 Copal Digital Sphygmomanometer. UA-251.
The Copal sphygmomanometer was made in Japan and supplied by:

Andrew Stephens,
Medical Electronics,
41 Dickson Road,
Blackpool.

The accuracy of this sphygmomanometer was checked before test 1 and test 2 by comparing readings with a standard sphygmomanometer. The readings were found to be similar.

D 1.2 Monark Bicycle.
The Monark bicycle ergometer was made by:

Monark Crescent AB,
Varberg,
Sweden.

The model number was 818. Calibration of the bicycle was carried out regularly by following the instructions in the Monark handbook. Calibration discs of a known weight were used in the calibration process.

D 1.3 T15 Tunturi Cardiotester.
The Tunturi cardiotester was made by:

Tunturipyora,
Untamonkatu 2,
20520,
Turku,
Findland.

Verification of the heart rate readings was made regularly by checking the heart rates at varying intensities using a stethoscope.

D 1.4 Skinfold Caliper.
The skinfold caliper was made by:

Holtain Limited,
Crosswell,
Crymuych,
Dyfed,
Wales, SA 41.

Calibration of the skinfold caliper was carried out using the standard calibration technique with accurately measured weights.

D 1.5 Sit and Reach Apparatus.
The Sit and Reach apparatus was made by:

The Works Department,
University of Glasgow,
Glasgow.

D 1.6 Centrifugal Analyser.
The Cobias-bio centrifugal analyser was made by:

Roche Diagnostics,
Wellyn Garden City.

D 1.7 Heart Rate Recorder.
The Sport Tester PE 3000 heart rate recorders were made by:

Polar Electric DY and supplied by:
Hampden Sports Limited,
Moray House College of Education,
Crammond Campus,
Edinburgh.

University of Glasgow
Department of Physical Education and Sports Science
Health Related Fitness Study

Name:_____ Week commencing:_____ Week No:____

Training Zone___ To ___

	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Exercise							
Training heart rate							
R.P.E.							
Alcohol consumption							
Tobacco usage							
Comments							

F1.1 EXAMPLES OF TUNE-UP SESSIONS.

F1.2 Tune-up Session.

1. WARM-UP

Jog 1 min,
1 min, of - Skip 1 length, jog ends, skip length, etc,
1 min, of - Cross gym, heels up 1 width - jog 1 width,
1 min, of - 10 knees up on spot, 10 stride jump and 10 jog on spot,
1 min, of - Cross gym, side slip 1 width, cross-over 1 width,

2. AEROBIC SECTION

(i) B B B

A

C C C

Inverted cycle x 10

A) Skip in volleyball court x 30, Tucked sit-up x 10,
B) Step-ups x 10, Stride jumps x 10,
C) Single leg squat thrust x 6

Perform A, B, or C,
Walk/jog/run 1 lap,
Repeat,
1 min, on each x 2

A

(ii) B B

Walk/jog/run on diagonal
Exercise A on side
Exercise B on end

1) A - Side slip, B - 5 Burpees
2) A - High knee skip B - Heels up
3) A - Jog backwards B - Elbow support cycle x 10
4) A - Knees up B - Single leg squat,

2 mins, 15 secs, on each

3. MUSCLE CONDITIONING

Press up	30 secs,
Sit-up	30 secs,
Back raise (wide/together/down)	30 secs,
Single leg step up	30 secs,

Repeat 15 secs, on each and then 30 secs, on each,

4. FLEXIBILITY

1) Hamstring stretch
2) Quad stretch
3) Calf stretch
4) Shoulder stretch
5) Trunk rotation
6) Sit and Reach Stretch

F1.3 Tune-up Session.

1. 1) Jog 1 min.
   2) Travel east - heels up
      Travel west - skip
      (lower gym - north/south) 1 min.
   3) Travel east - side step
      Travel west - run
   4) X gym - mini-strides/long strides alternate 1 min.
   5) 20 paces jog, 20 paces medium, 20 paces fast clockwise for 1 min.

2.

Inverted cycle x 10	Step-up OR	
Step-up x 10	Stride jump x 10	Prone stride x 10
	Small benches	

Walk/Jog — 2 mins in North Half, 2 mins, in South Half, X 2

Walk/Jog

Heels up on spot x 10	Back Raise x 10	Skip x 20 (Rope)
Knees up on spot x 10		

TAKE HEART RATE

3.

20 secs	20 secs	20 secs
1)Tucked sit up	Stride jump	X gym jog or walk
2)Alt leg raise (front lying)	Loop press up	X gym skip or walk
3)T position knee raise	split squat	X gym side step or walk
4)Side raise	Side raise	X gym cross-over or walk
5)Elbow support leg x-over	Inverted cycle	Knees up - punch air/jog on spot
6)Burpee	Elbow extension	X-gym jog or walk

4. STRENGTH
   1) Press up (thumbs touch) 20 secs)
   2) Elbow support cycle 20 secs)
   3) Single leg step up 20/20 secs) X 2
   4) Back raise wide/together down 20secs)
   5) Sit ups 20 secs)
   6) Leg raise on side 20/20 secs)

   FLEXIBILITY
5. 1) Snatch position
   2) Calf stretch - push into wall
   3) Butterfly stretch
   4) Forward/backward split
   5) Ankle grasp
   6) Shoulder stretch
   7) Hamstring Stretch

F1.4 Tune-up Session

WARM UP (5 mins)

1. Jog 1 min,
2. Bend & stretch 30 secs,
3. Slip step 30 secs
4. Elbow knee 30 secs,
5. Skip 30 secs,
6. 4 Corners 30 secs,
7. Cross-over 30 secs,
8. Jog 1 min, 5 mins,

AEROBIC 2 GROUPS A's and B's

1. Diagonal Work for 1 min, 10 secs to changeover
   A's turn left to do step ups at benches x 10 and
   continue on to diagonal pathway jogging,
   B's turn right to do sit-ups on floor x 5 and
   continue on to diagonal pathway jogging,
   Change over
   Second time through;
   A's do prone strices at benches x 10
   B's do stride jumps x 10
   Repeat Total of 8-9 minutes

TAKE PULSE

2. In pairs, Work in middle of gym doing exercise while partner jogs/walks one lap, Change over, Continuous work for 1 min, 10 secs, to change over,

   Exercises 1. Stride jumps
   2. Step kicks
   3. Sit ups
   4. Heels up,

   Total of 4-5 minutes

STRENGTH (5 mins)

1. Press ups 30 secs)
2. Sit ups 30 secs)
3. ½ Squats 30 secs) X 2
4. Back raise 30 secs)
5. Triceps 30 secs)

FLEXIBILITY (5 mins)

1. Ankle grasp
2. Calf
3. Quad,
4. Shoulders
5. Hamstring
6. Neck - sides and front only

Repeat to end,

F1.5 Tune-up Session.

SECTION A

1) 1 min. jog. Change direction
2) Skip 20 secs.
   4 corner stretch 20 secs. Change direction
3) Jogging with high knees. 20 secs.
   Bend and stretch 20 secs. Change direction.
4) Half pace run 20 secs.
   Stamp feet 10 secs.
   Punch arms 10 secs. Change direction.
5) Jog 20 secs.
   Twist 4 punch arms 4. 20 secs.
6) Skip 20 secs
7) Figure of 8 run/jog/walk 1 min. 5 mins.

SECTION B

Do activities *A B C* as many times as possible

A Front to back lying 5-10 reps
B Skip 20 turns reps.
C Step ups 10-15 reps. 3 mins.

SECTION C Use lengths.

1)	30 secs.	Slip step	20 secs.	Sit ups.
2)	30 secs.	Jog	20 secs.	Press ups.
3)	30 secs.	Skip	20 secs.	Alt. leg raise.
4)	30 secs.	Cross over run	20 secs.	Stride jumps

5) 20 secs. Medium intensity run/jog
   20 secs. Max. intensity run/jog
   20 secs. Low intensity run/jog/walk

SECTION D

Same arrangement as Section B - working for 3 mins.

A Squats 10
B Skipping 20
C Burpees 5-10

SECTION E REPEAT SECTION C

STRENGTH EX.

1) On front, alt. leg raise. 1 min. - as much rest as is needed
2) Alt. knee sit ups. 1 min. - as much rest as is needed
3) Side bends. 1 min. - as much rest as is needed
4) Wt. on feet and hands. Walk hands in and out, bend at elbows for seat press and repeat.
   1 min. - as much rest as is needed
5) Lying on R. side, raise R. leg. Lying on L. side, raise L. leg. 1 min. - as much rest as is needed.

SECTION F - FLEXIBILITY

1) Easy jog shuffle. 15 secs.
2) Calf stretch using wall. 10-15 secs. each side.
   Easy shuffle jog. 15 secs.
3) Thigh stretch. 10-15 secs. each leg.
   Easy jog shuffle.
4) Sit on floor. Stretch arms into air and hold X 2.
5) Half butterfly stretch. Right and left.
6) Hamstring stretch.
7) Stand up stretch to R. side and to L. side.
8) Jog shuffle 15 secs.

F1.6 Tune-up Session

WARM UP

1)	Jog	1 min
2)	Heels up x 10 - leg kick x 10 - stride jump x 5	1 min
3)	Jog clockwise - knees up in middle third	1 min
4)	K gym - mini strides/skip	1 min
5)	20 paces slow/20 paces fast	1 min

AEROBIC SECTION

A	B	C
I	I	I

1 min on each x 2

II	II	II

A) Prone strides x 10
Hop 4L 4R
Tucked sit up x 5
Walk/jog

B) Single leg step x 10
X over step
Back raise x 5
Walk/jog

Ci) Inverted Cycle x 10
High skip

Cii) Burpee x 5
Walk/jog

iii	Aii	i

1 min 15 sec, on each x 2

Skip	Skip

Ai) Step up x 10
ii) Prone strides x 10
iii) Inverted cycle x 10
Touch wall at end - jog to other end - repeat.

B) 15 sec, easy
15 sec, fast,

C) Skip (rope) x 20
Walk/jog length
Skip (rope) x 20

TAKE HEART RATE

Strength

1) Press up (thumbs touch)
2) Sit up
3) Squat
4) Tricep extension
5) Knee raise (T-position)
6) Back raise

25 sec, on each x 2

Flexibility

1) Shoulder stretch (at wall)
2) Quad stretch (at wall)
3) Calf stretch (at wall)
4) Side bend
5) Trunk rotation
6) Hamstring extension