The relationship between exercise tolerance and a single rate of perceived exertion as modified by training among older male and female subjects

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THE RELATIONSHIP BETWEEN EXERCISE TOLERANCE AND A SINGLE RATE OF PERCEIVED EXERTION AS MODIFIED BY TRAINING AMONG OLDER MALE AND FEMALE SUBJECTS

by

David Harry Hugh Williams

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy of the Loughborough University of Technology.

1st. December, 1995

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ABSTRACT

Most testing and training studies make objective measurements of responses to exercise by means of a wide range of physiological parameters of varying degrees of sophistication. However, rarely do such studies take as their central theme how the individual feels in response to a test or to exercise, before and after training. Some studies even conclude that a period of training has had no measurable effects on their subjects because the range of measurements made before and after training was unchanged - yet the subjects report that they felt better and could cope with exercise more effectively.

Thus, the aim of this thesis is to describe a series of studies which examined the physiological responses to exercise of middle-aged subjects with the emphasis placed on their subjective reaction to that exercise before and after training. Rather than employ a battery of psychological tests to assess such reactions the simple, but effective, expediency of using a single Rate of Perceived Exertion (RPE) was adopted.

The first Experimental Study (Exp.1) was exploratory in its attempts to reveal how best to distinguish between active and inactive middle-aged men. It employed a graded exercise test (GXT) but found a difference of only 33W between the two groups at similar heart rates when testing was carried out over a wide range of exercise intensities. The second Experimental Study (Exp. 2) examined, firstly, the differences in exercise intensities between active and inactive middle-aged men when heart rates were maintained at a constant level of 140 beat.min⁻¹ over 30 min (Exp. 2a). Differences averaged only 32W when using this test protocol. However, during the second part of the study (Exp. 2b), where subjects self-selected and maintained a power output using the criterion of 'Just Tolerable Discomfort' over 30 min, the active group worked, on average, at twice the load of the inactive group: 127W vs 64W respectively. This finding was deemed worthy of further investigation.

Consequently two training studies were carried out, each of 12 weeks' duration. For the first of these, Training Study 1 (TS1), a computerised data logging system was devised in order to administer the 30 min test to 35 middle-aged men: 24 experimental subjects and 11 controls. The single, prescriptive RPE was set at 14. The experimental group, whilst maintaining this rating throughout, was able to demonstrate a 50% higher power output following training. Measures of functional capacity, metabolic, hormonal and blood lipid responses were monitored in equating with this improved exercise capacity. Also, anthropometric measurements, including ultrasonic and water displacement techniques revealed hypertrophy of the thigh muscles of the
experimental subjects. This resulted in a 50% increase in the forces generated by these muscles when applied to a maximum voluntary contraction (MVC) test over 20s.

The limitations discovered during TS1 were overcome during the second Training Study (TS2) by enabling exercise intensities to be altered in keeping with the fluctuating sense of stress of maintaining an RPE of 14 over the time span of a 30 min test. The training-induced adaptations were also more clearly revealed by including a repeat test, post-training, at pre-training values. Of the 44 subjects: 16 males, 14 females and 14 controls, the males increased their power output by 30% following training, the females by 50%. The improved protocol enabled changes in functional capacity, metabolic, hormonal and blood lipid parameters, as well as in RPE itself, to be more clearly identified. Dietary analyses additionally provided a more incisive interpretation of calorific subdivisions especially as related to lipoproteins within the total picture of lipid metabolism.

During both training studies a Power Lactate Test was used as a model in order to identify metabolic changes in work intensity following training by means of a reference lactate value of 4 mmol.l⁻¹. Significant increases in exercise intensity occurred at this threshold following training whereas relative metabolic demand was stable at approximately 63%VO₂max. for both males and females whether before or after training. This exercise intensity was usefully identified with an RPE of 13.2, providing a benchmark by which the principle 30 minute tests could be assessed.

A carefully selected group of field tests, determined over distance and time, placed greater emphasis on the subjective control of predicted VO₂max. From these measurements, and particularly those of the two training studies, a strong relationship was identified between what may be regarded as a 'set point' RPE14 and relative work intensities as portrayed by a %RPE 14. The relationship was strong enough, especially following training, to be regarded as a dependence. These data suggested that a self-selected and self-regulated sense of stress was a useful frame of reference for the individual participant faced with a training regimen and was fully capable of monitoring adaptations in functional capacity and in metabolism, as modified by age and training. The method could be considered a particularly valuable model for the autogenic choice of the intensity of a training programme among older subjects whose level of fitness may be unknown.
ACKNOWLEDGEMENTS.

This work was completed in the Department of Physical Education, Sports Science and Recreation Management, Loughborough University.

I am deeply indebted to Professor Clyde Williams for making the production of this thesis possible. He generously made available the biochemical facilities of the Department and was able to perceive far more in my initial studies than I was able to discern myself. The result was that, through his unique guidance and considerable patience in answering my numerous queries, the research was able to burgeon. He continued to help resolve my numerous problems and was especially helpful in those related to the presentation of papers at International Conferences. Though it has been difficult to live up to his own exemplary standards of scientific excellence, I hope that, through his encouragement and particular flair in generating stimulation for his subject, I have become a better scientist than formerly.

I am also extremely grateful to Dr. Henryk Lakomy for the use of his specially adapted isometric chair and his helpful advice after reading the first draft of the section on anthropometric leg measurements and leg forces. Without his BBC microcomputer data logging programme for the first training study, subsequently modified for the second study, the 30 minute test would have been extremely difficult to administer. Further invaluable help was provided by means of a BBC programme which facilitated calculations of leg skinfold measurements, written by Mr. David Kerwin. Professor Peter Jones of the Human Sciences Department, kindly loaned the ultrasonics equipment and provided crucial training on its calibration and use. He also made available the two volumetric water tanks in order to measure thigh volumes.

Especial thanks are extended to Mr. Stephen Brooks for his meticulous guidance, often into the small hours of the morning, through the intricacies of the blood lipid, and catecholamine, assays and to Dr. Alan Nevill of the School of Sport and Exercise, Birmingham University, for the time and care that he took in analysing the considerable mass of data from the two 30 minute studies by means of the Biomedical Data Package. His interpretation of the results was invaluable.

I am more than gratefully appreciative of the enthusiasm and dedication of the large group of subjects who participated in the studies of this thesis; without their commitment these findings would have remained no more than a theoretical concept. Above all I would like to express my heartfelt thanks to my wife, Dorothy, to whom this thesis is dedicated. Her stoical support, together with her sustained patience and, at times, cooling of the fevered brow and what lay beneath, stretched the marriage vows to their limit. I am happy to report that they proved equal to the test.
PUBLICATIONS.

Unless otherwise indicated through acknowledgements or references to published literature, the work contained herein is that of the author. The findings presented in this thesis have been reported, in part, in the following publications:


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CHAPTER 1

1. INTRODUCTION

*Every adaptation is an integration.*

_Sir Joseph Barcroft (1934).

Exercise acts as a stimulus to the cardiovascular system and has the potential to induce adaptations. It provides a stress under which the ability of the heart to pump blood can be explored. In this regard, it can be viewed as a scientific instrument, manipulated in the laboratory by means of a spectrum of tests and work intensities. Repeated exposure to its influence can generate a 'training effect' which can provide a new threshold of physiological homeostasis. Under such conditions, the performance of the heart is very largely determined by adjustments within the peripheral circulation. When normal and sedentary young subjects are conditioned for 2-3 months, for example, increments in maximal cardiac output and systemic arteriovenous oxygen difference contribute about equally to the rise in maximal oxygen uptake (VO\textsubscript{2max})(Saltin et al., 1968; Rowell, 1974; Clausen, 1977). The increase in cardiac output has been found to be owing, entirely, to the increase in maximal stroke volume whereas the increase in arteriovenous oxygen difference has been attributed to the peripheral circulatory changes (Saltin and Rowell, 1980). In some subjects the increase in VO\textsubscript{2max} with physical conditioning is owing to the rise in maximal stroke volume and cardiac output almost completely; oxygen extraction is unaltered. This has been seen in middle-aged men (Hartley et al., 1969; Saltin et al., 1969) and middle-aged and elderly women (Klibom, 1971). Such responses present the case for identifying training-induced adaptations through heart rates and oxygen pulses alone as meeting two of the requirements of the Fick principle. And since, at common submaximal exercise intensities, oxygen uptake is as equally stable as arteriovenous oxygen differences at maximal intensity, comparisons between sedentary and trained middle-aged subjects should be possible on the basis of heart rates and pulse rates. As maximal heart rate is generally stable within any given age group (Rowell, 1986) it should also be possible for submaximal work intensities to provide an inherent safety factor, especially if the end point can be controlled by the subject, and more clearly reveal changes induced by training.

Therefore, on the premise that proportional and progressive physiological changes can be identified primarily by heart rates using a submaximal test of incremental intensity, the first of
the Experimental Studies (Exp. 1) was carried out. Its purpose was to identify differences in
heart rates in terms of power output when comparing active and inactive middle-aged men.

Though more can be learned about how a system operates when it is forced to perform,
through exercise, than when it is idle, the middle-aging process, with which this thesis is
concerned, superimposes its own physiological and psychological strictures. Among one of
the most influential physiological components is that of diminished compliance associated
with decreased chest wall capacity (Mittman et al., 1965; Turner et al., 1968) and increased
vascular stiffness (Lakatta, 1986) with the result that the dimensions of the heart are not easily
altered by training in older people (Saltin et al., 1969). This has led some workers to question
the trainability of older subjects and assert that physical training is futile after 50 to 60 years
unless the subject has been trained in earlier life (Hollman, 1964; Nocker, 1965; Kuroda,
1988). With progressively reduced heart rates and maximal oxygen uptakes after the age of
25 years (Robinson, 1938; Astrand, 1960; Pollock et al., 1978a; Hollman and Hettinger,
1980; Hollman et al., 1988) these limitations in functional capacity may be revealed at an
earlier age than that of the 50-60 year band, though the average improvement of 15% of
VO$_2$max. with physical conditioning appears to be common across a wide age range: 20 - 50
years (Saltin et al., 1968; Rowell, 1974; Clausen, 1977). Certainly, older subjects start with
lower values for VO$_2$max. and tend to improve less (Saltin, 1969). Much depends on the
starting, threshold level for each individual. It could be that the peripheral adjustments that
determine oxygen extraction take place rapidly and the long-term central circulatory changes,
which may involve morphological adjustments in the heart, progress more slowly. Neither the
VO$_2$max. test, therefore, nor an incremental test which progresses some way towards
achieving VO$_2$max., regardless of the subject's freedom in manipulating its end point, may be
the most suitable methods of determining differences between the sedentary, and the
trained, conditions of middle-aged subjects.

Rather than rely on the compliance of an ageing heart which may be a suspect measuring
instrument at increasing, or extreme, exercise intensities, a common, single submaximal heart
rate could be utilized as a criterion in determining peripheral responses. The focus would then
be upon a centrifugal factor, embodied in work intensities whose tolerance could be
examined over a set period of time rather than centripetal considerations of the heart's
plasticity. Thus, in developing the theme of Experimental Study 1 but focussing attention on
the changes in functional capacity revealed when a single heartrate was sustained over a set
period of time of 30 minutes, the second Experimental Study (Exp.2a) was developed and
applied to active and inactive middle-aged men.
An alternative to the tester-orientated approach would be to transfer the onus for load-setting and hence, of heart rate responses, into the hands of the subject. The physiological impact, it was felt, could be explored in a situation where a subject is required to choose his own exercise intensity using the term: 'Just Tolerable Discomfort'. In this psychological milieu, the subject would be in control but would be required to exercise a certain responsibility in determining his own endurance capacity. Such a situation is unusual in the laboratory where subjects are invariably presented with a task to which they may be asked to respond subjectively; rarely do they set their own work limits. The second part of Experimental Study 2 (Exp. 2b) was mounted using this method with the purpose of distinguishing, more clearly, differences between the trained and untrained conditions of middle-aged subjects and as an amplification of Experimental Study 1.

Results from the cross-sectional findings of Experimental Study 2b) were sufficiently interesting to merit further research by means of a longitudinal training study of sedentary, middle-aged men. It has been found that the further the initial VO\(_2\)max is below 45 ml.kg.min\(^{-1}\) then the greater the relative and absolute increment with conditioning (Rowell, 1986) although low, initial values can indicate, paradoxically, both 'slight' and 'high', trainability (Bouchard and Thibault, 1986). One of the questions to be addressed was whether VO\(_2\)max was a necessary measure in order to explain some of the special adaptations of aging man and woman and, if so, how best to administer it safely. If it is the single, most important determinant of endurance fitness (Shephard et al., 1968; Astrand and Rodahl, 1977; Sharkey, 1970), could it reveal a reliable training effect among subjects whose adaptation to training, because of the ageing process, may be in doubt? A further reservation is that changes in VO\(_2\)max have not always been found to be a valid index of adaptations to training.

Endurance performances, for example, have been shown to improve even after the response to training, as evidenced by VO\(_2\)max., had ceased (Daniels et al., 1978). Conversely, the reduction of the aerobic demands of submaximal training may enhance performance even in the absence of changes in VO\(_2\)max. (Daniels and Oldridge, 1971) leading to the conclusion that training-induced increases in endurance capacity may well be independent of VO\(_2\)max. (Bland and Williams, 1982). The inverse relationship between the percentage increase and the pre-training value for VO\(_2\)max. (Saltin et al., 1969) suggests that one of the virtues of the VO\(_2\)max. measure is not necessarily its validity as a criterion of functional capacity. Rather it could provide a more useful means of converting absolute values of submaximal intensity into readily comparable measures of relative exercise intensity within, and between, experimental groups of subjects of differing gender and training status. It is with this viewpoint in mind that the VO\(_2\)max. parameter was utilised during the two longitudinal training studies, Training
Study 1 and Training Study 2, subsequent to the Experimental Studies. The VO$_2$max. in these studies was predicted from a variety of field tests which provided the subjects with more time in order to achieve exhaustion, or near exhaustion, compared with, often brief, standardised laboratory tests of VO$_2$max. This, again, placed greater emphasis on a subjective control of energy expenditure during a demanding test.

The ability of an individual to sustain a high relative exercise intensity not only reflects the oxidative capacity of the skeletal muscle (Sjodin et al., 1976; Henriksson and Reitman, 1977) more accurately than VO$_2$max. but also permits the direct comparison of endurance performance between individuals with diverse VO$_2$max. values. Therefore it was considered desirable to utilise this parameter in view of its advantages and the age range of the subjects involved. But instead of obtaining it by means of a prescribed power output as in the normal course of laboratory practice, the rating of 'Just Tolerable Discomfort' was converted to the Borg Scale of Rate of Perceived Exertion (RPE) of 14 which it was designed to mimic. This single RPE was used during both training studies and provided an opportunity for psychological, as well as physiological, factors to influence performance. The administration of field, and laboratory, tests, repeated following training could, it was considered, provide a learning effect which has been found elsewhere (Daniels et al., 1977) under more standard conditions. Increased motivation following training may also enhance the relative effort of subjects using this single RPE14 and help to explain the findings of Experimental Study 2b.

Therefore the aim of the training studies presented in this thesis was to explore the implications of a self-regulated form of testing based on a single rate of perceived exertion of 14 using the Borg Scale (1962, 1985). Changes in the submaximal physiology of sedentary middle-aged men and women were examined following a regimen of training with the intention of identifying some of the mechanisms on which this self-determination of a given load may be dependent.
CHAPTER 2

2. REVIEW OF LITERATURE

'Stress is a biological concept, and whether it is viewed as psychological or as physiological is simply a function of the measurement paradigm a sport scientist elects to adopt'.

Rod K. Dishman, 1983.

2.1 INTRODUCTION

With the challenge of submaximal, dynamic exercise being manipulated endogenously by the subject, the regulation of the choice of workload by means of a suitable scale of effort takes on a crucial significance. Before being able to perform a subsequent bout of endurance exercise from the choice that he has made, the subject will have consciously, or subconsciously, critically evaluated his capacity to complete the work in the light of his level of fitness, his mental and physical preparedness, his skill and his age. This is an onerous task for a sedentary subject who is about to cross physiological and psychological boundaries which had previously been unknown to him. The adaptations which training provide may instil a new confidence which could yet have to be tempered by caution if a subject's new choice of power output is not to cause him embarrassment by not being able to be sustained. This omni-present human weakness can be overcome by the technology of a cybernetic 'governor', leaving the subject greater freedom of choice within the limits of a test to which he has committed himself. The precise form of this 'governor' is revealed in Training Study 2 (Chapter 7). It also provides the sports scientist with a more sensitive tool with which to identify those parameters that have most closely influenced the subject's choice. This review is consequently devoted to an overview of the scale of stress that was modified for use in these studies. Changes in the training status related to functional capacity, metabolism, gender, age and anthropometry are also considered, not as isolated physiological components but as elements cumulatively capable of modifying a subject's choice of power output when faced with having to complete an endurance test at an obligatory level of stress.

2.2 PERCEIVED EXERTION

Attempts to understand human performance require the integration of numerous scientific disciplines, not least those of psychology and physiology in relation to a study and understanding of perceived exertion. The dichotomy of physiological psychology, simply
defined as 'the study of physiological mechanisms of behaviour' (Morgan, 1943), can embrace the idea that perceptual cues serve as a primary source of information in physical performance, enabling individuals to regulate work intensity to the demands of a given activity or skill. Feedback from such cues can take the form of both general feelings of exertion and fatigue and also specific sensations varying in number and degree, such as muscular and joint pain, dyspnoea and heart arrhythmias (Borg and Noble, 1974). Despite these numerous, innate components, it appears to be generally accepted that the perception of stress is the 'gestalt' (Borg, 1962a) of many sensations and feelings related to the work either required, being performed, or just having been completed (Borg and Noble, 1974; Mihevic, 1981).

Kinsman et al., (1973) proposed an hierarchical schema in order to integrate the various levels of subjective symptoms accompanying the fatigue associated with prolonged cycle ergometry. This model has been applied to the perception of effort (Pandolf et al., 1975, 1978). It proposes that the undifferentiated, or overall, rating of perceived exertion represents a 'superordinate' level of subjective evaluation resulting from the cohesion of a number of discrete sensations but yet one place removed from the identification of specific sensory inputs. Certainly the search for a primary perceptual cue appears to represent a rather simplistic attempt to probe the complex psychobiological dynamics of the exercise response (Mihevic, 1981). The consensus of opinion favours a perceptual response to exercise as evaluated in terms of various modifying variables. These include exercise intensity, exercise modality, steady-state versus progressive exercise, and exercise duration.

Unless a physiological response is subject to conscious monitoring during exercise, it has been suggested that it is unlikely to act as a potent sensory cue for the perception of effort (Edwards et al., 1972). Yet perceptual psychologists regard perception as a sensory process dissociated from conscious awareness even though some individuals, notably elite distance runners, have been found to monitor somatic responses actively during exercise as a means of gauging and modulating exercise intensity (Morgan and Pollock, 1977). The solution appears to be both diffuse and complex, for whereas a conscious awareness of certain discrete physiological cues is likely to play its role in the evaluation of perceived exertion, the total exercise experience would appear to be determined by the conscious and unconscious integration of multiple physiological responses culminating in a conscious perception of effort.

It might be assumed that such effort could be embraced by primitive classifications using terms like 'light', 'moderate', 'very heavy' (Borg, 1986). Other attempts to quantify the boundaries of stress range from the purely anecdotal to the pseudo-precise (Williams, 1982), including well established relationships such as the 'Hurt - Pain - Agony' barriers associated
with swimming (Counsilman, 1970). But this type of classification has nothing to do with psychophysical scaling which is the scientific process of determining how the intensity of the perception grows with physical intensity and can best be described by mathematical models. A prerequisite for basic psychological studies was the development of methods that may be classified as ratio-scaling which provide measurements on a scale with a zero point and equidistant scale values, say from 0 - 10, depicting length, weight, temperature or work intensity, for example. Through the work of Stevens and his collaborators at Harvard (Stevens, 1953, 1957, 1966) and also of Ekman and his coworkers in Stockholm (Ekman, 1958, 1959, 1961) ratio methods were developed and applied to a large number of experimental studies. The methods were found to work well in most sense modalities (Stevens and Galanter, 1957; Stevens, 1971). These psychophysical methods passed through the fire of critical evaluation during the 1960s and survived physiological validations to become scientifically acceptable for general descriptions and comparisons among different modalities (Borg et al., 1967; Franzen and Offentoch, 1969). In other modalities studied with psychophysical ratio-scaling methods, power functions with exponents ranging from 0.33 (brightness) to 3.5 (subjective electrical intensity) were found to describe the variation of perceived intensity with physical intensity (Stevens and Galanter, 1957; Stevens, 1971). The first psychophysical studies of muscular work, performed in the late 1950s using short term exercise on a cycle ergometer (90rg and Dahlstrom, 1959, 1960) showed that perception of effort grows as a positively accelerating function of exercise intensity described by the expression:

$$R = a + cS^n$$

where $R$ = subjective force, $a$ = basic perceptual 'noise', $c$ = measure constant and $S$ = exercise intensity. The exponent 'n' was found to be 1.6 in this instance, whereas the subjective perception of handgrip force was determined at 1.7 (Stevens and Mack, 1959), that for isometric leg force 1.6 (Eisler, 1962), for weight lifting, 1.45 (Stevens and Galanter, 1957) and that for walking on the level, an exponent of 3 (Borg, 1961a, 1962a). Thus, during a variety of physical activities, it is apparent that there is no consistent proportional increase in subjective intensity in step with increasing power output.

These scaling methods, however, yield only ratios between subjective intensities, rather than absolute levels and consequently were of little help in practical work involving inter-individual differences. The advent of good ergometers helped to resolve the problem since studies could be conducted over work of longer duration (Borg, 1961a, 1962a; Borg and Dahlstrom, 1962; Hueting, 1965; Borg and Linderholm, 1967, 1970). Out of such work, Borg (1962a) devised a model in which he assumed the subjective range from a basic perceptual 'noise' level up to maximum intensity was the same for all subjects despite the fact that stimulus range
in terms of exercise intensity, for example, differed. Thus the constant 'c' in the above
equation, could be solved for each individual, as:-

\[ c = \frac{R_t - a}{\sin \theta} \]

where \( c \) = maximal level, \( R_t - a \) is the same for all subjects and \( \sin \) (work intensity, varied as a
function of time) determined for each individual. Though this model was subsequently
validated using heart rate as a criterion and was valuable for inter-individual comparisons, it was
of little assistance in applied studies. A simple scale was needed which could be used by
everyone rather than exclusively by trained university students.

Several different scales have been tried but the one most often in use is the scale for rating
perceived exertion (RPE). It started out, after trial and error (Borg, 1962b), as a 21-point
graded category scale on which every second number was anchored with expressions from 3
= 'very, very light' to 19 = 'very, very laborious' with 11 = 'neither light nor laborious'. The same
adverbs were entered on the lower part of the scale as on the upper in a symmetrical way and
the scale was found to possess high correlations with heart rates (\( r = 0.80 \) to 0.90) among
normal subjects (Borg and Linderholm, 1967) as well as in young individuals differing in
activity levels and body composition (Skinner et al., 1973). The scale was subsequently
modified to span 15 points which ranged from 6 to 20 to approximate one-tenth of heart rate
(Figures 3.1 and 3.2). The aim was to increase the linearity between RPE and power output
on the premise that the relationship between heart rate and power output was linear at
submaximal exercise intensities. The RPE scale was constructed to follow heart rate for work
on the cycle ergometer and for healthy middle-aged men performing moderate-to-hard work
so that heart rate should be about 10 times the RPE value. As with other age groups, the RPE
produced a very high correlation of \( r = 0.85 \) with absolute heart rates, when the work intensity
was varied from light, to heavy, work. However, using the same absolute power output,
correlations were low, varying from \( r = 0.20 \) to 0.50, especially with low loads. Not surprisingly,
the scale stimulated research into its scientific authenticity with studies focussing upon the
effect of gender (Mihevic, 1979; Mihevic and Morgan, 1980), fitness levels (Ekblom and
Goldberg, 1971; Patton et al., 1977) and type of exercise performed (Horstman et al., 1979)
on ratings of perceived exertion. Findings confirmed that RPE grows linearly with power
output and heart rate.

The heart rate for a certain workload is both a direct measurement of the degree of physical
stress and an indirect one of the physical working capacity (W). Analogous to the application of
heart rate measures, ratings of perceived exertion may be used to estimate W. To obtain
reliable and valid measurements, several exercise intensities should be administered with stepwise increases as in the tests designed by Sjostrand (1947) and Wahlund (1948). In the former, the test continues until the subject has reached a certain predetermined heart rate such as 170 beat.min\(^{-1}\) for young men and 150 beat.min\(^{-1}\) for middle-aged, or older, subjects. The construction of the rating scale makes it very simple to calculate the power output that a subject can manage at a reference level of, for example, 17 on the RPE scale. Since the rating of 17 roughly corresponds to the heart rate of 170 for most subjects between 25 and 50 years of age the WR17, or the power output at a rating of 17, corresponds to W170, or the power output at a heart rate of 170 beat.min\(^{-1}\) (Borg and Noble, 1974).

The reliability of the physical working capacity measurements based on ratings of perceived exertion were compared with those based on heart rates (Borg and Linderholm, 1970) in a group of 54 healthy male subjects. Intra-test correlations were determined from two different W130 measurements: the first, obtained by using the heart rate from 49 and 147 watt.min\(^{-1}\), and the second, by using the heart rate from 98 and 196 watt.min\(^{-1}\) from the same test. An overall reliability correlation coefficient of 0.91 was obtained whilst the WR13 was found to be 0.92. The error of a single measurement was thus only about 5% of the mean value, or 9.8 watt.min\(^{-1}\). Test-retest reliability coefficients were also determined in a group of 9 male and 10 female patients and a group of 12 male and 5 female healthy subjects (Borg and Linderholm, 1970). The W130 and W170 test-retest correlation coefficients were: \(r = 0.93\) and 0.98, respectively, in the patients and \(r = 0.88\) and 0.97, respectively, in the healthy subjects. The corresponding WR13 and WR17 correlation coefficients were \(r = 0.80\) and 0.94, respectively, in the group of patients and \(r = 0.91\) and 0.98, respectively, in the healthy subjects. Such correlations reveal that the reproducibility based on repeated measurements, is about as good for the WR data as for those of WHR. With these relationships in mind, the protocol for the first of the Experimental Studies was devised.

Although ratio scaling methods provide a means of monitoring functions, they fail short of any precise intensity levels by which individuals can be compared. The RPE scale, however, has the advantage of providing direct feedback of perceptual intensities and is consequently preferable in most practical situations and has been utilised throughout the present series of studies. However, it is sometimes desirable to obtain estimates of perceptual intensities that can be described both with regard to functional variation and 'absolute levels'. To do this, a category scale with ratio properties is needed. By placing verbal expressions on a numerical ratio scale, a combination of the two types of scales can be formulated. Borg (1982a) devised such a scale and called it a 'category-ratio' scale which is identified as follows:-
The advantage of Borg's (1962b, 1973) 15 point RPE scale is that it facilitates direct interindividual comparisons among exercise intensities or time periods. Clinicians and physiologists are no longer suspicious of the 'cardboard technology' (Noble, 1982) known as the 'Borg Scale'. Through its parsimony and versatility (Mihevic, 1981) it has become the most frequently employed and arguably, the most successful method for the measurement of perceived exertion. Yet its simplicity has often been misunderstood and misused. For example, ratings estimated during a stress test have been used in an attempt to control exercise intensity during training by the replication of similar efforts of exertion (Noble, 1982). This is probably beyond the remit intended for the scale. Other authors (Stamford and Noble, 1974; Robertson et al., 1979a; Robertson et al., 1979b) have devised a 9 point category scale for rating perceived exertion. Though values from this scale correlate highly with ratings obtained from the RPE scale (Borg, 1973), the results should be integrated cautiously with the RPE literature owing to the major differences between the structure of each of the scales, as already defined.

Perceived exertion, like pain, is a subjective, quite personal and extremely complex sensation. Defined as 'one's subjective rating of the intensity of work being performed' (Morgan, 1973), the important consideration is frequently not what the individual is doing but rather what he thinks he is doing. This seemingly arbitrary means of assessment has proven to be scientifically valid. Findings indicate that normal subjects are capable of consistently identifying differences in power output by means of Borg's psychophysical RPE category scale (Morgan, 1973). Furthermore, Bartley (1970) has suggested that greater understanding
of such perceptual sensitivity can be found by the inclusion of 'homeostatic' and 'comfort' systems. They embrace mechanisms of 'awareness' or 'experiential bodily comfort' and can be identified with sensory systems including pain, temperature, kinesthesia, touch and even some of the receptors involved in homeostatic processes such as carotid sinus receptors, sensory endings in the walls of the venae cavae and pulmonary veins (Bartley, 1970; Borg and Noble, 1974). If the average correlation coefficient between heart rate and RPE is approximately 0.82 ($r^2=67\%$), this leaves 33% of the variance which could well be explained by psychometric factors (Morgan, 1973) and which has been incorporated in the term 'Just Tolerable Discomfort' of Experimental Study 2.

2.2.1 CENTRAL AND PERIPHERAL FACTORS.

2.2.1a. HEART RATE (HR).

Despite Borg's (1961b, 1962a) original thesis of a multiple physiological 'gestalt' of integrated responses to account for the sensation of perceived exertion, he also acknowledged that perception of effort was dependent upon input from both 'musculature and the system of circulation' (Borg, 1962a). His early work was primarily devoted to the relationship of the RPE scale with heart rate using the cycle ergometer (Borg 1961a, 1962a, 1962b; Borg Linderholm, 1967), though it was equally applicable to treadmill exercise (Borg, 1973). Ekblom and Goldbarg (1971) were the first to argue that the greater local, or peripheral, muscular strain during ergometer cycling was the primary source of perceptual input and that this was applicable to both ergometer and treadmill exercise. The weight of their assertion has since been supported by a considerable mass of evidence as a comparison between selective central (Table 2.1) and peripheral (Table 2.2) factors reveal. Secondary central factors, according to these authors, consisted of 'pulmonary ventilation and circulation'. Although this central classification is unambiguous, it is as well to remember that the use of the term 'central' in the broader physiological and psychological literature refers to the central nervous system. A further consideration in attempting a quantitative weighting of either central or peripheral factors, is that a dozen different variables can be measured, all of which might be important to the assessment of perceived exertion but not one of which could provide a reductionistic 'explanation' (Borg, 1986). Only the sense of taste has shown that much of the perceptual intensity variation could be related to the afferent neurophysiological responses to the taste nerve (Borg et al., 1967). Not many situations occur where such simple 'explanations' are possible. Often there are many physiological functions to use for the deciphering of a multivariate combination. Unfortunately, much of the research has ignored the complexity and has been bivariate in nature (Mihevic, 1981) as illustrated by the dichotomy of central and peripheral components. This is acceptable under the aegis of a
TABLE 2.1
CENTRAL FACTORS

RPE related (●) or not related (Δ) to specific parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference</th>
<th>Related</th>
<th>Reference</th>
<th>Non-related</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>Bar-or et al., 1972</td>
<td>(●)</td>
<td>Pandolf et al., 1972</td>
<td>(Δ)</td>
</tr>
<tr>
<td></td>
<td>Edwards et al., 1972</td>
<td>(●)</td>
<td>Henriksson et al., 1972</td>
<td>(Δ)</td>
</tr>
<tr>
<td></td>
<td>Sargeant et al., 1973</td>
<td>(●)</td>
<td>Noble et al., 1973</td>
<td>(Δ)</td>
</tr>
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<td></td>
<td>Skinner et al., 1973</td>
<td>(●)</td>
<td>Pandolf et al., 1973</td>
<td>(Δ)</td>
</tr>
<tr>
<td></td>
<td>Winsman et al., 1976</td>
<td>(●)</td>
<td>Sidney et al., 1977</td>
<td>(Δ)</td>
</tr>
<tr>
<td></td>
<td>Borg, 1985</td>
<td>(●)</td>
<td>Van den Burgh, 1986</td>
<td>(Δ)</td>
</tr>
<tr>
<td></td>
<td>Borg et al., 1986</td>
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<td></td>
<td>Plus 4</td>
</tr>
<tr>
<td>VE, RR</td>
<td>Edwards et al., 1972</td>
<td>(●)</td>
<td>Stamford et al., 1974</td>
<td>(Δ)</td>
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<td></td>
<td>Morgan et al., 1973</td>
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<td></td>
<td>Robertson, 1982</td>
<td>(●)</td>
<td>Lollgen et al., 1980</td>
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<tr>
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<td>(●)</td>
<td>Henriksson et al., 1972</td>
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<td>Gamberale, 1972</td>
<td>(●)</td>
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<td>(Δ)</td>
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<td></td>
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<td>(●)</td>
<td>Pandolf, 1977</td>
<td>(Δ)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Plus 1</td>
</tr>
<tr>
<td>%VO₂max.</td>
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<td>(●)</td>
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<td></td>
<td>Ekblom et al., 1971</td>
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<td>(●)</td>
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<td>Eston et al., 1987</td>
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<tr>
<td></td>
<td>Hill et al., 1987</td>
<td>(●)</td>
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</table>

HR = Heart Rate. VE = Minute Ventilation. RR = Respiratory Rate. VO₂ = Oxygen Uptake. %VO₂max = Relative Exercise Intensity. Plus number = further supportive references, not included.
single paradigm of the rate of perceived exertion which is now well established. But it requires the acceptance of an understanding that, though local, peripheral signals may be potentiated by central factors (Catarelli, 1977) at one level of exercise intensities, the roles may be reversed at another. Since we already know that physiological response to quite dissimilar exercise modes is highly specific, it is not surprising that perceptual response is also mode specific varying, for example, between swimming, cycling, running and arm cranking. Likewise it seems logical that perceptual signals would vary with exercise intensity since the contributions of biomechanical, biochemical and cardiorespiratory factors are known to fluctuate as a function of intensity (Noble et al., 1986).

Two types of study may help to illustrate how experimental problems associated with the above are overcome. Firstly, RPE has been examined when heart rates, oxygen uptake and power output were held constant. Henriksson et al., (1972), for instance, studied concentric and eccentric work at two pedal frequencies of 30 and 60rpm using the cycle ergometer. They found that at the same power output, 30rpm was perceived as greater than 60rpm for both types of work. Pandolf and Noble (1973) studied the same problem, using concentric work only, and found that 40rpm work was subjectively judged to be more stressful than 60 and 80rpm. Thus some factor, other than heart rate, oxygen uptake and power output, must have accounted for the increased exertional perception at the lower pedal frequencies. Henriksson et al., (1972) speculated that muscle tension may be a factor in the higher exertional ratings since it must be doubled during the contractions to sustain the same power output at half the frequency. Pandolf and Noble (1973) pointed to the involvement of local, peripheral factors, possibly muscle and joint discomfort and/or anaerobic metabolites, as contributing to this exertion-rating phenomenon.

In the second type of study, it has been found that heart rate and oxygen consumption, recorded while walking and running at the same velocity, display similar patterns. Metabolic costs were greater for running at low velocities while the reverse was observed at high velocities. Walking and running heart rate, or oxygen consumption, were also seen to intersect at approximately 5mph (8km.hr⁻¹)(Boje, 1944; Noble and Borg, 1971). Noble et al., (1973a) re-examined these findings with the purpose of determining whether perceptions of exertion could be accounted for by concurrent physiological responses. Borg's (1962a) initial concept emphasised that subjective ratings closely followed the physiological strain exhibited by gross metabolic variables such as heart rate and oxygen consumption. The investigation by Noble et al., (1973b) found that such a general correspondence did exist when metabolic differences between modes were great, such as at low and high velocities. However, statistical analysis demonstrated that the Perceived Exertion Intersection Point occurred at a
significantly lower velocity than the Metabolic Intersection Point. This suggested that the physiological variable, heart rate, was not an exact mirror image of perceived exertion. Within the range of heart rates recorded, run-perceived-exertion was always lower than walk-perceived-exertion at the same heart rates. For example, at a heart rate of 150 beat.min⁻¹ running RPE was 10.3 compared with 12.1 for the walking RPE. Thus, again, some factor or factors other than heart rate must have been operating in one or other of the modes to serve as a basis for the perceptual differences. So perceived exertion is not always a function of metabolic equivalence alone but also of the stress placed on local musculature in the accomplishment of a task (Noble et al., 1973a; Table 2.2). When mechanical resistance increases during walking at higher velocities, though metabolic differences between walking and running is less acute, local muscular strain is enhanced, resulting in a higher perceived exertion.

These examples help to throw into perspective the relative contributions of central and peripheral physiological feedback but they do not explain the complexity or timing of those contributions. Although perceived exertion generally follows heart rate responses, for instance (Noble et al., 1973b), it has never been suggested that there is a causal relationship between RPE and heart rate (Borg and Noble, 1974). The equation: \( HR = RPE \times 10 \), was constructed more for the purposes of illustrating the validation of the scale than for exact productive use, though it has been found to function relatively efficaciously in a large group of middle-aged men (Grimby et al., 1972). Correlation coefficients between 0.77 and 0.90 (Borg, 1962b; Skinner et al., 1969; Bar-Or et al., 1972), computed over a wide range of exercise intensities, demonstrate that a high percentage of common variance is shared by perceived exertion and heart rate. The criticism here is that such relationships were created from non-random experiments, but studies have shown that the relationships remained when tests were administered in random order (Borg and Noble, 1974). A further modification was that when plots of the increase in heart rate and RPE with increasing exercise intensities, were superimposed on each other, it was clear that the prediction of heart rate from perceived exertion proposed by Borg (1962b, 1973), applied most reliably at higher exercise intensities (Horstman, 1977; Horstman et al., 1979).

The correlation between heart rate and RPE has been tested further by manipulative use of blocking agents in order to disrupt 'normal' conditions (Ekblom and Goldbarg, 1971). Beta-adrenergic receptors were blocked (BAB) with propanolol in 14 subjects, mean age 24yr (21-32yr.); parasympathetic receptors were then blocked with atropine (PSB) and both tests compared with controls (C). Maximal heart rate was the same in PSB as in C but, on average, 38 beat.min⁻¹ lower after BAB. For exercise intensities corresponding to 25%, 50% and 75%
VO₂\textsubscript{max}, the increase in heart rate with PSB was 28, 15 and 10 beat.min\textsuperscript{-1} respectively, higher than in C. After BAB, heart rates for a given absolute, submaximal load were lower than for C: for 25%, 50% and 75% VO₂\textsubscript{max}, heart rate was 13, 35, and 37 beat.min\textsuperscript{-1}, respectively, lower. For a given submaximal heart rate, such as 125 beat.min\textsuperscript{-1}, RPE varied from 8 with PSB, 11 with C and 15 with BAB. There were very high correlations between heart rates and RPEs for each of the three tests in support of a sustained linearity throughout. From such experiments it was clear that neither tachycardia nor bradycardia were the primary factors for the setting of RPE during exercise. It was confirmed that in many work situations, heart rate mirrored the physical strain subjectively experienced (Ekblom and Goldbarg, 1971). However, when heart rate was manipulated by autonomic blockade (Hartzell et al., 1986) or influenced by factors other than those of metabolism, such as excitement after cigarette smoking (Astrand and Rodahl, 1970; Goldberg et al., 1971), power output or oxygen uptake could be used as an alternative to heart rate for the evaluation of physical strain as reflected in a common linearity with RPE. A similar finding was made following cycle ergometer tests on a patient suffering from atrial fibrillation (Borg and Dahlstrom, 1962).

Experimental conditions that include both lowered (Bergh, 1980), and elevated (Rowell, 1974a), body temperature can provide abnormal environmental conditions in order to study, even more comprehensively, the relationship between RPE and heart rate. The RPE - heart rate relationships were affected differently by low body temperature during ergometer cycling as compared with breast-stroke swimming (Bergh, 1986). In the former exercise modality, the relationship was unchanged by lowered body temperature whereas in swimming, RPE was elevated at given levels of heart rate. It has been suggested that this was merely a result of the fact that the heart rate - oxygen uptake relationship was altered by low body temperature, providing a lower heart rate in relation to a given oxygen uptake (Holmer and Bergh, 1974). This could be attributed to an inhibition of sensory impulses, since low body temperature is known to induce a reduction of the nervous impulse frequency (Vanggaard, 1975). Such a mechanism is compatible with observations during rewarming from low body temperature by cycle ergometer exercise. In these experiments, subjects reported a momentary loss of sensation from the exercising legs (Bergh, 1986) comparable with the sensations experienced by some competitors during the transitional stages of triathlon competitions (Trew, 1992). Certainly at the lower levels of oxygen uptake, heart rate and workrate, there was a marked tendency towards lower ratings of perceived exertion associated with lowered body temperature. This trend, however, became less pronounced and was sometimes reversed at higher physiological values (Bergh, 1986). Conversely, at elevated temperatures, RPE was increased at most cycle ergometer work levels. In relation to heart rate, RPE was lower during heat stress though revealing a convergence at higher heart rates. This indicated
that the subjects perceived a given rate of work as being more strenuous during heat stress compared with cool conditions, as might be anticipated. The heart rate, however, appears to increase relatively more than RPE (Bergh, 1986) and this has been demonstrated by other workers (Pandolf et al., 1972; Annwall, 1975; Holmer and Arvidsson, 1975). After 5 minutes of recovery from exercise in the heat, RPE was found to be unaffected by heart rate in the range: 75 - 140 beat.min⁻¹. These data suggest that heart rate per se is not an adequate stimulus for the perception of exertion.

2.2.1b. OXYGEN UPTAKE (VO₂).

These fluctuating and equivocal relationships between heart rate and RPE are mimicked by those between oxygen uptake and RPE. Since there is a known linear relationship between heart rate and oxygen uptake, therefore it is not surprising that RPE is also linearly related to oxygen uptake (Borg and Noble, 1974). Consequently, in so far as heart rate and oxygen uptake reflect physiological strain, RPE can also be used as a 'strain indicator' (Borg and Noble, 1974) for one parameter equally as well as for the other. If the concept of perceived exertion is to be considered as a 'gestalt', 'accumulative hypothesis' (Noble et al., 1973b), or 'multiple integration of many factors' (Pandolf, 1982) and physiological processes, it may seem spurious to analyse the contribution of one parameter, such as oxygen uptake, out of the context of the summation of all the cardiorespiratory contributions. However, the 'gestalt' is a discrete, physiological-perceptual model which, if it is to be verified, requires certain physiological processes to be experimentally manipulated. Key parameters may therefore need to be assessed as to their role in the whole sensation of exertion, be it conscious or unconscious. Oxygen uptake is an especially pivotal parameter; it controls the functional behaviour of the heart in supplying the tissues' demands for oxygen; as the reciprocal of carbon dioxide production it is associated with the determination of pulmonary ventilation through the 'accelerator -brake' mechanism (Keele et al., 1973; Green, 1976; Guyton, 1976; Mountcastle, 1980) and by implication, the acid-base balance of the blood; as a relative proportion of maximal oxygen uptake it can reflect the trained or untrained state of the individual in terms of a given submaximal work intensity. Only at 75% VO₂max., for example, have oxygen uptake and heart rate been found to make significant contributions to central ratings (Ekblom and Goldbarg, 1971) though it is unlikely that oxygen consumption, as with heart rate, is sensed directly (Mihevic, 1981).

The use of such a relative work intensity, combined with oxygen uptake, has been found to be accurately predicted by RPE (Glass et al., 1992). Fifteen physically active, male subjects, mean age 22.4 (±3.1yr) completed a graded exercise test on a motor driven treadmill where oxygen uptake, together with heart rate and RPE, were measured each minute. An exercise
prescription was then developed from the RPE obtained, using the Karvonen (1957) formula to determine 75% of heart rate reserve. Two days later, the subjects were given a 10 minute exercise bout on a level treadmill. They were allowed to adjust the treadmill speed to achieve the required 'target RPE' in order to obtain the 75% heart rate reserve. They did this with no significant differences in either oxygen uptake or minute volume between the two tests and, after 6 minutes, heart rate for the 10 min. test was within 4 beat.min\(^{-1}\) of the graded test value. The study demonstrated that a subject's perceptual response can be used to prescribe accurately, exercise intensity during level treadmill running in keeping with the ACSM (1991) suggestion that: 'The individual participant's RPE response to a graded exercise may be employed in specifying the RPE level for conditioning'. Inadvertently, the study also illustrated that, since oxygen uptake at the same absolute, submaximal work intensity is unchanged (Varnauskas et al., 1966; Williams and Hamley, 1986) the RPE is fully capable of identifying such a criterion at a given 'setting'. The advantage of using RPE as a method of exercise prescription in this way is that its value 'in the field' enables the individual to readjust his pace, guided solely by his perception of effort, rather than having to stop in order to measure his heart rate.

Unfortunately, generalised assumptions are not always upheld when the exercising mode is changed or conditions are varied using the same apparatus. In order to study the effects of carrying excess weight on perceived exertion, 8 lean and 8 obese young men, mean age (±SD) 18.5 (±0.8yr) were tested to maximum on a cycle ergometer and on a treadmill. Each lean subject was given 'excess weight' in the form of a lead-weighted vest and belt equal to the excess weight, in terms of fat, of his matched obese counterpart. The average amount of weight needed was 19kg (range 16.8-22.0 kg). Both groups were then tested twice under the control conditions and twice, again, under the experimental conditions, that is, with lean subjects wearing excess weight. There were no significant differences observed between RPE-oxygen uptake relationships on the treadmill whilst the lean subjects reported higher RPEs on the ergometer than the obese. Of particular note was that all subjects rated the ergometer work to be harder than the treadmill exercise at the same oxygen uptake. And when 15 highly fit male subjects, mean age 20.2 (±1.1yr) performed three different work levels whilst ergometer cycling at equivalent power outputs for pedalling speeds of 40, 60 and 80rpm, metabolic costs, as revealed by oxygen uptake at each of the loads, did not differ significantly (Pandolf and Noble, 1973). The RPE responses at equivalent exercise intensities were generally negatively related to pedalling speed, though the differences were not statistically significant between 60 and 80rpm. The elevated RPE at 40rpm compared with the perceptual responses at higher pedalling speeds but equivalent power output, was particularly evident. Several investigations have demonstrated that muscular force needed to
### TABLE 2.2

PERIPHERAL FACTORS

RPE related (+) or not related (Δ) to specific parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Related</th>
<th>Reference</th>
<th>Not-related</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate</td>
<td>(+)</td>
<td>Ekblom et al., 1971</td>
<td>Kay et al., 1969 (Δ)</td>
</tr>
<tr>
<td></td>
<td>(+)</td>
<td>Edwards et al., 1972</td>
<td>Sargeant et al., 1973 (Δ)</td>
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<td></td>
<td>(+)</td>
<td>Gambarele, 1972</td>
<td>Stamford et al., 1974 (Δ)</td>
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<td></td>
<td>(+)</td>
<td>Pedersen et al., 1977</td>
<td>Robertson et al., 1979 (Δ)</td>
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<tr>
<td></td>
<td>(+)</td>
<td>Young et al., 1982</td>
<td>LolIgen et al., 1980 (Δ)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Plus 4</td>
<td></td>
</tr>
<tr>
<td>Proprio. L&amp;M.</td>
<td>(+)</td>
<td>Ekblom et al., 1971</td>
<td>Edwards et al., 1972 (Δ)</td>
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<tr>
<td></td>
<td>(+)</td>
<td>Henriksson et al., 1972</td>
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<td></td>
<td>(+)</td>
<td>Noble et al., 1973</td>
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<td></td>
<td>(+)</td>
<td>Pandolf et al., 1973</td>
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<td>(+)</td>
<td>Borg et al., 1974</td>
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<td>(+)</td>
<td>Stanford et al., 1974</td>
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<td></td>
<td>(+)</td>
<td>Ekblom et al., 1975</td>
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<td>(+)</td>
<td>Lollgen et al., 1975</td>
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<td>(+)</td>
<td>Pandolf et al., 1975</td>
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<td></td>
<td>(+)</td>
<td>Winsman et al., 1976</td>
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<td></td>
<td>(+)</td>
<td>Cafarelli, 1977</td>
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<td></td>
<td>(+)</td>
<td>Lollgen et al., 1977</td>
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<td>Pandolf, 1977</td>
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<td>(+)</td>
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<td>Noble et al., 1979</td>
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<td>Robertson et al., 1979</td>
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<td>(+)</td>
<td>Cafarelli, 1982</td>
<td>LolIgen et al., 1980 (Δ)</td>
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<td></td>
<td>(+)</td>
<td>Kostka et al., 1982</td>
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<td>(+)</td>
<td>Knuttgen et al., 1982</td>
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<td>(+)</td>
<td>Robertson, 1986</td>
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<td>(+)</td>
<td>Plus 5</td>
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</tbody>
</table>

Proprio. = Proprioceptive. L&M = Leg and Muscle Responses. Plus number = further supportive references, not included.
maintain equivalent power output increases as pedalling speed becomes slower but resistance greater (Dickinson, 1929; Banister and Jackson, 1967; Hoes et al., 1968). If individuals do perceive the muscular force necessary to overcome pedalling resistance, it is not surprising that a high pedalling speed, such as 80rpm, is subjectively preferred for most exercise intensities (Hoes et al., 1968). In this instance, with an unchanged oxygen cost, a distressed cardiorespiratory system could not have been the cause of the elevated RPE at equivalent exercise intensities. By maintaining a common metabolic intensity through the parameter of oxygen uptake, it has thus been possible to distinguish between central and peripheral contributions to RPE.

A singular method of determining the central contribution of oxygen uptake is to manipulate, and reduce it, by means of hypobaric hypoxia. Both Horstman et al., (1979) and Young et al., (1982) reported that RPE was reduced at selected relative exercise intensities following exposure to 4,300m, compared with sea-level measures. Maresh et al., (1993) examined this phenomenon further with persons native to different residential altitudes. The authors considered the experiment intriguing because moderate-altitude natives, compared with low-altitude natives, demonstrate adaptations such as increased tidal volume, lung capacity and minute ventilation (Dempsey et al., 1974; Dempsey et al., 1975), all of which may serve as physiological cues in perceived exertion. Subjects comprised 6 untrained, low-altitude (366m-740mm Hg) and 8 moderate-altitude (2,200m-585mm Hg) natives aged between 19-25yr. They were studied, and exercised, at five relative intensities of 35, 55, 75, 85, and 100% VO2peak both at their native altitudes and at a simulated altitude of 4,270m-447mm Hg.

Findings revealed that the pattern of local, or peripheral, RPE was greater than the central RPE in lowland subjects at their residential altitude and this is in agreement with previous studies (Cafarelli et al., 1977; Young et al., 1982). When compared at their respective residential altitudes, both groups reported similar differentiated RPE responses in terms of peripheral, central and overall RPE values. Heart rates and oxygen uptake were reduced to the same extent in both groups at 447mm Hg, compared with their residential values. According to these, and other, authors, such central measures, therefore, cannot be seriously considered in explaining the differences in RPE between the two groups and may not be sensed in the perception of effort (Pandolf and Noble, 1973; Cafarelli and Noble, 1976; Young et al., 1982). This would support the overview provided by Table 2.1 where there is more non-related support for these two parameters, than related.

2.2.1c. PULMONARY VENTILATION (VE) AND RESPIRATORY RATE (RR).

Thus, though heart rate and oxygen uptake correlate highly with RPE, there is considerable dispute as to the validity of such relationships, non-related studies often exceeding those
where relationships do exist (Table 2.1). No such equivocation appears to exist with pulmonary ventilation (VE) and respiratory rate (RR). Since heart rate, oxygen uptake and pulmonary ventilation have been linearly related to exercise intensity for some considerable time (Wahlund, 1948; Astrand and Rhyming, 1954; Astrand and Rodahl, 1986), there are equally high correlations between RPE and pulmonary ventilation. Consequently, RPE will increase at standard incremental exercise intensities or when going uphill, and will decrease when going downhill in step with VE (Pollock et al., 1986). Noble et al., (1986) and have provided explanatory models (Figure 2.1) which classify pulmonary ventilation as a ‘specific symptom’, capable of being directly sensed, whilst other responses, like oxygen consumption are ‘non-specific symptoms’ and are only indirectly sensed. Accordingly, subjects can ‘feel’ dyspnoea but oxygen uptake is probably only sensed through a generalised feeling of increased metabolism. Noble et al., (1986) suggested that such cues, or physiological symptoms, serve as signals for the setting of perceptual intensity, passing through a reference ‘filter’ which modifies their input depending on differentiated signals from other sources such as those of a psychological nature (Robertson et al., 1977). If this model is to be accepted, a full explanation of perceptual ratings will not be possible until the mechanisms associated with incoming physiological signals and how they are modified by other inputs to the system, are understood and evaluated. Robertson (1982) and Robertson and Metz (1986) proposed that central signals involving VE and relative VO₂ acted as ‘amplifiers’ or ‘gain modifiers’ that potentiate peripheral signals in proportion to the aerobic, metabolic demands. A series of manipulative experiments have been conducted in developing these hypotheses. Robertson et al., (1979c) used hyperoxic gas mixtures and erythrocythaemia separately to increase the arterial oxygen and arterial-oxygen difference and consequently reduce ventilatory drive at a fixed submaximal load and constant total body VO₂. It was found that pulmonary ventilation decreased from pre- to post-experimental manipulation at both 45% and 70%VO₂max. power output. Similar results were obtained from the red blood cell reinflation studies of Williams et al., (1981), and Cafarelli and Noble (1976) who used carbon dioxide enriched mixtures to alter ventilation.

These findings indicated that ventilation was not associated with a potent sensory signal at lower metabolic rates, ventilatory function beginning to contribute cues perceived as central signals at a critical metabolic rate falling between 50-70%VO₂max. A similar study (Robertson et al., 1982) used five male mountaineers (X age 33.2yr) who performed graded treadmill exercise prior to, and following, induced erythrocythaemia and hypoxia. Subjects were instructed to rate their feelings of ventilatory exertion, discomfort and stress originating in the chest region (RPE-C). The VE at a power output equivalent to 70%VO₂max, increased (p<0.01) from pre-infusion normoxia to pre-infusion hypoxia, then decreased (p<0.01) from
Figure 2.1

STIMULUS

Muscular Contraction → Physiological Responses

Organism

Specific Symptoms → Reference Filter
Non-specific Symptoms

RESPONSE

Local Rating
Overall Rating
Central Rating

After Noble et al., 1986

Figure 2.2

Exercise Stimulus → Physiological Precursor → Central Peripheral

Primary Signal → Neuromotor Activity
Sensory Cortex → Perceptual Response

Perceptual Feedback

Robertson and Metz, 1986
pre-infusion hypoxia to post-reinfusion hypoxia. Chest signals increased \((p<0.05)\) with hypoxia, then decreased \((p<0.01)\) following red blood cell reinfusion. Regression analysis confirmed that the higher the VE in response to changing haemoglobin concentration and inspired oxygen concentration, the higher the RPE-C. This indicates that the ventilatory response during dynamic exercise appears to be an important physiological precursor for central signals of exertion and a direct correspondence between ventilatory adjustments and RPE-C during high intensity exercise seems to be prevalent. Similar responses were subsequently found among women (Robertson et al., 1984). This work vindicates the earlier multiple regression study by Noble et al., (1973b) where 6 male students, mean age 21.3 (±2.5yr), completed 30 min of cycle ergometry for three trials at 48, 60 and 68%\(\dot{VO}_2\)max. under neutral (24°C) conditions and two trials at 48%\(\dot{VO}_2\)max. under hot (44°C and 54°C) conditions. Eight independent variables: VE, \(\dot{VO}_2\), RQ, RR, HR, VCO₂, TR, Tsk were recorded at 5, 15 and 30 minutes. Ventilation accounted for the greatest variance in RPE at 5 and 15 minutes and RR made the greatest contribution at 30 minutes. These results evoked the hypothesis that man does not directly attend to the changes in physiological processes \textit{per se}, but does attend to their externalisation as revealed by VE, RR and Tsk which can be directly perceived (Noble et al., 1973a). They would also suggest that the lower work intensities may provide perceptual signals that can be consciously monitored. The question of the threshold level at which perceptual responses occur appears to be one of degree. At metabolic rates less than 50%\(\dot{VO}_2\)max., some authors (Robertson and Metz, 1986) suggest that VE is related to the tissue oxidative requirement and likely does not provide strong sensory signals of ventilatory exertion. At exercise intensities greater than 50%\(\dot{VO}_2\)max., VE responses reflect both the tissue oxidative requirement and the need to buffer metabolic acidosis (Wasserman, 1978). It is at these higher exercise intensities that VE appears to provide strong central signals of exertion that are consciously monitored (Ekblom and Goldbarg, 1971; Eynde and Ostyn, 1986; Robertson and Metz, 1986; Hill et al., 1987).

In order to explore this acid-base: ventilatory relationship, Robertson et al., (1986) attenuated ventilatory buffering during combined arm and leg ergometry by inducing alkalosis through NaHCO₃ ingestion. Only at 80%\(\dot{VO}_2\)max. power outputs was VE lower \((p<0.01)\) under alkalosis compared with the CaCO₃ placebo. Chest signals (RPE-C) followed a similar response, whereas at 20, 40 and 60%\(\dot{VO}_2\)max., VE did not differ, presumably because metabolic acidosis is limited at these lower exercise intensities (Wasserman, 1978). Regression analyses confirmed that the higher the blood H⁺ concentration, the higher were both VE and RPE-C so that VE was seen as a reciprocal of RPE-C in terms of acid-base shifts. The conclusion was that ventilatory buffering of metabolic acidosis appeared to function as a precursor for exertional sensations that are of central origin (Robertson et al., 1986).
Horstman et al., (1979), in the previously reported altitude study, found that VE at 4300m, was greater and heart rate equal to that at sea level, while muscular strain was reduced as it was necessary to reduce pedal resistance in order to achieve the same relative exercise intensity. Within the range of 60 to 95%VO$_2$max., RPE was significantly less at high altitude than at sea level at the low end of the range, but there was no difference in RPE between the two conditions at the high end of the range. These results would lend support to Robertson's work with the suggestion that the functional relationship between perception of effort and peripheral factors, or muscular strain, is different from that between perception of effort and central factors, such as tachypnoea and tachycardia. Horstman and his colleagues proposed that peripheral factors were the dominant influence on the perception of effort at exercise intensities that do not greatly stress ventilation and circulation, but that once tachypnoea and tachycardia became of sufficient magnitude to be perceived as stressful, central factors exerted a greater influence on perception of effort.

As the product of VE is breathing frequency, or respiratory rate (RR), and mean tidal volume ($\overline{V_T}$), consideration has been applied to the question as to whether these components are involved in signalling central sensations of exertion (Robertson and Metz, 1986). Whereas RR, together with RPE-C were attenuated under induced alkalosis at 80%VO$_2$max., tidal volume did not differ between acid-base conditions, suggesting that, of the two, exertional sensations associated with RR were continuously monitored at the higher exercise intensity levels (Robertson et al., 1986). Regression analysis further established that adjustments during dynamic exercise would appear to reflect one of the primary physiological precursors for central sensations of ventilatory exertion (Robertson et al., 1986).

Though the neuromotor pathway for ventilatory sensations during exercise is not totally understood, it appears to involve RR, especially in its association with the sensation of breathlessness (Meakins, 1923; Meakins, 1934). Sherrington (1900) long since favoured the idea of muscular sensations linked to afferent feedback from intramuscular receptors but only recently has the hypothesis been supported by the revelation of a possible awareness of outgoing motor command within the CNS. This has been effected by reflex stimulation, or inhibition, of the alpha motor output and through considerable experimental work on collateral discharge, which is known to occur (McCloskey, 1978; Campbell et al., 1980; Gandevia et al., 1981; Burdon et al., 1982; Matthews, 1982; Killian and Jones, 1984; Killian et al., 1985). Thus, as RR increases during exercise, neuromotor signals reflecting changes in inspiratory muscle force and duration are believed to be sent to the sensory cortex (Killian et al., 1982). The exertional sensations are believed to include one or both of a feed forward system, where the increased central motor outflow that is required to sustain muscle contractile force.
is simultaneously monitored by both the motor and sensory cortex, and a feedback system where neuromuscular signals are transmitted from peripheral receptors in active muscles to the sensory cortex (Kostka and Cafarelli, 1982). Cafarelli (1982) has proposed that perceptions of exertion arising from the peripheral skeletal muscles involve the same sensory mechanisms, so it is possible that the primary signals for both central and peripheral sensations of exertion share the same neuromotor pathway (Robertson and Metz, 1986). From these studies, the general conclusion is that VE is the most satisfactory central measurement to correlate with RPE in the function of an intensity range from 68-100%VO₂ max. (Eynde and Ostyn, 1986) and provides a conscious sensation mediated by the peripheral 'motor command' (Killian, 1986) associated with respiratory rate.

From all the evidence that adjustments in VE and RR function as physiological precursors for central perceptions of exertion during dynamic exercise, Robertson and Metz (1986) proposed a model (Figure 2.2) where both central and peripheral factors were classified as providing exertional sensations. As these precursors change during exercise, they trigger concomitant responses in muscle contractile properties. The subsequent increase in motor unit involvement in either the respiratory, or peripheral, skeletal musculature is interpreted by the sensory cortex as the primary perceptual signal of exertion. Simple though this model is, it provides a valuable adjunct to the concept of Noble et al., (1986) (Figure 2.1) with its amplification of a neuromotor component and positive and negative feedback pathways.

2.2.1d. BLOOD AND PLASMA LACTATE CONCENTRATIONS.
Classification of a physiological response as a local, or peripheral, factor which could be important for the perception of effort, is based on the mediation of feelings of strain in the exercising muscles (Ekblom and Goldbarg, 1971). Significant among these is muscle lactate which, in the absence of its direct measurement, many authors have relied on increases in blood, or plasma, lactate levels as reflective of non-oxidative glycolysis (Table 2.2). The relationship between muscle and blood lactate concentrations following exercise was examined by Karlsson (1971) who concluded that the highest blood lactate concentration mirrored muscle lactate concentration fairly well after single bouts of exhaustive submaximal exercise. Unlike many of the parameters suggested as primary cues influencing perceived exertion, blood lactate closely resembles the growth of sensation for perceived exertion (Borg, 1962a; Gamberale, 1972). Even so, below an exercise intensity of about 65%VO₂ max, lactate does not appear to increase appreciably for normal subjects. Much would seem to depend on the subject's state of training, one of the main distinctions of a trained individual being the ability to exercise without raising the level of blood lactate appreciably. Without training, lactate may rise at only 40%VO₂ max.; with intense training,
lactate may, in extreme examples, be unchanged even at 90% $V_O^2_{max}$. (Despopoulos and Silbernagl, 1981). At low work loads, such as 30-60% $V_O^2_{max}$, concentrations have even been found to decrease in highly trained subjects (Hermansen et al., 1972). At the other end of the scale, concentrations at rest may vary depending on diet, age and training status (Aunola et al., 1982) so that threshold values could distort final concentrations unless delta data are used. However, rarely do resting values exceed 1mmol.l$^{-1}$ and this is true for the present series of studies. A critical point is the lactate threshold, or 'break-point', at about 2mmol.l$^{-1}$ (McDougall, 1977; Kindermann et al., 1979) which is defined as the exercise intensity where the rate of lactate production exceeds its rate of uptake or removal and is commonly known as the 'aerobic threshold'. The sharp, and exponential rise of lactate (Kindermann et al., op cit; Skinner and McLellan, 1980; Sjodin and Jacobs, 1981) occurring at a workload corresponding to 60-80% $V_O^2_{max}$, is arbitrarily chosen to mean a level of 4 mmol.l$^{-1}$ and is known as the 'anaerobic threshold'. In this thesis, such a point is termed the 'reference threshold'.

A significant relationship between perceived exertion and blood lactate has been observed for continuous and intermittent ergometer (Edwards et al., 1972) and treadmill, exercise (Morgan and Pollack, 1977). It has also been demonstrated (Ekblom and Goldbarg, 1971) that the relationship between lactate concentration and perception of effort remains constant despite the greater physiological strain associated with tasks requiring localised muscular performance. For instance, arm exercise is known to produce higher lactates than leg exercise at the same oxygen consumption (Astrand et al., 1968). Ekblom and Goldbarg (1971) found that, at a given absolute or relative oxygen uptake, RPE was higher in arm, than in leg, work. Yet, when blood lactate concentrations were used, RPE for arm and leg work was the same. Similarly, when cycling and running were compared, RPE was the same for given blood lactate concentrations. Gamberale (1972) studied arm weight lifting and cycle ergometer work. At approximately the same oxygen uptake of 0.83 l.min$^{-1}$ for arms and 0.89 l.min$^{-1}$ for legs, blood lactate levels were 3.2mmol.l$^{-1}$ and 1.8mmol.l$^{-1}$ respectively. These values corresponded to RPEs of 12.9 for arms, 9.9 for legs.

In contrast, Stamford (1973) studied cycle ergometer work at different pedal frequencies but equal power outputs and found that lactate and oxygen debt did not change, though RPE was significantly higher at 40, than 60rpm. It should also be noted that, in support of the lactate-RPE relationship, whereas most physiological variables grow linearly with workload, perceived exertion, when ratio-scaling methods are utilised, grows according to a positively accelerating function. Blood lactate is one of the few parameters following a similar exponential function.
At constant, low, submaximal workloads, it has been suggested (Johansson, 1985) that heart rate and blood lactate concentrations are both valid indicators of levels of perceived exertion, as they should reach an approximate steady state during prolonged exercise. To test this hypothesis, Johansson (op cit) exercised ten well-trained men (x age 27.3±8.5yr) at three, thirty minute bouts of randomly chosen work intensities of 50W, 100W and 200W at 60rpm. Perceived exertion was determined using Borg’s (1982a) 0-10 category-ratio scale (CR-10). Subjects exercised at approximately 20%, 33% and 60% of estimated VO$_2$max. (Astrand et al., 1977) at the three loads. During 50W and 100W, both heart rate and blood lactate concentration reached steady state after 5 minutes, but the ratings of perceived exertion grew by a negatively accelerating function. At 200W, heart rate was increasing over all the exercise period and was considered a valid indicator of perceived exertion. Blood lactate concentration stabilised after 10 minutes of exercise at about 3.5mmol.l$^{-1}$ and was considered partly responsible for the growing ratings of perceived exertion over time. This was because blood lactate accounted for 32% of the variance in perceived exertion, heart rate for 14%. Thus, almost half the variance in perceived exertion could be 'explained' by these two components with blood lactate accounting for the larger share.

Experimental manipulations of lactate concentration have yielded inconclusive results. Similar studies of hyperoxic conditions (Allen and Pandolf, 1977; Pedersen and Welch, 1977) have provided inconsistent results in relating perceived exertion and lactate responses and this may have been because single, rather than double-blind, techniques were used. Even so, they were consistent in demonstrating a decrement in lactate concentration at relatively low exercise intensities of approximately 50% VO$_2$max. for both ergometer and treadmill work. Horstman (1977) provided further supportive evidence in determining parallel reductions in lactate concentrations and perceived exertion during treadmill exercise in a cold environment. Previously reported altitude studies (Young et al., 1982; Maresh et al., 1993) provide an added dimension to the lactate responses. Young and coworkers’ (op cit) study of the perceptual and physiological responses to exercise at 85% VO$_2$max. whilst cycling for 30 minutes at sea level and during both acute high altitude (AHA; <2hr.) and chronic high altitude (CHA; 18 days) exposure at 4,300m induced an hypoxic reduction of VO$_2$max. of 27% for both conditions relative to sea level values. Plasma lactate concentrations after exercise at AHA exposure increased to the same extent as at sea level, that is, from about 1mmol.l$^{-1}$ at rest to 10mmol.l$^{-1}$. During CHA exposure, on the other hand, there was a significant reduction from 0.5mmol.l$^{-1}$ at rest to 1.8mmol.l$^{-1}$ following exercise. These observations suggest that sensations associated with the large increases of blood lactate at sea level and AHA could have influenced the peripheral factor and may have dominated the overall perception of exertion. Conversely, the conditions induced by CHA exposure may have accounted for the
reduced local RPE in parallel with the reduced lactate levels. Such findings support similar contentions by Allen and Pandolf (1977) and Pandolf (1978) that when a particular sensation, either local or central, is accentuated over others, it may dominate the overall RPE. The study by Maresh et al., (1993) revealed that plasma lactate was also reduced by similar percentages at each of five relative exercise intensities in both groups of low altitude and moderate altitude subjects at 4,300m. The significant correlation observed between plasma lactate and peripheral RPE in low altitude natives during hypobaric hypoxia suggests that lactate could be a marker of alterations in metabolic intermediates more closely involved in local RPE in unacclimatised subjects (Maresh et al., op cit). Edwards et al., (1972) and Noble et al., (1983) support these findings though other authors are in disagreement (Sargeant and Davies, 1973; Lollgen et al., 1980).

Though the preponderance of studies (Table 2.2) favour lactate concentration as a potent stimulant for perception of effort, the mechanism by which this influence is mediated is uncertain. One of the most common suggestions is that metabolic acidosis is the cause. The basis of this is that, together with the increased lactate production, there is a corresponding increase in hydrogen ion (H⁺) concentration. Taking the most extreme example, during exhaustive cycle ergometer exercise such as at VO₂max. lasting 5-10 min., about 94% of these hydrogen ions are the result of the accumulation of lactic acid, whilst the rest are owing to the accumulation of Glucose-6-phosphate (2%) and Glycerol-1-phosphate (3%) (Hultman and Sahlin, 1980). If these H⁺ ions were added to an unbuffered solution, the concentration of H⁺ would be approximately 35mmol.l⁻¹ and pH would decrease to 1.5. In muscle, however, pH only decreases to approximately 6.6 following brief maximal exercise as a result of H⁺ ions being taken up by the various buffering processes. The extent of the fall in intramuscular pH is, inevitably, largely determined by the buffering capacity of the muscle cell (Hultman and Sahlin, op.cit).

Several factors contribute to this buffering capacity. First, there are the physico-chemical properties of muscle as a result of H⁺ ion uptake by weak bases present in muscle and the blood (Siggaard-Andersen, 1976; Hainsworth, 1986). These include buffering by phosphate compounds, especially Pᵢ, by the carbon dioxide/bicarbonate system, and by peptides and proteins, especially carnosine (Davey, 1960). The second group of buffering processes are the result of reactions involved in ATP resynthesis during exercise associated with metabolism. The process of CP utilisation, IMP/NH₄ formation and oxidation of amino-acids, although limited during maximal exercise, would each result in the net uptake of hydrogen ions (Wootton, 1984). Finally, the potential influence of transmembrane fluxes of H⁺, and HCO₃⁻, ions during exercise must also be considered.
The concentration gradient of $H^+$ ions from muscle to blood is maintained by the removal of $H^+$ ions from the circulation through the buffering processes of the blood (Hainsworth, 1986). These include the uptake of $H^+$ ions by haemoglobin, plasma proteins such as cysteine and histidine (Hainsworth, 1986) and the bicarbonate buffering system ($H^+ + HCO_3^- = H_2CO_3 = H_2O + CO_2$) (Brooks and Fahey, 1985). The equilibrium of the reaction, and hence hydrogen ion uptake, is maintained to the right by the removal of $CO_2$ at the lungs through hyperventilation (Widdicombe and Davies, 1991). Consequently the plasma bicarbonate fraction of the blood, or 'alkali reserve' (Siggaard-Andersen, 1976), decreases as the pH falls.

Metabolic acidosis as a result of increasing lactate concentrations, is believed to act as a stimulus to free nerve endings in the muscle, leading to sensations of pain and discomfort (Kinsman et al., 1973; Stamford and Noble, 1974; Pandolf, 1978). Yet, when Paulus et al., (1974) manipulated blood pH by an infusion of NaHCO$_3$ to correct exercise-induced acidaemia during ergometer exercise at incremental exercise intensities to exhaustion, they found no effect on subjective feelings of fatigue. However, maximal CO$_2$ production and pCO$_2$ did increase significantly as a result of the reaction between HCO$_3^-$ and $H^+$. The subsequent findings of Robertson et al., (1979b) supported the conclusions that the influence of elevated blood lactate concentration on perceived exertion is not mediated by reduction in blood pH. In other words, blood lactate may reflect a mediation pathway other than blood acidaemia (Mihevic, 1981). This is not to deny the acceptability of the concepts put forward, rather than to focus attention on the validity of the design of the studies used to evaluate, systematically, such peripheral factors (Pandolf, 1982).

In contrast to the several studies which have implicated lactate concentration as a factor influencing perceived exertion, Stamford and Noble (1974), Kay and Shephard (1969) and Lollgen et al., (1980) have provided evidence which disputes lactate as a perceptual cue (Table 2.2). Stamford and Noble's (op. cit.) results cannot be fully integrated with previous reports, however, because of the limitations in employing a 1-9 scale rather than standard RPE, or ratio-scaling, procedures. The insignificant relationship ($r=0.15$) between perception of effort and arterial lactate concentration reported by Kay and Shephard (op. cit) may merely reflect the restricted perceptual values from the single 80%VO$_{2\text{max}}$ exercise intensity utilised. It has also been suggested (Mihevic, 1981) that the time lag between production of lactate by the muscle and its appearance in the blood, combined with the inability to assess lactate concentration at the level of the free nerve endings in the muscle, may be responsible for the lack of a high correlation. Even so, blood lactate is generally regarded as an accurate index of muscle lactate concentration, provided it is measured at peak concentrations (Gollnick and Hermansen, 1973). And whether the sampling times ranged from immediately
post-exercise (Edwards et al., 1972; Gamberale, 1972) or up to anything from 3-6 min following exercise (Pedersen and Welch, 1977; Gass et al., 1981) appears to be irrelevant in terms of determining the relationship between blood lactate concentration with perceived exertion.

In summary, the experimental investigations with different environmental conditions and various exercise tasks, indicate that the relationship between lactate concentration with perception of effort is a robust one. Whether trained, or untrained, subjects are involved, lactate concentration does not appear to reveal accelerated increases until exercise intensities of 50-65%VO₂ max are attained. Even so, comparisons of perceptual and lactate responses under normoxic, hypoxic and hyperoxic manipulations, indicate that lactate concentrations may provide markers of perception of effort at relatively lower intensities than those just indicated. Furthermore, although the mechanism of lactate's perceptual response has not been fully resolved owing to some limitations in experimental design, the muscular discomfort which accompanies lactate accumulation cannot be discounted as a source of sensory input which is readily available to conscious awareness.

2.2.1e. PROPRIOCEPTION.
Muscle contains muscle spindles with their two different types of afferent fibre (Groups Ia and II), Golgi tendon organs (Group Ib), free nerve endings and a few paciniform corpuscles. Joints contain free nerve endings and Ruffini spray endings, similar to those in tendons, in their ligaments (Matthews, 1972). In the present context, the question is whether these receptors, or sense-organs, situated within the tissues of the body, provide a conscious awareness whose 'kinaesthetic', or 'position sense', can be detected by degree of force and intensity on the Borg Scale. As long ago as 1896, Bloch considered that, while a movement is in progress, the impression of position is perceived with some accuracy, though deteriorating during immobility. Sherrington (1900, 1906, 1918) asserted that signals from receptors in both muscles and joints contributed to the full range of kinaesthetic sensations, and no distinction was made between them. No confirmatory experimental data were provided to support such a contention. However, by refining and extending the observations originally made a century earlier by Helmholtz, Brindley and Merton (1960) established that the discharges from muscle spindles in the extrinsic eye muscles are without influence on the conscious perception of the direction in which the eyes are pointing. Less rigorous experiments on moving joints after locally paralysing the joint afferents were taken to show that the stretching of limb muscles was also without sensory action (Browne et al., 1954; Provins, 1958; Merton, 1964, 1970; Gelfan and Carter, 1967). Granit (1972) appeared to put the seal on the controversy with his affirmation that, to a large extent, our motor acts belong to a subconscious organisation of
controls, only made part of conscious awareness if something goes wrong and relayed through the 'alpha-gamma linkage' (Granit, 1955). However, work on muscle vibration and finger anaesthesia led Goodwin et al., (1972a, 1972b, 1972c) to the conclusion that the proprioceptors involved can influence consciousness and do contribute to the role of assisting the cortical elaboration of a 'body image' (Fisher, 1973; Vander et al., 1994), its position and movement (Rasch, 1989).

The acceptance of the 'common-sense' classical view that muscle afferents do contribute to kinaesthesia carries with it the inclusion of Golgi and Ruffini endings of tendons and joints as well as the primary and secondary spindle endings of the muscles themselves. It may seem desirable to be able to subdivide position sense into its components, much as perceived exertion has its sub-units, since not all receptors reveal the same integrity of positioning. For instance, joint receptors are relatively inaccurate at providing an awareness of absolute position (Paillard and Brouchon, 1968; Goodwin et al., 1972b). This may be because the total number of afferent fibres to a joint is relatively small in comparison with the number of afferent fibres to muscles that act upon that joint. The cat knee-joint, for example, is supplied by fewer than 400 medullated fibres whereas, at a conservative estimate, there are some 4,000 medullated afferent fibres devoted to supplying the quadriceps, hamstring and sartorius muscles (Sherrington, 1894; Skoglund, 1956). This suggests that the delicacy of position sense could be appreciably improved by the utilisation of signals from muscle afferents along with those from joint afferents; but this should not be taken to imply that all the receptors convey the same type of information. What of the velocity of movement and the quantitative measure of speed, for instance? The problem for the neurophysiologist is how the information provided by a variety of different receptors is compounded by the CNS to produce a unitary picture of a limb position. The problem for the physiologist using the Borg Scale is resolved by the sensation of stress being referred to the whole limb with its joints, irrespective of whether the excited receptors happen to be related to the stretch of the muscle spindles, the tension relayed by the Golgi organs or the pressure receptors in joint capsules.

A valuable method of determining the contribution of any parameter is to compare the healthy, with the diseased state. Bar-Or and Reed (1986) examined 10 females and 14 males whose ages ranged from 9.5-20.5yr. All these adolescents suffered from neuromuscular and joint disease in which motor function was deficient and where proprioceptive, and other sensory information from their skeletal muscles, tendons and joints, were deemed to be affected. The major finding was that while the ratings of perceived exertion of the adolescents was higher than healthy adolescents at the same absolute workload, the differences in RPE
were virtually eliminated once it was plotted against the percentage of peak aerobic power. Based on correlational analysis, it was also found that the adolescents perceived exertion with similar 'acuity' as did healthy adolescents and adults. The strong association between RPE and %VO2max. was found to be in agreement with a wide span of studies including data published for individuals who vary in their fitness levels (Skinner et al., 1969), middle-aged versus the elderly (Sidney and Shephard, 1977) and cycling versus treadmill running (Ekblom and Goldberg, 1971). Even so, it is hard to assume that proprioceptive, and other exercise-induced peripheral sensory signals are of the same magnitude in a healthy limb as in a dystrophic, atrophic or spastic limb even when they all work at the same relative intensity. The authors concluded that the uniform RPE response of all the subgroups, healthy or diseased, was consequently suggestive of a central mechanism that controls RPE and these findings were supported by those of Cafarelli (1982).

A number of investigations associated with mechanoreceptor and proprioceptor feedback have focussed on the evaluation of varying pedalling frequencies during ergometer cycling and on a comparison of the perceptual responses of various exercise modalities. Some have compared perceptual ratings during ergometer exercise at equivalent exercise intensities but at different pedalling rates (Stamford and Noble, 1974; Cafarelli, 1977; Lollgen et al., 1977; Robertson et al., 1979a; Robertson et al., 1979c; Lollgen et al., 1980). The comparable metabolic and cardiac responses for various pedalling rates, despite significant perceptual differences, also provide the basis for the proposal that proprioceptive feedback, specifically Golgi tendon activity (Figure 2.2), is the underlying mechanism for perception of effort (Stamford and Noble, 1974). Such a suggestion is given added weight by the greater perception of effort for eccentric, in comparison with concentric, exercise at the same metabolic cost (Pandolf et al., 1978).

Confusingly, two comparisons of perceived exertion during continuous and intermittent exercise yielded different conclusions as to the validity of muscular strain and proprioceptive feedback as critical perceptual cues. Stamford and Noble (1974) found that, despite the significantly elevated metabolic cost for continuous work at 60 rpm, perceptual ratings for this condition did not differ from those recorded during intermittent work at 40 and 80 rpm. These data were interpreted as additional evidence for the importance of muscular strain, mediated by proprioceptive feedback mechanisms, as the critical input for perception of effort. Edwards et al., (1972); in contrast, concluded that efferent nervous information from the muscles and joints was not the primary determinant of perceived exertion, although it was suggested that the combination of exerted force and frequency of leg movements may play a role in determining the perceptual response. Such differences in results and conclusions may be
accounted for by methodological differences in terms of the criterion used for equating continuous and intermittent exercise such as the length of the exercise, and recovery periods for the intermittent work. The contrasting data helps to emphasize that many of the hypothesised factors signalling local, or peripheral, effort are nearly impossible to quantify (Pandolf, 1982). Only by accepting the all-embracing 'gestalt' concept and the contention by authors such as Bartley (1970) that RPE is, in part, a means of processing such perceptual information as sense of pain, temperature, kinaesthesis, touch and even some of the receptors involved in homeostatic processes, do such findings become comprehensible.

Comparisons of perceived exertion during cycle ergometry and treadmill exercise and for running and walking, have provided further support for the importance of local muscular strain as a perceptual cue (Ekblom and Goldbarg, 1971; Pandolf et al., 1975; Horstman et al., 1979). At given submaximal levels of oxygen consumption, perceptual ratings were found to be consistently higher during cycling in comparison with treadmill running (Ekblom and Goldbarg, 1971). As with the previous study (Bar-Or and Reed, 1986), these differences were eliminated when exercise intensity was expressed in relative terms. The findings of Horstman et al., (1979) depicting peripheral (legs) and central (respiration) ratings of perceived exertion whilst running or walking to self-imposed exhaustion at 80% VO_2max. on a treadmill revealed that the central, or respiratory, RPE was significantly lower during walking than running. Perception of leg exertion at exhaustion was 1.3 units greater than perception of respiratory exertion during the walking test; no differences were observed during running. A similar trend was revealed by Pandolf et al., (1975) during level and graded walking at equivalent energy expenditures where local muscular factors appeared to dominate the exertional perception over central responses. None of the perceptual differences was statistically significant, however.

A similar pattern emerges from the concentric and eccentric studies on the ergometer depicted by Robertson et al., (1979b) and that by Knuttgen et al., (1982) respectively. In the former, the differentiated perceptual responses for three pedalling rates of 40, 60 and 80 rpm at a constant power of 137W showed that the overall rating of perceived exertion was significantly lower than the peripheral rating for the legs and higher than that from the central, or chest, response at each pedalling speed. Knuttgen et al., (1982) investigated six male volunteers who were trained with eccentric contraction exercise for a 5 week period. Subjects exercised at 60 rpm on a specially designed cycle ergometer for which the pedal axle was driven in the reverse direction from normal cycling by an electric motor. The characteristic muscle soreness associated with this type of exercise was experienced by all subjects for 2-3 days after the initial, pre-training tests. Post-training, both oxygen uptake and heart rate
values were lower (p<0.05) and all RPE ratings, regardless of time, decreased with training. Peripheral RPE was higher than central RPE (p<0.05) and the overall RPE was higher than central RPE (p<0.05).

The pattern of thermal responses, all of which have been implicated as possible cues in the setting of exertional perception (Pandolf et al., 1972; Noble et al., 1973a; Kamon et al., 1974), was also measured in this latter study by Knuttgen et al., (1982). Weighted mean skin temperatures (Tsk) were lower (p<0.05) for the post-training evaluation after 15 minutes by about 1.5°C. It could be inferred that this difference may be associated with differences in peripheral blood volume or blood flow, after training. Concomitantly, the lowered Tsk after training may well be associated with the general reduction of all the differentiated ratings especially those related to the significant reductions in local RPE.

Observations from these latter studies would lead to the conclusion that local sensations from the exercising muscles and joints dominate the sensory process. In fact, Cafarelli et al., (1977) considered that, in no instance did central effort exceed local effort during exercise. This was explained (Cafarelli, 1977) by the fact that, the rapid rise in effort with the onset of exercise was not accompanied by immediate metabolic increases of the same magnitude, especially during the first 30 seconds of exercise. The effort sense was therefore peripherally determined first, with central input becoming progressively more dominant after this time threshold. A subsequent study (Cafarelli, 1978) found that although perception of effort continued to increase over a 6 minute exercise period at these submaximal exercise intensities on the cycle ergometer, the integrated EMG response remained constant across time and pedal frequency. This suggests that, while the efferent command to the working muscle is dependent on resistance, it is independent of time and pedal frequency. Such findings amplify, and are consistent with, the early proposals by Borg (1961b) that the perceptual response during short-term work is governed by local muscular sensations. Such a contention is verified by the plethora of studies which relate proprioceptive and general leg muscle sensations to RPE in a positive way (Table 2.2) though the precise manner in which sensory inputs are monitored and integrated to determine perceived exertion remains unclear.

2.2.2. TRAINING AND RELATIVE WORK INTENSITY.
Since submaximal oxygen consumption, ventilation and heart rate of trained individuals reflect the greater maximum aerobic power (MAP) of these subjects (Astrand and Rodahl., 1977; Patton et al., 1977), perception of effort would be expected to parallel the physiological responses to training and differentiate among individuals varying in fitness. That is, absolute
exercise intensities should be perceived as more strenuous for the less fit individual in correspondence with the greater physiological strain (Linderholm, 1967; Skinner et al., 1969). In fact the most conclusive point in favour of RPE as a measure of work intensity is that, following training, there is a significant decrease in RPE at a given workload (Rockefeller and Burke, 1979). It could be argued that Borg's (1962a) original scale, using middle-aged Swedish men working on cycle ergometers, is suspect in terms of validity and as related to a discrete age group. For instance, values of RPE in the range 11, 12 and 13, have been found to be 20 - 30 heart beats above the RPE x 10 value originally determined by Borg (Burke and Collins, 1984). For higher intensities, these same authors found RPE x 10 heart beats ± 10 beats. Burke and Keenan (1984), in contrast, reported male and female adult backstrokers as producing heart rates meeting the predicted RPE x 10. Yet such equivocal results do not diminish the utility of the scale, especially as changes in maximal heart rate with age (Astrand and Rodahl, 1986) are inevitable. Heart rate is still to be considered the primary physiological variable for estimating intensity (%VO₂max), but the RPE assists in adjusting that intensity to a level that is both suitable for eliciting a training effect and that is psychologically tolerable (Pollock et al., 1986b) - the 'Just Tolerable Discomfort' of Experimental Study 2 of the present thesis, for example.

Percentage of maximum oxygen uptake is the preferred criterion for prescribing appropriate exercise levels as indicated by the ACSM (1980) recommendation that healthy adults work at 50 - 85%VO₂max. during a training programme. And selected studies between 1969 to 1987 emphasise a positive relationship between RPE and relative work intensity which stand unapposed by studies to the contrary (Table 2.1). As a working model, particularly where laboratory techniques are unavailable, the difficulty lies in equating heart rates with oxygen uptake values. Pollock et al., (1979) compared exercise heart rates obtained at 70 and 85% intensities for VO₂max. with %HRmax. and %HRmax. reserve. Using %HRmax. yielded training heart rates consistently below the heart rate obtained for %VO₂max. In comparison, %HRmax. reserve nearly duplicated the metabolic parameters and this provided strong calibration evidence supporting the accuracy of the %HRmax. reserve method for exercise prescription. It also endorsed the validity of the formula of Karvonen (1957) as utilised during the present training studies. Further matching evidence between increases in both %VO₂max. and RPE has provided valuable working data (Burke and Meade, 1978; Burke and Keenan, 1984; Eston and Burke, 1984; Burke and Collins, 1984). These workers found that consistent RPE scores of 10 - 11 occur in the 60 - 70%VO₂max. range, 12 - 13 at about 70 - 80%VO₂max., and 14 - 15 at 80 - 95%VO₂max. Such category ratings can embrace a wide spectrum of participants from those subjects who never exercise (Stephens et al., 1985), cardiac patients (Foster, 1984), sedentary individuals at the onset of a training programme.
(Dishman, 1986) and highly trained athletes (Burke, 1986). The common denominator for them all is the level of fatigue as related to the onset of metabolic acidosis (Wasserman et al., 1973), more universally recognised as the 'anaerobic threshold' (AT) or reference point at which 4 mmol.l⁻¹ blood lactate is achieved.

Following observations of hundreds of adult participants in fitness programmes, Burke (1979) recommended that the optimal training stimulus for a running programme should be at RPE 12 - 14. Katch et al., (1978) further described the advantages of using AT to assess training intensity whilst Purvis and Cureton (1981), in implementing this method, found that RPE at AT (RPE_AT) was 13.1±0.9 (Mean±SD) for women and 14.2±0.9 for men. Using similar methods, Bellew and Burke (1983) reported 12.4±1.7 for women and RPE_AT of 13.6±1.5 for men; Small et al., (1982) revealed no significant differences in mean RPE_AT of 13.3±1.6 for young, and 13.5±1.5 for older, male distance runners. Vincent and Burke (1985) subsequently found an average RPE_AT of 14.2±1.5 in male distance runners and discovered significant differences (p<0.05) in time to exhaustion between work 5% above, and 5% below, the AT. These four latter studies indicate that, in order to work for long periods of time, individuals should work at, or below, AT which is customarily perceived to be 12- 14 on the RPE scale. This would help to confirm the empirical recommendation made by Burke (1979) based on healthy adults exercising in a fitness programme, that for comfort or 'preferred exertion' (Dishman, 1986) individuals should work at, or below, AT or at an RPE between 12 - 14.

Pollock et al., (1986b) supported these conclusions, emphasising that subjects should be properly instructed in the use of the RPE scale since it provides a valid adjunct to oxygen uptake and heart rate for prescribing exercise and monitoring intensity of training. They further deduced that an RPE 'training window' of 'somewhat hard' (RPE 12 - 13) and 'hard' (RPE 15 - 16) was consistent with metabolic intensities of 50 - 85%VO₂max. and 60 - 90%HRmax. reserve. It has been estimated that approximately 10% of the population cannot use the scale with any accuracy (Morgan, 1981) because of either under- or over-rating. But since RPE appears to remain consistent over time, such subjects may still find the scale of value as long as the tester, exercise leader, and participant are aware of this inconsistency (Pollock et al., 1986). Findings also suggest (op cit) that physiological determinants of RPE may differ slightly among test protocols: non-steady state methods, for instance, typified by incremental, or graded, exercise tests (GXT), may lead to a slightly higher heart rate to RPE relationship.
Even so, several rehabilitation programmes for cardiac patients have successfully used a combination of GXT and RPE values for controlling exercise intensities. In a study by Gutman et al., (1981), 20 coronary by-pass patients were tested on GXTs two, and eight, weeks after surgery. Subjects trained three times a week with the stricture not to exceed a certain peak heart rate. Heart rates during training were matched to similar heart rates during GXTs so that RPE could be evaluated at the same physiological level. The relationship between RPE and HR during the GXT and training was the same both at the two, and eight, week benchmarks although a shift had taken place in which the same RPE value was rated at a higher HR. Perceptual intensity was ESTIMATED on the RPE scale with the restrictions imposed by a stipulated peak HR.

The corollary to this protocol was that revealed from a study by Smutok et al., (1980) where participants were required to subjectively regulate their own treadmill speed at a stipulated RPE value. A certain perceptual intensity was required to be PRODUCED from RPE values made familiar as a means of guiding exercise intensity. In the first instance, 10 subjects performed a GXT in which their HR and RPE were registered at five different speeds. In two separate trials, subjects were requested to produce various RPE levels recorded at the previous GXT. No significant differences were found between the pre-determined and the subjectively-regulated speeds at equivalent RPE values. However, a significant difference was found for HR at equivalent RPE values between the two tests at RPE levels below 12 and HR levels below 150. This was taken to indicate that using only RPE as a single indicator of stress in exercise prescription, as in using only HR, was of limited value, though the difference in results obtained from these two studies might well be caused by the use of two different psychophysical methods.

Usually, subjective intensity is extrapolated from the RPE scale during exercise and is known as the ESTIMATION method, whilst the Smutok study illustrates the PRODUCTION method where subjects had to reproduce an exercise level that matched a certain subjective intensity. Though further research is needed to resolve the problem of the use of RPE in determining specific physiological demands (Burg and Ceci, 1986) there appears to be little doubt that the RPE rating is a potentially useful tool for exercise prescription (Purvis and Cureton, 1981; Birk et al., 1985). From the initial studies, the RPE scale has been shown to provide a high correlation with heart rate (Borg and Noble, 1974). Even more significantly, perceived exertion alone, or perceived exertion in combination with HR, has been found to predict VO2max. more accurately than predictions of VO2max. by HR alone (Morgan and Borg, 1980; Noble et al., 1981; Noble, 1982).

The RPE during rhythmic activity has been found to be clearly related to the %VO2max. (Skinner et al., 1969; Bar-Or et al., 1972; Mihevic, 1981; Robertson, 1982) though there is no evidence
that oxygen uptake or relative work intensity is directly monitored by the body (Mihevic, 1981). It has been suggested that \( \% \text{VO}_2\text{max} \) is only part of the central signal for perception of effort (Robertson, 1982) rather than the sole determinant of RPE since the relationship between RPE and \( \% \text{VO}_2\text{max} \) may be altered by such factors as state of training (Demello et al., 1985), drugs (Ekblom and Goldbarg, 1971; Davies and Sargeant, 1979; Sullivan et al., 1986), hypnotic suggestion (Morgan et al., 1976), pedal rate (Robertson et al., 1979d) and circadian rhythms (Hill et al., 1985).

The ventilatory threshold, a well studied phenomenon characterised by a disproportionate increase in ventilation with respect to \( \text{VO}_2 \), has been proposed as a more apposite measure than \( \% \text{VO}_2\text{max} \) for the prescription of exercise training for a wide variety of individuals (Purvis and Cureton, 1981; Brehm et al., 1985). One distinct value is that it is a measure related to exercise endurance performance (LaFontaine et al., 1981; Powers et al., 1983; Davis, 1985). Another is that it may be reliably determined using non-invasive methods (Calozzo et al., 1982; Gladden et al., 1985) in order to produce RPE\(_{AT}\). As already revealed, lactate levels are also known to be associated with RPE and it is difficult to separate the central effects of gas exchange kinetics which define the ventilatory threshold from the local effects of blood lactate; by way of confirmation, the latter has been found to be elevated at an intensity similar to that associated with the ventilatory threshold (Wässerman, 1984; Davis, 1985).

The RPE at the ventilatory, or lactate, threshold has corresponded to a perception of effort of 'somewhat hard' to 'hard' (13 - 15) on the Borg Scale (1962b, 1985) as determined by a number of authors (Dressendorfer et al., 1981; Purvis and Cureton, 1981; Bellew et al., 1983; DeMello et al., 1985). This relationship does not alter as a result of differences in gender (Purvis and Cureton, 1981; Bellew et al., 1983), nor over a period of days (Bellew et al., 1983) or within the same day from morning to afternoon (Hill et al., 1985). Since the \( \% \text{VO}_2\text{max} \) at the ventilatory threshold generally increases after training, and the RPE is related to \( \% \text{VO}_2\text{max} \), it might be anticipated that the RPE at the ventilatory, or lactate, threshold, is independent of the state of training.

Purvis (1980), for instance, found no change in the RPE at the ventilatory threshold after training but, unfortunately, her training protocol was not of sufficient intensity to alter the \( \% \text{VO}_2\text{max} \) at the ventilatory threshold. Simon et al., (1983), on the other hand, reported a significantly higher RPE at the ventilatory threshold in six trained, compared with six untrained, cyclists (13.5 vs. 10.5) whilst DeMello et al., (1985), using a cross-sectional design also, found no difference in the perception of effort at the lactate threshold in trained and untrained individuals. Hill et al., (1987) trained seventeen subjects (\( \bar{x} \) age range: 23.5 - 25.8yr.) comprising both men and women over six weeks with protocols of interval training and continuous running and cycling. Although means
for the trained subjects were significantly greater (p<0.05) than for the ten controls for VO$_2$max. (6%), work rate (20%), VO$_2$ (23%) and %VO$_2$max.(13%) at the ventilatory threshold, means for the RPE did not differ (2%). Thus, although the relationship between RPE and %VO$_2$max. was altered by training, with trained subjects revealing a lower RPE at a given %VO$_2$max., it was concluded that RPE at the ventilatory threshold was not affected by training, despite the fact that after training the ventilatory threshold occurred at a higher power output and was associated with higher absolute and relative metabolic cardiorespiratory demands.

In summary, RPE appears to be most closely related to the proportion of the maximal capacity required to perform a given power output. For, though maximum oxygen uptake may increase with training, RPE differences have not been observed when oxygen uptake was expressed in 'relative' terms, that is, as a percentage of maximum oxygen uptake. The HR:RPE relationship appears to remain unchanged with training, but the VO$_2$:RPE relationship is altered in terms of raw values though not in relative terms. This conclusion is also true of the ventilatory - lactate threshold which remains unaltered by training and which is a valuable 'action potential' in ensuring that RPE signals are more clear, and induce a greater training effect, at preferably higher work intensities.

2.2.3 AGEING
Since Robinson's (1938) contributions of comparative ageing profiles, many authors have pointed to the close relationship between good physical fitness, and the degree of habitual physical activity in middle-aged, and older, individuals (Strauzenberg, 1981). Ageing is characterised by functional and morphological changes which increasingly impair the general adaptive capacity. Without neglecting the significance of genetic determination, and particularly the suggestion that certain genotypes are more sensitive to training than others (Lortie et al., 1984), comparative tests, including those on monozygotic twins (Klissouros, 1973, 1976), have shown that the physiological process of ageing with reduced adaptability and degenerative processes, is not accelerated by constant use, as is the case with machines, but on the contrary, is slowed down and preserved (Shibayama and Ebashi, 1976; Strauzenberg, 1981). Even so, the decline of the physiological functions owing to ageing appears to be inevitable. Ageing is associated with a reduction in the reserve of virtually all organs and biological systems (Comfort, 1979; Kenny, 1982). Physical work capacity is also reduced with advancing age, including both the maximal attainable levels of external work and rate of aerobic energy expenditure together with the duration that any absolute submaximal load of exercise can be sustained (Robinson, 1938; Buskirk and Hodgson, 1987; Seals, 1993).
In young adults, the continuum which ranges from regular exercise to physical training, increases organ-system reserve, maximal and submaximal physical work capacity and favourably modifies risk factors for many chronic diseased states (Chapman, 1967; Astrand and Rodahl, 1977; Powell et al., 1987; Blair et al., 1989). There is increasing scientific evidence that similarly positive effects of chronic physical activity may occur in older subjects (Holloszy, 1983; Hagberg, 1987; Lakatta, 1993; Seals, 1993; Hagberg, 1994). If so, regular exercise could be an effective intervention for minimizing and delaying the increased prevalence of disability, loss of independence and of the quality of life, and morbidity associated with human aging.

In the present context, it is proposed that age- and training-related adaptations may be monitored efficaciously through the direct expediency of the use of the Borg RPE Scale. Thus, for a given RPE value, for example, heart rate decreases with increasing age; that is, exercise eliciting a given heart rate is perceived to be significantly more stressful for older subjects (Borg and Linderholm, 1967). Furthermore, the predictive power of performance capacity has been found to be equally good from measurements calculated from perceived exertion as for those calculated from heart rates. This has been found valid for other physiological variables related to age, such as: oxygen uptake, breathing frequency, anthropometry, catecholamines, blood lactate and blood pressure (Borg and Noble, 1974). And though the RPE Scale has been primarily devised for use during acute and chronic exercise, threshold values need to be considered as possible baseline variables as a consequence of the ageing process and which may modify quantitative data.

Thus, at REST, on average, cardiac output appears to decline with advancing age, owing primarily to a decrease in stroke volume (Granath et al., 1964; Strandell, 1964; Julius et al., 1967; Conway et al., 1971; Seals, 1993). Yet in healthy older men who have been rigorously screened for the presence of heart disease, cardiac output has not decreased with ageing owing to maintenance of, or even a slight increase in, stroke volume and left ventricular end-diastolic volume, or preload (Rodeheffer et al., 1984). Left ventricular end-diastolic volume in women, however, does not appear to increase with age (Lakatta, 1993), possibly as the result of the lack of oestrogen after menopause (Scheuer et al., 1987; Giraud et al., 1993). In addition, left ventricular contractility, as revealed by the ejection fraction, has not been found to decline with age but other left ventricular changes have been observed in increases in afterload, wall thickness, overall mass and a reduction in peak diastolic filling rate (Folkow and Svanborg, 1993; Lakatta, 1993).

Heart rate has not been found to change with age in the supine position, but appears to decline slightly in the upright position in both men and women (Docherty, 1990; Seals et al., 1994). Arrhythmias, on the other hand, have been observed to increase with age but they are not necessarily clinically significant (Lakatta, 1993).
In the population at large, arterial blood pressure rises with advancing age, and is especially accelerated after the menopause in women (Harlan et al., 1984; Ismaï et al., 1993) but in healthy, active, non-obese older individuals, elevations in arterial pressure are either non-existent or are minor (Rodeheffer et al., 1984; Ng et al., 1993). Where the rise in arterial blood pressure is observed, it has been found to be mediated by an age-related increase in systemic vascular resistance reputedly induced by a combination of structural and neurohumoral mechanisms. These include biochemical alterations in the arterial walls with increased collagen composition and accompanying reduced compliance and elasticity, resulting in increased stiffness in structure (Gerstenblith et al., 1976; Bader, 1983; Fleg, 1986). Sympathetic nerve activity to skeletal muscle (Sundlof and Wallin, 1978; Morlin et al., 1983; Seals et al., 1994) and whole body arterial plasma noradrenaline concentrations with spill-over rates, have been found to be elevated even in healthy older adults free of cardiovascular disease (Esler et al., 1981; Hoeldtke and Cilmi, 1985; Morrow et al., 1987) though the link between these neurohumoral and structural changes has not been determined.

The role of the pulmonary system in gas transport and exercise is a critical one - representing the first and last lines of defence in the regulation of arterial blood O₂ and CO₂ content and blood and tissue acid-base status (Dempsey and Seals, 1994). The major age-related changes in pulmonary system structure are reduced elastic recoil of the lung and stiffening of the chest wall (Turner et al., 1968). The lung connective tissue consists of elastin, collagen and proteoglycans and these elements are cross-linked in a unique way to provide the lung with its elastic recoil (Slonim and Hamilton, 1976). Unfortunately, the precise structural changes in the ageing lung have been difficult to determine as total elastin and collagen in the lung parenchyma and the length and diameter of its elastic fibres remains unaltered (Thurlbeck, 1991). Nor have the alveolar surface forces, produced primarily by lung surfactant, been shown to change with the normal aging process. Thus, more by elimination than anything else, it is generally propounded that the elastic recoil of the lung is reduced because of changes in the spatial arrangement of cross-linking of the elastin and collagen fibres such as loss of branching and the development of parallel orientation (Turner et al., 1968).

Alveolar-capillary surface area of the lung declines with age from about 70m² at 20yr to 60m² at 70-80yr and the total proportion of the lung formed by lung parenchyma declines by about 30% over the same period. The major source of lung capillarization and of the alveolar surface are the alveolar septa which decline in number with ageing so that the air space diameter increases (Brody and Thurlbeck, 1986). This is combined with calcification and reduced compliance of the pulmonary arteries as an ageing manifestation (Reeves et al., 1989). With the stiffening of the chest wall to add to these lung changes, the compliance of the total respiratory system may fall
slightly with age. The rib cage becomes more rigid, probably associated with: costal cartilage calcification, changes in rib to vertebral articulations, narrowing of intervertebral discs and a change in the shape of the chest (Brody and Thurtbeck, 1986; Crapo, 1993). Animal studies also suggest that, as with all locomotor muscles, the ageing diaphragm has a tendency to reduce in muscle mass through a reduction in the diameter of Type II fibres (Kallman et al., 1990). The translation of these structural changes culminates in an increased expiratory flow limitation causing airway narrowing or closure. It is reflected in the routinely measured decline in the FEV$_1$ and FVC. Both these measures show an increase through the early 20s, and a decline from the 30s onwards (Burrows et al., 1983).

It is as well to remember that most of these effects of ageing on pulmonary function are derived from cross-sectional studies so that there are several sources of uncertainty. Data of Ware et al., (1990), comparing cross-sectional, with longitudinal, data suggest that the annual rate of loss of pulmonary function in a longitudinal analysis is much greater than in the cross-sectional data. At age 75yr, reductions of FEV$_1$ and FVC were claimed to be as much as almost twice that of cross-sectional analysis, a result which begs more in-depth investigation.

**ACUTE EXERCISE** is characterised by the maximal oxygen consumption (VO$_{2\text{max}}$) as the ceiling by which submaximal intensities can be determined through relative percentages. Whether expressed as the maximal attainable power output or as the maximal oxygen consumption, this maximal capacity to perform dynamic exercise with large muscle groups declines with advancing age in humans (Robinson, 1938; Buskirk and Hodgson, 1987). The most generally agreed average rate of decrease in VO$_{2\text{max}}$ in non-physically trained subjects is about 10% per decade (Heath et al., 1981; Buskirk and Hodgson, 1987). Astrand and Rodahl (1986) indicated that at the age of 65yr the VO$_{2\text{max}}$ is about 70% of that of a 25yr old; in quantitative terms Hagberg (1987) identified a value of 0.45 ml.kg.min$^{-1}$yr$^{-1}$ decrease starting at 25yr and this confirms earlier studies (Binkhorst et al., 1966; Shephard, 1977). Both cross-sectional and longitudinal studies of sedentary women reveal a rate of decline of less than 0.30 ml.kg.min$^{-1}$yr$^{-1}$ (Hodgson and Buskirk, 1977; Plowman et al., 1979) whilst longitudinal studies (Hollman, 1966; Dehn and Bruce, 1972; Bruce, 1984) among sedentary men found an accelerated rate of decline of approximately 1.0 ml.kg.min$^{-1}$yr$^{-1}$. Such a loss over a 40yr span from ages 25 to 65 years would result in a VO$_{2\text{max}}$ of less than 10 ml.kg.min$^{-1}$ according to Shephard and Sidney (1978) and this is hardly an acceptable value for general application. Even so, such a decline could represent certain segments of, rather than the full, life span. A most recent study by Inbar et al., (1994) found VO$_{2\text{max}}$ values 5-12% lower throughout the age range studied compared with those reported by Dehn and Bruce (1972), Jones et al., (1985) and Wasserman et al., (1987). The relatively low VO$_{2\text{max}}$ and low cardiovascular fitness levels found in this sample of Israeli men may
partially explain the relatively slow VO₂max. age-related decline of 0.33 ml.kg.min⁻¹ yr⁻¹ when compared with American and European-based studies (0.45-0.50 ml.kg.min⁻¹ yr⁻¹). However, distortion in the latter groups may have provided non-representative data by including more active, and fitter subjects together with those involved in leisure time physical activity (>2hr.wk⁻¹) and without discriminating residency as between rural vs urban location (Inbar et al., 1994).

Longitudinal studies (Asmussen and Mathiasen, 1962; Booth, 1989) also show the decline in VO₂max. over a period from 20-90yr to be linear, falling to a basic metabolic rate of about 8 ml.kg.min⁻¹ at about 100yr (Booth et al., 1994). This finding coincides with, and may determine, the maximal life expectancy for humans. Other authors consider the age-related decline to be curvilinear in nature, with an accelerated reduction after 60 years of age (Buskirk and Hodgson, 1987; Rowe and Kahn, 1987). Of course, one of the major linchpins of this parameter is the ability, even questionable desirability, of measuring a 'true' VO₂max., especially in older subjects.

The rate of decrease in VO₂max. with age has been found to be 40-50% less in individuals who continue to perform vigorous aerobic-type exercise on a regular basis (Hagberg et al., 1985; Ogawa et al., 1992) and longitudinal studies suggest that VO₂max. can be fairly well maintained over 10-20 year periods of middle-age in men who continue to train vigorously (Kasch et al., 1990; Marti and Howald, 1990; Rogers et al., 1990) though a similar trend has yet to be identified in women.

The mechanism for the decline in maximal oxygen consumption from the third decade onwards is unknown but there is considerable controversy over what it could be. The one consistent finding with regard to the 'Fick' principle is that an age-related decrease in maximal heart rate of approximately one beat per year from the age of 25yr (Booth et al., 1994) reduces the maximal achievable cardiac output which contributes to the decline in VO₂max. (Rivera et al., 1989; Ogawa et al., 1992).

Recent findings indicate that total blood volume is lower in healthy older, compared to young, untrained men, and that these reductions are strongly related to the ageing decline in VO₂max. (Davy and Seals, 1994). There is also the strong suggestion that VO₂max. and total blood volume are highly correlated in postmenopausal women who differ in their levels of physical activity (Stevenson et al., 1994). Because total blood volume exerts a critical influence on maximal stroke volume and VO₂max. in young adults (Coyle et al., 1986; Convertino, 1991), the age-associated decrease could contribute to the lower maximal stroke volume observed in older subjects.
Changes in body composition appear to play an important part in the decline in VO\textsubscript{2}max. In that, at any given level of whole body weight, ageing is typically associated with a change in body composition such that fat-free mass in general, and skeletal muscle in particular, decrease, and the body fat content increases (Montoye et al., 1965; Poehlman and Horton, 1990). Accelerated loss of total muscle area and decrease in muscle fibre number begins at about 50 years of age with concomitant deterioration in associated motor units and motor neuron function (Booth et al., 1994). Because the absolute VO\textsubscript{2}max. is directly related to the size of the active skeletal muscle mass, these age-associated shifts in body composition and function will, independent of changes in cardiac pumping or peripheral oxidative capacities, cause VO\textsubscript{2}max. to decline with age. Although the exact magnitude of the contribution reported differs among studies (Fleg and Lakatta, 1988; Booth, 1989; Ogawa et al., 1992) it has been estimated that one third or more of the age-related decrease in VO\textsubscript{2}max. can be explained by these anthropometric changes (Ogawa et al., 1992).

The capacity of the older person to undergo peripheral, primarily active skeletal muscle, vasodilation during exercise is apparently impaired as revealed by an elevated level of systemic vascular resistance during exercise at the same %VO\textsubscript{2}max. in both untrained, and trained, subjects (Rodeheffer et al., 1984; Rivera et al., 1989; Ogawa et al., 1992). An increase in arterial stiffness with age (Gerstenblith et al., 1976; Lakatta, 1993) has been identified as a possible attributable factor. However, it should be remembered that, unlike arterial pressure, cardiac output is regulated as a function of the absolute power output, in terms of litres per minute of whole body oxygen consumption, during dynamic exercise. Thus, at the same relative submaximal load, which will be associated with a lower absolute level of oxygen consumption in the older subject owing to a lower VO\textsubscript{2}max., an elevated level of systemic vascular resistance would be an appropriate regulatory adjustment to generate the same arterial blood pressure response as the younger subject in the face of a lower cardiac output (Dempsey and Seals, 1994). In other words, regardless of age, any subject or population with a lower VO\textsubscript{2}max. should demonstrate a higher level of systemic vascular resistance during submaximal exercise performed at the same relative intensity. This would suggest that differences observed with age in the healthy human appear to be appropriately adaptive, rather than maladaptive (Dempsey and Seals, 1994).

It could be assumed that a suitable scale of stress would be capable of monitoring such adaptive responses and differences and that older individuals would respond to prolonged submaximal exercise at a higher level of stress if the above were true. Yet Hagberg et al., (1988), in focussing primarily on the metabolic and endocrine responses to 60 minutes of treadmill walking at approximately 70%VO\textsubscript{2}max. in young and older, healthy untrained men, found that the exercise task actually appeared to be less stressful in the older men, as indicated by smaller increases in
ratings of perceived exertion, plasma lactate and catecholamine concentrations. Reiling et al., (1994) considered that this finding for older subjects was because they undergo less of a loss of cardiovascular and thermal homeostasis by demonstrating less 'cardiovascular drift'. They studied healthy, non-obese, young and older men with similar chronic physical activity levels during 45 minutes of treadmill walking at approximately 65% peak oxygen consumption. As with the study by Hagberg et al., (1988), they observed smaller time-dependent increases in plasma lactate and noradrenaline concentrations during exercise in the older men, although there were no differences in ratings of perceived effort. The progressive rise in internal body (rectal) temperature throughout exercise was much smaller in the older men whereas the time-dependent adjustments in oxygen consumption, heart rate, arterial blood pressure and blood volume were similar in the young, and older, men. Such data indicate that during prolonged submaximal exercise performed at the same relative intensity, older men do not demonstrate a lesser cardiovascular drift, despite a smaller rise in internal body temperature. Or, alternatively, the older subjects actually demonstrated greater metabolic, or whole body oxygen consumption, and circulatory adjustments per unit increase in internal body temperature than the younger controls. These new findings suggest the possibility of an age-related change in the relationship between thermal, metabolic and cardiovascular regulation during prolonged exercise in the human (Dempsey and Seals, 1994).

**CHRONIC EXERCISE**

Over the last decade several studies have confirmed that previously sedentary older men and women demonstrate an increase in VO2max. in response to endurance training if the exercise stimulus is adequate enough (Seals et al., 1984; Seals and Chase, 1989; Hagberg et al., 1989; Makrides et al., 1990; Kohrt et al., 1991; Hagberg, 1994). Older individuals can elicit a similar relative increase in VO2max. as young adults when the training stimulus is sufficiently intense and prolonged but the absolute increase in terms of litres.min⁻¹ is generally smaller; thus, the similar percentage increases are owing to the lower pre-training baseline levels in older subjects. Using similar relative exercise intensities such percentage increases appear to be equivalent in older men compared to older women, but the absolute increases appear to be greater in the men, again owing to the lower baseline levels of the older women (Kohrt et al., 1991).

The magnitude of the percentage increase in VO2max. with endurance training varies markedly among older individuals. Seals et al., (1984) found an average increase of 30% following one year of vigorous training with a range of 2 - 49%, a finding confirmed by Kohrt et al., (1991). The reasons for this large variability, and hence in the range of VO2max. in older adults, have not been fully established. It has been reported that the baseline levels of VO2max., the intensity of the training, the reason for stopping the exercise test, and the skinfold thicknesses, all correlated with
the post-training VO₂max. levels and accounted for 62% of the variance (Thomas et al., 1985). The intensity and length of exercise training have always been reputed to exert a powerful effect on the magnitude of the VO₂max. increase and has been reconfirmed by a number of studies (Seals et al., 1984; Hagberg, 1987; Hagberg et al., 1989); the mode of exercise may also have an influence (Seals et al., 1984). Even so, not all data support the intensity of training as being such an important determinant for the increase of VO₂max. in older subjects (Badenhop et al., 1983; Foster et al., 1989; Belman and Gaesser, 1991) and in the study by Kohrt et al., (1991) the percentage increases in VO₂max. were not related to age, initial pre-training levels, or gender.

However, from a purely theoretical, physiological standpoint, it is perhaps worth stating that factors related to training stimulus, the subject's initial levels of fitness and genetic predisposition (Bouchard et al., 1992) should be considered as being highly influential among the critical determinants of the increase in maximal aerobic power among all healthy individuals whether young or old. Supporting such a generalised hypothesis are the findings related to degree of training intensity: very low intensity, for instance, may produce little or no increase in VO₂max. in older adults (Benestad, 1965; DeVries, 1970; Niinimaa and Shephard, 1978; Warren et al., 1993; Hagberg, 1994); moderate intensity training over one year was found to produce an average increase of 11% in VO₂max. in 100 men (Cunningham et al., 1987) whilst higher intensity levels, carried out over 6 - 12 months, have evoked approximately 20 - 30% increases in middle-aged and older men and women (Seals and Chase, 1989; Hagberg et al., 1989; Makrides et al., 1990; Kohrt et al., 1991).

The mechanisms responsible for increased VO₂max. values and adaptations wrought by submaximal endurance training can be classified under central circulatory, and peripheral oxidative, responses other than the obvious body composition-related mechanisms already considered. Between 50-100% of the higher levels of VO₂max. in older male endurance athletes compared to age- and gender-matched untrained controls, have been found to be owing to greater maximal cardiac output which is solely the result of a higher maximal stroke volume because maximal heart rate does not differ in the two groups (Hagberg et al., 1985; Fuchi et al., 1989; Rivera et al., 1989; Ogawa et al., 1992). These are cross-sectional findings but they have been confirmed by a recent longitudinal study (Spina et al., 1993a) and by findings reported for peak leg cycling stroke volume and cardiac output following intensive cycling training in older men (Makrides et al., 1990). Longitudinal investigations in older men after vigorous training (Ehsani et al., 1991) also indicate that the increase in maximal exercise stroke volume is associated with increases in left ventricular (LV) end-diastolic volume as well as enhanced LV systolic contractility during peak exercise. This former finding may (Levy et al., 1993), or may not (Schulman et al.,
be mediated in part by an increase in the peak early diastolic filling rate during exercise, which has been correlated with the magnitude of the increase in VO2max.

In contrast, the increase in VO2max in response to prolonged and intensive endurance training in older women is not associated with increases in maximal cardiac output nor stroke volume, LV end-diastolic volume, or LV contractile performance (Spina et al., 1993a; Spina et al., 1993b). This may be because the older, post-menopausal, women studied to date experience hormonal changes, particularly a lack of circulating oestrogen (Scheuer et al., 1987; Giraud et al., 1993). Young women, in comparison, demonstrate the same endurance training-evoked adaptations in cardiac pump function during maximal exercise as young, and older, men (Ehsani et al., 1991; Spina et al., 1992). Presumably, hormone replacement therapy studies could clarify this issue?

Studies which focus on the adaptations in active skeletal muscles indicate that an increase in maximal arterio-mean venous oxygen difference (a-v O2diff.) can account for up to 50% of the increase in VO2max in response to endurance training (Rivera et al., 1989; Ogawa et al., 1992; Spina et al., 1993a). Data from older women suggest that their entire training-induced elevation in VO2max is mediated by an increase in maximal a-v O2diff. (Spina et al., 1993b). Such elevated differences in both men and women are associated with increases in capillary density and oxidative enzyme activities in the trained skeletal muscles, suggestive of an enhanced capacity for oxidative energy production (Coggan et al., 1990; Coggan et al., 1992; Roger and Evans, 1993). Endurance training also appears to produce adaptations in the resistant arterioles of the active muscles resulting in peak vasodilatory capacity and peak reactive blood flow (Romanovski et al., 1981; Martin et al., 1990; Martin et al., 1991) and such adaptations have been found to relate strongly to increases in VO2max.

Under resting conditions, following longitudinal studies on training adaptations, older adults demonstrate reductions in heart rate, or a 'training bradycardia', together with lowered blood pressure (Benestad, 1965; Cononie et al., 1991). Experimental evidence suggests that the former is mediated by an increase in cardiac vagal tone and a reduction in intrinsic heart rate (De Meersman, 1993; Denahan et al., 1993). Plasma noradrenaline concentrations at rest have been reported to be unchanged after training (Cononie et al., 1991; Kohrt et al., 1993), but more sensitive measures of sympathetic activity, such as noradrenaline appearance rates and directly recorded muscle sympathetic nerve activity are, in reality, elevated in the endurance-trained state (Coyle et al., 1986; Poehlman et al., 1992).

In older men, the adaptations observed during submaximal exercise performed at the same absolute workload before and after training, are similar to those documented in young adults:
heart rate, systemic vascular resistance and plasma catecholamine levels are lower, stroke volume is higher and oxygen consumption, cardiac output and a-\(\bar{V}\) O\(_2\) diff. usually are unchanged (Seals et al., 1984; Spina et al., 1993a). Cross-sectional studies on older male athletes confirm, and support, these findings (Hagberg et al., 1985). As with \(VO_2\) max. responses, however, older women, in contrast (Spina et al., 1993a), reveal that after similar absolute workloads, the same rate of oxygen consumption is maintained at a lower level of cardiac output owing to a lower heart rate, unchanged stroke volume and higher a-\(\bar{V}\) O\(_2\) diff. The reason for the lack of an increase in stroke volume with endurance training in older women, has yet to be determined.

Finally, the lower heart rate and plasma catecholamine levels during submaximal exercise following vigorous training in older men and women have also been found to be related to the increase in \(VO_2\) max. (Kohrt et al., 1993). This is probably owing to the fact that the same absolute external power output represents a progressively lower relative exercise stress when measured against the greater training-associated increase in maximal exercise capacity.

In conclusion, age and training, when considered as separate influences, can be seen to drive the sense of stress in diametrically opposite directions when older individuals are working at the same absolute level of submaximal work intensities. When considered in juxtaposition, age and training, as measured at the same relative work intensity, can provide the older subject with a physiological environment even less stressful than that of the younger counterpart even though the mechanisms involved in, for instance, 'cardiovascular drift' are similar and metabolic demand per unit increase in internal body temperature is greater in the older person. A similarity in the sense of stress between older men and women may require careful revision in view of findings which reveal that training adaptations for older women are more weighted towards peripheral mechanisms, such as increases in a-\(\bar{V}\) O\(_2\)diff., rather than central circulatory changes through enhanced stroke volume, as with older men. The Borg Scale may require modifications in the light of these differences but equally, such changes should be regarded as reflecting appropriate adaptive, rather than maladaptive, morphology.
CHAPTER 3

3. GENERAL METHODOLOGY

No one believes an hypothesis except its originator, but everyone believes an experiment except the experimenter.

W.I. Beveridge, 1908

The present body of work comprises two Experimental Studies and two Training Studies with the addition of two ancillary studies in anthropometry and dietary analyses linked to the first and second training studies, respectively. Subjects who participated in these studies did so with the prior approval of the University Ethical Committee and after giving their informed, signed consent. Depending on the type of study that they entered, subjects completed a selection of the following tests:

3.1 TESTS OF FUNCTIONAL CAPACITY

3.1.1 PREPARATORY MEASUREMENTS

QUETELET INDEX: this is a body mass index (BMI) derived from the relationship between body mass and height:

\[
\text{BMI} = \frac{\text{body mass (kg)}}{\text{height}^2 (m)}
\]

The graded indices are common to both men and women and are classified as:

- \(<20 \text{ kg.m}^{-2}\) - leanness.
- \(20 - 24.9 \text{ kg.m}^{-2}\) - 'ideal'.
- \(25 - 29.9 \text{ kg.m}^{-2}\) - overweight.
- \(30 - 40 \text{ kg.m}^{-2}\) - obese.
- \(>40 \text{ kg.m}^{-2}\) - severely obese.

(Pacy et al., 1986).

These indices were calculated for each of the participants in the present studies.

ADIPOSITY: was obtained by measurements taken at four carefully marked anatomical sites of biceps, triceps, subscapular and suprailliac using the left side of the body and following the procedures of Marshall (1977). The Holtain caliper, exerting a constant pressure of 10 g.mm\(^2\) between its jaws was used, and was capable of taking readings to the nearest 0.1 mm. Adiposity percentage was calculated from regression equations produced by Durnin and
Womersley (1974), classified according to age, and which had been derived using Siri's (1956) equation.

**BLOOD PRESSURE:** following a 10 minute rest period, subjects' resting blood pressure was measured using an automatic sphygmomanometer (Bonn, Model D). This measurement was repeated 5 minutes after the cycle ergometer test of progressive intensity.

**VENTILATORY MEASURES:** vital capacity and forced expiratory volume in one second at BTPS were measured using a dry Spirometer (S-Model, Vitalograph Ltd.) Peak flow was obtained by means of the Mini Peak Flow Meter (Aimed Ltd., M3410).

**RESTING METABOLIC RATE:** whilst the subject rested quietly, a 6 minute sample of expired air was collected and analysed by standard Douglas bag methods. This enabled a resting Respiratory Exchange Ratio (R) value to be determined.

3.1.2 **HEART RATES:** during the pilot studies, resting, intra-test and post-test heart rates were recorded using a battery operated Cardiometer 275 (Cardionics Ltd.) with spongy electrodes soaked in electrolyte fluid to improve contact. Heart rates during the training study tests were monitored on a Rigel oscilloscope (Cardiac Monitor, 302) from 3 chest electrodes (3M Red Dot, 2255). The first of these was placed slightly inferior to the sternal angle, the remaining two in the V5 lead positions inferior to each nipple. The skin was thoroughly cleansed with an ethanol swab (Sterets, Seton Prebbles Ltd.) and slightly abraded to lower skin resistance prior to applying each electrode. During the 30 minute endurance tests, the Rigel cardiometer was interfaced with the microcomputer and a hard copy of the heart rates was generated and printed automatically every 15 seconds. Maximum heart rate for age was estimated using Astrand's (1960) equation:

\[
HR_{\text{max}} = 210 - (\text{age} \times 0.65)
\]

**CYCLE ERGOMETRY:** two cycle ergometers (Monark) were used throughout the studies. Both were friction - braked devices and therefore power output depended on both the resistive force and pedal frequency. The resistive force for all tests except the 30 minute endurance test was applied by means of an adjustable vertical arm (Model 818E). For the 30 minute test, a basket weight loading mechanism was utilised (Model 864). The load setting was manually administered by adding free weights to the basket, the weights ranging from 0.1 to 3.0 kg in size. The basket itself weighed 0.5 kg and therefore represented the lowest
frictional load available during testing. Both models possessed adjustable handlebars, saddle height and toe straps. Prior to each test, the saddle was adjusted so that the driving leg for each subject was 'athletically straight' on completion of the downstroke. These adjustments were made in order to standardise the posture of the subject on the two types of ergometer. Subjects were required to exercise at a pedal frequency of 60 rpm as indicated on the speedometer attached to the handlebars of each model. Calibration prior to each study was checked according to guidelines suggested in the Monark handbook (Astrand, 1980).

**WORKRATES**: these were calculated from the relationship between force, distance and time:

\[
\text{Work rate} = \frac{\text{Force} \times \text{Distance}}{\text{Time}}
\]

where force is the frictional load (e.g. 1.5 kg) multiplied by gravitational acceleration (9.81 m s\(^{-1}\)s\(^{-1}\)), and distance is the flywheel revolutions (e.g. 222) multiplied by the flywheel circumference (1.622 m) over the time period of 60 seconds. Thus the power output of a subject cycling against a resistance of 1.5 kg would be:

\[
(1.5 \times 9.81) \times (222 \times 1.622m) = 88.3 \text{ Nm.s}^{-1} \text{ or W}
\]

60s

To obtain a pedal frequency value, the number of flywheel revolutions was divided by a conversion factor of 3.7 (the ratio of flywheel revolutions to pedal revolutions) and thus a flywheel count of 222 in one minute would represent the desirable pedal rate of 60 rpm. In order to sample the precise number of flywheel revolutions, since cycling at precisely 222 on every occasion was extremely difficult, an electro-mechanical counter was attached to the flywheel of the cycle ergometer so that the exact rpm could be determined.

**EXPIRED AIR COLLECTION AND ANALYSIS**: as part of a familiarisation for all tests, a practice period was provided in order to enable subjects to become accustomed to a low resistance, two-way valve (Jakeman and Davies, 1979). Subjects breathed through this by means of a rubber mouthpiece (Harvard Equipment) wearing a noseclip. Wide bore (30mm), low resistance tubing (Falconia Ltd.) connected the valve to a two-way tap linked to a 150 litre capacity Douglas Bag (Harvard Equipment).

**OXYGEN ANALYSIS**: the oxygen content in expired air was analysed using a paramagnetic oxygen analyser (Sybron Taylor Servomex, Model 570A) with a digital read-out.
Figure 3.1

RATE OF PERCEIVED EXERTION

6
7 Very, very light
8 Very light
9 Fairly light
10 Somewhat hard
11 Hard
12 Very hard
13 Very, very hard
14 Maximal exertion

Borg, 1962

Figure 3.2

RATE OF PERCEIVED EXERTION

6 No exertion at all
7 Extremely light
8 Very light
9 Light
10 Somewhat hard
11 Hard (heavy)
12 Very hard
13 Extremely hard
14 Maximal exertion

Borg, 1985
accurate to 0.1%. The calibration, and gas analysis, procedures for this instrument can be found in Appendix 1.

CARBON DIOXIDE ANALYSIS: this was carried out using an infrared carbon dioxide analyser (Mines Safety Appliances Co., Lira Model 303). The meter reading displayed by the analyser was in an analogue form and was converted to a carbon dioxide percentage by means of a calibration curve supplied by the manufacturer. Calibration and procedural methods can be found in Appendix 1.

GAS VOLUMES: were determined using a dry gas meter (Harvard Equipment) with a digital read-out accurate to the nearest 0.1%. A thermistor, fitted inside the air inlet pipe, was linked to a thermometer (Edale, Type 2984, Model C) and enabled the temperature of the expired air to be measured.

RATE OF PERCEIVED EXERTION (RPE): during each expired air collection in the experimental studies, in the tests of progressive intensity and in the Lactate Power Tests, subjects were required to indicate the level of stress by means of the Borg Scale (1962a; Figure 3.1). This is a fifteen point graded category scale from 6 to 20 where 6 represented 'No exertion at all' and 20 'Maximal exertion' (Borg, 1985; Figure 3.2). The power output for the 30 minute tests of endurance was set by the unique method of using a single rate of perceived exertion. This required each subject and control to set a power output on the cycle ergometer at an RPE of 14 and to repeat this process following training (Figure 3.1).

3.2 THE LABORATORY STUDIES: these comprised 5 in number of which details are found in the relevant chapters.

3.3 THE 30 MINUTE CYCLE ERGOMETER TEST OF ENDURANCE PERFORMANCE.
For the second of the two experimental studies, a test was devised which required the subjects to cycle for thirty minutes on two occasions:-

1) At a pre-set power output which was extrapolated from the relationship between heart rates and exercise intensities obtained from Experimental Study 1 and:

2) At a self-selected power output using the criterion of 'Just Tolerable Discomfort' as a guideline.
For the former, subjects were set a specific exercise intensity and asked to maintain it for 30 minutes. For the latter, subjects were permitted five minutes in which time they could experiment freely with various exercise intensities. As previously indicated, an 'adjustable vertical arm', or extended sleeve, was adapted for, and attached to, the normal adjustable loading arm of the ergometer (Model 818E). This enabled the subject to make fine adjustments to his choice of exercise intensity over the five minute warm-up period without having to alter his posture unduly.

Once the warm-up period was completed, the subject was told the power output he had set and was then required to maintain it for the full 30 minutes. From the results of this latter test it was decided to adopt it as the focal test of subsequent training studies since it provided data which most cogently reflected the aims of each of the tests carried out.

THE COMPUTER SYSTEM: for the purpose of continuously monitoring the pedal frequency during such a 30 minute endurance test, a computerised data logging system was devised. A small D.C. generator (R.S. Components Ltd.) driven by the flywheel of the cycle ergometer, produced a voltage proportional to the speed of the flywheel. This voltage was fed into a microcomputer (BBC, Model B) via an internal analogue-to-digital converter. The system sampled at approximately 10Hz. Once converted into SI units the output voltage values, together with information concerning the applied frictional load, were used to calculate power output. During the test, the computer screen displayed:

i) Time elapsed (minutes and seconds).
ii) Pedal revolutions (rpm), updated every 2 s.
iii) Average pedal revolutions over a sampling period of 15 s (rpm/time), updated every 15 s.
iv) Average work rate (AWR) over a sampling period of 15 sec(work rate/time), updated every 15 s.
v) Cumulative average work rate (CAWR - total work done/total exercise time) updated every 15 s.

Every 15 s a hard copy of all the above information was generated on a Cannon Printer (Type PW-1080A).

VISUAL DISPLAY UNIT (VDU): constant feedback on performance was available to the subject throughout the test from the microcomputer screen positioned in front of the cycle ergometer (Figure 3.3). In addition to the data already outlined, performance was also displayed graphically on the VDU (Figure 3.4). The abscissa of the graph represented the
time constant divided into 30, one-minute periods. The ordinate represented the pedal revolutions in rpm. At intervals of 0.5 s a vertical line, proportional to the pedal revolutions, was built up providing an ongoing display of the subject's power output throughout the test. Once complete, the top of this vertical cursor reached a pair of horizontal lines if the subject had maintained the correct pedal revolutions of 60 rpm. As the test progressed, the space between the abscissa and these horizontal lines, representing rpm, was blocked in (Figure 3.4). This provided the subject with information as to the progress of the test and his precise performance during its prolonged completion in terms of immediate and completed exercise intensities. In addition, expired air samples and RPEs were collected every 5 minutes throughout the test as shown by Figure 3.3.

**CALIBRATION:** during the initial data collection, a discrepancy was discovered between the computer calculated value for power output and the associated mechanical check procedure used. It was found that the input signal from the small dynamometer driven by the cycle ergometer flywheel, varied according to the time allowed for the computer to warm-up. The method of overcoming this signal error was to ensure that the computer was switched on at least 30 min prior to each test and was set according to a calibration factor before each test.

**TEST-RETEST RELIABILITY:** 30 minute endurance test (T30).

This test revealed good test-retest reliability when 16 subjects (6 females and 10 males) self-selected exercise intensities at an RPE of 14 (T1) and repeated the test (T2) after an average of six days. There were no significant differences between Cumulative Average Work Rates (CAWRs) nor Average Work Rates (AWRs) of the two tests and high correlations were found between them at:

<table>
<thead>
<tr>
<th>Time</th>
<th>CAWR (W)</th>
<th>AWR (W)</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 min</td>
<td>144.9 ± 47.1W</td>
<td>146.5 ± 48.1W</td>
<td>r=0.96</td>
</tr>
<tr>
<td>10 min</td>
<td>145.6 ± 46.9W</td>
<td>147.0 ± 48.5W</td>
<td>r=0.96</td>
</tr>
<tr>
<td>20 min</td>
<td>145.6 ± 46.8W</td>
<td>147.1 ± 48.1W</td>
<td>r=0.96</td>
</tr>
<tr>
<td>30 min</td>
<td>145.3 ± 46.1W</td>
<td>147.1 ± 47.6W</td>
<td>r=0.96</td>
</tr>
</tbody>
</table>

(Mean ± SD)

These correlations were higher than those reported by Wilmore (1969) following test-retest exercise time to exhaustion (r=0.89) and for total work output (r=0.83) during a constant load test. They were better than those found by Katch and Katch (1972) for test-retest 'cumulated work performed' in a 10 minute 'drop-off' test and by Weltman and Regan (1982) for a test-retest of performance time and pedal revolutions in a constant load cycle ergometer test.
Figure 3.3 The 30min. test protocol utilised during the two training studies.

Time = 14min 50sec

Current RPM = 59.6  Aver. Revs. = 61.2
C.A.W.R. = 119.2  Work Rate = 120.3
Applied Load = 2kg  Heart Rate = 127

Figure 3.4 The VDU as seen by the subject in completing the 30 minute test.
(r=0.92). However, they were not quite as good as that reported by Boulay et al., (1984) for test-retest of total work output during a 90 minute 'Maximal Aerobic Capacity' test (r=0.99). Nevertheless, such high correlations failed to distinguish the heterogeneity of the group whereas using 95% agreement limits (Altman, 1991) the female group revealed better repeatability than their male counterparts as shown by the smaller range:—

<table>
<thead>
<tr>
<th>Males (Range in parentheses)</th>
<th>Females Units = Watts.</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 min</td>
<td>-20.0 to 10.4 (30.4)</td>
</tr>
<tr>
<td>10 min</td>
<td>-10.0 to 10.1 (20.1)</td>
</tr>
<tr>
<td>20 min</td>
<td>-8.3 to 10.1 (18.4)</td>
</tr>
<tr>
<td>30 min</td>
<td>-9.5 to 10.0 (19.5)</td>
</tr>
</tbody>
</table>

As the above calculations revealed no significant differences between CAWRs. and AWRs., it was decided to adopt the former measure in recording exercise intensities throughout the studies. This would more closely monitor the ability of the subject to maintain the correct output, it was felt, than an average power output which would tend to mask individual discrepancies within a minute-by-minute average.

A further study (Mayes, 1987), carried out in our laboratories between the two present training studies and using the same 30 minute test, required subjects to cycle at 80% VO2max. Significant differences were found in CAWRs at 10 and 20 minutes when the same test was repeated. The suggestion from this was that the test should be performed on two occasions in order to familiarise the subjects. However, it was also noted that, owing to the high reproducibility of the test (r=0.93), in 'extreme circumstances', the results from a single test would be adequate and acceptable. From these equivocal findings it was decided to continue with a single 30 minute test in the present series of studies for the following reasons:—

i) A slightly higher correlation of 0.96 had been found during the reproducibility tests for the present studies.

ii) The present test protocol differed substantially in that it examined the subjective choice of a power output as influenced by the individual's own level of fitness and degree of experience in completing a task of sustained endurance. In the pre-trained, sedentary state such a choice may well have embraced a degree of 'naivety' which it was desirable to monitor. This may well have been lost had an additional 30 minute test been permitted.
iii) It was considered that the preliminary tests of progressive intensity and the Power Lactate Tests, would have provided invaluable experience for the sedentary novice. He would have gained knowledge of the level of physical stress he was capable of enduring hand-in-hand with psychological responses from the obligatory feedback of the Borg rating scale. Thus, he should have been in a position to make valid deductions as to his level of fitness and his capacity to sustain a self-selected power output over the time course of 30 minutes.

3.4 FIELD TESTS AND TRAINING: for both training studies, subjects trained for 12 weeks, exercising regularly at least three times a week for at least 30 minutes on each occasion. During TS1, two of the training sessions were carried out under supervision in the laboratory on a cycle ergometer (Monark, Model 818E). For the third session, subjects were permitted to choose their own form of aerobic training. Such activities of a 'steady state' nature as walking, jogging, rowing ergometry, swimming and cycling were included. Because of the large numbers of subjects in TS2, only one training session each week was supervised in the laboratory.

Every three weeks, the training intensity on the cycle ergometer was increased according to a simple pulse rate formula after Karvonen et al., (1957):

\[ I = \frac{TP - RP}{MP - RP} \times 100 \]

where \( I \) = Intensity, \( RP \) = Resting Pulse, \( TP \) = Training Pulse and \( MP \) = Maximum Pulse.

In the early days of TS1 and at its conclusion, subjects were required to complete three training runs:

1) The 12 minute Cooper Run (Cooper, 1981).
2) A 2 mile (3219 metre) run.
3) A 5 kilometre run from which maximum oxygen uptake \( (V\text{O}_2\text{max}) \) was predicted (Ramsbottom et al., 1987).

For TS2, the three field tests of TS1 were replaced by a Multistage Fitness Test (Leger and Lambert, 1982) and a 2 mile run (after Bland, 1982). As before, these tests were incorporated into the programme during the early, and final, days of training. From both these tests it was possible to predict \( V\text{O}_2\text{max} \).
TRAINING DIARIES were kept by all subjects during both training studies and submitted at the conclusion of training. From the data supplied it was possible to determine overall changes in training intensity throughout the training period.

3.5 BLOOD SAMPLING AND ANALYSES:-

1) Power Lactate Tests.
Lactate and Glucose concentrations.
Duplicate 25ul aliquots of arterialised capillary blood samples were taken from the right thumb every four minutes during the tests using an Autoclix (Boehringer Mannheim) and calibrated micropipettes (Dade Diagnostic Inc.). Resting samples were obtained from a pre-warmed hand. The final sampling was taken three minutes following the conclusion of each test. These samples were immediately deproteinised in 2.5% perchloric acid at room temperature, centrifuged for 3 minutes (Eppendorf, 5414 and 5412) and stored at -20°C until assayed. Samples were analysed at the same time after the test period in order to avoid inter-assay error.

Blood lactate concentrations were determined by means of Olsen’s modified fluorimetric method (Maughan, 1982) and blood glucose concentrations using the GOD photometric method (Werner et al., 1970). Assay details for both methods are given in Appendix 2.

2) Thirty Minute Endurance Tests (T30).
Haemoglobin, Haematocrit, Lactates and Glucose.
Subjects rested for 5 - 10 minutes prior to the test, after which a 10ml sample of blood was drawn from the antecubital vein of the arm. A similar sample was taken one minute following the conclusion of the test. The samples were placed in tubes containing the anti-coagulant, lithium heparin. Duplicate 20ul samples were then taken from the tubes for the immediate determination of haemoglobin concentrations. Haematocrit was determined at the same time from centrifuged haematocrit tubes and read from the Hawksley Micro Scale. Aliquots (25ul) of blood were also withdrawn for the determination of lactate and glucose concentrations as previously described.

Plasma Catecholamines and Blood Lipids.
From the remaining 8-10ml of blood, plasma was separated by centrifugation at 4°C and 6000 rpm for 15 minutes (Burkard, Koolspin), 3ml were drawn off and treated with 200ul of a mixture of 100 mmol.l⁻¹ of GSH (reduced glutathione) to act as an anti-oxidising and chelating agent.
The treated plasma was stored at -20°C and assayed at a later date for adrenaline and noradrenaline by means of Liquid Chromatography with Electrochemical Detection (LCEC). The method relied on a simple liquid-solid extraction of the catecholamine onto alumina, followed by their elution with 0.1 M perchloric acid (Davies et al., 1981; Eriksson and Persson, 1982; Hallman et al., 1978).

The remaining plasma was also stored at -20°C in heparinised tubes for the analysis of blood lipids. Free fatty acids (FFA) were analysed by a photometric, colorimetric assay modified from Chomy et al., (1977), and glycerol was determined enzymatically from a fluorimetric assay modified from Laurell and Tibbling (1966). Total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) were determined by an enzymatic, colorimetric method after phosphate and magnesium chloride precipitation (Siedel et al., 1981) using commercially available reagents (Boehringer Mannheim).

Reliability of biochemical assays was determined, either by a Pearson product moment correlation coefficient of 0.999 between standards and optical densities or a coefficient of variation of assay in the region of 2%.

Details of all assay methods can be found in Appendices 2, 3 and 4 and their subsections.

3.6 STATISTICAL METHODS.

Standard parametric statistical techniques were used throughout (Cohen and Holliday, 1979). Unless stated otherwise, all values reported in the text and the tables refer to group means (x) and standard deviations (SD). Correlations between variables were assessed using the Pearson product moment coefficient. When test parameters before and after a training period were compared with each other, Student's t-test for correlated data was used. Paired t-tests for independent samples were used to evaluate differences in parameters between training and control groups; pooled t-tests determined differences within and between groups for biochemical measurements. A 4-Way Analysis of Variance (ANOVA) and Covariance (Biomedical Data Package [BMDP] 2V, Statistical Software Inc., 1440 Sepulveda Boulevard, Los Angeles, CA90025, USA, 1985) with repeated measures was used to determine interactions within and between groups (Winer, 1971). It was related to time (duration of test), age (30-40-50-60yr bands), group (experimental and control) and training (pre and post) for Training Study 1 (TS1), and between groups, training, gender and age for Training Study 2 (TS2). The null hypothesis (Ho) was rejected at the 0.05 level of significance, though that at the 0.01 level was also reported.
CHAPTER 4

4. EXPERIMENTAL STUDY 1

Physical training should aim at optimal effect with the least possible strain.

(1) Astrand, 1960

4. CARDIOVASCULAR RESPONSES OF TWO GROUPS OF MIDDLE-AGED MEN TO A CYCLE ERGOMETER TEST OF PROGRESSIVE INTENSITY.

4.1 INTRODUCTION.

In exercise tests which attempt to cater for sedentary and active individuals alike, the test protocols involve a graded progression from low levels of energy expenditure until fatigue, or attendant symptoms of stress, intervene (Bruce et al., 1963; Bruce and McDonough, 1969). Whereas middle-aged subjects, whether active or inactive, may not wish to push themselves to complete exhaustion, they may be willing to work along a continuum of increasing energy expenditure which they are able to terminate at any point where discomfort is experienced. Thus, some subjects are content to work strictly within submaximal limitations whilst others are prepared to push themselves in extremis. It seems to be a measure of a person's fitness that he is able to tolerate a higher incremental exercise intensity than his unfit counterpart and is also able to endure a higher subjective feeling of stress.

Therefore a test was chosen which was simple to administer and could relate heart rates to other parameters. The Physical Work Capacity test at a heart rate of 170 beats.min⁻¹ (PWC₁₇₀) is such a test (Sjöstrand, 1947; Wahlund, 1948; Balke, 1954; Borg and Noble, 1974). It is designed to measure cardio-respiratory endurance and is based on heart response to a graded sequence of three consecutive workloads using a cycle ergometer and aiming to bring the subject's heart rate to 170 beats.min⁻¹ by the conclusion of the test. Only the values of the last two workloads are used for the calculation of the PWC₁₇₀. Because some of the subjects' predicted maximum heart rate values in this study were below, or close to, 170 beats.min⁻¹ according to Astrand's (1960) equation, the test was modified, as indicated in the method.

Therefore, the purpose of this initial study was twofold, namely to:-

a) Select, or devise, a suitable ergometer test for middle-aged, male subjects whose level of fitness may be recondite and:
b) Determine the most clear-cut differences between active and inactive subjects of this age group by means of such a test and with special reference to heart rates.

4.2 METHODS.

4.2.1 SUBJECTS.
Forty middle-aged men were divided into two groups, 20 active and 20 inactive, according to their lifestyles. Their ages and physical characteristics are shown in Table 4.1. The active group consisted of those subjects who trained at least twice a week for at least thirty minutes on each occasion; many were in regular training on a daily basis. Others were included whose life-styles encompassed some form of active exercise. Activities were consequently varied and ranged from bi-daily cycling, recreational jogging, regular swimming, athletics, tennis, squash and weight training to training for the half-marathon. The level of habitual activity of the inactive group was negligible, ranging from the purely sedentary to once-a-week golf. Many were content to allow their occupations to provide exercise for their needs.

4.2.2 PROTOCOL.
Subjects were familiarised with ergometer cycling on a day prior to the test together with practice in wearing the mouthpiece and noseclip and establishing the correct saddle height. They arrived at the laboratory on the day of the test in the post-absorptive state, not less than two hours since their last meal, when they were first weighed and measured. Four skinfold readings were then taken in order to determine body fatness, followed by a ten minute rest period when resting heart rates were measured using a battery operated cardiometer (Cardionics Ltd.) with spongy electrodes, as described in 3.1.2. Subjects were given a five minute warm-up period on a cycle ergometer (Monark 818E) when the method of signalling both during, and at the conclusion of, the test was agreed since noseclip and mouthpiece were worn continuously throughout the test. The method of indicating the stress level on the Borg Scale (RPE) was also established. The test load on the ergometer started at 0.5 kg; thereafter the load increased every two minutes by a further 0.5kg until the subject signalled that he only wished to continue for a further minute.

Expired air samples were collected during the final minute of each power output using the standard Douglas bag technique and analysed for oxygen and carbon dioxide as outlined in Section 3.1.2. Pulmonary ventilation was measured by means of a Dry Gas Meter (Harvard Equipment). Rates of Perceived Exertion were also collected during this final minute whilst heart rates were monitored continuously.
TABLE 4.1

Physical characteristics of Active and Inactive male subjects.

(Mean ±SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Active</th>
<th>Inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>46.8 ±7.7</td>
<td>46.1 ±6.0</td>
</tr>
<tr>
<td>Range</td>
<td>(34 - 64)</td>
<td>(36.6 - 62)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>177.0 ±5.7</td>
<td>177.6 ±5.8</td>
</tr>
<tr>
<td>Range</td>
<td>(165.3 - 185.9)</td>
<td>(166.2 - 188.5)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>73.9 ±8.0</td>
<td>79.4 ±12.1 **</td>
</tr>
<tr>
<td>Range</td>
<td>(58.3 - 87.1)</td>
<td>(64.3 - 106.8)</td>
</tr>
<tr>
<td>Adiposity (%)</td>
<td>19.4 ±3.2</td>
<td>24.1 ±3.5 **</td>
</tr>
<tr>
<td>Abs. Body Fat (kg)</td>
<td>14.5 ±3.5</td>
<td>19.4 ±5.3 **</td>
</tr>
<tr>
<td>Lean Body Mass (%)</td>
<td>80.6 ±3.2</td>
<td>75.9 ±3.5 **</td>
</tr>
<tr>
<td>Lean Body Mass (kg)</td>
<td>59.5 ±5.4</td>
<td>60.1 ±7.8</td>
</tr>
<tr>
<td>Quetelet Index (kg.w/ht²)</td>
<td>23.5 ±1.8</td>
<td>25.3 ±3.3 *</td>
</tr>
<tr>
<td>Lean Body Mass (kg.w/10cm.ht)</td>
<td>3.4 ±0.2</td>
<td>3.4 ±0.4</td>
</tr>
</tbody>
</table>

* Significant difference between groups (p<0.05)

** Significant difference between groups (p<0.01)
Prior to the study, the reliability of the test was established on a test-retest basis using 10 male subjects who repeated the test following a seven day interval. All subjects cycled for eight of the loads, that is, up to and including 4kg. When workrates were compared, a perfect correlation ($r = 1.000$) was found between the data at the 95% level of confidence.

Inferential statistics in the form of a one-tailed paired 't' test for correlated data on independent samples was used for analysis. A Pearson product moment correlation was used to examine the relationship between variables.

4.3 RESULTS.

The two groups were homogeneous in age and height (Table 4.1) but the inactive group was heavier ($p<0.01$) and this was reflected in the higher Quetelet Index. This BMI classified the active group as 'ideal', the inactive as 'overweight'. The inactive group also possessed a significantly larger fat mass both in absolute, and relative, terms. The active group, however, revealed a greater percentage of lean body mass ($p<0.01$) though there were no differences in absolute lean body mass nor in the lean body mass to height ratio.

Maximal oxygen uptake ($VO_2max$) was estimated from a modified Astrand - Ryhming Nomogram (1954), using nine data points to reduce error (Maritz et al., 1961) and fitting straight lines by the method of least squares (Figure 1, Appendix 5). Oxygen uptake was extrapolated to the age-predicted maximal heart rate (Astrand, 1960) and revealed a higher $VO_2max.$ for the active group ($p<0.01$, Table 4.2). Relative oxygen uptake measures at a common physical work capacity of 170 bpm. heart rate (PWC$_{170}$) were virtually identical at 91.4% (active) and 89.7% $VO_2max$ (inactive). Similarly, oxygen uptake at each of the exercise intensities throughout the test were identical except in the later stages when inactive subjects were unable, either to maintain the workrate, or continue with the test (Figure 3, Appendix 5; Table 4.3).

The higher peak oxygen uptake of the active group was matched by higher peak pulmonary ventilation ($p<0.01$) and ventilatory equivalents and reflected the significantly higher final exercise intensities between the groups. Thus, at a common PWC$_{170}$, the active group were able to ventilate 20 litres per minute more (Figure 2, Appendix 5) than their inactive colleagues. These changes reflect increased metabolism as revealed by high, final R values, both groups reaching 1.15 (Figure 4, Appendix 5).
### TABLE 4.2

Final Average Physiological Responses of the Two Groups.

(Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Active</th>
<th>Inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Estimated VO2max</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(l.min⁻¹)</td>
<td>3.5 ±0.6</td>
<td>2.9 ±0.4 **</td>
</tr>
<tr>
<td>(ml.kg.min⁻¹)</td>
<td>47.4 ± 8.3</td>
<td>36.5 ±8.0 **</td>
</tr>
<tr>
<td>Estimated %VO2max at PWC170</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak VO2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(l.min⁻¹)</td>
<td>3.0 ±0.4</td>
<td>2.5 ± 4 **</td>
</tr>
<tr>
<td>(ml.kg.min⁻¹)</td>
<td>40.9 ±4.6</td>
<td>32.3 ±5.2 **</td>
</tr>
<tr>
<td>Peak VE (l.min⁻¹)</td>
<td>104.4 ±22.3</td>
<td>79.0 ±25.7 **</td>
</tr>
<tr>
<td>VEFF (Vent.Equiv.)</td>
<td>34.2 ±6.8</td>
<td>30.9 ±6.6 *</td>
</tr>
<tr>
<td>Heart rates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resting</td>
<td>64 ±11</td>
<td>75 ±9 **</td>
</tr>
<tr>
<td>Peak</td>
<td>172 ±11</td>
<td>170 ±11</td>
</tr>
<tr>
<td>HRmax (age)</td>
<td>180 ± 5</td>
<td>180 ± 4</td>
</tr>
<tr>
<td>Final Workrate (W)</td>
<td>227.0 ±36.5</td>
<td>188.7 ±30.9 **</td>
</tr>
<tr>
<td>Resp.Exch.Ratio (R)</td>
<td>1.17 ±0.01</td>
<td>1.18 ±0.09 *</td>
</tr>
<tr>
<td>Rate of Perceived Exertion (RPE)</td>
<td>17.1 ±1.4</td>
<td>15.9 ±2.2 *</td>
</tr>
</tbody>
</table>

* Significant difference between groups (p<0.05)
** Significant difference between groups (p<0.01)
When heart rates were plotted against exercise intensities, significant differences were found between six of the mean heart rates for the same absolute exercise intensities (Figure 4.1; Table 4.3) whilst at PWC$_{170}$ the active group were working at 32.6 watts above that of the inactive group. High linear relationships were found between these parameters: active ($r=0.989$); inactive ($r=0.988$) and when matched with the equally high relationships between oxygen uptake and workrates (Figure 3, Appendix 5), a resulting oxygen pulse in millilitres per beat:

\[
\text{Oxygen Pulse} = \frac{mL\text{O}_2 \text{ consumed/min}^{-1}}{\text{HR (beat/min}^{-1})}
\]

revealed differences ($p<0.01$) between nine of the measures that were compared:

<table>
<thead>
<tr>
<th>Oxygen Pulse (ml bt$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
</tr>
<tr>
<td>Active:</td>
</tr>
<tr>
<td>n</td>
</tr>
<tr>
<td>Inactive:</td>
</tr>
</tbody>
</table>

There were also significant differences between the mean Rates of Perceived Exertion of the two groups ($p<0.05$; Table 4.2).

In order to determine the relationship between heart rate and other key parameters, a correlation matrix was compiled with the following results:

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>WR</th>
<th>VO$_2$</th>
<th>VE</th>
<th>VEFF</th>
<th>RER</th>
<th>RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>0.99</td>
<td>0.98</td>
<td>0.93</td>
<td>0.80</td>
<td>0.98</td>
<td>0.46</td>
</tr>
<tr>
<td>Inactive</td>
<td>0.99</td>
<td>0.99</td>
<td>0.95</td>
<td>0.78</td>
<td>0.97</td>
<td>0.63</td>
</tr>
</tbody>
</table>

4.4 DISCUSSION.

The main finding of this study was that the relationship between heartrates and exercise intensities was found to be unique in revealing distinct differences between active and inactive middle-aged men. These differences were displayed, not solely at the PWC$_{170}$ level but throughout the total period of the test. Therefore the purposes of the study could be said to have been satisfactorily achieved.
### TABLE 4.3

**Active and Inactive group values during each stage of the Test of Progressive Intensity**

(Mean ±SD)

<table>
<thead>
<tr>
<th></th>
<th>Active n</th>
<th>20</th>
<th>20</th>
<th>20</th>
<th>20</th>
<th>20</th>
<th>19</th>
<th>15</th>
<th>6</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inactive n</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>16</td>
<td>11</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td><strong>Work rates (W)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>x</td>
<td>29.7</td>
<td>58.8</td>
<td>88.9</td>
<td>118.5</td>
<td>149.0</td>
<td>177.1</td>
<td>205.6*</td>
<td>235.3</td>
<td>267.5</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>1.6</td>
<td>2.8</td>
<td>3.9</td>
<td>4.3</td>
<td>5.3</td>
<td>7.7</td>
<td>13.1</td>
<td>12.5</td>
<td>12.6</td>
</tr>
<tr>
<td>Inactive</td>
<td>x</td>
<td>29.5</td>
<td>58.5</td>
<td>87.9</td>
<td>117.2</td>
<td>146.4</td>
<td>173.9</td>
<td>197.4</td>
<td>225.2</td>
<td>245.6</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>2.1</td>
<td>2.8</td>
<td>4.9</td>
<td>5.8</td>
<td>6.0</td>
<td>6.5</td>
<td>11.2</td>
<td>18.6</td>
<td></td>
</tr>
<tr>
<td><strong>Heart rates (beat.min⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>x</td>
<td>87**</td>
<td>99**</td>
<td>110**</td>
<td>126**</td>
<td>141***</td>
<td>155*</td>
<td>165</td>
<td>169</td>
<td>174</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>10.9</td>
<td>12.9</td>
<td>14.4</td>
<td>14.7</td>
<td>16.0</td>
<td>14.0</td>
<td>14.3</td>
<td>12.4</td>
<td>15.7</td>
</tr>
<tr>
<td>Inactive</td>
<td>x</td>
<td>98</td>
<td>109</td>
<td>123</td>
<td>139</td>
<td>153</td>
<td>163</td>
<td>170</td>
<td>175</td>
<td>179</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>9.9</td>
<td>10.3</td>
<td>11.7</td>
<td>12.4</td>
<td>12.4</td>
<td>10.1</td>
<td>7.8</td>
<td>10.6</td>
<td></td>
</tr>
<tr>
<td><strong>Oxygen Uptake (L.min⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>x</td>
<td>0.7</td>
<td>1.1</td>
<td>1.3</td>
<td>1.7</td>
<td>2.0</td>
<td>2.4</td>
<td>2.8*</td>
<td>3.1</td>
<td>3.5*</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.2</td>
<td>0.2</td>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Inactive</td>
<td>x</td>
<td>0.7</td>
<td>1.0</td>
<td>1.4</td>
<td>1.7</td>
<td>2.0</td>
<td>2.4</td>
<td>2.6</td>
<td>2.9</td>
<td>2.8</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.5</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Significance of difference between data: * p<0.05; ** p<0.01
Tests of a predictive, or progressively comparative, protocol tend to reveal limitations, either in the choice of parameter (Rowell et al., 1964), in the conservatively short duration of the test itself (Bruce, 1972) and, above all, in the comparatively few data points extrapolated from the physiological responses measured (Astrand et al., 1954). Many tests also fail to consider the age of the subject where levels of fitness may be unknown.

The present test is characterised by a commonly accepted revolution rating of 60 rpm, a low starting load and progressively low incremental stages which did not place the untrained subject at a mechanical disadvantage since choices in speed and loading were not required. The aim of the test was, therefore, to employ a procedure incorporating two-minute work bouts which were reasonably taxing but not unique to the inexperienced, older subject.

The more common exercise test mode favours the active individual who finds work on a cycle ergometer, rowing ergometer and treadmill often familiar through his own form of energetic pursuit. By requiring subjects to cycle at a constant speed and providing a progressive increase in power output, an exercise pattern was established which was not unfamiliar to the inactive individual and yet allowed the active to reveal some of his training-induced physiological advantages. Furthermore, the intimidation of the test was partially offset by providing every subject with the opportunity to terminate the test whenever he wished, since a pre-determined end point was not obligatory. The efficacy of the test was reflected in the fact that all twenty active subjects completed six of the stages, only one subject dropping out at the seventh, whilst twenty inactive subjects successfully completed five of the loads, four dropping out at the sixth (Table 4.3).

The value of this type of submaximal testing, either as a predictive test of maximal oxygen uptake or in relationship to a given heart rate, such as that of the PWC170, is that, not only are numerous linear relationships able to be utilised and studied (Astrand et al., 1986; Lamb, 1984; McArdle et al., 1986) but a comparatively large number of data points ensure both the accuracy and validity of the regression lines produced. A comparison between the slopes of the lines of the inactive, and active, groups in predicting the maximal oxygen uptake, for instance (Figure1, Appendix 5), could well mimic differences found between pre- and post-training measures of either individual or group responses, regardless of gender (Shangold et al., 1988).
Certainly the predicted VO\textsubscript{2max} of 2.9 l.min\textsuperscript{-1} for the inactive group and 3.5 l.min\textsuperscript{-1} for the active (Table 4.2) closely agree with changes in baseline levels following training found in the literature. Saltin et al., (1969) measured 42 male subjects aged between 34 - 50yr and discovered that maximum oxygen uptake values increased from 2.89 l.min\textsuperscript{-1} to 3.44 l.min\textsuperscript{-1} following 8 - 10 weeks of training. Furthermore, when using the regression equation calculated from 700 observations of VO\textsubscript{2max} (Dehn and Bruce, 1972) for the 40 - 69 yr old age group:–

\[
\text{VO}_2\text{max} = -0.362 \text{ (yr)} + 52.741
\]

the calculated value of 36.1 ml.kg.min\textsuperscript{-1} for the present inactive group closely equates with the prediction of 36.5 ml.kg.min\textsuperscript{-1} (Table 4.2). The benefits which regular exercise bestow are reflected in the comparative differences of 17.1% between the predicted maximal oxygen uptake values of the two groups and the 16.7% improvement between the measured values. Other authors, reporting data from up to 6 months of various training regimens among the 44 - 66 yr old age range, revealed 10 - 20% improvements in maximum aerobic work capacities (Naughton et al., 1965; Hanson et al., 1968; Ribisl, 1969; Tzankoff et al., 1972).

There were no significant differences between each of the oxygen consumptions of the active and inactive groups (Figure 3, Appendix 5) during the first six exercise intensities and this result is consistent with the findings of a majority of authors (Varnauskas et al., 1966; Saltin et al., 1968; Clausen et al., 1969; Kilbom et al., 1969; Saltin et al., 1971; Girandola et al., 1973; Byrd et al., 1974; Fox et al., 1977) though not all researchers agree that endurance training has little or no effect on oxygen uptake at standardised submaximal work intensities (Fox et al., 1975; Patton et al., 1977). This absence of a difference in the oxygen uptake between the two groups during the major part of the test and up to its conclusion in all but two of the measurements, emphasises the fact that both the active and inactive subjects were equally skilled at the task of ergometer cycling. The result suggests that the choice of test protocol was appropriate in that neither the active nor inactive individuals were able to claim any advantages while performing the test. Also by virtue of ergometer work being weight supported, the significantly heavier mass of the inactive group (Table 4.1) would prove less of a handicap than in other forms of testing, such as in treadmill running, where body mass is not so advantageously supported. This meant that the test could more closely reflect the condition of the identical lean body tissue of each of the two groups (Table 4.1).

The similar oxygen uptake for all but two of the exercise intensities (Table 4.3) combined with similar volumes of pulmonary ventilation (Figure 2, Appendix 5) reflect the ability of the tissues of both groups to extract the same amount of oxygen from equal volumes of inspired air. This is confirmed by ventilatory equivalents which reveal no significant differences throughout the
test. However, although high linear relationships exist between the two groups when ventilation is plotted against heart rates (Figure 2, Appendix 5), the lines diverge as the power output rises. Thus, at the same heart rate of 170 beats per minute (PWC170), the active group are able to ventilate 20 litres above that of the inactive group in order to extract a larger volume of oxygen and work at a 33 watt higher power output. Conversely, if the responses of both groups are measured at the same power output of 204 watts (Figure 4.1), the active group is able to extract a substantially higher oxygen uptake from a ventilation identical with that of the inactive group at 82 l.min⁻¹, yet does so with a lower heart rate (Table 4.3).

Although high correlations were found between heart rate and other physiological parameters, as revealed by the correlation matrix, the heart rate to power output relationship is unique in that the regression lines are parallel throughout the test. This indicates that, at any given absolute power output, the active group is able to work at a significantly lower heart rate of approximately 10 beat.min⁻¹ below that of their inactive colleagues (Table 4.3). Alternatively, at the same heart rate, the active group reveals a 33 watt higher power output at all stages of the test (Figure 4.1).

Such cardio-respiratory advantages as are attributed to the active group, can be considered under both central and peripheral adaptations. In terms of the Fick Principle, this means a consideration of cardiac output mechanisms and arterio-venous oxygen differences respectively. One of the most noticeable effects of regular activity, or training, is that of the heart rate response to exercise and this is manifest in a decrease in the heart rate at rest (Table 4.2) and during submaximal exercise (Figure 4.1). It has been postulated that the maximal heart rate is probably the same in active, as in inactive, middle-aged men (Andersen et al., 1965) as illustrated by the peak values of both groups (Table 4.2). However, by virtue of an enhanced venous return and greater sympathetic drive, inducing greater contractility in the heart of the trained performer, a lower heart rate during submaximal exercise could be attributed mainly to greater stroke volume (Rowell, 1962; Saltin et al., 1968; Ekblom et al., 1968; Saltin and Rowell, 1980; Hollman et al., 1988). In the present context, higher oxygen pulses for all nine workrates of the active group support this contention.

It may also be speculated that, if the cardiac output of an inactive individual and an active individual of equal body mass are actually the same at a given submaximal oxygen uptake as some (Mitchell et al., 1957; Huonker et al., 1989), but not all (Bevegard et al., 1963) data suggest, the higher splanchnic blood flow of the active individual indicates that he may be sending a slightly smaller fraction of his cardiac output to the working tissues. Clearance rates of radioactive Xenon¹³³ from working quadriceps muscle of trained and untrained subjects
Figure 4.1 The heart rate responses between active and inactive subjects during the modified PWC\textsubscript{170} test.
support this suggestion (Grimby et al., 1967). As muscle blood flow appears to be the same in two individuals at a given percentage of VO\(_2\)max (Rowell, 1969), this raises the question as to whether the greater endurance of the active individual, as presently illustrated, may be related, in part, to the maintenance of a higher visceral blood flow.

There is also evidence to suggest that trained muscles are able to extract more of the oxygen offered to them and can therefore function with a reduced blood flow during submaximal exercise (Clausen et al., 1969; Armstrong and Laughlin, 1984). That an organism's maximum capacity for exercise is enhanced through regular activity means that a given submaximal oxygen consumption demands less change in the metabolic pattern and in the circulatory regulation. This may be brought about by an increased capillary bed resulting in enhanced diffusing capacity between capillary and muscle cell and a consequent widening of the arterio-venous oxygen difference (Cotter and Hudlika, 1976; Evans, 1985). This observation need not be confounded by findings which suggest that improvements in maximum oxygen capacity of trained middle-aged men are restricted solely to an increase in stroke volume (Hartley et al., 1969; Saltin et al., 1969) since adaptations at maximal level often mask physiological benefits revealed at submaximal work intensities (Davis et al., 1979).

For instance, studies carried out between young and old men (61 - 83 yr) by means of right heart catheterization (Granath et al., 1964; Granath and Strandell, 1964) revealed lower cardiac output and oxygen uptake relationships of the older men indicating a higher total arterio-venous oxygen difference. The lower muscle blood flow following endurance exercise training, mentioned above, has also been clearly demonstrated both in normal subjects (Stenberg, 1971) and in patients with coronary heart disease (Clausen et al., 1969; Clausen and Trap-Jensen, 1970; Varnauskas et al., 1970). Further evidence comes from studies using plethysmographic techniques, showing that blood flow in the calf immediately after a submaximal exercise test is lower in the trained, than the untrained, state (Elsner et al., 1962; Treumann et al., 1968). The working tissues, it is suggested, compensate for the lower blood flow in the trained state by extracting more oxygen and this concept has gained more recent support (Bjorntorp, 1992). The overall effect is probably a better matching of the cardiac output and oxygen consumption (Rowell, 1974a; Armstrong and Laughlin, 1984); the heart becomes better 'educated', as it were (Williams, 1986). The result is that lower heart rates occur during submaximal exercise after regular activity (Andrew et al., 1966; Clausen et al., 1973) as revealed by the present data (Figure 4.1).

Of course, the reduction in the blood flow to the working muscles after training might be fundamental for the altered circulatory regulation by inducing a reduced general sympathetic
vasoconstriction during exercise. This could provide a psychogenic advantage for the active group, not available to the inactive group, and account for the latter's higher heart rates. Yet the disparity in heart rates was recorded while both groups were performing an exercise task familiar, and common, to both groups. Thus the higher heart rates could be regarded as primarily caused by the differences in activity levels rather than by differences in the degree of psychogenic stress present during the test.

In contrast to the observations reported in the literature that the correlation between Rate of Perceived Exertion and heart rate fall between 0.75 and 0.90 (Borg, 1962a; Borg et al., 1967; Frankenhaeuser et al., 1969; Skinner et al., 1969) the findings of this study revealed only modest correlations (Cohen and Holliday, 1979) of 0.63 for the active group and 0.46 for the inactive group. More recent research has proposed that the physiological precursors for the perceptions of physical exertion possess both peripheral and central origins (Pandolf, 1983). Of the former, alterations in the contractile and energy-producing properties of skeletal muscle dominate (Cafarelli, 1982; Kostka and Cafarelli, 1982). Among the latter precursors thought to signal central perceptions, evidence is strongest in support of the role of pulmonary ventilation acting as an amplifier or 'gain modifier' (Robertson, 1982) that potentiates the peripheral signal. Certainly the correlations between RPE and VE provide higher values of 0.71 for the active, 0.58 for the inactive, group. During exercise at intensities greater than 50% VO₂max., VE responses reflect both the tissue oxidative requirement and the need to buffer metabolic acidosis. At this exercise intensity, VE appears to provide strong central signals of exertion that are consciously monitored (Robertson et al., 1986).

As estimated %VO₂max. for active and inactive subjects at PWC₁₇₀ was 91.4% and 89.7% respectively (Table 4.2), it is surprising that ratings of perception were not considerably higher especially since, at high levels of work intensity, cycle ergometry is notorious for the pain subjects may experience in the quadriceps muscles. This would support Bartley's (1970) contention that RPE is a means of processing such perceptual information as sense of pain and that perceived exertion would not always seem to be a function of metabolic equivalence alone because it is also influenced by the stress placed on local musculature (Counsilman, 1970; Gibson et al., 1979). The lack of a more sensitive response by both groups may be accounted for simply by inexperience in using the Borg Scale.

Such changes in the metabolic pattern are best illustrated by the R values which reveal high linear relationships when plotted against workrates (Figure 4, Appendix 5). A surprising fall towards a desirable resting value of 0.85 from 0.87 (Active) and 0.89 (Inactive) is revealed by the second power output. This was probably induced because of an initial mismatch between
required energy expenditure and oxygen demands resulting in hyperventilation and an 'oxygen deficit' (Astrand et al., 1970) by both groups at the first load. From the fifth workrate onwards there were differences (p<0.05) in the R value despite similar exercise intensities, whilst at an R value of 1.15, which is deemed to indicate that VO$_2$max has been achieved (Issekutz et al., 1961), the active group members were working at approximately 90 watts above that of the inactive group. This subtle but distinct shift in the carbon source of energy has clearly distinguished the two groups, especially when related to their power output capacities. Because of lack of fitness, the inactive group has deprived itself of an efficacious aerobic system with the result that it is poorly supplied with tissue mitochondria and therefore has limited use of enzymes of the TCA cycle and B-oxidation pathways (Davies et al., 1979; Newsholme et al., 1983). Energy required for the test is thus primarily restricted to the anaerobic conversion of glycogen to lactate. Since this is a very inefficient means of producing ATP, it has to occur at a higher rate than the active group and more readily causes fatigue, most probably because of an accelerated build-up of lactic acid (Dill, 1974; Gollnick and Saltin, 1982) as protons escape into the bloodstream.

SUMMARY

It would appear that when active and inactive middle-aged men are exposed to a test, familiar to both and not to the advantage of either group, then many of the physiological differences reported between similar groups elsewhere in the literature, are also evident while using the test protocol described in this present study.

Although anticipated linear relationships were clearly demonstrated between, and within, physiological variables, in only one relationship between heart rates and power output were there consistent differences between active and inactive groups throughout the test. This is valuable in that the fitness level of an individual may be determined regardless of limitations in duration or 'performance'. It also provides distinct differences of over ten heart beats per minute at any given power output. This means that both purposes of the study have been met.

Nevertheless, the difference of 33 watts between the two groups at similar heart rates is not very impressive as a reflection of the ability of the active individual to display the sustained endurance qualities acquired through the regimen of an active lifestyle. Thus the test of progressive intensity cannot be said to have been altogether successful in this regard. It was not possible to attain physiological 'steady state' with exercise intensities limited to two minutes in duration (Nagle, 1973) so that an imbalance was likely to have existed between anaerobic - aerobic metabolic contributions in favour of the former. Consequently it was
questionable whether aerobically acquired advantages could be clearly represented by the active individuals. Therefore it would appear evident that the ergometer test should be modified further to take account of this factor.
CHAPTER 5

5. EXPERIMENTAL STUDY 2

If an experiment requires statistical analysis to establish a result, then one should do a better experiment.

Ernest Rutherford, 1924

5. THE RESPONSES OF ACTIVE AND INACTIVE MEN TO TWO 30 MINUTE TESTS OF CONSTANT INTENSITY.

5.1 INTRODUCTION.

The initial requirement of Experimental Study 1 was to devise a test which could determine training-induced differences between active and inactive male subjects. The Progressive Intensity Test chosen did show consistent differences throughout when heart rate, as the dependent variable, was plotted against power output, the independent variable (Figure 4.1). However, the difference of 33 watts - a little more than 0.5 kg as an ergometer power output - was hardly convincing evidence for accepting the test as a model. Furthermore, the exercise intensities, constantly increasing every two minutes, could be considered to be at the lower limit of the time span for acquiring a desirable 'steady-state' condition (Fox, 1979; Astrand and Rodahl, 1986) when oxygen uptake matches the oxygen requirement of the tissues.

A test which could reflect this aerobic, homeostatic condition, could probably distinguish the active subjects more clearly by virtue of their type of training which favoured steady-state regimens. An endurance-orientated test, prefaced by a familiarisation period and duplicated in order to provide 'habituation', or negative conditioning, in order to eliminate, as much as possible, the anxiety of subjects as an important source of error (Shephard, 1987), seemed appropriate.

Gollnick (1982) has suggested that light, prolonged exercise requiring 30 to 50% VO$_2$max. can be sustained for, from one, to several, hours, whilst moderate exercise eliciting an oxygen uptake of from 65 to 75% of the VO$_2$max. can be continued for about three hours by well motivated individuals. Yet, whereas treadmill testing can be readily sustained for up to two hours by well trained subjects at 80% VO$_2$max. (Jooste and Strydom, 1976), cycle ergometer testing, to which the present work has been primarily confined, becomes distinctly uncomfortable, often well before an hour, to those subjects unconditioned to prolonged cycling. Various workers have used ergometer tests ranging from 5-60 minutes' duration
when examining middle-aged subjects (age, 37-45 yr.), whether of sedentary, or active, disposition and using relative work intensities of, from 45 to 85% HRmax. or VO₂max. (Elder, 1969; White and Ismail, 1976; Davis et al., 1979; Denis et al., 1984).

Bearing in mind that the well trained heart consumes much less oxygen and substrate per contraction than the untrained heart (Heiss et al., 1977) and because relationships between oxygen uptake, cardiac output and heart rate are linear over a wide range, heart rate can be used as a measure of exercise intensity as far as testing and training are concerned in older and unfit subjects. About 80% of VO₂max., for example, is attained at a heart rate of 200 minus age, 70% at 190 minus age and 60% at 180 minus age. The lower threshold of effectiveness has been regarded as 50% VO₂max. for 30 to 40 minutes since endocrine stimulation with a significant increase in sympathico-adrenergic activity is only seen with intensities above this level (Nazar, 1971; Milhom, 1982). Karvonen et al., (1957) and Hollman and Venrath (1962) have defined minimal intensity levels needed to elicit an improvement in cardiovascular function, that is, a heart rate of 135-150 beat.min⁻¹ or approximately 60% of maximal heart rate capacity. Billings et al., (1960) used the time to reach a pulse rate of 150 beat.min⁻¹ as an evaluation of submaximal exercise, though with male subjects whose ages averaged 27.5yr.

From the above considerations, a realistic test-duration, particularly for inactive subjects, was judged to be 30 minutes. This would equate to an exercise session comprising fast walking, or jogging, 5 kilometres and expending about 300 kcal (1255.8 kJ) of energy (Pollock et al., 1978b), a work output capable of producing optimal responses when performed at a training session on 3-5 occasions per week (Cureton, 1969). It is also a time span which, in its later stages (20-30 min.) is believed to utilise an increasing proportion of fat metabolism, accounting for as much as 30% of total metabolism according to some authors (Wahren, 1979). Even over a century ago, this amount of exercise was believed to provide a prophylactic for coronary heart disease (Heberden, 1802).

A major difficulty in exercise tests involving a fixed power output is that a level of work which is light for active subjects may be maximal, or exhausting, for inactive, or sedentary, subjects. However, the results of Experimental Study 1, especially the relationships between power output and heart rates, provided guidelines of differentiation. A further consideration in the interests of: the level of fitness, the feelings and the safety of the subject, is in permitting the subject to determine his own work intensity. This can be done by devising terminology related more to subjective interpretation - that is, qualitative, rather than using quantitative measures of heart rate or power output. Therefore, the present study consisted of two cross-sectional
experiments. The first used a heart rate of 140 beat.min\(^{-1}\) which translated into a Rate of Perceived Exertion (RPE) of between 'Somewhat hard' and 'Hard' on the Borg Scale (Figures 3.1 and 3.2) and converted into a desirable 60% exercise intensity (Pollock et al., 1986b) for each group. The second required subjects to select an exercise intensity according to their interpretation of the term 'Just Tolerable Discomfort'. Thus the following aims were formulated for these studies:-

1) To determine the endurance capacity of active and inactive subjects by means of a 30 minute ergometer test whose load was pre-set (PSL) from a heart rate of 140 beat. min\(^{-1}\) using the data from Figure 4.1.

2) To compare exercise intensities, self-selected by the subjects, at an intensity of 'Just Tolerable Discomfort' (JTD) and maintained over a period of 30 minutes.

3) To compare the relationship between heart rate and Rate of Perceived Exertion (RPE) within each of the two tests.

5.2 METHODS.

5.2.1 SUBJECTS.
Seven active, and seven inactive, subjects participated in the two cycle ergometer tests. Their ages, physical characteristics and resting physiological parameters, are shown in Table 5.1. Individual members of the active group trained in a wide variety of sports including: swimming - four sessions per week, covering 1846 metres (2000 yd) on each occasion; twice-daily running over 5-11 kilometres (3-7 miles); training for half marathons, marathons and orienteering; regular squash and badminton; regular cycling; training for the National Veterans' Decathlon. They were thus highly trained 'athletes'. The inactive group exercised intermittently, if at all, on a recreational basis. They did not consider themselves sedentary, especially the four physical educationists in the group, but felt that they did not train sufficiently intensively to be part of the active group.

5.2.2. PROTOCOL.
As part of a familiarisation process and in order to minimise the effects of anxiety, subjects visited the laboratory a few days prior to the main tests. The experiments were fully explained to them and they each signed an Informed Consent form. The saddle height was determined for each subject and he was permitted a 10-15 minute practice period at various loads of his own choosing during which time he was asked to rate the degree of stress by means of the
TABLE 5.1

Physical, and Resting Physiological, Characteristics of Active and Inactive Male Subjects.

(Mean±SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Active</th>
<th>Inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>50.4 ±6.9</td>
<td>44.7 ±5.4</td>
</tr>
<tr>
<td>Range</td>
<td>(41.6 - 61.4)</td>
<td>(35.6 - 52.2)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>181.5 ± 4.1</td>
<td>179.5 ±5.7</td>
</tr>
<tr>
<td>Range</td>
<td>(175.7 - 187.1)</td>
<td>(173.7 - 188.1)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>76.7 ± 5.7</td>
<td>82.4 ±9.7</td>
</tr>
<tr>
<td>Range</td>
<td>(70.2 - 84.8)</td>
<td>(71.3 - 99.6)</td>
</tr>
<tr>
<td>Quetelet Index (kg/wt/h²m)</td>
<td>23.3 ±1.8</td>
<td>25.5 ±1.7 *</td>
</tr>
<tr>
<td>Heart Rates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resting</td>
<td>60 ± 7</td>
<td>69 ± 6 *</td>
</tr>
<tr>
<td>HRmax(age)</td>
<td>177 ± 4</td>
<td>181 ± 4</td>
</tr>
<tr>
<td>Expired Air. Analysis (Rest):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VE (l.min⁻¹)</td>
<td>9.80 ±1.20</td>
<td>9.10 ±1.10</td>
</tr>
<tr>
<td>VO₂ (l.min⁻¹)</td>
<td>0.32 ±0.03</td>
<td>0.32 ±0.04</td>
</tr>
<tr>
<td>VCO₂ (l.min⁻¹)</td>
<td>0.29 ±0.02</td>
<td>0.30 ±0.04</td>
</tr>
<tr>
<td>R (Resp.Exch.Ratio)</td>
<td>0.91 ±0.06</td>
<td>0.94 ±0.06</td>
</tr>
<tr>
<td>VE.VO₂ (Vent.Equiv.)</td>
<td>30.6 ±2.30</td>
<td>28.4 ±4.80</td>
</tr>
</tbody>
</table>

* Significant difference between groups (p<0.05)
Borg Scale (1962b). He was made familiar with wearing the mouthpiece and noseclip and encouraged to maintain pedal revolutions of 60rpm as reflected in a rating of 222 on the flywheel rate-meter. All fourteen subjects found that one familiarisation visit was sufficient in order to prepare them for the test conditions and test environment.

Subjects presented themselves for each of the respective tests in the post-absorptive state of not less than two hours since their last meal. They were weighed and measured and then seated on the Monark cycle ergometer (Model 818E) at the correctly adjusted saddle height as described in Section 3.1.2. This procedure was identical for both tests except that prior to the first test, a six minute resting, expired air sample was taken in order to determine the subject's Resting Metabolic Rate (RMR). A pre-test resting heart rate value was also averaged over a five minute period using a battery-operated cardiometer (Cardionics Ltd.) during the sampling of the resting expired air collection. Following this, the subjects exercised for 30 minutes, maintaining the power output calculated for them, or that which had been self-determined.

TEST 2a. PRE-SET LOAD (PSL).

The power output was pre-set and was calculated by means of the regression equations produced from Figure 4.1 at a pre-determined heart rate of 140 beat.min⁻¹ for both active and inactive groups. This resulted in exercise intensities of 158.3W and 128.9W for active and inactive groups, respectively. Power output was then determined from a rearrangement of the workrate formula shown in 3.1.2, namely:

\[
\text{Workload} = \frac{\text{WFI} \times 60}{9.81 \times \text{Revs.} \times 1.622\text{m}}
\]

and were found to be 2.7 kg for the active, 2.2 kg for the inactive, group, assuming constant flywheel revolutions of 222.min⁻¹. Provided the heart rate was maintained at 140 beat.min⁻¹ throughout the test, the exercise intensity was calculated as 68% for the active subjects and 63% HRmax reserve, for the inactive subjects using the Karvonen et al., (1957) formula (Section 3.4) and the data of resting heart rates and HRmax for age from Table 5.1. Once the test had started, heart rates were recorded every 15s and subsequently averaged over a minute, with expired air collections, flywheel revolutions and RPEs taken, successively, at: 0-1min, 4-5min, 9-10min, 14-15min, 19-20min, 24-25min, 29-30min. Recovery heart rates were taken over a period of 5min post-test, whilst subjects remained seated on the ergometer. Subjects were permitted to remove mouthpiece and noseclip between collections and up to 90sec before each sampling. This ensured that appropriate breathing patterns were
### TABLE 5.2

**WORKRATES (W)**

(Mean ±SD)

#### PRE-SET LOAD

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.7 kp)</th>
<th>INACTIVE (2.2 kp)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(\bar{x})</td>
<td>(\bar{x})</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>SD</td>
</tr>
<tr>
<td>5'</td>
<td>160.5</td>
<td>125.1</td>
</tr>
<tr>
<td>10'</td>
<td>161.1</td>
<td>129.0</td>
</tr>
<tr>
<td>15'</td>
<td>160.1</td>
<td>129.0</td>
</tr>
<tr>
<td>20'</td>
<td>160.4</td>
<td>128.6</td>
</tr>
<tr>
<td>25'</td>
<td>159.1</td>
<td>128.1</td>
</tr>
<tr>
<td>30'</td>
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<td></td>
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<tr>
<td></td>
<td>0.01</td>
<td>0.01</td>
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</tbody>
</table>

**JUST TOLERABLE DISCOMFORT**

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.1 kp)</th>
<th>INACTIVE (1.1 kp)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(\bar{x})</td>
<td>(\bar{x})</td>
</tr>
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<td>0.05</td>
<td>0.05</td>
</tr>
</tbody>
</table>

*\(p = \) Significant differences between Active and Inactive Groups.*
established prior to each collection. Water was freely available for imbibing and a fan cooled the subject during anticipated rises in core temperature. Expired air samples were analysed using standard Douglas bag techniques to provide measures of VE, VO₂, VCO₂ together with derived quotients of R values and Ventilatory Equivalents.

**TEST 2b. 'JUST TOLERABLE DISCOMFORT' (JTD).**

This test required the subject to cycle for 30 minutes at a SELF-SELECTED POWER OUTPUT using the phrase 'Just Tolerable Discomfort' as a guide. Subjects were permitted 5 minutes in order to experiment freely with various exercise intensities until a suitable intensity had been determined. An extended sleeve was adapted for, and attached to, the adjustable loading arm of the cycle ergometer in order to prevent subjects having to change posture during the self-loading procedure. However, the actual power output in absolute terms of precise kilograms, was masked from the subject's view. Once the power output had been established, the protocol for this test was identical with that of Test 1 except for the omission of the resting expired air sample.

**5.3 RESULTS.**

There were no differences in age, height and mass between the two groups (Table 5.1), though the inactive group, on average, was 5.7yr younger and 5.7kg heavier. The Quetelet Index also rated the inactive group in the 'overweight' category and the active group as 'ideal'. The resting R values of both groups were high at 0.93 and 0.94, active to inactive, respectively. However, the R value in this instance, was more a pre-exercise value rather than a true resting value and anticipated the approaching test with increased ventilation and above average oxygen and carbon dioxide values of 0.32 l.min⁻¹ for the former, 0.30 l.min⁻¹ for the latter for both groups. 0.25 l.min⁻¹ of oxygen consumed is a more average value for an adult's resting metabolism with the production of 0.20 l.min⁻¹ of carbon dioxide (Matthews, 1967) as a direct result. This provides a more desirable R value of 0.80, indicating a slightly greater metabolism of fat over carbohydrate at rest. No differences were found in minute ventilation nor in ventilatory equivalents between the two groups but resting heart rates were lower (p<0.05) for the active group.

**PRE-SET LOAD (PSL):** There were no differences in heart rates between the two groups in completing this test (Table 5.3, Figure 5.1). However, whereas the active group maintained the desirable 'threshold heart rate of 140 beat.min⁻¹, within one or two beats, from 10 min onwards, that of the inactive group rose steadily from the commencement of the test until, from 20 min onwards, a heart rate of 152-154 beat.min⁻¹ was being maintained. Thus, an elevated work intensity of 74% had to be sustained by this group, compared with its estimated
TABLE 5.3

HEART RATES (beat min\(^{-1}\))

(Mean ± SD)

<table>
<thead>
<tr>
<th>PRE-SET LOAD</th>
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<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time</td>
<td>1'</td>
<td>5'</td>
<td>10'</td>
<td>15'</td>
<td>20'</td>
</tr>
<tr>
<td>ACTIVE (2.7 kp)</td>
<td>x</td>
<td>116</td>
<td>129</td>
<td>136</td>
<td>138</td>
<td>141</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>14</td>
<td>17</td>
<td>17</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>INACTIVE (2.2 kp)</td>
<td>x</td>
<td>116</td>
<td>132</td>
<td>140</td>
<td>146</td>
<td>152</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>8</td>
<td>10</td>
<td>11</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>p</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

‘JUST TOLERABLE DISCOMFORT’

|               | Time          | 1'            | 5'            | 10'           | 15'           | 20'           | 25'           | 30'           |
| ACTIVE (2.1 kp) | x             | 101           | 115           | 118           | 119           | 121           | 122           | 123           |
|               | SD            | 18            | 29            | 32            | 32            | 34            | 35            | 38            |
| INACTIVE (1.1 kp) | x             | 105           | 107           | 110           | 109           | 110           | 109           | 111           |
|               | SD            | 9             | 11            | 9             | 11            | 11            | 11            | 11            |
|               | p             | NS            | NS            | NS            | NS            | NS            | NS            | NS            |

NS = No Significant differences between Active and Inactive Groups.
63% and the 68% of the active group, during the final ten minutes of the test. There were no differences between the heart rates during the five minutes of recovery though, by its conclusion, the active group's mean heart rate had returned to 86 beat.min\(^{-1}\) whilst that of the inactive group was at 96 beat.min\(^{-1}\). There were differences in exercise intensities (p<0.01; Table 5.2; Figure 5.2) averaging 31.6W, and oxygen uptake (p<0.01; Table 1, Figure 1, Appendix 6A) but not in minute ventilation (Table 2, Appendix 6A), ventilatory equivalents (\(\overline{V}_{\text{E}}\) range 26.3 to 27.3), or R values (R range 1.04 to 1.08), between groups. Very high correlations (Cohen and Holliday, 1979) were found between heart rates and RPEs. of 0.97 (Active) and 0.98 (Inactive; Figure 4, Appendix 6A) within the two groups.

'JUST TOLERABLE DISCOMFORT' (JTD): At this self-selected power output, there were no differences between the heart rates of the two groups during the test (Table 5.3; Figure 5.3) nor during the recovery period. The mean heart rate for the inactive group rose from 105 beat.min\(^{-1}\) in the first minute to 110 beat.min\(^{-1}\) after ten minutes and maintained this level, within two or three beats, until the conclusion of the test. That of the active group rose to 101 beat.min\(^{-1}\) in the first minute, reached 118 beat.min\(^{-1}\) by the tenth minute, and concluded at 123 beat.min\(^{-1}\). Exercise intensities for the active group during the last ten minutes of the test were calculated at 52.2%, and for the inactive group at 36.4% HRmax. reserve. By the fifth minute of recovery, the heart rates were virtually identical at 86 beat.min\(^{-1}\) for the active, 87 beat.min\(^{-1}\) for the inactive, group. There were differences in exercise intensities between the two groups (p<0.05; Table 5.2, Figure 5.4). The active group worked, on average, at twice the load of the inactive group: \(\overline{x}\) 126.7W. vs. 63.8W., throughout the test. This difference was mirrored by the minute ventilation (p<0.05; Table 2, Appendix 6A) and oxygen uptake (p<0.05; Table 1, Figure 2, Appendix 6A), though not by the ventilatory equivalents (\(\overline{V}_{\text{E}}\) range: 23.0 to 25.7 between groups) nor the R values (R range: 0.93 to 1.06). Correlations between heart rates and RPEs. were high for the active group (r=0.83) but not as high as for the inactive group (r=0.98; Figure 5, Appendix 6A).

MECHANICAL EFFICIENCY (M.E.) was calculated every 10min during each of the tests (Appendix 6) and for each of the groups with the following results:-

<table>
<thead>
<tr>
<th>PRE-SET LOAD:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minute</td>
</tr>
<tr>
<td>Active</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Inactive</td>
</tr>
</tbody>
</table>
Pre-set Load

![Graph showing heart rate versus time for active and inactive subjects.](image)

Figure 5.1 Time-related heart rates at pre-set loads for active and inactive subjects during the completion of T30.

![Graph showing work rate versus time for active and inactive subjects.](image)

Figure 5.2 Time-related work rates for the two groups of subjects during the 30 min. ergometer test.
'JUST TOLERABLE DISCOMFORT':-

<table>
<thead>
<tr>
<th></th>
<th>Active</th>
<th>Inactive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>23.7%</td>
<td>23.9%</td>
<td>23.7%</td>
</tr>
<tr>
<td></td>
<td>23.7%</td>
<td>24.8%</td>
<td>24.1%</td>
</tr>
</tbody>
</table>

The oxygen cost for these tests, from which the M.E. was derived, was higher for the active group: PSL (p<0.01); JTD (p<0.05); Appendix 6A, Figures 1 and 2 respectively. Yet M.E. during JTD was similar for both active and inactive groups in keeping with similar heartrates.

RELATIVE EXERCISE INTENSITY (%V0₂max.):- Four of the active subjects each completed a maximum oxygen uptake test on the cycle ergometer. It was thus possible to determine their relative exercise intensities, that is, the oxygen cost of the two tests in relationship to the subjects' mean oxygen uptake. For the PSL, the four subjects worked at 64% VO₂ max. and at 68.5% VO₂ max. for the load of JTD. Mean maximum heart rate for the four was 179 beat.min⁻¹ which compares favourably with the mean maximum heart rate for age of 177 beat.min⁻¹ (Table 5.1). For the two tests, %HRmax. reserve was 75.5% (PSL) and 81.5% (JTD). A paired t-test for correlated data revealed no differences between %VO₂ max. and %HRmax. reserve for the group following the two tests. Modest correlations were found between these parameters for the PSL (r=0.61) and for the JTD (r=0.69).

5.4 DISCUSSION.

PRE-SET LOAD.
The ability of both active and inactive subjects to maintain, and complete, a pre-set load at a pre-determined heart rate for 30 minutes on a cycle ergometer fulfilled one of the principle aims of this study. It also helped to justify the findings of Experimental Study 1 (Exp.1) as revealed by Figure 4.1. Both groups sustained the required power output throughout the test period (Figure 5.2) and did so at an average of 31.6W between the respective exercise intensities of the two groups. During the first five minutes, power output rose slightly above this level (35.4W), and fell slightly below it during the last five minutes (28.1W). Thus, ratings fluctuated around the predicted 33W difference determined by Exp.1 but they did so at considerably greater cardiac cost for the inactive group. The question arises as to why this should be so.

Unlike Experimental Study 1, where the time-course of each incremental stage was too short for true steady state conditions to prevail, this test was based on a chosen workload which it
'Just Tolerable Discomfort'

![Heart Rate vs. Time Diagram](image)

Figure 5.3 Time-related heart rates at 'Just Tolerable Discomfort' for active and inactive subjects during T30.

![Work Rate vs. Time Diagram](image)

Figure 5.4 Maintained work rates over the 30 min. test by the two groups of subjects.
was anticipated would induce a heart rate of 140 beat.min⁻¹, able to be maintained throughout the test by both groups. The steady state concept implies that the rate of oxygen uptake in the lungs corresponds to the tissues' oxygen demands and that such comparatively easily measured functions as heart rate and pulmonary ventilation, have attained stability. Yet whereas the active group achieved this rate 18 min into the test and maintained it, within two beats, to its conclusion, the inactive group reached it after 9 min and thereafter 'drifted' fourteen beat.min⁻¹ beyond it until the test was terminated (Figure 5.1).

The answer would seem to lie between the inter-relationships of the Fick principle - that fundamental equation of exercise:

\[ \text{VO}_2 = Q(\text{HR} \times SV) \times (a-V) \text{O}_2 \text{diff.} \]

and therefore to changes that are referable both to central cardiovascular adjustments and to peripheral adaptations occurring within the skeletal muscles themselves. After 20 min of the test, for instance, and using regression formulae devised by Faulkner et al., (1977) and confirmed by other authors (Tanner, 1949; Ekelund and Holmgren, 1967; Rowell, 1974a), the cardiac output of the two groups was found to be 218 ml.kg.min⁻¹ for the active, 186 ml.kg.min⁻¹ for the inactive, group:  

Active: \( Q = 5.2(\text{slope}) \times 29.3 \ (\text{VO}_2 \text{ml.kg.min}^{-1}) + 66 \ (\text{intercept}; \text{ml.kg.min}^{-1}) \)  
\[ = 218.4 \text{ml.kg.min}^{-1} \]

Inactive: \( Q = 5.9 \times 23.2 + 49 \)  
\[ = 185.9 \text{ml.kg.min}^{-1} \]

These data, combined with the differences between heart rates revealed by Figure 5.1 and the considerable difference in oxygen pulse: 0.21 ml.br⁻¹ (active), 0.15 ml.br⁻¹ (inactive), provide useful evidence in support of a larger, more economical, stroke volume for the active group. Although the inactive group was working at a significantly lower workload, lower oxygen uptake and lower cardiac output, it could not maintain such an output without an elevated heart rate and pronounced 'cardiovascular drift'. This has also been termed a 'secondary rise' in heart rate which appears to occur after the first ten minutes of exercise (Nielsen et al., 1984; Shephard, 1987) as in the present test. It has been attributed to a fall in central blood volume, which may cause a decreased filling pressure of the heart, and thereby a decreased stroke volume. The rise has also been positively related to the increase in plasma catecholamines as a result of sympathetic nervous activity (Nielsen et al., 1984). The underlying symptoms, therefore, do not appear to be easily identifiable. What is clear is that the relative exercise intensity in terms of fractional heart rate utilisation was far higher for the inactive group by the conclusion of the test (74%) than both the predicted value (63%) and that of the active group (69%).
Since the cardiovascular, thermoregulatory and metabolic demands of exercise occur in proportion to the relative work intensity (Saltin, 1973a; Rowell, 1974a), it would appear axiomatic that the active group were experiencing a significantly higher work rate and oxygen uptake with far less discomfort than their inactive counterparts and this is confirmed by the fact that after five minutes of recovery, the heart rates of the inactive group were ten beat.min\(^{-1}\) higher. It is also reflected in the averaged RPEs which were progressively higher for the inactive group every five minutes from 15 min to the conclusion of the test (Inactive: 14.8: 15.0: 15.4: 15.4. Active: 14.4: 14.6: 14.9: 15.0). The measured \(VO_2\text{max}\). of 3.7 l.min\(^{-1}\) of four of the active group also reveals an enhanced oxygen carrying capacity for this group resulting in a 64% \(VO_2\text{max}\). work intensity for the PSL test.

Even without this confirmation, the higher oxygen uptake (p<0.01; Table 1, Appendix 6A) of the active group suggests the availability of a far more efficient oxygen extracting system by this group. However, it cannot be assumed that an improved ability to extract oxygen is the sole pre-requisite of the skeletal muscles, particularly during similar relative exercise intensities (Seals et al., 1984). A more convincing explanation for a widened arterio-venous oxygen difference is that the well-trained person directs less of the total cardiac output to regions of the body where oxygen extraction is poor (Ekblom et al., 1968; Shephard, 1987), such as the viscera and skin.

Without the activation of thermoregulatory mechanisms, it is believed that even moderate exercise in humans would be limited to fifteen minutes or less (Nadel, 1977). Certainly during the present test, the active group expended a total of 335kcal(1403kJ), compared with the inactive group’s 283kcal(1183kJ), in completing the test. Both groups revealed similar average R values: active - 1.05, inactive - 1.06, with carbohydrate as the dominate substrate. They also worked at similar mechanical efficiencies of 24.1% (active), 22.5% (inactive). Thus, similar percentages of heat were produced by the two groups: 75.9% active, 77.5% inactive.

It could be hypothesised that both groups were faced with a similar problem of a competition between skin and muscle for blood flow (Rowell, 1977), particularly after the first ten minutes of the test. The solution would seem to be to merely increase the cardiac output (Brouha, 1960). This could solve the problem in the active skeletal muscles where vasodilation is corrected by autoregulation - that is, a combination of increased cardiac output and regional vasoconstriction. However, this is not the main source of the difficulty since, despite very high blood flow in vasodilated, exercising skeletal muscle, there is little increase in muscle blood volume (Asmussen, 1943). Where both active and inactive subjects would have had great difficulty, would have been in counteracting the shifts in blood volume that attended
vasodilation in the highly compliant and capacious region of the skin. Thus, a rise in skin blood flow, as would occur during the present test, appears to be associated with a marked increase in cutaneous volume (Shepherd, 1966; Webb-Peploe et al., 1968; Rowell, 1974b). This could facilitate evaporative heat loss and maintain a stable core temperature but venous return may have been jeopardised in the process. Of course, exercise can be an added stimulus to cutaneous vasoconstriction (Brown et al., 1970) and muscle contraction does help to empty cutaneous veins so that the average venous volume decreases (Henry et al., 1950) and central blood volume 'sumps' are maintained. Even so, data suggest that there is still a progressive rise in skin blood flow and skin blood volume during endurance exercise in a cool environment. These factors would make a major contribution to the upward drift of heart rate and downward drift of stroke volume over the thirty minute test period (Ekelund, 1967). Equally relevant is that over this time span, muscle blood flow has been found to fall as skin blood flow rises (Johnson et al., 1975). The main point is that whatever the total increase in skin blood flow might be, it is sufficient to alter ventricular preload, afterload (Keul et al., 1981; Slutsky, 1981; Suga et al., 1982), and thoracic blood volume during exercise of thirty minutes in a cool environment. Apparently this is true despite the defensive influence of vasoconstriction: of the skin (Johnson et al., 1973), of inactive skeletal muscle, and of splanchnic and renal vascular beds (Rowell, 1974b).

With training, the active group would, undoubtedly, have experienced some alleviation of these conditions. Increased capillary density (Saltin et al., 1977), combined with increased mitochondria (Holloszy et al., 1977) which have also been found in older individuals (Suominen et al., 1977a and b), provide adaptations which appear to permit the muscle to extract a consistent amount of oxygen from a much lower blood-volume-threshold during endurance testing. Furthermore, less skin blood flow would be needed because sweating would occur sooner and in greater quantities, cooling the skin and increasing heat transfer per unit of blood flow (Nadel et al., 1974). There is also less superficial fat after training, so that heat transfer through the subcutaneous tissues is less dependent on blood flow (Shephard, 1987).

From the above discussion, it would appear that the inactive group did not possess the necessary adaptations to cutaneous, and active skeletal muscle, circulations, nor to central cardiac mechanisms in order to maintain a constant cardiac output except by a progressive rise in heart rate as revealed by the pronounced 'cardiovascular drift' of Figure 5.1.
'JUST TOLERABLE DISCOMFORT'.
At the conclusion of the test of 'Just Tolerable Discomfort' (JTD) it was found that, despite no differences between heart rates (Table 5.3; Figure 5.3), the active group's power output was twice that of the inactive group (127W. vs. 64W.; Table 5.2; Figure 5.4). This was accompanied by differences in oxygen uptake (p<0.05; Table 1; Figure 2, Appendix 6A) and in minute ventilation (p<0.05; Table 2, Appendix 6A).

Not only had the inactive group a lower 'heart rate reserve' in terms of heart beats available for any given power output compared with the active group (112 vs 118 beat.min⁻¹) but when coupled with a calculated lower stroke volume (97ml vs. 120ml; Faulkner et al., 1977) after 20 min into the test period, it is apparent that this group was inherently limited in its working capacity. It is also, perhaps, more than fortuitous that the active group has autogenerated a exercise intensity conveniently at that level which is considered 'the lower level of effectiveness' (Nazar, 1971; Heiss, 1977; Milhorn, 1982; Nilsson, 1982), namely 52% HRmax. reserve. Even so, the power output could not be completed without five beats 'drift' from a heart rate of 118 beat.min⁻¹ after ten minutes of the test to 123 beat.min⁻¹ at its conclusion. In comparison with the former PSL test, it would appear that maximum stroke volume had been reached at 120 ml but that the required constant cardiac output could only be maintained with a slight increase in heart rate (Ekelund, 1967; Nadel, 1977). The inactive group, on the other hand has, seemingly, self-selected an exercise intensity of 36% HRmax. reserve which has required no such compensatory adjustments in heart rate in order to maintain cardiac output. This is half the intensity of the former test (74%) and half the workrate (60W vs 120W) and reveals a stroke volume 'reserve' compared with the PSL test (97 vs 101ml) which circumvents the need for an elevated heart rate from ten minutes onwards. Although such an exercise intensity failed to reach a threshold necessary to produce a 'training effect' (Barry et al., 1966; Ekblom et al., 1968; Holloszy et al., 1976), the choice of load is surprising in being half that of the active group.

Also of interest is the choice of a power output of 128W by the active group, a rate which closely mimics the 129W of the inactive group during the PSL test. In other words, 'Just Tolerable Discomfort' is a term that is translated by active, trained subjects to a 50% exercise intensity and this equates to a 70% exercise intensity for an inactive group whose workrate was predicted from a heart rate of 140 beat.min⁻¹. Other measures tend to reflect, either the disparity in power output, or similarity in heart rates, between the groups. For instance, there were no differences in R values, at 1.02 for the active, and 0.96 for the inactive, groups. This would suggest a continuing, or similar, dependence on glycolysis and a large contribution of carbohydrate to energy metabolism. However, the calculated energy expenditures of 276kcal
(1155kJ) for the active group and 168kcal (703kJ) for the inactive, reflected the differences in exercise intensities whilst the differences in oxygen pulse of 0.20ml.b•t\(^{-1}\)(active) and 0.13ml.b•t\(^{-1}\)(inactive), together with the calculated stroke volumes shown above, go some way in explaining the comparable heart rates of Figure 5.3. The lower work levels of this test, compared with those of the PSL, were yet completed at a higher, average mechanical efficiency of 24.3% for the inactive group which more closely matched the 23.6% of the active group. Presumably, at the lowered work intensities of this present test, the competition between skin and muscle for blood (Rowell, 1977) was far less intense and could be met with only minor adjustments to the cardiovascular system of the inactive group.

From the discussion so far, it is very difficult to assess how much of the significant differences in exercise intensities, oxygen uptake and minute ventilation between the two groups was owing to the lack of central and peripheral adaptations of the inactive individuals and how much was the result of a difference in the timing of psychogenic recruitment of the sympathetic nervous system in response to the physical challenge of the test. Familiarisation with the exercise task would not appear to have created a problem since both active and inactive groups were equally experienced with the test. It does raise the question about where familiarisation ends and training begins (Davies et al., 1970). In the present study, the doubly higher workrates of the active individuals were recorded while performing an exercise task which was familiar to both active and inactive groups. Therefore, these higher workrates were mainly owing to the differences in training status per se, rather than to differences in the degree of psychogenic stress present during the test. For, although the degree of stress was determined by the terminology used, it was at a common level for both groups and its interpretation was dependent on the physiological status of the subjects.

In terms of the sensory feedback of this common level of stress, relationships were strongest between heart rate and rate of perceived exertion for the inactive group (Figure 4, Appendix 6A). With a correlation coefficient of 0.98 they were identical to those of the previous test (Figure 3, Appendix 6A) and were probably associated with the need to recruit and maintain more motor units (Freund et al., 1979). With a change in the vegetative system of the active group as a result of training and as revealed by the increased stroke volume, reduced cardiac work and decreased myocardial oxygen consumption (Huonker et al., 1989), it could be speculated that similar relationships would not be so close (r=0.83; Figure 4, Appendix 6A).

If this is true, other sources of sensory cortical response to the exercise stimulus may need to be considered. Minute ventilation (Ve) has already been mentioned as a central amplifier or 'gain modifier' that potentiates the peripheral signal (Ekbloom and Goldberg, 1971; Robertson,
As well as serving the prime function of providing gaseous exchange at the alveolar level in support of cellular oxidative metabolism, ventilatory adjustments also buffer metabolic acidosis (Wasserman, 1978). The 'acid gas', carbon dioxide, raises the pCO₂, lowers the blood pH and itself, may be a major 'accelerator' of the ventilatory system (Lundholm and Svedmyr, 1966; Roberts, 1980), though not all authors agree that it serves as an effective stimulation during mild exercise (Whipp, 1983). In the present instance, there was a difference in minute ventilation (Table 2; p<0.05; Appendix 6A) between the two groups but not in %CO₂ produced, averaging 3.9% for the active, 4.0% for the inactive, group over the test period. Thus, lower ventilatory volumes were driven by equally high common percentages in CO₂ for the inactive group. This is reflected in the higher correlation coefficients between RPE and VE for the inactive group (0.71 vs. 0.62) and the considerably higher relationships between RPE and %CO₂ of the inactive (r=0.96), over the active (r=0.60), group. Rather than support the active group’s choice of power output, it would seem that the VE%CO₂ ventilatory mechanisms more strongly endorse that of the inactive subjects.

So far, explanations for the substantial difference in workrates, coupled with common levels of stress, heart rates and %CO₂ production appear to be elusive. Of the numerous central signals of exertion, only ventilatory function has been found to be consciously monitored (Robertson et al., 1979c; Mihevic, 1981; Robertson, 1982) whilst peripheral sensations of exertion have been signalled by metabolic acidosis (Allen and Pandolf, 1977; Kostka and Cafarelli, 1982; Young et al., 1982; Noble et al., 1983). However, these responses were found at higher relative work intensities of 80%VO₂max. rather than the lower intensities of the present study, and among young subjects whose ages averaged 23 years. Because ventilatory buffering of metabolic acidosis is limited at the lower exercise intensities (Wasserman, 1978), VE was found not to differ between acid-base conditions below 60%VO₂max. Assuming HRmax. reserve to closely equate with VO₂max., such a criterion would embrace the 52% and 36%HRmax. reserve of the present active, and inactive, groups respectively. Even so, it would not account for the decrease in alkali reserve for the older person (Mori, 1936; Astrand, 1956) nor for the possibility that training may elevate those reserves. Such enhanced reserves could account for the buffering of the significantly elevated ventilatory measures of the active group over the inactive group at the same %CO₂ and at the same sense of stress. The term 'Just Tolerable Discomfort', however, was interpreted significantly differently by the two groups as the test progressed. The active group rated it, on average, at an RPE of 13.1, the inactive at 11.3 (p<0.01) so it would be a mistake to assume that the two groups were responding psychogenically in quite the same way.
Yet there are comparable demands on the circulatory systems of the two groups as reflected by the similarity in heart rates. It is noticeable that these are at the level of, or just above, 100 beat.min\(^{-1}\) where vagal tone is believed to be released (Robinson et al., 1966) and sympathetic activity to the heart becomes increasingly important. With it, by implication, plasma noradrenaline begins to rise with leakage from sympathetic nerve endings (Christensen and Brandsborg, 1973; Escourri et al., 1984; Rowell and Johnson, 1984). This threshold heart rate of 100 beat.min\(^{-1}\) is associated with a proportionally inverse fall in splanchnic, and renal, blood flow and also in mixed venous content (Hansen et al., 1978). The rise in heart rate is also closely correlated with plasma renin activity and with a rise in sympathetic activity to the kidneys (Kotchen et al., 1971; Finberg et al., 1977; Galbo, 1983). This benchmark heart rate appears to be capable of initiating a cascade of responses from the autonomic nervous system in controlling autoregulation in the tissues and effectively readjusting total peripheral resistance. The comparable heart rates do not appear to have occurred by chance. Because of the central and peripheral benefits bestowed by training, the active group would seem to have experienced a delay in the effect of the sympathetic, chronotropic fibres (Linden, 1968; Linden et al., 1970) during which time the workrate has increased to twice that of the inactive group. Further supportive evidence for the comparable cardiovascular stress of the two groups is provided by virtually identical recovery heart rates of 86 beat.min\(^{-1}\) for the active, 87 beat.min\(^{-1}\) for the inactive, group at five minutes post-exercise.

**SUMMARY.**

The results of this two-part study have shown that, firstly, when two groups of men, one active and the other inactive, maintained an exercise intensity at a common heartrate, a difference of 32W was achieved in the power output of the active group over their inactive contemporaries. This confirmed the findings of Experimental Study 1 and met the demands of the first aim of the study.

Secondly, when the same two groups self-selected an exercise intensity using the term 'Just Tolerable Discomfort', the active group chose a power output twice that of the inactive group. That a 100% difference between the exercise intensities of these two groups was found over a five minute practice period using the same reference level of stress was both significant and surprising. It was even more surprising that these differences were able to be maintained over the full thirty minute test period without significant changes in the heartrates. Since both groups had previously completed a similar test at a higher, pre-set power output over the same time span, there was little doubt that each group possessed the endurance capacity to maintain its chosen load over the test period. Nevertheless, all subjects found it extremely
difficult to set a suitable exercise intensity according to the dictates of the term, 'Just Tolerable Discomfort', especially during the initial five minute practice period. There was general concern that an exercise intensity might be chosen which it would be difficult to maintain over the full time. This apprehension could account for the conservative power output chosen but does not explain the significant difference between them. Hence the second aim of the study was completed.

Finally, in meeting the third aim of the study, correlations between heart rates and Rates of Perceived Exertion were found to range from 'high' to 'very high' during the two studies. These were found to be valuable guidelines in determining the precise level, and subsequent use, of the RPE scale.

CONCLUSION.
The finding that a highly trained, active group of middle-aged men was able to choose a workrate twice that of a group of inactive men of similar age whilst maintaining similar heart rates and using the same scale of stress, appeared to be worthy of further study and research. Using an inactive, or sedentary, group as representative of a baseline level of fitness and training them over a prescribed period of time, it should be possible to assess a relative percentage workrate improvement in accordance with the 100% increase in workrate as revealed by the active group in this experimental study. Proposed guidelines could include an adaptation of the 30 minute test already delineated in order to facilitate data collected from associated measurements of functional capacity and biochemical analyses as suggested by the above discussion. The data from these parameters could reveal something of the way in which sensory uniformity can be maintained in the face of rising work intensities.
CHAPTER 6

6. TRAINING STUDY 1.

I know one who set himself a task of sawing wood half an hour every day, and was nearly cured (of heart disease).

R. Heberden, English Physician (1802).

6.a) THE EFFECT OF ENDURANCE TRAINING ON THE FUNCTIONAL CAPACITY OF A GROUP OF MIDDLE-AGED MEN AS MODIFIED BY A SINGLE RATE OF PERCEIVED EXERTION.

6.1 INTRODUCTION.

Experimental Study 2a has shown that highly trained individuals who have adopted regular exercise as a life-style, have been able to reveal significantly higher exercise intensities than their untrained contemporaries. This has been achieved by the use of the criterion of a common heart rate so the finding was not unique considering the improved cardiac capacity that training is able to bestow directly (Ekblom et al., 1968; Clausen, 1977; Wolfe and Cunningham, 1982) nor the peripheral, tissue adaptations which are capable of alleviating central mechanisms indirectly (Wallace, 1974; Andersen, 1975; Holloszy and Booth, 1976). However, a corollary to the study (Experimental Study 2b) showed that trained individuals doubled their power output over untrained individuals when asked to work at a common level of stress. Both trained and untrained subjects also showed no differences in heart rates whilst completing their respective loads during this second experiment. In order to explore, more fully, the training-induced adaptations capable of modifying a sense of stress which could, in turn, affect an individual's choice of exercise intensity, a training study was devised. Its purpose was to examine whether an increase in habitual physical activity could enable an individual to perform more work at the same level of perceived exertion. It did so by means of:-

a) Pre-training measurements of functional capacity, including metabolic, hormonal and blood lipid responses. These measurements would then be compared with similar measurements, post-training.

b) Examining the anthropometry of the thigh and the forces it was capable of generating before and after training. The specific muscle group of the thigh is particularly relevant to the
type of testing and training involved. It was hypothesised that changes in its morphology and fluctuations in the motor units it was able to recruit, may well be identified with accompanying levels of stress. As such, it was considered a particular germane focus of enquiry.

6.2 METHODS.

6.2.1 SUBJECTS.
Thirty-five men entered the study, 24 subjects and 11 controls with mean ages (±SD) of 44.4±8.2yr. and 42.8±6.9yr. respectively, and with similar age ranges of 31-58yr. for subjects and 31-54yr. for controls. They were recruited from academic, technical and related staff of two local institutions of higher education and were either sedentary or only participated in exercise on a recreational basis. Prior to training, both subjects and controls attended the laboratory in the post-absorptive state, not less than two hours prior to their last meal, in order to complete a familiarisation cycle ergometer test as devised by Experimental Study 1. They were weighed and measured; resting blood pressures and resting electrocardiograms were also taken together with ventilatory measures and percentage body fat. The subjects were fully acquainted with all procedures and thoroughly informed regarding the study before signed consent was obtained. The protocol for the study is outlined in Chapter 3.

6.2.2 PROTOCOL.

TRAINING PROGRAMME.
The subjects trained for 12 weeks during May to November. They exercised regularly at least three times a week for 30 min. on each occasion. Two of the training sessions were carried out on a cycle ergometer under supervision. For the third session, subjects were permitted to chose their own form of aerobic training which was recorded in a training diary. Such activities as brisk walking, jogging, swimming and cycling were included. Every three weeks the training intensity on the cycle ergometer was increased according to the Karvonen (1957) formula (Chap.3.4). As an obligatory part of training, subjects were required to complete three field tests both in the early days of training and at its conclusion. From a 5 kilometre run it was possible to predict VO\textsubscript{2max}; a 2 mile (3219 metre) run together with a 12 min. Cooper (1981) test also helped to establish a desirable 'steady-state' form of exercise which characterised the whole training programme.

TESTING.
Both subjects and controls were required to complete two laboratory tests before, and immediately after, the 12 week training period. The tests elicited both physiological and
TABLE 6.1

Physical, and Ventilatory, Measures of Subjects and Controls, pre- and post-training.

(Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>24</td>
<td>11</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>44.4 ± 8.2</td>
<td>42.8 ± 6.9</td>
</tr>
<tr>
<td>Range</td>
<td>(31-58)</td>
<td>(31-54)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174.7 ± 6.3</td>
<td>178.6 ± 6.4</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>72.1 ± 7.8</td>
<td>76.1 ± 9.6</td>
</tr>
<tr>
<td>Post</td>
<td>71.5 ± 7.6</td>
<td>75.9 ± 10.3</td>
</tr>
<tr>
<td>Quetelet Index (kg.wt/m²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>23.6 ± 2.6</td>
<td>24.0 ± 2.4</td>
</tr>
<tr>
<td>Post</td>
<td>23.4 ± 2.5</td>
<td>24.0 ± 2.7</td>
</tr>
<tr>
<td>Adiposity (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>20.8 ± 4.4</td>
<td>18.5 ± 2.7</td>
</tr>
<tr>
<td>Post</td>
<td>19.2 ± 4.3</td>
<td>17.3 ± 3.3**</td>
</tr>
<tr>
<td>Vital Capacity (litres)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>5.3 ± 0.9</td>
<td>5.4 ± 0.9</td>
</tr>
<tr>
<td>Post</td>
<td>5.2 ± 0.9</td>
<td>5.5 ± 0.7</td>
</tr>
<tr>
<td>FEV₁ (litres)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>4.2 ± 0.8</td>
<td>4.2 ± 0.9</td>
</tr>
<tr>
<td>Post</td>
<td>4.1 ± 0.8</td>
<td>4.4 ± 0.9</td>
</tr>
<tr>
<td>%FEV₁</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>79.5 ± 6.5</td>
<td>77.5 ± 7.6</td>
</tr>
<tr>
<td>Post</td>
<td>78.4 ± 7.0</td>
<td>78.9 ± 8.7</td>
</tr>
<tr>
<td>Peak Flow (l.min⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>607.8 ± 65.0</td>
<td>644.0 ± 73.2</td>
</tr>
<tr>
<td>Post</td>
<td>612.9 ± 71.7</td>
<td>633.4 ± 62.0</td>
</tr>
</tbody>
</table>

Significance of difference between data: * p<0.05; ** p<0.01
psychological responses in order to determine the effects of training. The first was a Power Lactate Test of 16 min (T16) duration which measured both functional capacity, and metabolic, responses. Loads for this test were selected for each individual on the basis of results from the initial familiarisation test and were progressively raised every 4 min.

The second test required subjects to tolerate a workload of their own choosing for 30 min (T30). They self-selected the load according to a rating of 14 on the Borg Scale of Rate of Perceived Exertion (RPE). They did this by sitting on the ergometer (Model 818E) and cycling at 60 rpm over a period of five minutes. An extended, adjustable arm enabled them to experiment with various exercise intensities without altering their posture. The power output was masked from view so that the subject's only guideline was his own sense of psychological stress as conveyed by the muscular tension of any particular load. A visual aid was provided by means of the Borg Scale, placed directly in front of each subject and highlighting the RPE of 14. After the five minute trial period, the chosen load was transferred to the basket weight-loading ergometer (Model 864) and linked into the computerised data logging system as described in Chapter 3.3. By means of this system it was possible to determine precise changes in functional capacity following the training period. The test before training was identified as Test 1 (T1); the test load after training (T2) was re-established by repeating the above process, using the same RPE but modified by the subject's psychological and physiological responses to the sense of stress acquired through the training process.

6.2.3 BIOCHEMICAL ANALYSES.
Methods of analyses of plasma lactates, glucose, free fatty acids, glycerol, catecholamines and lipoproteins are described under Section 3.5 of the General Methodology and assay procedures under Appendices 2, 3, 4. Such analyses are in keeping with the explanation of, and enquiry into, a single RPE as a 'multiple integration of many factors' (Pandolf, 1982).

STATISTICAL ANALYSES.
When test parameters before and after the training period were compared with each other, Student's t-test was used. Paired t-test was used to evaluate differences in parameters between training, and control, groups. Pearson product moment coefficient was used to assess correlations between variables. A 4-Way Analysis of Variance (BMDP: Biomedical Data Package Statistical Software, Programme 2V) with repeated measures was used to investigate the results of the 30 minute study. The between-subject factors were age (30-40, 50-60 yr age groups) and group (experimental and control) and the within-subject factors were time (duration of test) and training (pre and post).
TABLE 6.2
Physiological responses pre- and post-training.
(Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects</th>
<th>Controls</th>
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<tbody>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td>Haemoglobin (g.dl⁻¹) Pre</td>
<td>14.9 ± 0.6</td>
<td>14.6 ± 1.0</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>14.6 ± 0.9</td>
</tr>
<tr>
<td>Haematocrit %</td>
<td>Pre</td>
<td>43.9 ± 2.0</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>47.3 ± 2.8 **</td>
</tr>
<tr>
<td>Blood Pressure (mm. Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic Pre</td>
<td>133 ± 10.7</td>
<td>124.7 ± 10.9</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>130 ± 7.9</td>
</tr>
<tr>
<td>Diastolic Pre</td>
<td>88.6 ± 9.7</td>
<td>83.4 ± 3.8</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>79.8 ± 6.5 **</td>
</tr>
<tr>
<td>R (Resp. Exch. Ratio) Pre</td>
<td>1.00 ± 0.3</td>
<td>1.01 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>0.96 ± 0.1 **</td>
</tr>
<tr>
<td>Heart rate (beat.min⁻¹) (Rest)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>69 ± 10</td>
<td>62 ± 9</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>63 ± 9 **</td>
</tr>
<tr>
<td>Maximum Oxygen Uptake (VO₂max)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(l.min⁻¹) Pre</td>
<td>2.88 ± 0.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>3.26 ± 0.7 **</td>
</tr>
<tr>
<td>(ml.kg.min⁻¹) Pre</td>
<td>39.9 ± 11.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>45.6 ± 9.7 **</td>
</tr>
</tbody>
</table>

Significance of difference between data: * p<0.05; ** p<0.01
Running Performances

** p < 0.01

![Graph showing improvements in running performances](image)

Figure 6.1. Improvements in field tests (Mean ± SD) following 12 weeks of training.
6.3 RESULTS.
a) Functional capacity, metabolic, hormonal and blood lipid measurements.
Over the 12-week training period there was a small but significant decrease in body weight (p<0.05), significant decreases in percentage body fat, diastolic blood pressure, resting heart rates, and R values (p<0.01) by the experimental subjects (Tables 6.1 and 6.2). The Quetelet Index averaged 23.8 kg.m⁻² for both experimental group and controls, providing a classification of 'ideal'. Ranges over the pre- and post-training period extended from well below the leanness, to the overweight, category for the subjects (17.9 - 28.5 kg.m⁻²) with a slightly narrower band for the controls (19.6 - 27.4 kg.m⁻²).

The 4-Way ANOVA revealed that, though there were significant decreases in percentage body fat for both experimental and control, groups, the 'potential' for reducing body fat was greater among the 50 year-old experimental subjects than any other age group (F₁,₂₉ = 2.83; p<0.076), probably because this group had more fat to lose. Training was also found to have reduced both resting heart rates (F₁,₂₉ = 6.87; p<0.01) and systolic blood pressure for the experimental group (F₁,₂₉ = 5.47; p<0.05) compared with the untrained controls.

Haemoglobin concentrations were maintained at normal levels throughout the testing period (Table 6.2) though haematocrit increased for both subjects and controls (p<0.01).

The only ventilatory measure to change was FEV₁, which was lowered (p<0.05) for the subjects but as a result of an improvement in the 5 kilometre run (x 27.3 vs 24.9 min; p<0.01) VO₂max was increased by 14.3%. Improvements were also produced for distance and time in the 12min Cooper Test (x 1.4 vs 1.6 miles; p<0.01) and 2 mile run (x 17.2 vs 15.3 min; p<0.01; Figure 6.1). Such improvements could have been anticipated from the progressive increase in exercise intensity (66-83%) over the training period together with the accompanying increases in individual, and weekly, training energy expenditures (Table 6.4).

The Power Lactate Test.
Though there were no changes in oxygen uptake for the four incremental loads of the Power Lactate Test (T16) for either subjects or controls following training, the experimental group revealed significant decreases in lactate concentration for three of the four exercise intensities after training (Figure 1, Appendix 7). For instance, at the fourth workload, pre-training values fell from 5.5 mmol.l⁻¹ to 3.9 mmol.l⁻¹ (p<0.01) post-training together with a
### TABLE 6.3

16 min. LACTATE TEST (T16)

Rate of Perceived Exertion (RPE) correlated with other parameters of stress.

<table>
<thead>
<tr>
<th>RPE</th>
<th>HR</th>
<th>VO₂</th>
<th>VE</th>
<th>[La]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre (n=24)</td>
<td>0.997</td>
<td>0.990</td>
<td>0.981</td>
<td>0.950</td>
</tr>
<tr>
<td>Post</td>
<td>1.000</td>
<td>0.999</td>
<td>0.999</td>
<td>0.964</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre (n=11)</td>
<td>0.997</td>
<td>0.994</td>
<td>0.985</td>
<td>0.954</td>
</tr>
<tr>
<td>Post</td>
<td>0.998</td>
<td>0.998</td>
<td>0.991</td>
<td>0.924</td>
</tr>
</tbody>
</table>

RPEs and %VO₂ max. during the four incremental loads:

<table>
<thead>
<tr>
<th>RPE</th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre (Subjects)</td>
<td>9.4</td>
<td>11.7</td>
<td>13.5</td>
<td>15.5</td>
</tr>
<tr>
<td>Post</td>
<td>7.9**</td>
<td>9.3**</td>
<td>11.2**</td>
<td>13.2**</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre (Controls)</td>
<td>8.6</td>
<td>11.2</td>
<td>13.2</td>
<td>15.0</td>
</tr>
<tr>
<td>Post</td>
<td>8.5</td>
<td>10.2</td>
<td>12.9</td>
<td>15.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>%VO₂ max.</th>
<th>Pre</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre (Subjects)</td>
<td>38.9%</td>
<td>47.6%</td>
<td>61.1%</td>
<td>74.7%</td>
</tr>
<tr>
<td>Post</td>
<td>34.7%</td>
<td>42.9%</td>
<td>54.9%</td>
<td>65%</td>
</tr>
</tbody>
</table>

HR = Heart Rates, VE = Minute Ventilation, [La] = Lactate concs.

Significant difference: ** p<0.01
Figure 6.2. a) Relative exercise intensity (%VO$_2$max.) before and after 12 weeks of training as determined by the reference lactate concentration.

b) Changes in RPE as a factor of both relative work intensity and reference lactate concentration.
decrease in accompanying heart rates (161 vs 147 beat.min⁻¹; p<0.01). Thus, whereas before training this ultimate load was completed at 75% VO₂ max, following training it required only 65% VO₂ max (Figure 6.2a). Furthermore, at the same reference lactate tolerance of 4 mmol.l⁻¹ it was possible to increase the power output substantially post-training, from 138W to 167W with little change in relative work intensity: 63% vs 65% VO₂ max (Figure 6.2a). A fifth exercise intensity, presented to subjects following training in order to measure their new work capacity and lactate tolerance, resulted in a workrate of 227W at a lactate concentration of 8.5 mmol.l⁻¹ and at an intensity of 82% VO₂ max. (Figure 1, Appendix 7).

The 30 Minute Ergometer Test.

When oxygen uptake was plotted over the 30 min. exercise period (Figure 2, Appendix 7), there were differences (p<0.01) between T1 and T2 at each of the sampling points for the experimental group and this amounted to a 49% higher workload than in the pre-trained condition (116W vs 173W; F1,29 = 41.16; p<0.001) as shown by Figure 6.3. These changes were matched by increases in pulmonary ventilation (F1,29 = 17.78; p<0.001) and heart rates (F1,29 = 9.42; p<0.01; Figure 6.4). The oxygen uptake of the controls also increased following the 12 week training period but the resulting power output improved by only 8%. The most important interactions between experimental, and control, groups were found to be those of oxygen uptake (p<0.001), heart rates (p<0.01) and exercise intensities (p<0.001) with far greater differences revealed by the experimental group as a result of training.

During all 30 min tests, heart rates were found to be higher for the 30 year-olds than the 50 year-olds and there was a steady, but positive, increase, over the duration of the test common to both groups; this is apparent from Figure 6.4. The initial acceleration in heart rate, pre-training, was also not so great as that revealed during the post-training test.

Respiratory Exchange Ratio (R) values for the experimental group increased significantly (p<0.01) at 15 min. (1.02 pre- to 1.06 post-training) and remained elevated to the conclusion of T2. For the controls there were no changes throughout (R 1.05 pre-, 1.03 post-12 weeks), though both subjects and controls showed a decrease in R values over the duration of the test and this was true for all age groups.

Very high correlations were found between RPE and heart rates, pulmonary ventilation and lactates during T16 for both subjects and controls before and after the 12 week training period (Table 6.3). Following training there were significant changes in RPE over the time of the 30 minute test (T30) for both subjects and controls (F6,174 = 2.98; p<0.01) but the RPE
### TABLE 6.4

**12 WEEKS OF TRAINING**

Workrate, Energy Expenditure and Training Intensity

(Mean Values)

<table>
<thead>
<tr>
<th>SUBJECTS (n = 24)</th>
<th>WEEKS 1-3</th>
<th>WEEKS 4-6</th>
<th>WEEKS 7-9</th>
<th>WEEKS 10-12</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Workrates (W)</strong></td>
<td>121.4</td>
<td>147.9</td>
<td>165.2</td>
<td>179.2</td>
</tr>
<tr>
<td><strong>VO₂ (l.min⁻¹)</strong></td>
<td>1.85</td>
<td>2.25</td>
<td>2.36</td>
<td>2.60</td>
</tr>
<tr>
<td><strong>kcal.min⁻¹</strong></td>
<td>9</td>
<td>11</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td><strong>kJ.min⁻¹</strong></td>
<td>39</td>
<td>47</td>
<td>49</td>
<td>54</td>
</tr>
<tr>
<td><strong>%Intensity</strong></td>
<td>65.6%</td>
<td>73.7%</td>
<td>80.3%</td>
<td>83.0%</td>
</tr>
</tbody>
</table>

**30 min. Training:**

<table>
<thead>
<tr>
<th>kcal.</th>
<th>278</th>
<th>338</th>
<th>354</th>
<th>390</th>
</tr>
</thead>
<tbody>
<tr>
<td>kJ.</td>
<td>1161</td>
<td>1412</td>
<td>1481</td>
<td>1632</td>
</tr>
</tbody>
</table>

**3x Week Training:**

<table>
<thead>
<tr>
<th>kcal.</th>
<th>833</th>
<th>1013</th>
<th>1062</th>
<th>1170</th>
</tr>
</thead>
<tbody>
<tr>
<td>kJ.</td>
<td>3483</td>
<td>4236</td>
<td>4443</td>
<td>4895</td>
</tr>
</tbody>
</table>

1 kilocalorie = 4.184 kilojoules.
Training intensity determined using formula after Karvonen et al., (1957). (General Methodology).
Energy expenditure calculated on the basis of: 1 litre of Oxygen = approx. 5 kcal./21 kJ. (Shephard, 1987; McArdle et al., 1986).
for subjects increased compared with that of the controls ($F_{6,174} = 2.65; p<0.01$) and of the pre-training values. This can be readily observed for the subjects in Figure 6.5.

**Metabolic, Hormonal and Blood Lipid Responses.**

The post-training 30 min test (T2) revealed elevations in the differences in plasma lactates, plasma glucose and plasma catecholamines for the experimental group ($p<0.01$; Figures 6.6 and 6.8) compared with the pre-training test (T1). Resting lactate values, pre and post-training, were 0.76 and 0.61 mmol.l$^{-1}$ respectively for the experimental group, 0.70 and 0.85 mmol.l$^{-1}$ for the controls, providing the differences in concentrations shown in Table 6.6. Plasma glucose levels fell, pre-training, for the subjects, from 4.06 to 3.63 mmol.l$^{-1}$ ($p<0.01$) during T1 but increased during T2 from 4.30 to 4.60 mmol.l$^{-1}$ ($p<0.01$). For the controls, however, resting values of 4.24 and 4.42 mmol.l$^{-1}$ prior to T1 and T2 respectively, fell to concentrations of 3.95 and 4.15 mmol.l$^{-1}$ ($p<0.01$).

No differences were found, within or between either of the groups for plasma FFA and plasma glycerol (Figure 6.7; Table 6.6). Nevertheless, these data mask differences that occurred in absolute values between resting and final measures of FFAs. following T1 (0.36 and 0.49 mmol.l$^{-1}$; $p<0.01$) and T2 (0.36 and 0.50 mmol.l$^{-1}$; $p<0.01$) for subjects whereas controls remained unchanged. Resting glycerol concentrations also increased, doubling at the conclusion of each of the two tests: for example, T2 values for the subjects rose from 0.10 pre to 0.24 mmol.l$^{-1}$ ($p<0.01$) post-test but this was common for both experimental subjects and controls with similar concentrations throughout.

Absolute concentrations of noradrenaline for the subjects doubled following training (Table 1, Appendix 7A); a resting value of 1.8 nmol.l$^{-1}$ rose to 6.1 nmol.l$^{-1}$ ($p<0.005$) by the conclusion of T1 whilst during T2, a resting concentration of 2.2 nmol.l$^{-1}$ increased to a final 13.8 nmol.l$^{-1}$ ($p<0.005$). Adrenaline concentrations rose for both tests for the experimental group: T1, 0.58 to 1.50 nmol.l$^{-1}$ ($p<0.005$); T2, 0.55 to 2.27 nmol.l$^{-1}$ ($p<0.005$) and these results showed differences over the controls ($p<0.05$) after the post-training test, T2 (Figure 6.8; Table 6.6).

The experimental subjects increased their pre-training total cholesterol following training ($p<0.01$) and also revealed higher values than the post-trained controls ($p<0.05$; Table 2, Figure 1, Appendix 7A). These data were confirmed by the 4-Way ANOVA which also emphasised an increase in LDL-C ($F_{2,28} = 5.49; p = 0.0097$) as a factor of age rather than of training. There were increases in HDL-C ($p<0.05$) for both the trained subjects and the controls. The HDL/LDL fraction was identical for both groups pre-training at 28%, increasing to 29% for the subjects and 32% for the controls, post-training.
TABLE 6.5

Measures of Functional Capacity during the 30 min. test (T30), pre-training (T1), and post-training at new loads (T2).

(Mean ± SD)


<table>
<thead>
<tr>
<th>Variable</th>
<th>Test</th>
<th>Subjects</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Workrates (W)</td>
<td>[T1]</td>
<td>115.6 ± 32.7</td>
<td>126.6 ± 32.7</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>173.9 ± 30.3 ***</td>
<td>137.3 ± 27.8 (NS)</td>
</tr>
<tr>
<td>Heart rates</td>
<td>[T1]</td>
<td>139 ± 21</td>
<td>138 ± 23</td>
</tr>
<tr>
<td>(beat.min⁻¹)</td>
<td>[T2]</td>
<td>157 ± 16 **</td>
<td>139 ± 20 (NS)</td>
</tr>
<tr>
<td>VO₂ (l.min⁻¹)</td>
<td>[T1]</td>
<td>1.8 ± 0.4</td>
<td>2.0 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>2.5 ± 0.4 **</td>
<td>2.1 ± 0.4 (NS)</td>
</tr>
<tr>
<td>VO₂ (ml.kg.min⁻¹)</td>
<td>[T1]</td>
<td>25.0 ± 5.6</td>
<td>26.3 ± 6.6</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>35.0 ± 5.6 **</td>
<td>27.7 ± 5.3 (NS)</td>
</tr>
<tr>
<td>VE (l.min⁻¹)</td>
<td>[T1]</td>
<td>44.6 ± 13.4</td>
<td>51.4 ± 14.4</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>67.6 ± 20.0 ***</td>
<td>54.1 ± 11.5 (NS)</td>
</tr>
<tr>
<td>R Values</td>
<td>[T1]</td>
<td>1.02 ± 0.07</td>
<td>1.05 ± 0.09</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>1.06 ± 0.07 **</td>
<td>1.03 ± 0.07 (NS)</td>
</tr>
<tr>
<td>Oxygen Pulse</td>
<td>[T1]</td>
<td>0.18 ± 0.01</td>
<td>0.19 ± 0.01</td>
</tr>
<tr>
<td>(ml.br⁻¹)</td>
<td>[T2]</td>
<td>0.22 ± 0.01 **</td>
<td>0.20 ± 0.01 (NS)</td>
</tr>
<tr>
<td>RPE (Rate Perc. Exert.)</td>
<td>[T1]</td>
<td>13.7 ± 1.5</td>
<td>13.6 ± 0.7</td>
</tr>
<tr>
<td></td>
<td>[T2]</td>
<td>14.8 ± 1.5 **</td>
<td>13.3 ± 1.6 (NS)</td>
</tr>
</tbody>
</table>

Significant difference between tests: * p<0.05; ** p<0.01; *** p<0.001
NS = Not Significant.
6.4 DISCUSSION

The main finding of this study was that after 12 weeks of progressively increasing aerobic training, 24 middle-aged men demonstrated their ability to complete a 30 min ergometer test at a 50% higher power output than before training. They did this despite maintaining the same, single RPE of 14 for each of the tests, both before (T1), and after (T2), training.

The ability of the group to make these changes at the same RPE was part physiological, part psychological. The physiological components were established through the group's trainability as revealed in significant improvements in commonly accepted forms of fitness testing in the field, namely the 12 min Cooper test, a 2 mile (3219m) and a 5 km, run from which it was possible to predict VO2max. (Ramsbottom et al., 1987) in attempting to overcome some of the limitations of previous methods (Rowell et al., 1964) as already delineated (Chapter 4). It also avoided the trauma that the VO2max. presents in the laboratory, especially to the older, sedentary individual.

The Power Lactate Test.

This test was invaluable in monitoring standard functional changes in response to submaximal exercise. It was a useful model in reflecting metabolic adaptations induced by the training, and in providing the essential practice which is known to improve the applicability of the psychological aspects of the study, that is, the gradations of the RPE scale, particularly at the lower levels between 9 - 13 (Eston and Williams, 1988). From among the 'gestalt' of sensations (Borg and Noble, 1974) believed to influence the choice of RPE, blood lactate concentrations are suspected of providing local, peripheral signals (Pandolf, 1985) whilst pulmonary ventilatory responses during exercise intensities greater than 50% VO2max., as with the fourth and fifth loads of T16, pre- and post-training, are considered to be capable of providing central signals of exertion that are consciously monitored (Robertson and Metz, 1986). Even so, the fall in ratings of stress (p<0.01), post-training (Table 6.3, Figure 6.2b) would suggest a diminished sensory drive with a training-induced lowered VE in response to acid-base shifts (Rowell, 1969). However, the close coupling between blood lactate, RPE and relative work intensity is reflected in a linearity which did not diminish following training (Figures 6.2a and b).

Nor did the relative work intensity vary by more than 2% at the 4 mmol.l-1 lactate level following training, indicating a 'preferred exertion' (Dishman, 1994) averaging 64% at the level of lactic acid 'turnover' (Stainsby and Brooks, 1990). This balance of lactate tolerance between production and removal, was equivalent to an RPE of 13.2 (Figure 6.2b) and averaged 60% VO2max. under pre- and post-training conditions. Thus, a recommended training prescription
of between 60 - 64% \( \text{VO}_2\max \), as suggested by these findings, would provide a more precise training intensity than is generally advocated. For example, the ACSM (1991) recommend as wide a range as 40 - 85% \( \text{VO}_2\max \). in an attempt to embrace a large population of fitness requirements. The lactate reference point, or anaerobic threshold (AT), of 13.2 RPE\(_{\text{AT}}\) provides further validity for the testing procedures by falling within the training guidelines of RPE 12 - 14 recommended by a number of authors (Katch et al., 1978; Burke, 1979) though Purvis and Cureton (1981) err towards the higher limit of RPE\(_{\text{AT}}\) 14.2 ± 0.9 for men. It could also be deduced (Table 6.3) that training was capable of lowering RPE by a factor of 2 points as revealed by a comparison of the ratings of the latter three work intensities, pre- and post-training (Figure 6.2b) and in keeping with the findings of previous authors (Ekblom and Goldbarg, 1971).

**The 30 Minute Ergometer Test.**

Whereas the common denominator for subjects completing T16 was a predetermined workload, that for the 30 min endurance test (T30) was a predetermined single rating of exertion of 14, between 'hard' and 'somewhat hard' on the Borg Scale (Borg, 1985), bearing in mind the special relevance of the scale for healthy, middle-aged men (Borg, 1973) and the guidelines advocated by earlier authors (Purvis and Cureton, 1981). It is an interesting phenomenon that, after 12 weeks of acute training, subjects should, after 5 min of reassessment, chose a load virtually 50% higher than before training (116W vs 173W; \( p<0.001 \). Figure 6.3). The group maintained this load within 4W over a 30 min period without any indication of a change in the magnitude of the 'fatigue curve' in the form of a diminished power output. In terms of the relative oxygen demands of the test, the untrained subjects were found to be working at 62% \( \text{VO}_2\max \). throughout T1 and this translated to just below the 4 mmol.l\(^{-1}\) lactate tolerance level of T16, 3.2 mmol.l\(^{-1}\) as measured at the conclusion of the test (Table 6.6), 3.9 mmol.l\(^{-1}\) in absolute terms (Table 1, Appendix 7A). Following training, such an innate inhibitory mechanism appeared to be lifted: subjects maintained a relative work intensity of 77% \( \text{VO}_2\max \). for the duration of the test and this resulted in a final lactate concentration difference of 7.7 mmol.l\(^{-1}\), 8.3 mmol.l\(^{-1}\) as an absolute concentration.

An explanation for such an elevated lactate concentration following training may lie in the mechanisms associated with blood lactate production and its clearance. It would be a mistake to interpret blood lactate accumulation as solely reflective of muscle lactate production. The intestine, liver, skin, heart and non-exercising fibres of skeletal muscle itself, have all been found to be capable of releasing, or using, lactate (Stainsby and Brooks, 1990). With all these tissues available for lactate removal together with pathways enhanced through training, it would not be surprising if the removal of lactic acid from the blood should rise as blood lactate
Figure 6.3 Cumulative Average Work Rates (CAWR) over the 30 min. ergometer test during T1 and T2 for both subjects and controls.

Figure 6.4 Time-related heart rates for both subjects and controls during T1 and T2.
concentration and activity rise. This would provide the well known phenomenon of lowered blood lactate following training (MacRae et al., 1992) as illustrated by the post-training Power Lactate Test values (Figure 1, Appendix 7). If this has been found to be true for a test of progressive exercise intensity, it would seem to be even more valid for endurance exercise sustained at a given power output over 30 minutes. A 'steady-state' condition could be considered capable of providing an environment in which lactate removal exceeded its production. The consistent power output (Figure 6.3) and oxygen uptake (Figure 2, Appendix 7) confirm the stable exercise intensity and mechanical efficiency. Yet increasing heart rates (Figure 6.4) and RPEs (Figure 6.5) suggest that the biological and psychological stresses are anything but uniform during this test. It has also been proposed that when blood adrenaline rises, glycolysis is stimulated causing an increase in muscle lactate production and a decrease in its removal from other, buffering tissues, resulting in a net increase (Stainsby and Brooks, 1990). Such a scene appears to have been set during the post-training, 30 min test with adrenaline concentrations doubling their pre-training values (Figure 6.8) together with increased (p<0.01) glucose levels (Figure 6.6). Increased blood lactate concentrations during a 30 min post-training test would thus appear to be a combination of an increased rate of lactate appearance and a decreased rate of lactate clearance, a reversal of the normal diminished rate of lactate appearance following training. The subjective response to these conditions are reflected in steadily rising RPEs.

A further distinction was the ability of the experimental group to maintain a relative work intensity of 77% VO\textsubscript{2}max. throughout the 30 min post-training test (T2) at a putative RPE of 14. The explanation for this achievement appeared to be dependent on a multiplicity of conditions, not least that of the initial level of fitness (Satin, 1969). At starting VO\textsubscript{2}max. values of 39.9 ml.kg.min\textsuperscript{-1} (2.88 l.min\textsuperscript{-1}) in completing T1, the experimental group fell between the categories of 'sedentary' and 'normally active' (Rowell, 1969), the further the initial VO\textsubscript{2}max. below 45 ml.kg.min\textsuperscript{-1}, the greater the reputed, relative and absolute, increment with conditioning. That the subjects were capable of improving their VO\textsubscript{2}max. by 14.3% following training would appear to have been primarily as a result of an ability to raise their power output progressively every three weeks during training. Consequently they achieved an improvement which closely paralleled that found among several studies of normal young men, aged 20 - 30 years, after 2-3 months of training (Satin et al., 1968; Rowell, 1974a; Clausen, 1977). Something of an 'age-sparing' capacity would appear to have been acquired by this progressive overload form of training, enabling the post-training test to be completed at a 15% higher relative work intensity than before training.
Figure 6.5. Fluctuations in Rates of Perceived Exertion (RPE) for subjects pre (T1) and post (T2) 12 weeks of training.
This higher tolerance of exercise intensity was not achieved without cost. As already indicated, consistently uniform oxygen demands, minute volumes and work intensities during T1 and T2 were paid for by steadily rising heart rates and RPEs. Despite working at 20 beat.min\(^{-1}\) above T1, the trained subjects demonstrated a similar 'cardiovascular drift' of, from 6 - 8 beat.min\(^{-1}\), from 10 min to the conclusion of T2. This was a similar pattern to that shown when they completed T1 before training. They did enhance their stroke volume through training as revealed by an improved oxygen pulse (p<0.01; Table 6.5). Yet it would appear that the dual role of the blood as a transporting medium for blood gases and as a heat transfer mechanism, could not fulfil the tissues' requirements in their competition with the skin (Rowell, 1977) except by means of an accelerated heart rate.

All subjects, whether during T1 or T2, rated the exercise intensity lower after 5 min following the commencement of the test. The stipulated RPE of 14 was not reached until just beyond 20 minutes with a final rating of 14.3 at the conclusion of T1. In contrast, during T2, a rating of 14.1 was reached after 10 min and rose to 15.9 at 30 min (Figure 6.5). The untrained subjects thus erred on the side of caution in their desire to complete the test with a load which, presumably, they felt able to manage for the full 30 min. The trained subjects experienced no such inhibitions, though it is apparent that they were sensitively aware of the increasing demands of the test as their constantly increasing RPEs reveal.

Of the age groups with the highest relationships between heart rates and RPEs, that from 40 - 50 years revealed the highest number of correlations above 0.7 with very high correlation coefficients (r = 0.9) increasing from 2 to 6 post-training. Correlations between VE and RPE, however, revealed decreased sensitivity throughout the three age groups: 30 - 40 year olds; 40 - 50 year olds; 50 - 60 year olds. This decreased sensitivity to the ventilatory response over the rising age continuum, may well reflect the inability of men over 40 years to respond to increasing metabolic acidosis in the same way as younger men, possibly as a result of decreased alkali reserves, long known to be an attribute of the aging process (Mori, 1936). Thus, the proportional increase in expired carbon dioxide as a product of bicarbonate buffering would be diminished, as would the respiratory compensation or total pulmonary ventilation. The result would be a reduced sensitivity in terms of both ventilatory stress and derived perceived exertion. For though it is unlikely that heart rate is consciously perceived during exercise, when ventilatory responses have been manipulated by, for instance, hypercapnia (Cafarelli and Noble, 1976), hypoxia (Robertson et al., 1979b) and hyperoxia (Pedersen and Welsh, 1977), potent sensory signals have been monitored by means of RPE. It is possible that mechanoreceptors in the chest wall, lungs and airways, may provide the mediators for such sensitivity. Wolkove et al., (1981) reported that signals from the
TABLE 6.6

Differences in plasma metabolites, within and between pre (T1) and post (T2) training 30 min Tests following 12 weeks of training.

(Mean±SEM)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>n=24</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lactates (mM)</td>
</tr>
<tr>
<td>Pre</td>
<td>3.2±0.4</td>
</tr>
<tr>
<td>Post</td>
<td>7.7±0.9 **</td>
</tr>
<tr>
<td>Glycerol (mM)</td>
<td>Adrenaline (nM)</td>
</tr>
<tr>
<td>Pre</td>
<td>0.1±0.02</td>
</tr>
<tr>
<td>Post</td>
<td>0.2±0.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Controls</th>
<th>n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lactates (mM)</td>
</tr>
<tr>
<td>Pre</td>
<td>4.3±1.1</td>
</tr>
<tr>
<td>Post</td>
<td>4.7±0.9</td>
</tr>
<tr>
<td>Glycerol (mM)</td>
<td>Adrenaline (nM)</td>
</tr>
<tr>
<td>Pre</td>
<td>0.1±0.03</td>
</tr>
<tr>
<td>Post</td>
<td>0.1±0.02</td>
</tr>
</tbody>
</table>

** Significantly higher post-training (p<0.01)
mechanoreceptors to the sensorium were consciously monitored only when the tidal volume exceeded 700 ml. As peak tidal volume is achieved at 50% VO\textsubscript{2}max., it appears that only when the metabolic rate increases to this level, and beyond, does the intensity of the VE signal also increase. The onset of these signals has been found to fall near the 4 mmol.l\textsuperscript{-1} lactate threshold for most individuals (Robertson, 1982) so the buffering of metabolic acidosis may then provide an increased pulmonary ventilation as a positive signal of perceived exertion.

In the present study, it may be hypothesised that the untrained subjects unconsciously matched their chosen relative power output of 63% VO\textsubscript{2}max. by means of conscious ventilatory responses coincidental with a 4 mmol.l\textsuperscript{-1} metabolic equivalent and an RPE of 14 as a term of reference. Following training, were the same relative work intensity to be chosen, it would no longer provide the ventilatory 'drive' since pulmonary volume would have fallen for the same absolute power output. By gradually raising the load, a point would be reached where similar sensations of perceived exertion would be monitored through the ventilatory mechanisms. That point was found to be at the work intensity of 77% VO\textsubscript{2}max., inducing, predictably, a new lactate tolerance threshold in the region of 7 mmol.l\textsuperscript{-1}. The experimental group benefitted through the enhanced ventilatory scale of perception which provided no such training 'dose response' for the controls whose ventilation remained unchanged following the 12 week period.

It is difficult to propose an alleviation of the sense of stress during T2 by means of any 'glucose sparing' metabolism in order to account for a raised work intensity at the same RPE when the balance between glycolytic, and oxidative, phosphorylation is so clearly biassed towards the former pathway. The experimental group may well reveal an ability to tolerate a higher lactate threshold but the presence of lactic acid is symptomatic of the rate of glycogen degradation in the working muscle (Sahlin, 1992) and can be regarded as a powerful influence in stimulating RPE during the latter half of T2 (Johansson, 1985; Maresh et al., 1993). Only unless an ability to remove lactate is revealed can fat be utilised as an energy substrate (Costill, 1986). Furthermore, the significant increase in adrenaline at the conclusion of T2 (Table 6.6, Figure 6.8) provides substantial evidence that muscle glycogenolysis has been stimulated (Jansson et al., 1986) especially when coupled with the contractile process of exercise itself, a known stimulator of glycogenolysis (Cohen, 1981; Richter et al., 1982). An enhanced utilisation of glucose in the working muscles during T2 is given additional support by R values higher than those of T1 (p<0.01; Table 6.5). The increased glucose differences (Figure 6.6) which mimic these changes may be, in turn, coincidental with, and balanced by, enhanced hepatic gluconeogenesis (Isseruk, 1981). This is reflected in the glucose
Figure 6.6 Plasma lactate and glucose differences pre and post 12 weeks following T1 and T2 for both subjects and controls.
precursors, not only of increased lactate concentrations but those of glycerol (Saltin and Gollnick, 1983) which rose over the test period (Figure 6.7).

Yet FFA concentrations did rise (p<0.01) from resting values, slightly more so during T2 than T1, and where increased plasma FFA concentrations have been identified, this always means an increased rate of FFA utilisation (Pruett, 1971). Plasma FFA levels during exercise are determined by the combined effects of several factors not least those of noradrenaline as a powerful stimulator of FFA mobilisation (Issekutz, 1964) and lactate as an equally powerful inhibitor (Issekutz et al., 1975). Both noradrenaline and lactate concentrations increased during the completion of T2 (p<0.01; Table 6.6) but which concentration may have dominated the metabolism remains equivocal in the absence of intra-test sampling. The ability of released noradrenaline to stimulate lipolysis may have been inhibited, not only by the lactate concentration, but by the fact that the source of noradrenaline concentrations during the relatively heavy exercise of 77% VO₂max. could be owing, primarily, to the 'spillage' from the tonically active sympathetic nerve terminals (Rowell, 1969; Levick, 1991) rather than from glandular secretion. Alternatively, since lipolysis has been found to be activated after 20 min during this type of exercise intensity (Pruett, 1970), the incipient rise in FFA concentration may only just have begun to be monitored in the plasma by the conclusion of T2. Whatever the answer, it is clear that the regulation of fat and carbohydrate metabolism during moderately heavy exercise depends on a very complicated balance from among many factors.

As a reflection, both of the type of training in which the experimental subjects were able to choose a higher power output for a given RPE, and the lipid environment in which that improvement was effected, the lipoprotein profile was measured. It showed that there were no significant differences in resting cholesterol levels for either training, or control, groups (Figure 1, Appendix 7A). The subjects revealed a higher concentration following training compared with the controls (206 mg.dl⁻¹ to 185 mg.dl⁻¹ or 5.3 mmol.l⁻¹ and 4.7 mmol.l⁻¹, respectively). These values fell within acceptable reference ranges (Schettler, 1982; Ball and Mann, 1988) and similar levels have previously been equated with large groups of middle-aged men who participated in jogging and marathon running respectively (Hartung et al., 1980). There was an increase in HDL-C (p<0.05) for the subjects but this could not be attributed to training alone since there was a similar increase for the controls. Seasonal variation may play a major role in lipoprotein concentration (Huttunen et al., 1979) though the recreational form of exercise of the controls, carried out as a lifestyle, cannot be discounted. In terms of the relationship between training and HDL-C, it has been suggested that studies terminating after only 8 - 16 weeks of training can measure only transitory changes in lipoproteins (Wood et al., 1983). Numerous studies would support this contention (Lopez,
**Figure 6.7** Differences between plasma free fatty acids and glycerol concentrations among subjects and controls over the training period as revealed by the 30 min ergometer test.

**Figure 6.8** Catecholamine differences in response to the training period and to the 30 min ergometer test.
1976; Wood and Haskell, 1979) including data from the 'Vasaloppet' ski race (Carlsson and Mossfeldt, 1964) where a marked decrease in serum triglyceride values was found without significant changes in the cholesterol concentration of any of the lipoprotein fractions.

The present lipoprotein profile would be represented by an increased HDL-C subfraction with no change in TC nor in LDL-C. This would place it in a comparable position to the study by Thomas et al., 1984 (Table 3, Appendix 7A) with which it has similarities in both training intensity and duration. With HDL-C as the carrier of apolipoprotein C-II, an activator of lipoprotein lipase, the enzyme responsible for triglyceride uptake by the cell (Miller, 1979), a high HDL-C would appear to favour triglyceride uptake, resulting in lower plasma levels. The key mechanism which may stimulate the need for enhanced triglyceride and thus trigger the production of more HDL-C could be the more precise quantification of the intensity and duration of exercise per se with its varied facility in lowering intramuscular triglyceride stores. Why free fatty acids failed to increase significantly in the exercising subjects of the present study may reflect the balance between carbohydrate and fat metabolism as a factor of training demand. Dietary influences may also play their part, though this aspect was not considered, manipulated nor analysed with the present subjects. Alternatively, since training alone cannot account for HDL-C concentration, age may be a contributory factor. However, when a study by Hudson et al., (1987) compared three groups of subjects: untrained, sprint-trained and endurance-trained with 12 subjects in each group (6 males, 6 females), average ages 21, 23 and 23 years respectively, only the TC of the untrained group was lower (p<0.05) than the sprint-trained group. Since there were no changes in HDL-C, it was concluded that training status has little or no influence on HDL-C. Though the 4-Way ANOVA in the present study did reveal increases in LDL-C as a factor of age rather than of training, the overall impression was that training was not an underlying factor influencing the present lipoprotein findings.

SUMMARY

This study has shown that the methods of training and testing were valid in determining the trainability of a group of middle-aged men. In the laboratory a Power Lactate Test of 16 min duration (T16) provided a useful model in assessing metabolic changes and setting criteria of relative work intensity and RPE at a 4 mmol.l⁻¹ reference lactate threshold. The main test of the study consisted of an ergometer endurance ride, sustained over 30 min at an exercise intensity self-selected by the subjects' sensory response to a single RPE of 14. When completing this test, pre-training, both experimental subjects and controls were found to exercise in a similar manner to the T16 model in terms of metabolism, RPE and relative exercise intensity. Following training, however, the experimental group selected a power output 50% above pre-training values. This choice was accompanied by a considerably higher
relative exercise intensity, together with increased hormonal concentrations and a metabolism dominated by glycolysis.

CONCLUSION
A group of sedentary, middle-aged men were capable of increasing their power output by 50% when completing a 30 min ergometer test following a period of acute training. They achieved this by means of a self-selected choice of exercise intensity using a single RPE of 14. Whereas the pre-training test was completed in what may be regarded as a 'comfort' zone of stress, post-trained subjects displayed a capacity to endure a 'just tolerable discomfort' level of intensity as revealed by increases in functional capacity and accelerated carbohydrate metabolism. This ability to perform significantly more work at a single RPE illustrates the adaptations possible when the level of habitual activity is increased. It also emphasises the advantages of a subject- rather than a tester-orientated choice of exercise intensity when examining middle-aged, and older, subjects.

LIMITATIONS
It has been noted that, though certain parameters were maintained at constant values throughout T30, that of the Rate of Perceived Exertion could not be controlled owing to limitations in the test protocol. Nor was it possible to comment with confidence regarding measures of some of the more crucial training-induced changes, such as blood lipids, other than by speculation. These shortcomings would appear to call for further investigation.
6. b) ANTHROPOMETRIC ADAPTATIONS FOLLOWING TWELVE WEEKS OF AEROBIC TRAINING.

6.5 INTRODUCTION.
A clear interplay between both central and peripheral adaptations as a result of endurance training has been found to exist, particularly in those studies which have utilised a single leg training model (Saltin et al., 1976). Improved heart rate and blood lactate responses to standard exercise after training have occurred but only during exercise with the trained leg. Peripheral changes of skeletal muscle metabolism are also believed to be important determinants of the improvements in endurance found as a consequence of training (Gollnick et al., 1973; Davies and McDonough, 1982) and these improvements are often in excess of the modest training-induced increases found in maximum oxygen uptake. They are usually accompanied by reductions in the rate of perceived exertion (RPE; Borg, 1985) when the same absolute power output is repeated, post-training, and this would suggest modifications to the sensorium of the cortex through afferent pathways.

Furthermore, these central neurogenic pathways are strengthened through what has been called an 'hierarchical chain of command' (Edwards, 1981; Newsholme and Leech, 1983), passing from the motor cortex, through the pyramidal and extra-pyramidal network and culminating in the spinal 'motor pool'. The excitation-contraction coupling (Davies and McDonough, 1982) is stimulated and this is manifest in an improved generation of force. Almost any strength training method has been found to result in an increase in muscle strength but only if the frequency of exercise and loading intensities are progressively increased (Hellebrandt et al., 1947; Komi, 1986). Synonymous with such increased strength is skeletal muscle hypertrophy, the two being conjoint consequences of functional overload (Steinhaus, 1955; Goldberg et al., 1975; McDonough and Davies, 1984).

During the present study, the experimental subjects were required to increase the load on the ergometer flywheel progressively every three weeks, thus meeting the above progressive overload criterion. The most dominant prime movers of the training regimen undertaken by the experimental group are known to be the m. quadriceps (Thompson, 1985) so changes in forces and volumes of this muscle group could best be identified by measurements of the thigh. Therefore the purpose of this study was to utilise a water displacement technique based on the Archimedian principle in order to validate calculated thigh volumes used in combination with anthropometric techniques (Jones and Pearson, 1969). Ultrasonic measuring techniques, in turn, were also used in order to validate the
skinfold measurements integral to the anthropometry. Forces generated by muscles of the thigh were determined by means of a 20s isometric maximum voluntary contraction (MVC) and, from the accumulated non-invasive data, it was proposed to examine the relationship between central and peripheral mechanisms as mediators of the training stimulus. It was speculated that any changes in the forces developed by the thigh muscles would relate to changes in the RPE associated with the 30 min cycle ergometer endurance test.

6.6 METHODS.

In order to meet the requirements of the purpose outlined above, the thirty-five males of the study, 24 subjects and 11 controls, were required to complete a battery of tests and measurements comprising:-

i) ANTHROPOMETRIC MEASUREMENTS: The anthropometric determination of leg fat and muscle plus bone volumes from a previously validated method by Jones and Pearson (1969) (Figure 6.9). The protocol for the required calculations can be found under Appendix 8.

ii) WATER DISPLACEMENT. Reproducibility of the calculated thigh volumes was carried out by comparing the values with those obtained by means of a water displacement method with apparatus especially designed for the purpose (Jones and Pearson, 1969). This consisted of two tanks which were so constructed as to enable a) total leg volumes up to the gluteal furrow and b) shank plus foot volumes, to be determined (Figure 6.10). Reliability of repeated displacements was found to be ± 50ml. (Jones and Pearson, 1969). Further testing, using displacement of objects of known weight, provided a Coefficient of Variation (Coeff.of Var.) of 1.27%; twenty repeat measurements of a single male subject's leg provided Coeff.of Var. of 1.41% for shank plus foot displacements and 1.78% for that of total leg volumes.

iii) A TWENTY SECOND VOLUNTARY ISOMETRIC CONTRACTION OF THE QUADRICEPS MUSCLES. A purpose built, isometric chair (Figure 6.11) with adjustable force transducer (HBM: Hottinger Baldwin Messtechnik, Darmstadt, Germany 7) ensured a standardised posture for each subject. The accuracy of the position of the leg for repeated measurements was obtained by means of a goniometer placed over the lateral femoral-tibial joint. The position of the ankle cuff was determined by measurements from the floor to each end of the linkage joining cuff and transducer. A strain gauge amplifier (CIL Electronics Ltd., Worthing, W.Sussex) relayed data to a chart recorder which served as a visual cue for each subject. Prior to use, the loadcell on the amplifier was calibrated by means of an 800N weight. A test-retest reliability study using 17 subjects: 6 females, 11 males (‡ age 35.9 ±12.3yr.) with
Leg Anthropometry

\[ \frac{1}{3} h(a + \sqrt{ab} + b) \]

Jones & Pearson. 1969

Figure 6.9  Anthropometric model: the six truncated segments of the leg and the sites of measurement.

Water Displacement

\[ \text{Thigh Volume} = \frac{x - y}{2} \]

Figure 6.10  The method of determining thigh volumes by means of water displacement.
Figure 6.11  The purpose-built, isometric chair used to determine forces generated by the m. quadriceps following training.
an average of 4 days between tests, was carried out on this test. For the right leg, correlations were very high (Table 6.7) ranging from 0.97 to 0.91 over the 20 s period; for the left leg, correlations were high to very high, in the range 0.88 to 0.96 (Cohen and Holliday, 1979).

iv) ULTRASONIC MEASUREMENTS OF SUBCUTANEOUS ADIPOSE TISSUE.
In order to reinforce skinfold measurements and replace those which were notoriously difficult of access, such as the posterior thigh and medial and lateral calf, ultrasonic measurements were utilised. This enabled more precise anthropometric volumes to be calculated. Subcutaneous adipose tissue thickness (SCATT) measurements were made using a 5MHz transducer coupled to an EchoScan 1502 (Par Scientific Instruments ApS, Denmark) in A-Scan mode. The transducer was coupled to the dermis with aquasonic gel which helped to isolate the appropriate peaks on the display oscilloscope. Calibration was effected by means of a perspex block (P104-50-50) of known thickness (20mm) and known ultrasonic velocity (2700ms). From this information it was possible to determine the Transit Time (TT) through the perspex block and calibrate it according to the ultrasonic velocity of human tissue (1450ms) as follows:-

\[
\text{Transit Time}(TT) = \frac{\text{Perspex Thickness}}{\text{Velocity of ultrasound}} 	imes \frac{\text{Velocity of Human Tissue}}{\text{Tissue}}
\]

\[
= \frac{20}{2700} \times 1450
\]

\[
= 10.7 \text{mm}
\]

SCATT measurements could then be extrapolated from the various 'echoes', or peaks, revealed by the display.

All measurements, whether skinfold or ultrasonics, were taken by one observer, as suggested by Burkinshaw, Jones and Krupowicz, (1973). Prior to the study, 10 repeat skinfold measurements on the anterior thigh of four subjects provided a mean Coeff. of Var. of 0.98%; 10 ultrasonic measurements on the same site of the same subjects resulted in a Coeff. of Var. of 2.5%. For the skinfolds, the same Holtain caliper was used throughout, having a jaw surface area of 0.09m² and exerting a pressure of 10g.mm². The technique used was that described by Marshall (1977), usually resulting in a stable reading up to values of 20mm. Skinfold
TABLE 6.7

TEST - RETEST RELIABILITY FOR 20 s LEG EXTENSION TEST

(Forces in Newtons ± SD)

RIGHT LEG
n = 17

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th>POST</th>
<th>(r = 0.92)</th>
<th>NS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 s</td>
<td>625.1 ± 148.8</td>
<td>649.8 ± 154.3</td>
<td>(r = 0.92)</td>
<td>NS</td>
</tr>
<tr>
<td>5 s</td>
<td>624.5 ± 168.1</td>
<td>622.4 ± 167.4</td>
<td>(r = 0.97)</td>
<td>NS</td>
</tr>
<tr>
<td>10 s</td>
<td>603.4 ± 148.2</td>
<td>620.5 ± 172.3</td>
<td>(r = 0.94)</td>
<td>NS</td>
</tr>
<tr>
<td>15 s</td>
<td>575.8 ± 142.9</td>
<td>591.9 ± 164.3</td>
<td>(r = 0.93)</td>
<td>NS</td>
</tr>
<tr>
<td>20 s</td>
<td>421.2 ± 130.3</td>
<td>461.2 ± 141.4</td>
<td>(r = 0.91)</td>
<td></td>
</tr>
</tbody>
</table>

LEFT LEG
n = 17

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th>POST</th>
<th>(r = 0.96)</th>
<th>NS</th>
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</thead>
<tbody>
<tr>
<td>1 s</td>
<td>604.2 ± 176.9</td>
<td>619.1 ± 169.9</td>
<td>(r = 0.96)</td>
<td>NS</td>
</tr>
<tr>
<td>5 s</td>
<td>580.5 ± 168.6</td>
<td>611.8 ± 171.2</td>
<td>(r = 0.94)</td>
<td></td>
</tr>
<tr>
<td>10 s</td>
<td>568.2 ± 163.9</td>
<td>596.7 ± 156.8</td>
<td>(r = 0.93)</td>
<td>NS</td>
</tr>
<tr>
<td>15 s</td>
<td>543.9 ± 157.7</td>
<td>569.7 ± 154.2</td>
<td>(r = 0.88)</td>
<td>NS</td>
</tr>
<tr>
<td>20 s</td>
<td>427.9 ± 129.9</td>
<td>435.1 ± 139.8</td>
<td>(r = 0.89)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Significant differences between means: * p<0.05; NS = Not Significant.
thicknesses at the various sites were measured on the left leg according to standard procedures (Weiner and Lourie, 1981).

6.7 RESULTS

Correlation coefficients between skinfold and ultrasonic measurements of SCATT ranged from modest ($r = 0.59$) to high ($r = 0.81$) as shown by Table 6.8 with the more consistently high correlations found between measurements of the medial and lateral calf. The ratios of the caliper reading to the ultrasound reading for medial and lateral calf were also lower (range 1.56 - 1.78) than anterior and posterior thigh (range 2.2 - 2.0), illustrating the variation in compressibility (caliper reading divided by ultrasound reading). This suggests that sites on the calf were more compressible than elsewhere but for any of the eight readings, Coeff. of Var. was 19.2 - 31.5%. A similar pattern was revealed by the controls. Both methods actually measure subcutaneous adipose tissue plus dermis but in the case of the caliper measurements, this was a compressed double layer. Therefore, in order to obviate the need for applying a correction factor for compressibility and dermis thickness (Brown and Jones, 1977) for skinfold data and in view of the high correlations between ultrasonic measurements and a wide range of sampling techniques (Jones, Davies and Norgan, 1986) including cadaver and soft-tissue radiographs (Jones, 1970), it was decided to adopt the ultrasonic measurements in order to calculate the relevant leg volumes.

Very high correlations were found between leg volumes determined by water displacement and those calculated from anthropometric measurements (range: $r = 0.87$ to $r = 0.95$; Table 6.9). These results compare very favourably with those determined in the initial study (range: $r = 0.99$ to $r = 0.93$) by Jones and Pearson (1969) and contribute in validating the anthropometric method for partitioning the volume of the leg into six segments which are comparable to truncated cones. Subcutaneous fat volume was estimated by subtracting the muscle plus bone value from the total leg volume as described under Appendix 8. There were no significant differences in thigh fat for the subjects (Pre: $0.88 \pm 0.27$ vs Post: $0.90 \pm 0.20$ litres) whereas controls did reveal a significant increase over the 12 week period (Pre: $0.81 \pm 0.27$ vs Post: $0.93 \pm 0.32$ litres; p<0.05).

Particularly relevant to the purpose of this study was the significant increase in the thigh volumes as calculated by anthropometric techniques following 12 weeks of training (Pre: $10.5 \pm 1.5$ vs Post: $10.6 \pm 1.6$ litres; p<0.05). These differences were supported, and confirmed, by water displacement volumes (Pre: $10.7 \pm 1.7$ vs Post: $11.0 \pm 1.7$ litres; p<0.05; Table 6.9). The slightly higher water volumes could be accounted for by the fact that the SCATT would be included in these measurements. There were no changes for the controls, either in
### TABLE 6.8
RELATIONSHIP BETWEEN SKINFOLDS AND ULTRASOUND

**SUBJECTS**
(n = 24)

*(MEAN ± SD)*

<table>
<thead>
<tr>
<th>Site</th>
<th>Skinfolds (mm)</th>
<th>Ultrasonics (mm)</th>
<th>Corr.Coeff. (r)</th>
<th>Significance (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anterior Thigh</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>13.7</td>
<td>6.6</td>
<td>0.77</td>
<td>NS</td>
</tr>
<tr>
<td>SD</td>
<td>3.9</td>
<td>1.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>12.5**</td>
<td>6.00**</td>
<td>0.70</td>
<td>NS</td>
</tr>
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<td>SD</td>
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<tr>
<td><strong>Posterior Thigh</strong></td>
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<td>5.5**</td>
<td>0.59</td>
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<tr>
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</tr>
<tr>
<td><strong>Medial Calf</strong></td>
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</tr>
<tr>
<td>Pre</td>
<td>8.0</td>
<td>4.9</td>
<td>0.72</td>
<td>0.01</td>
</tr>
<tr>
<td>SD</td>
<td>2.5</td>
<td>1.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>7.2**</td>
<td>4.6**</td>
<td>0.81</td>
<td>0.01</td>
</tr>
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<td>SD</td>
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<td><strong>Lateral Calf</strong></td>
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</tr>
<tr>
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<td>4.5</td>
<td>0.76</td>
<td>0.01</td>
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<tr>
<td>SD</td>
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<td>1.1</td>
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<td></td>
</tr>
<tr>
<td>Post</td>
<td>7.5**</td>
<td>4.4 (NS)</td>
<td>0.72</td>
<td>0.01</td>
</tr>
<tr>
<td>SD</td>
<td>2.0</td>
<td>1.0</td>
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</tr>
</tbody>
</table>

Significant differences between pre- and post-measurements: * p<0.05; ** p<0.01; NS = Not Significant.

Significance (p) = between skinfolds and ultrasonics.
Skinfolds: a double layer of compressed tissue.
### TABLE 6.9

THIGH VOLUMES DETERMINED BY WATER DISPLACEMENT (WD) AND BY ANTHROPOMETRY (A)

**SUBJECTS**

(n = 24)

(Mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Leg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>18.7</td>
<td>(15.3)</td>
<td>15.2</td>
<td>0.95</td>
</tr>
<tr>
<td>SD</td>
<td>2.3</td>
<td>(2.3)</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>19.0*</td>
<td>(15.6)</td>
<td>15.4*</td>
<td>0.92</td>
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<tr>
<td>SD</td>
<td>2.2</td>
<td>(2.2)</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td><strong>Shank</strong></td>
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<td></td>
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<tr>
<td>Pre</td>
<td>8.0</td>
<td>(4.5)</td>
<td>4.7</td>
<td>0.94</td>
</tr>
<tr>
<td>SD</td>
<td>0.8</td>
<td>(0.8)</td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>8.0</td>
<td>(4.6)</td>
<td>4.7</td>
<td>0.94</td>
</tr>
<tr>
<td>SD</td>
<td>0.8</td>
<td>(0.8)</td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td><strong>Thigh</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Pre</td>
<td>10.7</td>
<td></td>
<td>10.5</td>
<td>0.91</td>
</tr>
<tr>
<td>SD</td>
<td>1.7</td>
<td></td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>11.0*</td>
<td></td>
<td>10.6*</td>
<td>0.87</td>
</tr>
<tr>
<td>SD</td>
<td>1.7</td>
<td></td>
<td>1.6</td>
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</tbody>
</table>

Significance (p): differences between Water Displacement (WD) and Anthropometry (A).

Measurements in parentheses: water displacement after calculated volume of foot (1/2 l x b x h) has been removed.

Differences between pre- and post-measurements: * p<0.05
anthropometric volume calculations (Pre: 11.0 ± 2.1 vs Post: 11.1 ± 2.2 litres) or water displacement volumes (Pre: 11.3 ± 2.2 vs Post: 11.4 ± 2.3 litres), over the 12 week period.

Following training, there was an increase in the forces generated by the right and left legs of the experimental subjects when measurements were totalled and compared at every second over the 20s MVC (Figure 6.12, Figure 6.13 and Table 6.10). This meant that 36 measurements out of the combined total of 40 measurements of both right and left leg tests showed increases. However, statistically significant increases (both \( p<0.05 \) and \( p<0.01 \)) were revealed by only 47.5% of the measurements, 3 by the right leg and 16 by the left. The controls revealed no such increases (Figure 6.14 and Figure 6.15). The dominant leg for both subjects and controls was the right: 13 for the experimental subjects, 9 for the controls. There was no change in the torque (length of moment arm [m] x Newtons) of either leg of the subjects after training (Appendix 8A) during the first second of the tests, nor in those of the controls.

It was apparent that forces generated by the right leg of the controls (Figure 6.14) during the first second were greater than those of the subjects (416N vs 393N). This was reflected in a greater torque (Pre: 128.3 ± 29.9Nm vs Post: 127.6 ± 36.4Nm) compared with those shown by the experimental subjects (Appendix 8A). Correcting for the controls' greater body mass (Table 6.1) still produced a higher quotient than that of the subjects (Pre: 1.69 ± 0.34Nm vs Post: 1.69 ± 0.47Nm). Left leg forces of the controls, on the other hand, were very similar to those of the experimental subjects at Pre: 116.3 ± 32.3Nm vs Post: 115.7 ± 33.5Nm measures (cf. Appendix 8A). The rate at which peak force was achieved did not vary significantly between pre- and post-training values for either subjects or controls but the controls were faster, averaging 1.4 s for both legs with the experimental group at 1.6 s. When the fatigue index was calculated:

\[
\text{Fatigue Index} = \frac{\text{Peak Force} - \text{End Force}}{\text{Peak Force}} \times 100
\]

there was a significant average percentage decrease for both right (Pre: 23.3% vs Post: 17.1%; \( p<0.01 \)) and left (Pre: 23.7% vs 16.0%; \( p<0.01 \)) legs of the experimental subjects, before and after training respectively, with no change for either leg of the controls.
TABLE 6.10

FORCES GENERATED DURING A TWENTY SECOND ISOMETRIC CONTRACTION OF THE QUADRICEPS MUSCLES PRE- AND POST-TRAINING
(Forces in Newtons ± SD)

SUBJECTS
(n = 24)

RIGHT LEG

<table>
<thead>
<tr>
<th>Time (s)</th>
<th>PRE</th>
<th>POST</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>393.1 ± 78.7</td>
<td>382.9 ± 75.6</td>
<td>NS</td>
</tr>
<tr>
<td>5</td>
<td>332.4 ± 86.1</td>
<td>346.2 ± 74.4</td>
<td>NS</td>
</tr>
<tr>
<td>10</td>
<td>318.7 ± 86.4</td>
<td>330.3 ± 77.0</td>
<td>NS</td>
</tr>
<tr>
<td>15</td>
<td>307.4 ± 79.2</td>
<td>333.4 ± 67.2</td>
<td>**</td>
</tr>
<tr>
<td>20</td>
<td>303.1 ± 81.3</td>
<td>316.3 ± 62.0</td>
<td>NS</td>
</tr>
</tbody>
</table>

LEFT LEG

<table>
<thead>
<tr>
<th>Time (s)</th>
<th>PRE</th>
<th>POST</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>387.4 ± 84.2</td>
<td>386.2 ± 68.2</td>
<td>NS</td>
</tr>
<tr>
<td>5</td>
<td>329.5 ± 87.9</td>
<td>346.3 ± 70.2</td>
<td>NS</td>
</tr>
<tr>
<td>10</td>
<td>306.1 ± 88.8</td>
<td>335.5 ± 69.1</td>
<td>**</td>
</tr>
<tr>
<td>15</td>
<td>302.0 ± 90.3</td>
<td>320.1 ± 67.4</td>
<td>*</td>
</tr>
<tr>
<td>20</td>
<td>297.0 ± 84.8</td>
<td>325.3 ± 67.4</td>
<td>**</td>
</tr>
</tbody>
</table>

Significant differences between means: * p < 0.05; ** p < 0.01; NS = Not Significant.
Figure 6.12  Time-related forces (N) generated by the subjects pre and post-12 weeks during right leg extension.

Figure 6.13  Time-related forces (N) generated by the subjects pre and post-12 weeks during left leg extension.
Figure 6.14 Time-related forces (N) generated by the controls pre and post-12 weeks during right leg extension.

Figure 6.15 Time-related forces (N) generated by the controls pre and post-12 weeks during left leg extension.
6.8 DISCUSSION

The key finding of this study was that, following maximum voluntary isometric contractions, the non-specific form of submaximal training resulted in, virtually a 50% increase in the forces generated by the thigh muscles. This increase in strength 'per se' and strength endurance in particular, could account for some of the peripheral influences which enabled more work to be accomplished at the same RPE following training.

When heart rate was plotted as a function of power output during the initial, graded familiarisation test of this study there appeared to be no indication of a limitation of cardiovascular delivery of blood to the working muscles since the relationship was found to be linear. This was equally true both before and after training except that for the latter, there were significant reductions in heart rate at the same absolute exercise intensities. These differences ranged from 6 beat.min\(^{-1}\) at the first exercise intensity to around 17 beat.min\(^{-1}\) at the seventh exercise intensity. A similar, linear pattern was seen during the Power Lactate Test accompanied by a reduced heart rate following training from 161 to 147 beat.min\(^{-1}\) at the fourth exercise intensity. Such results are typical for both healthy individuals and coronary artery disease (CAD) patients (Wilmore et al., 1980; Hagberg, 1986). The reduction of the resting heart rate by 6 beats (p<0.01; Table 6.2) is also redolent of characteristic improvements elicited by training in healthy individuals. Such beneficial changes in the resting heart rate are attributed to altered autonomic nervous system activity, particularly to an increase in parasympathetic tone and possibly, to a reduction in the intrinsic rate of the sinus node (Hagberg, 1986) as well as to increased stroke volume (Kanstrup and Ekblom, 1978; Seals et al., 1984).

These central adaptations have been closely identified with the peripheral changes in skeletal muscle oxidative capacity with evidence in support of a 'muscle - heart' reflex (Hollander and Bouman, 1975). The development of such a centripetal reflex implies a reversal of the chain of command proposed earlier (Edwards, 1981). Not only could it provide a reciprocal inhibition of the heart but may be considered as a potent stimulus in modifying the RPE following training. The brain may provide the initial stimulus but may receive 'command' signals from the muscles to assess and modify its scale of effort in the light of afferent impulses from the periphery. All the loads of T16, for example, showed decreases (p<0.01, Table 6.3) in RPE following training whereas the controls revealed no such consistent differences.

It was considered feasible to utilise an MVC test in order to determine some of these peripheral adaptations and identify improvements in strength as a result of: increased hypertrophy, possible recruitment of new motor units and changes in resistance to fatigue in
the quadriceps muscles. The specificity of the submaximal form of training would appear to detract from the validity of such testing yet almost any strength training method has been found to result in an increase in muscle strength but only if the frequency of exercise and loading intensities are progressively raised (Komi, 1986). The varied training regimens of the experimental subjects and, in particular, the progressive increase in exercise intensity from 66 - 83% HRmax. reserve during the training period would appear to meet the required criteria. In addition, stimulation of the three main types of motor unit would be likely to occur including fast oxidative glycolytic (Type Ila) fibres as an evolution of both the training and age of the subjects (Edstrom and Larsson, 1987).

Inherent within the training-induced aerobic adaptations already outlined is the development of a more dense capillary bed (Cotter and Hudlika, 1976; Ingjer, 1979) indicative of an improved blood supply to all fibre types. During maximal voluntary contractions, blood flow will be occluded, but this does not eliminate the contribution of muscle fibres which have been developed as a result of anaerobic - aerobic metabolism through training. Nor does it diminish the possibility of a greater residual blood volume within the maximally contracted muscle, better able to enhance the muscle's buffering capacity and whose responsibility is to sequester and remove potentially harmful protons (Burke et al., 1990). Evidence for the existence of a greater buffering capacity is suggested from the results of T16 with significant decreases in lactate concentrations over the three of the four exercise intensities (Appendix 7, Figure 1). Such an endogenous mechanism, developed through training, could, it is suggested, alleviate the negative effects of acidosis on maximal, isometric force generation not least through the calcium ion binding qualities of muscle (Hultman et al., 1981) on the contractile process itself. Moreover, the similar relative work intensities of 63 and 65% VO2max., achieved before and after training at the same lactate concentration of 4 mmol.l⁻¹ (Figure 6.2a), appears to establish a desirable working equilibrium between lactate production and lactate clearance - a helpful environment for the muscle at any training intensity, regardless of the age of the subjects.

Even so, the question may not arise as to whether the buffering system has been enhanced through training when as short a maximum voluntary contraction as 20 seconds is utilised since the buffering mechanism may not need to be effective until after the conclusion of the contraction. Some authors (Keul, 1973; Astrand and Rodahl, 1986) consider that the anaerobic energy production is quantitatively predominant for exercise lasting up to about 20 seconds. This would emphasise the source of metabolism from the high-energy phosphate pool with accelerated contributions of the Embden-Mayerhof pathway (McGilvery, 1973) but not from oxidative phosphorylation sources. This might pre-suppose that only fast glycolytic
(Type IIb) fibres would be involved in such contractions. Yet biopsy studies have revealed (Burke et al., 1971; Renstrom, 1981) that the maximal voluntary strength of human skeletal muscle is related to fibre composition in only a minor way. Rather, total cross-sectional area is generally agreed to be the major factor (Steinhaus, 1955; Shephard, 1987; Klausen, 1990).

It was not possible to carry out invasive measurements in the present study. Instead, previously validated anthropometric and water displacement techniques (Jones and Pearson, 1969) provided very high correlations between thigh volumes determined by these two methods (Table 6.9). In order to resolve some of the controversy over the choice between skinfold and ultrasonic measurements in carrying out the anthropometric calculations (Haymes et al., 1976; Borkan et al., 1982; Jones et al., 1986) both techniques were applied, the ultimate choice resting with the ultrasonics as providing more directly available data. Results from these measurements showed that the proportional relationship between percentage increase in volume and percentage increase in force over the twelve week training period was 1:7.6%. This compares favourably with studies where isometric training over the same training period produced a 1:7% relationship between increases in percentage muscle cross-sectional area and percentage isometric force (Lindh, 1979; Jones and Rutherford, 1987).

Dynamic training, also over twelve weeks, has produced a similar effect, for although strength gain was less than with isometric training, it was still greater than the change in muscle area (Ikai and Fukanaga, 1970; Young et al., 1983; Jones and Rutherford, 1987). Thus, the present finding helps to confirm those studies where an increase in strength was greater than could be accounted for by a change in the volume/cross-sectional area of the muscle. Moreover, it does so for a group of men whose age range extended to almost 60 years.

Findings from concentric, dynamic training regimens of short duration (Horber et al., 1985; Jones and Rutherford, 1987) additionally showed small, but consistent, increases in the radiological density of muscle. One of the key reasons for this, it has been suggested, would be a decrease in the fat content of the muscle. It would not be unreasonable to suppose that the reduction in skinfold, supported by ultrasonic, measurements (p<0.01; Table 6.8) in the present study, could reflect concomitant decreases in fat within the muscle itself. A consequence of such decreases would be an increase in the force per unit area/volume.

Figures 6.12 and 6.13 show a reduction in the Fatigue Index by 6.2% for the right, and 7.7% for the left, leg compared with the pre-trained state. This would suggest a greater contribution from the more fatigue-resistant Type I and IIA fibres resulting in a slowing of relaxation (Jones, 1981) though some authors believe there are no clear differences in maximum isometric stress between fast glycolytic and slow oxidative, muscles (Alexander and Vernon, 1975).
However, it would be logical to assume that a greater proportion of fast glycolytic (Type IIb) fibres would be associated with the initial dynamic contraction during the first few seconds of the test where no changes were observed following training (Appendix 8A).

Thereafter, it could be speculated, Type I and IIa fibres would tend to predominate. The higher frequency fatigue demonstrated pre-training, has been related to impaired membrane excitation, particularly associated with an accumulation of K+ or, conversely, a depletion of Na+ (Bezanilla et al., 1972) in the inter-fibre space, or especially in the extracellular fluid contained in the transverse tubular system (Bigland-Ritchie et al., 1979). It would seem axiomatic that the longer the tetanic contraction, the greater the increase in K+ concentration and the longer the decay time (Hnik et al., 1976). The higher K+ concentrations are believed to have profound effects on neuromuscular transmission and on muscle metabolism (Hnik and Vyskocil, 1981).

The muscle spindle as the controlling mechanism between the alpha motor neurone and the gamma loop system (Green, 1974) would appear to be especially vulnerable. The threshold to mechanical stimuli of both nuclear bag and nuclear chain spindles (Kidd et al., 1971; Matthews, 1972), together with Paciniform corpuscles (Akoyev and Elman, 1974) is lowered when K+ is increased. This means that the sensitivity of these encapsulated mechanoreceptors is enhanced and non-proprioceptive afferents responsible for cardiovascular, respiratory and pain stimuli, are rapidly affected (Hnik et al., 1969; Rybicki et al., 1985; Tallarida et al., 1985). It is a well known fact that substances which have been found to give rise to pain when they are injected into the blood supply of a muscle, do not do so when they are injected directly into the body of the muscle (Moore et al., 1934). It would appear as though a barrier (Kidd and Charlesworth, 1972), perhaps that provided by the capsule of the muscle spindle itself, could prevent an inhibitory concentration of K+ from accumulating in the immediate vicinity of the receptor ending as a direct result of the adaptation provoked by training. The result could be, not only a greater generation of force per se but an increased ability to tolerate fatigue and a reduction of the fatigue index as illustrated in Figures 6.12 and 6.13. The range of physiological responses following training would therefore be part adaptation, and part tolerance.

Pain can arise in muscles subjected to severe and prolonged activity, and it arises earlier in untrained than in trained subjects (Kidd and Charlesworth, 1972). Furthermore, if a muscle is strengthened by training, a given tension can be developed at a smaller fraction of its maximum voluntary force (Shepherd, 1987). As a working hypothesis, it could be argued that the barriers which reduce the rate of K+ accumulation in the effective environment of a receptor ending not only provide a contributory reduction in the rate of fatigue during a maximum voluntary isometric contraction but convey afferent impulses capable of reducing
the sense of pain, or stress, at a given absolute power output. These changes could, in some measure, account for the ability of the present group to work at a 50% increase in power output (Figure 6.3) without any change in the rate of perceived exertion following twelve weeks of training.

SUMMARY
A variety of anthropometric techniques have proved to be equally valid in determining changes in thigh volumes related to a submaximal form of training of increasing intensity. The resulting training-induced adaptations of the peripheral musculature generated increased force during maximum, voluntary isometric contractions and these changes have been found to be equivalent, in part, to that induced by weight, and isometric, training. Increases in muscle strength was, surprisingly, greater than could be explained by increases in muscle size alone but this was found to be consistent with current findings and has been attributed to rapid adaptations within the muscle during the first 6 - 12 weeks of training. Various stimuli that could account for these adaptations and which were relevant to the findings of the study, were considered. They included mechanical stress and changes in the electrolyte flux associated with the integrity of the muscle spindle and subsequent contractile force of the extrafusal fibres. It was hypothesized that, in relationship to the sense of stress, afferent impulses from the peripheral tissues may well provide centripetal, controlling pathways which could modify the generally accepted centrifugal chain of command. If valid, such a mechanism could help to explain the alleviation of the sense of stress when subjects work at the same absolute power output following short term training.

CONCLUSION
The results have revealed training adaptations at a peripheral level to complement those identified centrally where a 30 min endurance test was used. Increases in maximum forces of the thigh reflect changes in muscle morphology which appear to be associated with changes in a sense of stress. As such, peripheral adaptations of this nature may influence central - cardiac behaviour by, for instance, lowering heart rate. They may also, by impinging on the sensorium of the brain, be capable of manipulating a single RPE so that 50% more work is accomplished following training before its particular sensitivity is reached. The more conservative viewpoint is that improvements in the peripheral musculature are simply one of a number of a 'gestalt' of influences on a single Rate of Perceived Exertion.
CHAPTER 7

7. TRAINING STUDY 2.

Living systems are worn out by inactivity and developed by use.
A. Szent-Gyorgyi (1942).

7. A SINGLE PERCEPTION OF STRESS AS A DETERMINANT OF TRAINING-INDUCED CHANGES AMONG MIDDLE-AGED MALE AND FEMALE SUBJECTS.

7.1 INTRODUCTION.
In order to overcome some of the major shortcomings of Training Study 1 (TS1) as outlined at the conclusion of Chapter 6, a second Training Study (TS2) was carried out. It included females as a means of examining gender-related comparisons of responses to submaximal work intensities (Flint et al., 1974; Drinkwater, 1984) with those of males. It also incorporated modifications within the 30 minute test protocol which enabled loads to be readjusted during the testing itself, dependant on the subject’s fluctuating sense of fatigue and in keeping with the requirement to maintain a Rate of Perceived Exertion (RPE) of 14. This was a refinement not available during TS1. In addition, the pre-training loads of this same test were repeated, immediately post-training. It was believed that, by this modification, physiological adaptations bestowed by the short-term training regimen, could be more closely monitored and more clearly delineated by being compared with the pre-training ‘threshold’ values. Finally, in order to demonstrate the influence of diet, as measured rather than controlled, on the various types of blood lipid concentrations (Beaumont et al., 1970), all subjects were required to complete a seven-day weighed food history (Marr, 1971).

The purpose of this second training study, therefore, was to examine the responses of male and female subjects to a single Rate of Perceived Exertion of 14 by means of:-

a) Adaptations in functional capacity as revealed by The Power Lactate Test and The 30 min Ergometer Test and as related to power output and cardio-respiratory responses before and after 12 weeks of training.
b) The influence of metabolic, hormonal, blood lipid and dietary responses before, and following, the 12 week training regimen and hence on the resulting RPE.

7.2 METHODS.
7.2.1 SUBJECTS.
Forty-four subjects entered the study, 16 males with mean ages (±SD) of 45.6 ±7.3 years, 14 females aged 42 ±6.6 years and 14 controls (7 males and 7 females) aged 40 ±7.5 years. Ages ranged from 30 - 60 years for the subjects and 27 - 53 years for the controls (Table 7.1). As with TS1, they were recruited from academic, technical and related staff of two local institutions of higher education, and from professions and businesses within the town of Loughborough. Initially, subjects were either sedentary or participated in physical activity on a recreational basis only. Prior to training, both subjects and controls attended the laboratory in the post-absorptive state, at least 2 hours after their most recent meal and before completing a familiarisation test. They were weighed and measured; resting blood pressure and resting electrocardiograms were also taken together with ventilatory measures and percentage body fat. Details of the methodology can be found in Chapter 3.

7.2.2 PROTOCOL.
TRAINING PROGRAMME.
The subjects trained for a total period of 12 weeks. They exercised regularly at least three times a week for 30 min on each occasion, details being recorded in a training diary. One of the training sessions was carried out under supervision in the laboratory on a cycle ergometer (Monark, Type G1H). For the remaining two sessions, subjects were permitted to choose their own form of aerobic training. Such activities as brisk walking, jogging, cycling, swimming, aerobics and rowing ergometry were included. Subjects were encouraged to increase the frequency of their weekly training if they so wished. Every three weeks the training intensity in cycle ergometry was increased according to the Karvonen formula (Chap.3, Section 3.4).

As a required part of the training, two field tests were obligatory in the early days of training and at its conclusion. They consisted of a 2 Mile (3219 metre) Track Run (after Bland, 1982) and a Multistage Fitness Test (MFT) which is a 20 metre indoor shuttle-run test (Brewer et al., 1988). The 2 Mile test was 'modified' in that, to the initial study of 32 subjects, the data of 31 further subjects were added (Figure 1, Appendix 9). This provided a regression equation of:

\[ V_{O_2\text{max}} = 94.25 - (3.14 \times \text{2 Mile time}) \]

with a strong, negative correlation between maximum oxygen uptake (VO₂max.) and performance time \( r=-0.85; p<0.01 \). Both tests provided criteria by which each subject could readily determine the level of his, or her, fitness and training since, from each test, maximum
TABLE 7.1
Physical, and Ventilatory, Measures of Subjects and Controls, pre- and post-training.
(Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Males</th>
<th>Females</th>
<th>Control (M)</th>
<th>Control (F)</th>
</tr>
</thead>
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<tr>
<td>n</td>
<td>16</td>
<td>14</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>45.6 ± 7.3</td>
<td>42.0 ± 6.9</td>
<td>41.9 ± 7.0</td>
<td>38.0 ± 8.7</td>
</tr>
<tr>
<td>Range</td>
<td>(36 - 60)</td>
<td>(30 - 52)</td>
<td>(33 - 53)</td>
<td>(27 - 53)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>177.8 ± 5.2</td>
<td>162.3 ± 5.7</td>
<td>173.3 ± 9.1</td>
<td>162.9 ± 6.0</td>
</tr>
<tr>
<td>Weight (kg) Pre</td>
<td>76.9 ± 7.9</td>
<td>63.3 ± 9.7</td>
<td>67.9 ± 12.4</td>
<td>61.4 ± 6.5</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>75.8 ± 7.9</td>
<td>63.0 ± 9.7</td>
<td>68.1 ± 12.7</td>
</tr>
<tr>
<td>Quetelet Index (kg.m².m) Pre</td>
<td>24.4 ± 2.6</td>
<td>23.9 ± 2.6</td>
<td>22.6 ± 3.2</td>
<td>23.1 ± 1.8</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>24.4 ± 2.6</td>
<td>24.0 ± 2.8</td>
<td>22.6 ± 3.1</td>
</tr>
<tr>
<td>Adiposity (%) Pre</td>
<td>20.4 ± 4.1</td>
<td>34.5 ± 3.6</td>
<td>16.8 ± 4.6</td>
<td>29.6 ± 8.1</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>19.6 ± 4.1</td>
<td>32.5 ± 3.8</td>
<td>16.3 ± 4.7</td>
</tr>
<tr>
<td>Vital Capacity (L) Pre</td>
<td>5.1 ± 0.8</td>
<td>3.6 ± 0.7</td>
<td>4.9 ± 0.7</td>
<td>3.9 ± 0.4</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>5.0 ± 0.9</td>
<td>3.7 ± 0.5</td>
<td>4.9 ± 0.6</td>
</tr>
<tr>
<td>FEV₁ (litres) Pre</td>
<td>4.2 ± 0.7</td>
<td>3.1 ± 0.7</td>
<td>4.1 ± 0.6</td>
<td>3.3 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>4.1 ± 0.8</td>
<td>3.2 ± 0.5</td>
<td>4.2 ± 0.5</td>
</tr>
<tr>
<td>%FEV₁ Pre</td>
<td>83.0 ± 4.3</td>
<td>85.0 ± 6.4</td>
<td>84.0 ± 1.8</td>
<td>83.6 ± 6.0</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>82.9 ± 4.8</td>
<td>86.1 ± 4.9</td>
<td>85.3 ± 2.9</td>
</tr>
<tr>
<td>Peak Flow (L.min⁻¹) Pre</td>
<td>627.0 ± 62.6</td>
<td>478.9 ± 68.3</td>
<td>603.6 ± 48.2</td>
<td>487.4 ± 88.5</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>644.8 ± 72.2</td>
<td>510.1 ± 47.9</td>
<td>593.6 ± 58.2</td>
</tr>
</tbody>
</table>

Significant difference between data: * p<0.05; ** p<0.01
oxygen uptake could be predicted. Also track times and number of shuttle runs could be compared.

**TESTING.**

Both subjects and controls were required to complete a battery of laboratory tests before, and immediately after, the twelve week training period. The tests comprised both physiological and psychological responses in order to determine the effects of training. Changes in metabolism were measured by means of a Power Lactate Test of 20 min for males (T20) and 16 min for females (T16). Exercise intensities (5 for men, 4 for women) were selected for each individual on the basis of the previously completed familiarisation test, and were progressively raised every 4 min.

The 30 min endurance test was utilised in order to determine adaptations in functional capacity, metabolism, hormonal and blood lipid responses. The subjects were provided with the additional facility of being able to raise or lower the power output during the test itself, depending on the fluctuating sense of stress as determined by the RPE of 14. The initial, pre-training, test was known as T1 and, with its identical power output, was repeated immediately following training by each subject and identified as TR. A resetting of the power output, using the same RPE, was then carried out. This final, re-rated, test was completed in the light of the subject's newly-acquired fitness and was known as T2.

Details of test protocols, methods of analysis and statistical treatment can be found in Chapter 3, Sections 3.3-3.6.

**7.3 RESULTS.**

There were no significant changes in age, height, body weight or Quetelet Index for either the experimental group or the controls following the 12-week training period (Table 7.1), though there were decreases in percentage body fat for both male and female subjects (p<0.01). Both groups fell within the 'ideal' index quotient and this was typical of British middle-aged subjects (Rosenbaum et al., 1985) though the range (20.1-29.2 kg.m²) included both 'lean' and 'overweight' in all groups. There were no changes in the Ventilatory Measures of Vital Capacity, FEV₁ and %FEV₁ but data was above the predicted norm of 77% (Lowe et al., 1968) in %FEV₁, averaging 85%. Peak Flow, however, increased for the experimental groups: p<0.05 for males, p<0.01 for females. Resting diastolic blood pressure decreased for the experimental group following training (p<0.05; Table 7.2) whilst maximum oxygen uptake increased by an average of 11.7% (6.2% males, 17.2% females; Appendix 9, Figures 2 and 3) based on the improvements in the 2 mile track run and MFT (Figures 7.1 and 7.2).
**TABLE 7.2**

Physiological responses pre- and post-training.  
*(Mean ± SD)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Males</th>
<th>Females</th>
<th>Control (M)</th>
<th>Control (F)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>16</td>
<td>14</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td><strong>Haemoglobin (g.dl⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>13.8 ± 1.0</td>
<td>12.5 ± 0.9</td>
<td>13.6 ± 1.0</td>
<td>11.5 ± 1.9</td>
</tr>
<tr>
<td>Post</td>
<td>14.2 ± 0.5</td>
<td>12.6 ± 0.7</td>
<td>14.5 ± 2.3</td>
<td>12.7 ± 0.9</td>
</tr>
<tr>
<td><strong>Haematocrit (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>41.8 ± 4.7</td>
<td>37.0 ± 4.4</td>
<td>40.5 ± 5.4</td>
<td>35.1 ± 4.8</td>
</tr>
<tr>
<td>Post</td>
<td>42.7 ± 2.7</td>
<td>38.8 ± 3.3</td>
<td>42.3 ± 3.3</td>
<td>37.9 ± 4.0</td>
</tr>
<tr>
<td><strong>Blood Pressure (mm.Hg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>133 ± 12</td>
<td>131 ± 17</td>
<td>133 ± 11</td>
<td>128 ± 10</td>
</tr>
<tr>
<td>Post</td>
<td>130 ± 11</td>
<td>124 ± 11</td>
<td>131 ± 13</td>
<td>124 ± 9</td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>85 ± 11</td>
<td>86 ± 14</td>
<td>78 ± 12</td>
<td>79 ± 7</td>
</tr>
<tr>
<td>Post</td>
<td>80 ± 9 *</td>
<td>78 ± 6 *</td>
<td>79 ± 9.</td>
<td>77 ± 7</td>
</tr>
<tr>
<td><strong>R (Resp. Exch. Ratio)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>0.90 ± 0.2</td>
<td>0.93 ± 0.1</td>
<td>0.91 ± 0.2</td>
<td>1.08 ± 0.3</td>
</tr>
<tr>
<td>Post</td>
<td>0.89 ± 0.1</td>
<td>0.90 ± 0.1</td>
<td>0.90 ± 0.1</td>
<td>0.91 ± 0.1</td>
</tr>
<tr>
<td><strong>Heart rate (beat.min⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Rest)</td>
<td>62 ± 8.0</td>
<td>63 ± 9</td>
<td>60 ± 8</td>
<td>63 ± 13</td>
</tr>
<tr>
<td>Post</td>
<td>59 ± 8.0</td>
<td>60 ± 8</td>
<td>61 ± 9</td>
<td>58 ± 12 *</td>
</tr>
<tr>
<td><strong>Maximum Oxygen Uptake (VO₂)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(l.min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>3.37 ± 0.6</td>
<td>1.68 ± 0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>3.53 ± 0.6 **</td>
<td>2.01 ± 0.3 **</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(ml.kg.min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>43.8 ± 7.5</td>
<td>26.5 ± 7.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post</td>
<td>46.5 ± 7.2 **</td>
<td>31.9 ± 7.6 **</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Significant difference between data: * p<0.05; ** p<0.01
Figure 7.1 Two Mile performances of the Experimental Groups.

Figure 7.2 Multistage Fitness Test results of the Experimental Groups.
The male group lowered their 2 mile time from 16.1 to 15.2 min. (p<0.01), the females from 21.7 to 19.9 min. (p<0.01); the shuttle run number for the MFT was increased from an average of 62.6 to 73.9 by the males (p<0.01), 35.7 to 42.3 by the females (p<0.01). Correlation coefficients between the 2 mile run and the MFT pre- and post-training for the males was 0.92 and 0.93 and for the females, 0.91 and 0.95 respectively. These improvements reflected the dose-response effects of the training intensity: 66-85% for the male group and 67-76% for the females over the training period as monitored by the Karvonen (1957) heart rate data (Table 7.4). Work capacity in terms of energy expenditure increased within the 'moderate work' range (5-7.4 kcal.min⁻¹) for the female group, and within the 'very heavy work' category (>10 kcal.min⁻¹) for the male group (Table 7.4; Durmin and Passmore, 1967).

The Power Lactate Tests (T16 and T20).

When the power lactate tests were repeated following training there were decreases (p<0.05 and p<0.01; Appendix 9, Figures 4 and 5) in all lactate concentrations for the experimental groups with no changes for the controls. During the final power output for the male group, concentrations decreased from 7.5 mmol.L⁻¹ pre-training to 5.6 mmol.L⁻¹ post-training (p<0.01); the same power output for the females showed a concentration of 6.3 mmol.L⁻¹ pre-training reduced to 4.3 mmol.L⁻¹ post-training (p<0.01). At a common reference point of 4 mmol.L⁻¹ of lactic acid, exercise intensities improved by 23W for the male group following training (141 to 164W) and by 18W for the females (79 to 97W). When data were expressed in terms of relative work intensity (Figures 7.3 and 7.4) the female group worked at 66% VO₂ max. pre- and post-training, the male group at 58% pre- and 61% VO₂ max. post-training at the 4 mmol.L⁻¹ lactate reference level.

At each power output for both experimental groups, %VO₂ max. was lowered following training (Table 7.3c) and this was matched by significant decreases in Rates of Perceived Exertion (Table 7.3b). No similar changes were found among the controls except at the single, initial load for the female group. Correlation matrices also revealed very high relationships between RPEs. and other parameters of stress for both experimental subjects and controls (Table 7.3a). The 4 mmol.L⁻¹ lactate level was found to occur at the same RPE of 13.2 for both males and females. However, whereas the males exercised at the same relative intensity of 60% VO₂ max. both pre- and post-training at this rating (Figure 7.5a), the females exercised at 75% VO₂ max. pre-training and 66% VO₂ max. post-training (Figure 7.5b).

The 30 Minute Ergometer Test.

The relationships in functional capacity between the three 30 min tests are shown in Table 7.5. For the male group, at the same RPE of 14, the trained subjects established an increased
TABLES 7.3 a) and b)

20 min. (T20) Male and 16 min. (T16) Female Lactate Tests.

a) Rate of Perceived Exertion (RPE) correlated with other parameters of stress.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>HR</th>
<th>VO₂</th>
<th>VE</th>
<th>[La]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>Pre</td>
<td>0.996</td>
<td>0.994</td>
<td>0.991</td>
</tr>
<tr>
<td>(n = 16)</td>
<td>Post</td>
<td>0.999</td>
<td>1.000</td>
<td>0.995</td>
</tr>
<tr>
<td>Females</td>
<td>Pre</td>
<td>0.993</td>
<td>0.996</td>
<td>0.984</td>
</tr>
<tr>
<td>(n = 14)</td>
<td>Post</td>
<td>0.992</td>
<td>0.999</td>
<td>0.996</td>
</tr>
<tr>
<td>Controls</td>
<td>Pre</td>
<td>0.999</td>
<td>0.997</td>
<td>0.988</td>
</tr>
<tr>
<td>Males</td>
<td>Post</td>
<td>0.991</td>
<td>0.995</td>
<td>0.991</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>Females</td>
<td>Pre</td>
<td>0.990</td>
<td>0.995</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>Post</td>
<td>0.997</td>
<td>0.991</td>
<td>0.971</td>
</tr>
</tbody>
</table>

b) RPE at each of the workloads pre- and post-training:

<table>
<thead>
<tr>
<th>Load 1</th>
<th>Load 2</th>
<th>Load 3</th>
<th>Load 4</th>
<th>Load 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td>Pre</td>
<td>8.9 ± 1.3</td>
<td>10.6 ± 1.2</td>
<td>12.1 ± 1.3</td>
</tr>
<tr>
<td>Males</td>
<td>Post</td>
<td>7.9 ± 1.3 **</td>
<td>9.2 ± 1.4 **</td>
<td>11.1 ± 1.2 **</td>
</tr>
<tr>
<td>Females</td>
<td>Pre</td>
<td>9.0 ± 1.1</td>
<td>10.9 ± 1.0</td>
<td>12.6 ± 1.5</td>
</tr>
<tr>
<td>Males</td>
<td>Post</td>
<td>7.1 ± 1.1 **</td>
<td>9.2 ± 1.4 **</td>
<td>11.4 ± 1.5 **</td>
</tr>
<tr>
<td>Controls</td>
<td>Pre</td>
<td>8.6 ± 1.4</td>
<td>10.3 ± 1.3</td>
<td>12.3 ± 1.1</td>
</tr>
<tr>
<td>Males</td>
<td>Post</td>
<td>8.9 ± 1.6</td>
<td>10.3 ± 1.8</td>
<td>12.1 ± 1.6</td>
</tr>
<tr>
<td>Females</td>
<td>Pre</td>
<td>8.6 ± 1.3</td>
<td>10.6 ± 1.1</td>
<td>12.9 ± 0.9</td>
</tr>
<tr>
<td>Males</td>
<td>Post</td>
<td>7.3 ± 0.5 *</td>
<td>10.6 ± 1.1</td>
<td>13.1 ± 1.2</td>
</tr>
</tbody>
</table>

Significant difference: * p <0.05; ** p <0.01
TABLE 7.3 c)

20 min. (T20) Male and 16 min. (T16) Female Lactate Tests.

c) %VO2max. at each workload, pre- and post-training:-

<table>
<thead>
<tr>
<th>Load</th>
<th>Subjects</th>
<th>Pre</th>
<th>Load 2</th>
<th>Load 3</th>
<th>Load 4</th>
<th>Load 5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre</td>
<td>34.7%</td>
<td>41.4%</td>
<td>52.4%</td>
<td>64.9%</td>
<td>72.7%</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>28.4%</td>
<td>35.5%</td>
<td>46.4%</td>
<td>58.0%</td>
<td>69.5%</td>
</tr>
<tr>
<td></td>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre</td>
<td>38.6%</td>
<td>50.6%</td>
<td>68.9%</td>
<td>86.2%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>30.6%</td>
<td>41.4%</td>
<td>55.5%</td>
<td>68.6%</td>
<td></td>
</tr>
</tbody>
</table>
Figure 7.3 and 7.4 Relationships between lactates and %VO₂max before and after training for both males and females.
Figure 7.5  
a) Relationships between %VO$_2$max. and RPE as determined by the reference lactate level (Males).

b) Relationships between %VO$_2$max. and RPE as determined by the reference lactate level (Females).
power output, 38.2W higher (p<0.01) than pre-training when completing T2, a 28.5% increase (Figure 7.6). A similar pattern was seen with the female group, though at lower absolute exercise intensities (Figure 7.7); T2, re-rated at an RPE of 14, was established 28W higher than T1, a 48% increase in power output (p<0.01). These increases in power output for T2 were matched by increased: heart rates (p<0.05; Figures 7.8 and 7.9), pulmonary ventilation (p<0.01) and oxygen uptake (p<0.01). The TR revealed the classic decreases in these parameters, except for oxygen uptake which, generally, remained unchanged (Appendix 9, Figures 6 and 7). For the controls there were no changes in exercise intensities following the 12 week period nor in any of the parameters of functional capacity comparable with the experimental groups, that is: heart rates, pulmonary ventilation or oxygen uptake.

Before training the 4-Way ANOVA revealed no differences in power output between the experimental group and the controls following T1. After training, the group interaction differed \( (F_{1,32}= 18.43; p<0.001) \) as a result of the higher choice of exercise intensities by the subjects. Heart rates between groups differed post-training \( (F_{6,192}= 6.58; p<0.01) \): over the 30 min period of the test, the heart rates of the experimental group increased whilst the controls remained unchanged. The older age groups (40-50yr and 50-60yr) showed higher heart rates over the test period, post-training, than the younger group (30-40yr) and the controls \( (F_{12,192} = 3.72; p<0.001) \) whose heart rates did not change after the 12 week period. After training, the female group displayed a greater increase in heart rates than their control counterparts but not so great as the male experimental group \( (F_{6,192}= 3.30; p<0.01) \). There were increases in pulmonary ventilation between the experimental groups compared with the controls, post-training \( (F_{1,32}= 5.46; p<0.05) \) and the male group displayed an increase over the test period, post-training \( (F_{5,160}= 6.44; p<0.001) \), not shown by the females. Similarly, over the time course of the test itself, the controls showed increases in oxygen uptake not revealed by the experimental groups \( (F_{5,160}= 3.50; p<0.05) \). Between pre- and post-training measurements, however, oxygen uptake increased for the experimental group \( (F_{1,32}= 9.49; p<0.01) \) whilst the controls were unchanged.

7.4 DISCUSSION.

a) Adaptations In Functional Capacity.

The main finding of this second study was that, following the stipulated training, both male and female middle-aged subjects increased their work capacity by an average of 40% when exercising at a single RPE of 14. This corroborated the findings of the first training study. However, equally important was the finding that, by including a repeat 30 minute test immediately following training at the same power output as before training, it was possible to detect a 2 point discrimination to account for the above improvement in power output. The
sensitivity of the changes during the three 30 minute tests, particularly following training, were found to equate closely with a common relative exercise intensity which was sufficiently consistent among both male and female subjects to suggest a %RPE 14 as a working model.

The Physical Characteristics of the Subjects.

A comparison between the physical characteristics of the present study and of Training Study 1 (TS1, Chapter 6) revealed an homogeneity of the men involved in both experimental groups and controls. There were similar decreases in percentage body fat (p<0.01) and comparable patterns in ventilatory measures (cf. Tables 6.1 and 7.1) and physiological responses (cf. Tables 6.2 and 7.2) following training. Both male and female groups were taller and heavier than their 'reference' (Behnke, 1969; Behnke and Wilmore, 1974) values (Male: 151 cm and 70 kg; Female: 142 cm and 57 kg) and these criteria would place all experimental subjects in the 'overweight' category, providing finer discrimination than the Quetelet Indices. However, a summary of data from earlier female studies provides mean values (Height: 164±0.9 cm; body mass: 61.5±1.4 kg) to which the female group more closely conform (Profant et al., 1972; Drinkwater et al., 1975; Voigt et al., 1975; Hossack et al., 1982; Hudson, 1991). In terms of percentage body fat, the values before training for both males (>20%) and females (>30%) classified them as 'obese' according to some authors (Davidson et al., 1979; MacKeen et al., 1982) and this rating was not improved after training for the females, though there was a decrease (p<0.01) in their percentage body fat. Unfortunately, height and weight tables and 'norm' values fail to take into account the body compositional changes with age. With bone mineral and muscle mass losses after 30-40 years in sedentary subjects, the decrease in lean body mass and increase in fat mass tend to be masked by the total increase in body mass. For instance, one of the few longitudinal studies of sedentary, middle-aged subjects over a 12 year span (32-44yr) revealed a gain of 6.4 kg and more than an equivalent gain in body fat (Males: 16-22%) (Chien et al., 1975). The comparatively high percentage of body fat among the present female experimental group, can be detected by comparison with their control counterparts (Table 7.1).

In absolute values, pre-trained females were carrying 21.8 kg of fat which was reduced to 20.5 kg post-training (p<0.01) whilst males carried 15.7 kg and 14.9 kg (p<0.01) respectively. The implications of these data were that weight-supported exercise, such as that of the laboratory testing and training on the cycle ergometer, did not disadvantage the females whereas the field tests favoured the males with their larger Fat Free Mass (Pre: 61.2 kg. Post: 60.9 kg vs. Females: Pre: 41.5 kg. Post: 42.5 kg). The stable weights, combined with decreases in percentage body fat suggest a shift in body composition (Getchell and Moore, 1975). The increase in Lean Body Mass could represent muscle hypertrophy for both males.
and females as a result of the training regimen and as noted in similar studies elsewhere (Pollock et al., 1971; Kollias et al., 1973).

Testing and Training.

An unforeseen disadvantage of the Multistage Fitness Test (MFT) for both males and females from among the older age groups, was the discomfort experienced by some subjects in negotiating the unsympathetic change in direction at each end of the 20 metre course. Because of this, and despite significant improvements in both 2 mile run times and MFT scores (Figures 7.1 and 7.2), the former data were used to predict VO$_2$max values (Appendix 9, Figure 2).

The validity of the 2 mile run as a predictor of VO$_2$max appeared to be justified in comparison with results from the literature where sedentary subjects were trained over a short term period. This is particularly true when the combined pre-trained, male and female, results are considered: 35.2 ml.kg.min$^{-1}$. This would place the data within one standard deviation of the age curve value of 39 ml.kg.min$^{-1}$ (Robinson, 1938; Ribisl, 1969; McDonough et al., 1970) and, more recently, that of 36 ml.kg.min$^{-1}$ of Hossack and Bruce (1982). Yet it is obvious that the initial male values of TS2 at 43.8 ml.kg.min$^{-1}$ (Table 7.2), were considerably higher than those of other investigators whose subjects were found to have a mean of 30.7±4.5 ml.kg.min$^{-1}$ (Naughton and Balke, 1964; Cureton and Phillips, 1964; Adams, 1972) and compared with TS1 at 39.9 ml.kg.min$^{-1}$ (Table 6.2). This higher critical threshold of physical activity may have made it more difficult to obtain the substantial gains reported by others after training (Saltin et al., 1968: 35% and 96%; Saltin, 1969: 18%) and helps to illustrate the maxim that the degree of improvement in aerobic power is inversely related to the subject's initial level of fitness. The 6.2% increase in VO$_2$max of the males, post-training, for instance, compares unfavourably with the 14.3% increase by the males in TS1 (Chapter 6) and the 17.2% increase by the females.

The pre-training mean (±SD) VO$_2$max of 26.5(±7.6)ml.kg.min$^{-1}$ (Table 7.2) of the female group was lower than the 29.2(±2.5)ml.kg.min$^{-1}$ of six comparable studies (Drinkwater et al., 1975; Plowman et al., 1979; Hossack et al., 1981; Hossack and Bruce, 1982; Notelovitz et al., 1986; Notelovitz et al., 1988) but a 'physical activity homeostasis', through training, restored the balance with a mean of 31.9(±7.8)ml.kg.min$^{-1}$. The low pre-training value was skewed by the fact that three subjects registered measurements less than 20 ml.kg.min$^{-1}$ and one less than 15 ml.kg.min$^{-1}$. These low values could be owing to a multiplicity of inter-related factors, not least, some women's 'distaste for straining themselves physically' (Astrand, 1956). Two of
### TABLE 7.4

12 WEEKS OF TRAINING

Workrate, Energy Expenditure and Training Intensity.

*(Mean Values)*

<table>
<thead>
<tr>
<th></th>
<th>WEEKS 1 - 3</th>
<th>WEEKS 4 - 6</th>
<th>WEEKS 7 - 9</th>
<th>WEEKS 10 - 12</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males (n = 16)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Workrates (W)</td>
<td>130.7</td>
<td>151.9</td>
<td>169.6</td>
<td>187.8</td>
</tr>
<tr>
<td>VO₂(l.min⁻¹)</td>
<td>2.05</td>
<td>2.38</td>
<td>2.46</td>
<td>2.72</td>
</tr>
<tr>
<td>kcal.min⁻¹</td>
<td>10</td>
<td>12</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>kJ.min⁻¹</td>
<td>43</td>
<td>50</td>
<td>52</td>
<td>57</td>
</tr>
<tr>
<td>% Intensity</td>
<td>65.5%</td>
<td>69.7%</td>
<td>76.4</td>
<td>84.7%</td>
</tr>
<tr>
<td><strong>30 min. Training.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>kcal.</td>
<td>308</td>
<td>357</td>
<td>369</td>
<td>408</td>
</tr>
<tr>
<td>kJ.</td>
<td>1287</td>
<td>1494</td>
<td>1544</td>
<td>1707</td>
</tr>
<tr>
<td><strong>3x Week Training.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>kcal.</td>
<td>923</td>
<td>1071</td>
<td>1107</td>
<td>1224</td>
</tr>
<tr>
<td>kJ.</td>
<td>3860</td>
<td>4481</td>
<td>4632</td>
<td>5121</td>
</tr>
<tr>
<td><strong>Females (n = 14)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Workrates (W)</td>
<td>63.0</td>
<td>81.8</td>
<td>93.6</td>
<td>103.0</td>
</tr>
<tr>
<td>VO₂(l.min⁻¹)</td>
<td>1.08</td>
<td>1.18</td>
<td>1.43</td>
<td>1.50</td>
</tr>
<tr>
<td>kcal.min⁻¹</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>kJ.min⁻¹</td>
<td>23</td>
<td>25</td>
<td>30</td>
<td>31</td>
</tr>
<tr>
<td>% Intensity</td>
<td>66.6%</td>
<td>70.6</td>
<td>72.1%</td>
<td>75.5%</td>
</tr>
<tr>
<td><strong>30 min. Training.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>kcal.</td>
<td>162</td>
<td>177</td>
<td>215</td>
<td>225</td>
</tr>
<tr>
<td>kJ.</td>
<td>678</td>
<td>741</td>
<td>898</td>
<td>941</td>
</tr>
<tr>
<td><strong>3x Week Training.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>kcal.</td>
<td>486</td>
<td>531</td>
<td>644</td>
<td>675</td>
</tr>
<tr>
<td>kJ.</td>
<td>2033</td>
<td>2222</td>
<td>2693</td>
<td>2824</td>
</tr>
</tbody>
</table>

1 kilocalorie = 4.184 kilojoules.
Training intensity determined using formula after Karvonen et al., (1957).
Energy expenditure calculated on the basis of: 1 litre of oxygen = approx. 5 kcal/21 kJ.
(Benedict and Cathcart, 1913; McArdle et al., 1986; Shephard, 1987).
5.0 - 7.4 kcal.min⁻¹ (Moderate work); >10 kcal.min⁻¹ (Very Heavy work). (Durnin and Passmore, 1967).
the three women possessed percentage body fat well above the average for the group at 37.7% and 37.6%; another was overweight, whilst the 46% improvement in VO\textsubscript{2}max. of one 50 year old female over the training period (13.9-20.3 ml.kg.min\textsuperscript{-1}) suggests a 'postmenopausal syndrome' as a possible cause. This is the phenomenon where postmenopausal women have been found to improve their VO\textsubscript{2}max. by a larger percentage over premenopausal women when trained at the same time (Gill et al., 1984; White et al., 1984; Cowan and Gregory, 1985; Notelovitz et al., 1985). Even so, such low initial measures have been accounted for by the American Heart Association (1972) which places them in the 'Poor' category (<17 ml.kg.min\textsuperscript{-1}) and they are not unusual in specific studies and classifications (Notelovitz et al., 1986). After training, two of these subjects improved sufficiently to be classified in the upper ranges of 'Fair' (17-23 ml.kg.min\textsuperscript{-1}), the third into 'Average' (24-30 ml.kg.min\textsuperscript{-1}) whilst the group, as a whole, was rated 'Good' (31-41 ml.kg. min\textsuperscript{-1}).

Both resting heart rates and resting blood pressures were reduced following training (Table 7.2) and these beneficial changes may be owing to the ameliorating effect of training on the ageing process itself. Male heart rates fell 3 beat. min\textsuperscript{-1} but this was not so large as the 6 beat. min\textsuperscript{-1} (p<0.01; Table 6.2) of the males of TS1 and could reflect the difference in initial baseline levels of fitness before training commenced. Resting heart rates of the female experimental group fell by only 2 beat. min\textsuperscript{-1} following training but this was offset by the 6 beat. min\textsuperscript{-1} (p<0.05) reduction of the female controls.

**Age Groups.**

That the older age groups between 40-60 years showed higher heart rates than the 30-40 year-old age groups when completing T2, is an interesting phenomenon. The decrease in heart rate with age has been found to be similar for the sedentary person, the average performer and the top athlete (Sagiv, 1993). Peripheral vascular resistance is also increased owing to a decrease in the elasticity in the walls of both the heart and arteries as well as increased stiffness and fibrosis within the aortic and mitral valves. This will elevate afterload and impair left ventricular emptying so that the workload of the myocardium will be increased with a consequent reduction in stroke volume (Lakatta, 1986; Fleg, 1992b; Sagiv, 1993). The combined, and anticipated, decrease in maximal heart rate and exercising stroke volume reduces cardiac output, so limiting work capacity. For a given absolute power output, the heart rates of older age groups can be expected to be higher than younger groups as has been illustrated by T2 of this, and T2 of the first, training study (Chapter 6). These changes in compliance may also partially explain the distinctive 'cardiovascular drift' of the males of both studies.
The most consistent reduction in diastolic blood pressure which was not mimicked by the controls, displayed as much as 5mm decreases in pressure for this study (p<0.05) and for the males of TS1 (p<0.01). These findings match those of earlier studies (Choquette and Ferguson, 1973; Tipton et al., 1979; Hagberg et al., 1983) and could suggest an hierarchy of adaptations with the present type of submaximal, 'steady state' training and testing in which modifications to the elasticity of the arteries precede those of the compliance of the heart. A positive correlation between body fatness and blood pressure has also been reported in the literature (Montoye et al., 1965) but this was only true for diastolic blood pressure when data from both TS1 and TS2 were collated. Taken collectively, both systolic and diastolic blood pressure improved for the 54 subjects involved in TS1 and TS2:-

<table>
<thead>
<tr>
<th></th>
<th>Systolic (mm Hg)</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>132 ±13</td>
<td>87 ±11</td>
</tr>
<tr>
<td>Post</td>
<td>129 ±9</td>
<td>79 ±7</td>
</tr>
</tbody>
</table>

(p<0.05) (p<0.005)

though greater improvement occurred with diastolic pressure. This supports another study where subjects who spent more hours of their total energy output in active leisure, developed lower diastolic pressure (Montoye et al., 1972) or, at the other extreme, where ageing was found to have a pronounced influence on diastolic, but not on systolic, blood pressure following maximal exercise (Ho and Lee, 1981). The present overall, post-training blood pressure profile matches, more favourably, that found in an Anglo-American comparison (Reid et al., 1967) of 40-49 yr old Englishmen:- 127 ±18: 79 ±13 but falls short of those of subjects from other cultures, such as 47 yr old Chinese pedicab men:- 118±15: 76±9 (Systolic: diastolic; Chiang et al., 1968) or Korean diving ama:- 108±2: 71±2 (Kang et al., 1963) where chronic exercise was espoused as a lifestyle.

The Power Lactate Tests.

During the power lactate tests, blood lactate concentrations were reduced for the experimental group following training at all the exercise intensities for both male and female subjects (Appendix 9, Figures 4 and 5). These results compared favourably with the significant decreases of the second, third and fourth workloads during TS1 (Appendix 7, Figure 1) which meant that at the same power output, post-training, lactate concentrations for all subjects were reduced by approximately 1 mmol·l⁻¹. This difference could, in part, be owing to the higher VO₂max. in both training studies since it has been shown that metabolic changes, in terms of lactate concentrations, occur in relation to the %VO₂max. at which the individual is exercising (Hermansen and Saltin, 1967). Thus, at a given absolute workrate, the
# TABLE 7.5

Measures of Functional Capacity during the 30 min. test (T30), pre-training (T1), post-training at pre-training loads (TR) and post-training at new loads (T2).

(Mean ± SD)

Subjects: Males [M]: n = 16   Females [F]: n = 14

<table>
<thead>
<tr>
<th>Variable</th>
<th>Test 1 (T1)</th>
<th>Test R (TR)</th>
<th>Test 2 (T2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Workrates (W)</td>
<td>[M] 134.0 ± 31.0</td>
<td>132.7 ± 28.5</td>
<td>** 172.2 ± 35.4</td>
</tr>
<tr>
<td></td>
<td>[F] 64.1 ± 24.7</td>
<td>66.4 ± 24.4</td>
<td>** 94.9 ± 24.3</td>
</tr>
<tr>
<td>Heart rates (b.min⁻¹)</td>
<td>[M] 142.1 ± 16.2</td>
<td>** 129.7 ± 13.4</td>
<td>* 154.8 ± 9.3</td>
</tr>
<tr>
<td></td>
<td>[F] 147.9 ± 23.3</td>
<td>** 129.1 ± 24.6</td>
<td>** 159.8 ± 17.6</td>
</tr>
<tr>
<td>VO₂ (ml.kg.min⁻¹)</td>
<td>[M] 26.6 ± 5.2</td>
<td>** 24.9 ± 4.6</td>
<td>** 32.5 ± 5.4</td>
</tr>
<tr>
<td></td>
<td>[F] 17.9 ± 4.7</td>
<td>17.1 ± 4.4</td>
<td>** 23.5 ± 4.4</td>
</tr>
<tr>
<td>VE (L.min⁻¹)</td>
<td>[M] 50.8 ± 13.7</td>
<td>** 45.2 ± 9.9</td>
<td>** 69.9 ± 19.1</td>
</tr>
<tr>
<td></td>
<td>[F] 30.6 ± 9.8</td>
<td>** 25.7 ± 6.6</td>
<td>** 41.7 ± 11.5</td>
</tr>
<tr>
<td>R Values</td>
<td>[M] 1.01 ± 0.07</td>
<td>** 0.94 ± 0.09</td>
<td>** 1.01 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>[F] 1.01 ± 0.08</td>
<td>** 0.94 ± 0.05</td>
<td>** 0.96 ± 0.07</td>
</tr>
<tr>
<td>Oxygen Pulse (ml.br⁻¹)</td>
<td>[M] 0.19 ± 0.01</td>
<td>** 0.20 ± 0.01</td>
<td>** 0.21 ± 0.01</td>
</tr>
<tr>
<td></td>
<td>[F] 0.12 ± 0.01</td>
<td>** 0.13 ± 0.01</td>
<td>** 0.15 ± 0.01</td>
</tr>
<tr>
<td>RPE</td>
<td>[M] 14</td>
<td>*** 12.2 ± 0.7</td>
<td>*** 14</td>
</tr>
<tr>
<td>Rate Per.Ex.</td>
<td>[F] 14</td>
<td>*** 11.6 ± 0.8</td>
<td>*** 14</td>
</tr>
</tbody>
</table>

Significant difference between tests: * p<0.05; ** p<0.01
Non-parametric Sign Test: *** p<0.0001
The relative physiological stress of the activity would be reduced, post-training, and account for the lowered blood lactate concentrations.

The corollary to this phenomenon is that revealed by significant increases in exercise intensities at the same reference lactate of 4 mmol.l⁻¹: 23W(16.3%) for the males in the present study; 18W(22.8%) for the females and 29W(21%) for the males of TS1 (Chapter 6). Findings of a similar nature have been reported by other workers who have noted significant increases in the workrate corresponding to a reference lactate, or threshold, following endurance training (Williams et al., 1967; Davies et al., 1979; Sjodin and Schele, 1982; Yoshida et al., 1982). Changes in the metabolic profiles of the muscles themselves may account for these differences. It has been widely documented that endurance training can lead to an increase in skeletal muscle oxidative enzyme activity (Gollnick et al., 1973; Henriksson and Reitman, 1977; Fournier et al., 1982) and increased concentration of mitochondria (Gollnick and King, 1969; Holloszy et al., 1971). Collectively, these changes result in the ability of muscles to degrade more pyruvate oxidatively, converting less to lactate (Sjodin and Schele, 1982). Invariably, such changes are coupled with the facility of other organs and tissues to take up and oxidise lactate (Hurley et al., 1984) and for glycogen to be 'spared' through greater synthesis of fat as a metabolic substrate and as an inhibitor of glycolysis (Karlsson et al., 1974). Although falling R values reflect something of this metabolic transposition with significant decreases at the fourth power output for both males (1.1 vs. 1.0: \( p<0.01 \)) and females (1.2 vs. 1.1: \( p<0.01 \)), though not for the males of TS1 (1.1 vs. 1.1), at no time did quotients fall below unity. Given the incremental nature of the power lactate test and the experimental subjects' limited training, this was not surprising.

But whereas the males of TS1 (Chapter 6) and the females of the present study revealed no changes in oxygen uptake, the males of TS2 showed lower measures (\( p<0.01 \)) for all but the fifth power output. This could reflect an improved mechanical efficiency, a diminished aerobic intensity for a given absolute power output and less reliance on mechanisms leading to lactate production. Since heart rates were also lowered at all exercise intensities (\( p<0.01 \)), this could denote an improved stroke volume or an enhanced oxygen extracting ability by the tissues from a lowered blood volume. It could be combined with a better distribution of blood flow. It did not, however, result in a greater diminution of lactate concentration compared with other groups - that is, 1 mmol.l⁻¹ at the same absolute power output post-training (140W: males of TS1; 141W: males of TS2; 79W: females of TS2). Where it did reveal an advantage was in the final power output; although post-lactate concentrations were lowered by the same amount of 2 mmol.l⁻¹ for all groups, the males of TS2 were working at the augmented power output of 190W compared with the 178W of the males of TS1 and 98W for the females of TS2.
Comparatively, when seven, physically active female subjects (x age 20.9±2.2yr.) trained for six weeks, three times a week at 80%VO₂max and above, their final power output of 150W revealed a lowered post-training lactate difference of almost 4 mmol.L⁻¹ (Mayes et al., 1987). None of the present middle-aged groups was able to initiate such a high training intensity although they had done so by the ninth week of training. Thus, though the potential for aerobic adaptation appears to be similar, that of the younger group, possibly through more quickly generated peripheral mechanisms combined with a larger and more efficacious buffering system, was both quicker and greater.

Despite an increase in the exercise intensities at the same absolute lactate reference values, there was very little change in the %VO₂max at these exercise intensities. For the males of TS1, pre- and post-training relative work intensities were 63% and 65%VO₂max. respectively (Figure 6.2a) whilst the males of TS2 showed 58% and 61%VO₂max. respective values (Figure 7.3). The females remained at 66%VO₂max. both before and after training (Figure 7.4). This is in contrast to reports indicating a decrease in blood lactate concentration at both absolute and relative workrates following endurance training (Karlsson et al., 1972; Hurley et al., 1984; Henritze et al., 1985) but in agreement with others (Satlin et al., 1969; Yoshida et al., 1982). In both these latter studies the %VO₂max at which the 4 mmol.L⁻¹ lactate threshold occurred was unchanged at an average of 60%VO₂max. post-training, despite changes in VO₂max. and submaximal blood lactate concentrations at a given absolute workrate, as in the present data.

Younger individuals have revealed an identical relative work intensity of 65%VO₂max. pre- and post-training at the 4 mmol.L⁻¹ lactate level. This was only after six weeks of training (Mayes et al., 1987) but with an effective increase of 13% in VO₂max. So neither age, nor training in terms of enhanced percentage maximum oxygen uptake, would appear to alter the pattern of relative work intensity at the 4 mmol.L⁻¹ lactate level. A common value of 60%VO₂max. has been observed, though this must be measured against the considerable increase in power output at this intensity, post-training. The use of a single lactate reference value equally masks differing relative intensities at common exercise intensities. For example, at the fourth power output of each of the present studies, the males of TS1 were working at 74% and 65%VO₂max. pre- and post-training respectively (Figure 6.2a); the males of TS2 at 65% and 58%VO₂max. and the females at 87% and 69%VO₂max. (Figures 7.3 and 7.4). The trained state was, therefore, not merely responsible for the observed lower blood lactate level but of a lowered relative exercise intensity in completing a given task. It was mirrored by a significantly lowered sense of stress as revealed by the lowered RPE values at each of the exercise intensities, post-training (Figure 6.2b, Figures 7.5a and b).
As a model for defining a relationship between the 4mmol.l⁻¹ lactate level and RPE, the Power Lactate Test provided some interesting information. All groups, whether the males of TS1 (Chapter 6) or the males and females of the present study, were found, by extrapolation, to rate the prescribed lactate level at an RPE of 13.2. However, whereas the males of both studies exercised at 60% VO₂max. (Figures 6.2b and 7.5a) during both pre- and post-training tests in order to achieve this rating, the females exercised at the much higher intensity of 75% VO₂max. pre-training, 66% VO₂max. post-training (Figure 7.5b). Lactate production and clearance from the blood would therefore not appear to be similar for males and females in terms of relative exercise intensity in the pre-trained condition when completing a GXT. It could be suggested that training provides a useful 'tool', especially for females, in alleviating a relative exercise intensity and moving it towards a level common to both males and females. This may have greater significance for the 30 minute endurance test where ample time is available to resolve the mechanisms of lactate production and assimilation than the transience of the graded exercise test.

The 30 Minute Ergometer Test.
This training in the sensitivity of a sense of stress during various incremental exercise intensities was a valuable attribute for subjects to have acquired before setting and determining the power output for each of the two 30 minute tests. That this was a training-induced sensitivity could be suggested by the absence of any significant differences in RPE by the controls during either the Power Lactate Test or the 30 min. test, post-twelve weeks. The advantage of the TR test was that, by reproducing the pre-training power output, the training-induced adaptations could be revealed. Such adaptations were classical in form with decreased heart rates, pulmonary ventilation and blood lactate concentrations (Christensen, 1931; Astrand, 1956; Rowell, 1962; Saltin et al., 1969; Tzankoff et al., 1972). There were no significant changes in oxygen uptake except for four values for the males (Figure 6; Appendix 9). However, there was a general trend towards lower oxygen demands for both sexes during TR and this could be accounted for variously by improved mechanical efficiency (Gemmell et al., 1930; Robinson and Harman, 1941; Cotes and Meade, 1959) and the decreased oxygen cost of breathing resulting in lowered Ventilatory Equivalents as revealed by other authors (Yeng et al., 1985). This oxygen cost of breathing has been assessed at 4 ml.l⁻¹ of VE by Shephard (1966) at exhaustion. This would amount to a saving of 22.4 ml. and 19.6 ml. of oxygen for males and females respectively; even allowing for the submaximal nature of TR, this would be far in excess of anything depicted in these present results. The answer could be found in the combined fall in heart rates (13 beat. min⁻¹ for males, 19 beat. min⁻¹ for females) and pulmonary ventilation. In other words, the oxygen cost of breathing has been
Figures 7.6 and 7.7  Cumulative Average Work Rates following each of the three 30 minute endurance tests for both males and females.
reduced and the same, or a lowered, oxygen demand has been more efficiently extracted from the blood by the tissues. Whether the volume of blood passing through these tissues has been reduced, following training, is, again, a debatable point since the lowered heart rates have been balanced by an increased stroke volume as revealed by an increased (p<0.01) oxygen pulse (Table 7.5).

The male heart rates of TR are not only significantly lower than before training but in their range of 119 beat. min⁻¹ to 130 beat. min⁻¹ from 5 - 30 min of the test, approach the values of the active group of Experimental Study 2 (Chapter 5) during the test of 'Just Tolerable Discomfort': 115 beat. min⁻¹ to 123 beat. min⁻¹ (Table 5.3; Figure 5.3). A re-rating of 14 in completing T2 raised the heart rates, on average, 13 beats higher than T1 with, seemingly, no additional feelings of discomfort. Such a response supports the belief that perceived exertion is not dependent on the conscious perception of heart rate during exercise (Ekblom and Goldbarg, 1971; Davies and Sargeant, 1979). In terms of power output, the trained male subjects during T2, established a 30% increase of 40W above T1 (Figure 7.6). Interestingly, the final average power output of 172W is virtually the same as the final power output of TS1 at 173W indicating that the training effect of 12 weeks of progressively increasing intensity is similar in absolute terms despite varied threshold levels of maximum oxygen uptake. Since male subjects rated TR as 12.2 on the Borg Scale, this elevated exercise intensity of T2 amounted to a two point increase (p<0.0001) in the re-setting of the RPE following training.

A similar pattern is seen with the females though heart rates are characterised by little 'cardiovascular drift' during each of the tests except to rise slightly after 15 min. of T2 (Figure 7.9). This is in contrast to the male group where 'cardiovascular drift' was apparent after 5 min of each of the three tests (Figure 7.8) - and this despite lowered pulmonary ventilation and oxygen uptake after training when completing TR (Table 7.5). This rise in heart rate in the absence of an increased power output has been reported in studies where heart rate increases during steady-state exercise (Ekblom, 1969; Brewer, 1986). It is often regarded as a consequence of the decrease in stroke volume secondary to a peripheral displacement of central blood volume owing to thermoregulatory demand (Rowell, 1974a). It may be that the females were more adept at both the initial selection of an appropriate power output in keeping with their improved stroke volumes, and in readjusting that load during each of the tests in response to cardiorespiratory cues. The TR for the females was 20 beat. min⁻¹ below T1 with T2 varying from 8 - 12 beat. min⁻¹ above it. Between T2 and TR the differences were in the region of 28 beat. min⁻¹, comparable to the differences among the males at 26 beat.min⁻¹. However, the average female heart rates for the three tests were higher than those of the males by 17 beat. min⁻¹ (T1), 10 beat. min⁻¹ (TR) and 12 beat. min⁻¹ (T2) despite working at almost half the absolute power output of the males (Table 7.5; Figure 7.9). Power
Figures 7.8 and 7.9  Heart rates following each of the three 30 minute endurance tests for both males and females.
output for the females established T2, 29W (x 66 - 95W) higher than TR (Figure 7.7) which was rated at 11.6 on the Borg Scale. Thus a two point discrimination for females accounted for a 48% increase in power output; that for the males, a 30% increase.

Although the possession of a higher maximal oxygen uptake gave the male subjects a clear advantage over the females, the ability to sustain a high relative exercise intensity did not appear to have been influenced by gender. Following an identical twelve week regimen which resulted in a similar training status for both males and females, the relative exercise intensity was identical at 54%VO₂max. for TR. This is reinforced by several studies which have claimed that metabolic parameters measured during submaximal exercise are better predictors of endurance performance than VO₂max. (Kinderman et al., 1979; Sjödin et al., 1982). Such improved adaptations appeared to depend, in no small measure, on the ability of subjects to delay the accumulation of blood lactate at a given %VO₂max. This training-induced adaptation was initially revealed by the incremental Power Lactate Tests of this study and that of TS1 (Chapter 6), and endorsed by the lactate concentrations of TR. The %VO₂max. an individual could tolerate during each of the 30 min tests would therefore appear to be determined more by the lactate-delaying facility provided by training than VO₂max. per se.

The large differences between the mean VO₂max. values for males and females (Post-training: males 43%, or 1.5 l.min⁻¹ higher; Table 7.2) could be considered, in part, to be owing to the higher relative body fat of the females. When VO₂max. is expressed relative to lean body mass, a smaller percentage difference, or no difference at all, between the sexes has been reported (Flint et al., 1974; Astrand and Rodahl, 1977). In studies using cycle ergometry, however, such as those under consideration, the influence of body fat on performance is minimised because body weight is fully supported. Differences in VO₂max. between males and females, therefore, were mainly a function of the larger muscle mass utilised during the exercise by the significantly taller and heavier males and was reflected in terms of their increased endurance potential for 30 min of ergometry exercise. This was confirmed by the higher workrates (p<0.01) and oxygen uptake values (p<0.01) of the males compared with those of the females in completing T2 (Table 7.5). But when each group's oxygen-dependent-power output was expressed relative to the VO₂max., no significant difference was found between males and females in terms of %VO₂max., as has already been noted. Therefore, despite the males possessing higher VO₂max. values than the females, they were unable to tolerate a higher percentage of VO₂max. during T2. The poor relationships between VO₂max. and %VO₂max. during the post-training test, T2 (TS1: males- r=-0.44; TS2: males-r=-0.28; females-r=-0.75), further emphasises the fact that the ability to tolerate a high percentage of VO₂max. is independent of VO₂max. in these studies.
Particularly relevant to these findings is the knowledge that whatever physiological parameters have determined a given \( \% \text{VO}_2 \text{max} \), they have been equally efficacious in registering a common RPE in completing each of the 30 min endurance tests. Changes in the relative exercise intensity thus appear to have been capable of influencing, in some way, the individual's sense of stress. This reliance on the individual's responses is both a unique and valuable tool in designing safe training protocols for older subjects.

**SUMMARY**

The study revealed adaptations commensurate with those of Training Study 1 (Chapter 6) in following the same training period and intensity. Experimental groups, comprising both males and females, showed a 40% increase in power output following the acute training regimen. This was accompanied by classical adaptations in functional capacity and this could be observed by means of a repeat 30 minute test (TR) not available to the first training study (TS1). The model of the Power Lactate Test also revealed a training-induced delay in the accumulation of blood lactate and reductions in RPE at all exercise intensities for both males and females following training. An extrapolated RPE of 13.2 was found to be consistent with a 4 mmol.L\(^{-1}\) lactate reference level for both males and females and this was identified with a common relative exercise intensity following training. The findings of this graded exercise test anticipated the new data showing that the form and intensity of training was capable of reducing the RPE by 2 points on completion of TR. Measures of functional capacity were sufficiently sensitive to follow this pattern of change. They were aided by a test protocol in which subjects were permitted to alter power output according to the changeable sense of perception during the pre-training and final, post-training, 30 minute tests.
7b) METABOLIC, HORMONAL, BLOOD LIPID AND DIETARY RESPONSES.

7.5 INTRODUCTION.
The purpose of this section of the study was to determine the influence of the specific form of training on the carbohydrate and fat metabolism of the male and female subjects when completing the three 30 minute endurance tests. Whereas at the same absolute power output, physically fit subjects may possess the energy stores of adipose tissue at their disposal as a source of metabolic fuel, depending on the level of exercise intensity, sedentary subjects with their preferential anaerobic metabolism, have only limited access to these energy deposits (Wood et al., 1976; Enger et al., 1977; Moffat and Gilliam, 1979). In determining the relative contributions of the two fuels it would appear axiomatic that by measuring the concentrations of lactate as the result of glycolysis and glycogen degradation and of FFA as reflective of the utilisation of blood lipids, an appropriate division of energy contribution could be resolved. Unfortunately, the method of sampling at the conclusion of each of the tests in the present study, provided only limited information from which accurate calculations could be made. Nor was it possible to determine whether a concentration was measuring utilisation, or production, by the muscle. A further, inherent complexity is the inhibition that the concentration of one substrate may impose on another. However, by measuring related concentrations of catecholamines (Klepping et al., 1966; Taylor et al., 1971) and of a major gluconeogenic precursor such as glycerol (Felig et al., 1975) it was felt that a more informed deduction could be made.

7.6 METHODS.
As for section 7.2

7.6.1 BIOCHEMICAL ANALYSES.
Methods of sampling and analysing the blood lactates and glucose, plasma free fatty acids, glycerol, catecholamines and lipoproteins are described under section 3.5 of the General Methodology.

7.6.2 DIETARY ANALYSES.
The nutrient intakes of all experimental subjects and controls were assessed using the weighed inventory technique (Marr, 1971). This technique has been shown to provide a representative and valid description of an individual's nutritional intake, especially for protein, fat and carbohydrate (Adelson, 1969). Subjects completed a 7 - day dietary 'history' to assess
their normal nutrient intake and typical meal patterns, both in the early days of the study and during its conclusion. Each individual was provided with a weighing balance (Soehnle Digital Scales) and a food diary in which all food and drinks consumed over 7 consecutive days were recorded. Diets were then coded using the MAFF/MRC Food Composition Tables (Paul and Southgate, 1978) and analysed by a computerised food composition programme to establish the nutrient profile of each individual's normal diet.

7.6.3 STATISTICAL METHODS.
A 4-way ANOVA measured interactions between groups, training, gender and age for all parameters related to the 30 minute endurance tests (Section 3.6).

7.7 RESULTS.
R Values.
R values over the time of the test and between pre- and post-training tests, decreased for the experimental group but this was also true of the controls (F1,32 = 3.64; p<0.05), primarily as a result of lower values for the female controls.

Lactates.
Lactate concentrations reflected both the metabolic demands of each of the three 30 min tests and the varying degrees of stress tolerance before, and after, training. Concentrations between T1 and TR fell for the experimental group (p<0.01), only to rise above T1 levels by the conclusion of T2 (p<0.01; Table 7.6; Figures 7.10a and b). No changes were found between T1 and T2 for the controls: males 4.6 and 4.8 mmol.l⁻¹ and females 2.6 and 2.5 mmol.l⁻¹, respectively. The ANOVA showed that, though there were no differences, pre- and post-exercise, between the experimental group and the controls before training, after training the experimental group's lactate concentrations increased (F1,32 = 9.18; p<0.005) over the controls whose values did not differ from pre-training concentrations.

Glucose.
Blood glucose concentrations for the experimental group were higher than for the controls (F1,32 = 6.36; p<0.01). This was true whether pre- or post-test or pre- and post-training values were considered and averaged 5.2 mmol.l⁻¹ for the experimental subjects, 4.8 mmol.l⁻¹ for the controls. The female experimental subjects also showed higher values than the males at the conclusion of the pre-training test (Table 7.6) and compared with the controls, post-training (5.5 vs 4.8 mmol.l⁻¹, F1,32 = 6.63; p<0.01). Following training, the experimental groups revealed no changes in glucose concentrations though the 4-Way ANOVA showed that blood glucose values rose over the age range of the groups.
Catecholamines.
There were differences in noradrenaline, post-training, between experimental groups over controls (F_{1,31} = 22.99; p<0.001) and between T1 and T2 for both males and females (p<0.01; Figure 7.11a). TR values were unchanged for the males compared with T1 but were lower for the females (p<0.05). Adrenaline concentrations were higher as a result of training between the experimental groups and the controls (F_{1,31} = 59.26; P<0.001). This was owing to stepwise increases for TR and T2 over T1 concentrations by the males (Figure 7.11b), whereas females remained unchanged following each of the three tests.

FFA and Glycerol.
Both FFA and glycerol concentrations showed similar profiles (Figures 7.12 and Appendix 9A, Figure 1). There were decreases from T1 to TR by the females (p<0.01) and increases between T1 and T2 (p<0.01) and from TR to T2 (p<0.01) for both males and females. The effect of the tests themselves on FFA concentrations was significant for both experimental groups (F_{1,31} = 75.12; p<0.01).

Cholesterol and Lipoproteins.
No changes were found in plasma total cholesterol (Table 7.6; Appendix 9A, Figure 3) nor in HDL-C or LDL-C subfractions. However, an increase in the HDL-C moiety for the 50 - 60 year old age group was discovered (F_{2,31} = 3.76; p<0.05) but this finding was inconclusive in view of the small sample size of the controls.

Dietary histories.
The seven-day weighed dietary histories revealed no changes in basic nutrients but an increase in total energy intake was found among males over females (11 vs 7 MJ; F_{1,26} = 17.45; p<0.001) between the pre- and post-training values (Appendix 9A, Figure 2). The results also showed no percentage daily energy changes in either protein, fat, carbohydrate or alcohol (Figure 7.13). However, a difference in protein (F_{1,26} = 5.62; p<0.05) as a result of gender, pre- and post-training was found with males at 15.5% and females at 16.7%. These values were considered high compared with the National Advisory recommendation of 11% (NACNE, 1983). There was also a difference in carbohydrate as a result of gender (F_{1,26} = 5.53; p<0.05) attributed to the lower percentage of the male controls (45.2%) compared with the female controls (48.5%).
### TABLE 7.6

Glucose, lactate, hormonal and blood lipid values following each of the 30 minute endurance tests including lipoprotein concentrations pre- and post-training.

**Mean ± SE<sub>M</sub>**

<table>
<thead>
<tr>
<th>Metabolite</th>
<th>Test 1 (T1)</th>
<th>Test R (TR)</th>
<th>Test 2 (T2)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glucose</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>5.02 ± 0.23</td>
<td>4.96 ± 0.22</td>
<td>5.48 ± 0.22</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>5.20 ± 0.24</td>
<td>5.13 ± 0.23</td>
<td>5.48 ± 0.30</td>
</tr>
<tr>
<td><strong>Lactates</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>4.09 ± 2.20</td>
<td>2.95 ± 1.52</td>
<td>6.51 ± 4.02</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>3.38 ± 1.63</td>
<td>2.02 ± 0.66</td>
<td>4.95 ± 1.97</td>
</tr>
<tr>
<td><strong>Adrenaline</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(nmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>0.61 ± 0.07</td>
<td>0.95 ± 0.20</td>
<td>1.10 ± 0.24</td>
</tr>
<tr>
<td>(nmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>0.67 ± 0.11</td>
<td>0.59 ± 0.18</td>
<td>0.61 ± 0.08</td>
</tr>
<tr>
<td><strong>Noradrenaline</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(nmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>5.20 ± 1.05</td>
<td>4.10 ± 0.43</td>
<td>8.50 ± 1.28</td>
</tr>
<tr>
<td>(nmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>3.50 ± 0.64</td>
<td>2.60 ± 0.33</td>
<td>5.30 ± 0.69</td>
</tr>
<tr>
<td><strong>FFA</strong></td>
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<td></td>
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<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>0.44 ± 0.05</td>
<td>0.36 ± 0.03</td>
<td>0.50 ± 0.05</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>0.60 ± 0.17</td>
<td>0.36 ± 0.06</td>
<td>0.66 ± 0.07</td>
</tr>
<tr>
<td><strong>Glycerol</strong></td>
<td></td>
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<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>0.42 ± 0.20</td>
<td>0.32 ± 0.13</td>
<td>0.43 ± 0.15</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>0.25 ± 0.04</td>
<td>0.15 ± 0.02</td>
<td>0.28 ± 0.03</td>
</tr>
<tr>
<td><strong>Total Cholesterol</strong></td>
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<td></td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>5.46 ± 0.25</td>
<td>NS</td>
<td>5.65 ± 0.31</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>5.28 ± 0.27</td>
<td>NS</td>
<td>5.14 ± 0.20</td>
</tr>
<tr>
<td><strong>HDL-C</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>0.64 ± 0.08</td>
<td>NS</td>
<td>0.67 ± 0.08</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>1.05 ± 0.07</td>
<td>NS</td>
<td>0.97 ± 0.11</td>
</tr>
<tr>
<td><strong>LDL-C</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[M]&lt;/sub&gt;</td>
<td>4.82 ± 0.22</td>
<td>NS</td>
<td>4.98 ± 0.28</td>
</tr>
<tr>
<td>(mmol.l&lt;sup&gt;-1&lt;/sup&gt;)&lt;sub&gt;[F]&lt;/sub&gt;</td>
<td>4.24 ± 0.23</td>
<td>NS</td>
<td>4.11 ± 0.19</td>
</tr>
</tbody>
</table>

*M = Male.  F = Female.
NS = Not Significant.
Significance of difference between tests: * p <0.05; ** p.<0.01
Figures 7.10 a) and b) Lactate concentrations at the conclusion of each of the three 30 minute endurance tests for both males and females.
7.8 DISCUSSION.

The new findings of this section of the study are that, by improving the protocol to include a repeat 30 min test (TR) following training at pre-training power output levels and by enabling the power output to be adjustable during the test itself, it has been possible to identify a more sensitive contribution of blood lipids. With lactate accumulation serving as a possible inhibitor and increasing noradrenaline concentration as an equally powerful stimulator of FFA, the contribution of blood lipids during TS1 (Chapter 6) was equivocal. During TS2, however, and particularly during T2, the influx of FFA into the blood appears to have exceeded the efflux resulting in a rise in concentration above the standard resting value of about 0.3 mmol.l⁻¹ (Carlson and Pernow, 1959; Figure 7.12a and b). Such a contribution may partially underly the ability of both male and female experimental subjects to exercise at a re-rated RPE of 14 despite clear indications of a dominating glycolytic metabolism mediated by increased lactate concentrations (Figure 7.10a and b). (Carlson and Pernow, 1959; Figure 7.12a and b)

Oxidative capacity.

Though the training resulted in only a modest improvement of 11.7% in VO₂max, it was within the parameters suggested by the intensity and duration of training (Ekblom, 1969; Davies and Sargeant, 1975) and may mask as much as a doubling of the oxidative capacity of the muscles (Gollnick et al., 1973). Certainly, the higher lactate concentrations of the pre-trained muscles contrasts with the lowered concentrations in the immediate post-trained state of TR (Figure 7.10a and b). It appears likely that the skeletal muscles' increased content of mitochondria, mitochondrial enzymes, capillaries and myoglobin could be responsible for this change (Holloszy and Booth, 1976; Saltin and Rowell, 1980), rather than an alleviation of the 'hypoxic' condition of the untrained muscles per se (Karlsson et al., 1972; Hagberg, 1984), the working skeletal muscles themselves probably becoming one of the major sites of lactate clearance (Stanley et al., 1986). From such improvements, the working muscles become a more effective source of lipid utilisation (Gollnick, 1977; Brooks and Fahey, 1985) particularly since, through training, the inhibitory effect of a lactate concentration of between 6 to 8 mmol.l⁻¹ of lactic acid (Issekutz et al., 1965; Boyd et al., 1974) will probably have been lifted.

Ventilation.

Interestingly, the training-induced decreases in ventilation appear to follow similar decreases in blood lactate concentrations (Tables 7.5 and 7.6). Presumably the demand for buffering bicarbonate has been reduced, compared with the pre-trained status; carbon dioxide levels would thus have been lowered and ventilation levels diminished to the 'aerobic threshold' (Kindermann et al., 1979; Sjodin and Jacobs, 1981) of the 2.0 mmol.l⁻¹ achieved by the females (Figure 7.10b). The significant relationships in the present data between changes in VE, R values, heart rates and blood lactate concentrations (Tables 7.5 and 7.6) and the
similarity of the patterns they provide, would lend support to the view of Klausen et al., (1982) that ventilation and the changes in ventilation during training are related to muscle metabolism. Since ventilation and respiration rate are readily available to conscious monitoring during exercise (Horstman et al., 1979; Pedersen and Welch, 1977), these may have provided the most dominant perceptual cues in the re-rating of TR at an RPE of 12.2 for the males and 11.6 for the females.

**Lactates.**

Whereas the reduced blood lactates for both males and females in completing TR (Figure 7.10a and b) suggest a diminution in the mechanisms leading to the production of lactic acid, the increased lactates of T2 could reflect a faster clearance as a result of a more expansive capillary bed and improved blood flow (Anderson and Henriksson, 1977; Ingjer, 1979). The lowered anaerobic metabolism of TR was confirmed by falling R values (Table 7.5) which, though not low enough to indicate that fat metabolism was being increased, yet supported the concept of a 'glucose sparing' mechanism. If blood lactate concentration of 4 mmol.l⁻¹ is a good predictor of endurance performance (Sjödin and Jacobs, 1981; Williams and Nute, 1983), then the concentrations being lowered towards the 2 mmol.l⁻¹ value after TR emphasises the highly responsive changes of lactates to training (Sjödin et al., 1982) which would have created a more aerobic environment for the exercising muscle tissue. Of course, it could be proposed that the higher lactates of T2 also revealed an imbalance between the rate of lactate production and its clearance (Donovan and Brooks, 1983; Donovan and Pagliassotti, 1990) as a result of concentrations which were continuing to rise. The methodology which enabled subjects to adjust the power output in accordance with the criterion of an RPE of 14 would tend to preclude such a proposition. It could therefore be supposed that lactate concentrations had been modified according to the set sense of stress whose intensity would be reflected in the relative work intensity and could be identified as %RPE14.

During endurance exercise such as that of a 30 min ergometer test, the accumulation of blood lactate is detrimental to performance; consequently, the ability to delay such an accumulation would be a welcome benefit. That benefit could be acquired through training and would be revealed by an alleviation of the subjective sense of stress. That TR was completed at an RPE of 12 suggests that those training-induced benefits had been achieved. It is reinforced by the fact that TR was completed at 54% VO₂max. by both males and females alike, whilst before training these relative exercise intensities were at the higher value of 62% and 68%, males to females, respectively. Thus the effect of training was to reduce the relative exercise intensity to a common level of %RPE12 when working at the same absolute power output as before
training. This is in agreement with other studies in which no differences have been found between the %VO$_2$max of males and females exercising at different exercise intensities (Hermansen and Saltin, 1967; Bland, 1982; Maughan and Leiper, 1983; Jakeman, 1986; Ramsbottom, 1986). Nevertheless, the concept that similar relative exercise intensities induce similar metabolic responses in terms of blood lactate concentrations (Hermansen and Saltin, 1967; Mayes, 1987) did not hold true in the present data where male and female blood lactate concentrations were different following TR: 2.95 mmol.l$^{-1}$ for males and 2.02 mmol.l$^{-1}$ for females (p<0.05). For T2, however, where relative exercise intensities rose to an average 72% VO$_2$max for both males and females, there was no difference in blood lactate concentrations between male and female values (Table 7.6) so data conformed to that more commonly found among the above, and more recent (Steed et al., 1994), authors. The results were also in close agreement with TS1 where male subjects produced a %RPE14 of 77% VO$_2$max in completing the post-training 30 min test (T2).

Catecholamines.

The 'steady state' nature of the three 30 minute endurance tests revealed increased concentrations of catecholamines (Figures 7.11a and b) which mirrored the changes in functional capacity as reflected by power output and heart rates (Table 7.5). The level of circulating noradrenaline exceeded that of adrenaline, on average, sevenfold and eightfold after T1 and T2 respectively, for both males and females. Following TR, however, there was a decrease to a fivefold relationship. Such a blunting of sympathetic nervous activity has previously been reported in healthy individuals (Winder et al., 1978) and has been identified as a major cause of the reduction in heart rate during submaximal exercise following training (Hagberg, 1986).

Noradrenaline, normally related to the 'spillage' from sympathetic postganglionic terminals (Rowell, 1986; Levick, 1991), has also been identified with the relatively rapid stimulation of 'hormone-sensitive lipase' and, together with adrenaline, is responsible for the initiation of lipolysis at the onset of exercise (Carlson et al., 1963; Havel et al., 1963). Without the inclusion of the repeat test, TR, the modifications bestowed by training would have been masked, as in TS1 (Chapter 6). Despite the maintenance of pre-training exercise intensities, a less intense sympathetic response has occurred in keeping with the lowered relative exercise intensity of 54% VO$_2$max, for both males and females. Similar responses have been found by earlier authors (Scheuer and Tipton, 1977) and associated with varied exercise intensities (Korge et al., 1974; Bloom et al., 1976).
Catecholamines, glucose and FFA mobilisation.

The lowered hormonal response to exercise after training is believed to be partly owing to higher circulating blood glucose concentrations (Luyckx et al., 1978). However, glucose homeostasis was maintained with test TR, levels never falling below 4.96 mmol.l⁻¹ and showing no change from the concentrations of T1 and T2 (Table 7.6) for either males or females. It may be that the levels of catecholamines, as illustrated in this study, together with glucagon concentrations, increase during exercise but that the magnitude of the increase is significantly less in endurance-trained, than in untrained, individuals (Winder et al., 1979). The close matching of the fluctuating levels of noradrenaline with power output, heart rates, pulmonary ventilation, FFA and glycerol mobilization - the latter as a valuable precursor of FFA mobilisation (Paul, 1975) - tends to confirm exercise as a valuable rate-limiting agent in lipolytic synthesis. Noradrenaline concentrations also appear to closely follow the changes in RPE which are known to be significantly correlated with such stress markers (Skrinar et al., 1983).

The disparity between male and female adrenaline concentrations (Figures 7.11a and b) would suggest that greater 'aggression' stress (Newsholme and Leech, 1983) has been imposed by the significantly higher absolute exercise intensities of the males, despite common relative exercise intensities. Yet the modest acceleration of the catecholamine response has provided a more pronounced lipolytic metabolism for the females (Table 7.6; Figure 7.12b). This would suggest that the sensitive balance between the intensity of exercise training and the enhanced mobilisation of free fatty acids into the blood stream may not be identical between male and female subjects of this age group.

Cholesterol and Lipoproteins.

Neither male nor female subjects revealed changes in total cholesterol or any of its subfractions (Appendix 9A, Figure 3) despite a pronounced training effect as identified by an increase in maximum oxygen consumption, a fall in both heart rate and minute volume at the same absolute, submaximal workrate (TR), and an average 14% improvement in training intensity. Though values fell within suggested reference ranges (e.g. 3.5-7.8 Total Cholesterol; Ball and Mann, 1988), concentrations at the lower end of the scale for TC and LDL-C would have been desirable considering the numerous epidemiological studies demonstrating an inverse relationship between HDL-C and the incidence of atherogenesis (Yaari et al., 1981; Wilson et al., 1988; Manninen et al., 1988). Females did reveal a significantly higher HDL-C (p<0.01) over the males, probably attributable to pre-climacteric advantages (Notelovitz et al., 1982) but it would appear axiomatic that, with variables such as body weight and diet remaining constant, lipoproteins, especially HDL-C, would not change (Lipson et al., 1980; Wood et al., 1988). The decrease in percentage body fat (Table 7.1)
Figures 7.11 a) and b) Noradrenaline and Adrenaline concentrations at the conclusion of each of the three 30 min. endurance tests.
Figure 7.12 a) and b) Free Fatty Acid values for both males and females following the three 30 min. endurance tests.
without any change in HDL-C confirms similar findings (Savage et al., 1986; Hagan et al., 1986) whereas the possible increase in HDL-C associated with subjects in the fifth decade of life has previously been found with age-related increases in TC and plasma lipids (Fredrikson et al., 1967; McTaggart and Ribas-Cardus, 1976). The eight comparative studies (Appendix 7A, Table 3) show that at least three studies of 12 weeks’ duration, or below, were capable of reducing total cholesterol so a clear pattern of cause and effect is far from resolved.

Whether duration, intensity, frequency, acute or chronic, forms of training and their various combinations are able to produce a desirable increase in HDL-C may be determined by elevated levels of tissue lipoprotein lipase (LPL). This enzyme is believed to adapt well-trained individuals for rapid and efficient mobilisation of lipids for energy during exercise (Taskinen et al., 1980). It may also contribute to decreased triglyceride levels since HDL-C as the carrier of apolipoprotein C-II, an activator of LPL, could be responsible for triglyceride uptake by the cell (Miller, 1979). These lipid, lipoprotein and LPL changes are recognised as chronic training effects; however, even an isolated, prolonged exercise bout may elevate postheparin LPL activity (Kantor et al., 1984). The crucial mechanisms in stimulating the cascade of these reactions could thus be the sensitive juxtaposition of the various ingredients of a training programme. Untrained subjects, for instance, walking at 30% VO2max. have revealed increases in HDL-C after an hour (Pay et al., 1992) whilst subjects at the other end of the training continuum who trained for over a year of 30 min. daily walking at 68%HRmax (Stensel, 1992) failed to elicit any changes in TC or its subfractions. Since, in the present study, lipolysis does appear to have been activated, and bearing in mind the known large intra-individual variability in measured concentrations (Demacker et al., 1982), it is suggested that the anticipated changes in lipoproteins may have been masked by progressively high exercise intensities linked to comparatively short training sessions - a combination proving inappropriate for all but the 50 - 60 year old age range. The accumulating wealth of evidence would appear to indicate that only regular, aerobic exercise, carried out as an ingredient of everyday life, can unequivocally ensure beneficial lipoprotein levels for middle-aged men and women.

SUMMARY.

By being able to adjust the power output to the fluctuating sense of stress during the 30 min test the experimental subjects were able to show a clear pattern of change. Such a pattern was characterised by comparable changes in, for example, heart rates and pulmonary ventilation supported by similar profiles in lactates, noradrenaline, FFA and glycerol. The repeat test, TR, was particularly valuable in showing changes in catecholamines which
Figure 7.13 Dietary percentages for both males and females pre- and post-training.
suggested a 'down-regulation' of physiological responses to exercise after training and with it a reduction of RPE to 12. Lower FFA and lactate concentrations during exercise after training also indicated the achievement of a more favourable match between mobilisation and utilisation, better able to contribute to an environment in which tissues were capable of increasing power output by 40% with no change in the sense of stress.

CONCLUSION.
Improvements in test protocol were invaluable in identifying some of the training adaptations which enabled more work to be accomplished after training at the same, single RPE as before training. A Power Lactate Test also served as a useful model in establishing both a desirable RPE and a relative work intensity at a lactate reference level of 4 mmol.l⁻¹. Yet this test, as a graded exercise test, revealed its limitations when compared with the principle steady-state test of 30 minutes' duration. The sensitivity of this test was enhanced by the new protocol so that each subject could maintain an RPE of 14 throughout its duration. This provided a more clear cause-and-effect determination of changes in stress following training than was possible with the first training study (TS1). It identified changes in functional capacity, mirrored by clear patterns of metabolic, hormonal and blood lipid responses among both males and females. Thus the 'tool' of training with the specificity of its intensity, frequency and duration, was able to lower the RPE of the experimental subjects following the repetition of the same absolute workload as before training and elevate functional performance when the set RPE of 14 was restored. It was not of a suitable form to alter lipoprotein concentrations favourably, despite a dietary stable background.
8. GENERAL DISCUSSION

Ratings of perceived exertion have been described as the 'single best indicator of the degree of physical strain' and provide a psychological complement to physiological responses to exercise. *Borg (1982b).*

8.1 Development of the Testing Protocol.

The progressively developmental nature of the studies of this thesis involved a total number of 133 subjects, total average age of 44.3yr. (±3.4), (Mean ± SD), range: 27-64yr. Of these, the 23 females included in the final training study averaged a slightly younger age of 40yr. (±2.8) and slightly narrower age band: 27-53yr. than the male group: 45.3yr. (±2.6), range: 31-64yr. The principal aim of the studies was to develop a suitable test which could most clearly determine differences between the trained and untrained state of such middle-aged subjects and which, consequently, could be of universal application in identifying the fitness profile of any middle-aged individual.

An initial, preliminary Experimental Study (Exp.1, Chapter 4), using a common physical work capacity of 170 beat.min⁻¹ (PWC₁₇₀), produced similar relative oxygen uptake values of 91.4% for an active group, and 89.7% for an inactive group with differences (p<0.05) of 1.2 units between RPEs. of the two groups: 17.1 (±1.4) Active, 15.9 (±2.2) Inactive. Correlations between heart rates and exercise intensities for the two groups not only proved to be highly linear (r=0.99) but differences in heart rates between the two were consistent throughout the test at approximately 12 beat.min⁻¹ for any given power output. This was interesting and useful in that differences between the trained and untrained state of middle-aged male subjects could be measured at a single power output merely by means of heart rate alone. In terms of power output, using heart rate as the dependent variable, differences were found to be 33W between the 20 active, and 20 inactive, subjects. Thus, at the same heart rate, a trained subject from this age group could be found to be working at about a 0.5 kg power output above that of his untrained counterpart. Even so, although 33W was a valuable, quantifiable criterion, it appeared to be a limited training adaptation in terms of a 'dose-response' for someone advised to adopt training as a life-style.
With this limitation in mind, a second Experimental Study (Exp.2, Chapter 5) was mounted using data from Exp.1. The graded exercise test (GXT) was replaced by two endurance tests over thirty minutes, with consideration for the prophylactic value of endurance training and testing for this age group, such as the stimulus to increase the mass and density of mitochondria in skeletal muscle, the enhancement of the oxidation of free fatty acids with their glycogen-sparing effect (Astrand, 1992) to name but two adaptations which can affect a subject's sense of stress. For the first of these tests a Pre-Set Load was extrapolated from a heart rate of 140 beat.min⁻¹ as calculated from the data of Figure 4.1 of Exp.1. This resulted in differences in actual exercise intensities (p<0.01), averaging 31.6W (159.8W Active, 128.2W Inactive) at 68.4% and 63.3% of %HRmax. reserve (Karvonen et al., 1957). Though this was no more than the difference found during Exp. 1, and helped to justify the findings of that test, it revealed that the relatively untrained individuals were able to sustain such a power output and heart rate over a thirty minute period without undue stress as revealed by an average RPE of 14 (Figure 3, Appendix 6A). The greatest differences in power output, however, were found during the second endurance test where subjects were required to chose a load equivalent to a stress experienced as 'Just Tolerable Discomfort'. With no difference in heart rates between the two groups, the active subjects were found to have completed the thirty minute test at virtually twice the power output of the inactive subjects (127W vs. 64W; Figure 5.4)

From these results, a twelve week training study was mounted using, as a focal test, a thirty-minute endurance ergometer ride whose power output was determined by means of a self-selected choice, using an RPE of 14 as capable of reproducing some of the findings of the test of 'Just Tolerable Discomfort'. The justification for such a choice of rating was manifold but was supported by the suggestions of Pollock et al., (1986b) that an RPE 'training window' between 'somewhat hard' (RPE 13) and 'hard' (RPE 15) was capable of producing work intensities of 50 - 85% VO₂max. and 60 - 90% HRmax. reserve, a recommendation approved by the ACSM (1980) and at the upper level consistent with a desirable RPE_{AT} (12 - 14; Dishman, 1986; Chapter 2, Section 2.2.2). The 'production' method delineated by Smutock et al., (1980), enabled subjects to arrive at an exercise intensity in keeping with the required rating. The choice of a single, sustainable power output over thirty minutes was a modification of the ACSM (1991) guidelines that an individual's RPE response to a graded exercise test (GXT) may be utilised in specifying the RPE level for conditioning.

The pre-training choice of power output in the present studies, once completed for 30 minutes, was then carried into the training protocol and formed the threshold level which was raised every three weeks thereafter during the training period. The method relied, not solely on the subject's perceptual response in accurately prescribing exercise intensity (Glass et al.,
1992), but explored the subject's potential in a form of autogenic prescriptive testing and training. It also examined the reputed weakness of the relationship between heart rate and RPE which has been reported to be less strong, for the maintenance of a single exercise intensity (Mihevic, 1981).

The results of the second Experimental Study (Exp. 2) revealed that a cycle ergometer test of 30 minutes duration did not disadvantage the inactive subjects, whether they were required to sustain a power output imposed upon them by virtue of a chosen heart rate or whether they were free to self-select their own power output since, in both instances, they were able to complete the tests without undue distress. Equally acceptable, the test enabled the active subjects to reveal their training-induced advantages, both physiologically and psychologically. Similar claims have been made for the GXT of Exp.1 but that study revealed that a complete comparison between the two groups, active and inactive, was not possible in view of the inactive group's inability to sustain a similar 'performance' as that of the active group. The administration of a steady-state 'endurance' test which both groups were able to complete in its entirety, would appear to be more valid and more comparable in view of the age of the subjects and in relationship to the format of their training.

A 'frame of reference' (Skinner et al., 1973) by which subjects could identify an RPE of 14 in terms of a power output suitable for their 30 min. test, was provided by a familiarisation, and a Power Lactate Test, both of which were GXTs. Although it has been stressed that subjects should be properly instructed in the use of the RPE scale (Pollock et al., 1986b) this was not done obtrusively. Subjects were left free to chose a wide range of ratings since every change in power output required an obligatory re-rating during the preliminary tests. The total number of progressive ratings could thus vary from thirteen to fifteen in number, together with the five minute trial period, pre-30 min. test, and additional re-assessments every 5 minutes (TS1), and every 3 minutes (TS2), during the 30 min. test itself.

8.2 Maximum Oxygen Uptake.

The general guideline that healthy adults should work at a recommended relative work intensity of 40 - 60% VO$_2$max. (ACSM, 1991) contains the inherent flaw in its assumption that all subjects are equally committed and sufficiently motivated to complete a VO$_2$max. test from which such a relative work intensity can be accurately deduced. This is an unwise assumption to make, particularly in terms of safety when testing a middle-aging population as in the present series of studies and especially when as many as three such tests may be necessary in order to obtain a 'true' VO$_2$max. (Lakatta, 1993). In addition, it may be extremely difficult to assess the precise fitness level of subjects who present themselves at the laboratory on the first occasion.
Figure 8.1 Changes in $\dot{V}O_2$max of the 40 male, experimental subjects according to age, pre- and post-training, from the combined training studies.
To overcome these problems, the three laboratory tests were carried out first, so ensuring a certain basic level of fitness. These were followed by specifically designed field tests for each of the training studies as outlined in Chapter 3, Section 3.4. They enabled the VO$_2$max. to be estimated more by the control of the subjects than of the tester whilst the training intensity, as determined by the work on the cycle ergometer and raised every three weeks over the 3 month training period, utilised the HR$_{max}$. reserve by means of a formula devised by Karvonen et al., (1957) and known to virtually replicate VO$_2$max. for age (Pollock et al., 1979).

As can be seen from Figure 8.1, which combines the data of both male experimental groups of each of the training studies, and Figure 8.2 of the female experimental group, there is a negative rate of decline when VO$_2$max. is plotted against age. Both figures illustrate the linear decline of VO$_2$max. typified by longitudinal studies for both genders (Asmussen and Mathieson, 1962; Adams, 1972; Notelovitz et al., 1986; Booth, 1989) though not an age-related curvilinear decline espoused by some authors (Buskirk and Hodgson, 1987; Rowe and Kahn, 1987). Nor are the numbers of female experimental subjects sufficiently large to determine whether loss of aerobic power is related to menopause 'per se', though post menopausal women (X age 57 yr.) have been found to improve their VO$_2$max. by 19%, 7% higher than premenopausal women (X age 41 yr.) when both groups trained over a 9 week period (Cowan and Gregory, 1985). Although no precise data was culled regarding the menopausal condition of the present group of females, six of them could be considered within the menopausal age band (46 - 55 yr.; Notelovitz, 1986). They all improved their VO$_2$max. values, post-training, averaging a 20.3% increase though one 51 yr. old woman revealed little post-training change (32.1 vs. 32.2 ml.kg.min$^{-1}$). Nevertheless, the overall percentage increase in VO$_2$max. by the women was 17.2% compared with a 6.2% increase by males in TS2, and a 14.3% increase in TS1 (x 10.3%).

Even a one year training protocol of moderate intensity has been known to improve the VO$_2$max. of 100 retired men by only 11% (Cunningham et al., 1987) whilst higher intensity training over 6 - 12 months produced increases of from 20 - 30% in 70 - 79 yr. old men and women (Hagberg et al., 1989; Makrides et al., 1990). The limitation with such average data is that it masks individual differences as discovered by Seals et al., (1984) and Kohrt et al., (1991). Twelve months of vigorous training of older subjects (60 - 70 yr.), whilst evoking an average increase of 30% VO$_2$max., was found to reveal a 2 - 49% range. It is apparent that the same training programme may result in almost no change in VO$_2$max. for some subjects, whilst others gain as much as 1 litre of oxygen uptake. The initial, pre-training, level can account for as much as 25% of the variance in the response of VO$_2$max. to a programme (Bouchard et al., 1992). This factor has been stressed throughout both of the present training studies; the lower the initial VO$_2$max., the greater the increase in training as illustrated by the females of TS2 (Table 7.2). Yet neither age, nor gender, have been found to be attributable to individual
differences in the trainability of phenotypes involved in endurance performance. Increasingly
greater cognisance is being given to the highly familial, and primarily genetically, determined
factors governing the individual VO_2max. profile and its sensitivity to training (Bouchard,
1986; Bouchard et al., 1988).

The percentage decrease of VO_2max. for the 30 - 60 yr. age span of the male group when the
two studies were combined (Figure 8.1), averaged 12.3% per decade in the pre-trained
condition and was very similar at 11.5%, post-training. This amounted to a loss of 0.55
ml.kg.min^{-1} per year and 0.53 ml.kg.min^{-1}yr^{-1}, pre- and post-training respectively. These
values are slightly higher than the 0.45 ml.kg.min^{-1}yr^{-1} identified by Hagberg (1987) but well
within the 1.0 ml.kg.min^{-1}yr^{-1} found by other authors (Hollman, 1966; Dehn and Bruce, 1972;
Bruce, 1984).

Female rate of decline of VO_2max. (Figure 8.2) averaged 9% pre-training, 8.8% post-training
with decrements per year of 0.24 ml. and 0.28 ml., pre- and post-training, respectively. These
data conform to both cross-sectional and longitudinal findings of a rate of decline of less than
0.30 ml.kg.min^{-1}yr^{-1} for sedentary women (Hodgson and Buskirk, 1977; Plowman et al.,
1979) whilst both male and female changes roughly agree with the 10% per decade decrease
in VO_2max. over the 30 - 70 year age span (Astrand and Rodahl, 1986). It is apparent that in
terms of both percentage decrease and decrease per ml.kg.min^{-1}yr^{-1}, the loss for males is
greater than that for females and this can be identified by the steeper slope of the regression
lines of Figure 8.1 compared with those of Figure 8.2. Even so, in absolute terms, male values
are consistently higher than those of the females and this is true when the average training
improvements of 5.1 ml.kg.min^{-1} (male) and 4.4 ml.kg.min^{-1} (female) are taken into
consideration, though a narrowing of the age-related differences can be observed as 60
years is approached.

Unfortunately, the data suffer from low correlations so that the males, for instance, with pre-
and post-training values at -0.542 and -0.563 respectively, can account for only 29% and 32%
of the variance through the age factor when considering VO_2max. Yet such data are still
above that found in similar studies (r = -0.4; Notelovitz et al., 1986; r = -0.3; Hudson, 1991).
The female data is even further handicapped by small numbers. However, the key finding with
both gender groups is that for any given age between 30 yr. and 60 yr., training can improve
VO_2max. by about 5 - 6 ml above pre-training values. If death rates are characterised by a finite
oxygen uptake, the active lifestyle typical of the form of training shown by these studies,
present a favourable risk ratio and may be regarded as capable of increasing longevity.
Despite these limitations, the comparatively close relationship of the VO_2max. data of these
studies with that found in the literature, substantiates the methods based more on field tests
Figure 8.2 Changes in VO₂ max of the 14 female experimental subjects from the second training study, pre- and post-training, according to age.
than those normally carried out in the laboratory. The greater onus on subject-controlled testing does not relieve the exercise physiologist of his responsibilities in preparing an inactive subject for the rigours of a 'modified' VO2max. test but it does share the burden of testing, biased as it is towards greater involvement and integrity of the subject.

8.3 Relative Work Intensity (%VO₂max.)

The establishment of the validity of VO₂max. by the methods used, also confirmed the authenticity of the relative work intensities from which they were derived. The RPE and %VO₂max. relationships have been identified as possible, strong central signals (Skinner et al., 1973; Robertson, 1982; Pandolf, 1983; Pandolf et al., 1984); the lactate, or reference, threshold, regarded as conveying positive, peripheral signals, is also believed to have its foundation in the same relationship (Hill et al., 1987). So a 'whole-body' perception of effort may well be associated with RPE - %VO₂max. Of particular interest in the present context of the validity of the 30 min. ergometer test, is that the reference threshold is thought to reflect the highest submaximal level of oxygen consumption that can be sustained during prolonged exercise (Dempsey and Seals, 1994). And an RPE of between 13 - 15 has been found to correspond to that reference threshold (Bellevue et al., 1983; DeMello et al., 1985) with a metabolic intensity of between 50 - 85% VO₂max. (Pollock et al., 1986b).

Such a large range of relative intensities may be more precisely identified from the Power Lactate Tests of the present studies. The lactate reference point from this test averaged 63% VO₂max. (Range 58 - 66% VO₂max.; Figures 6.2a, 7.3 and 7.4) for both TS1 and TS2, supporting the claim that RPE is related to the relative work load, irrespective of the level of conditioning (Bar-Or et al., 1972). Despite the reservation of the test as a graded exercise test, it provided a valuable benchmark by which the T30 tests could be assessed. Such a relative work intensity corroborated the trainability of the experimental groups since, at the same relative work intensity there were substantial increases in absolute exercise intensities post-training: 21% and 16% for the males of TS1 and TS2, respectively, and a 23% increase for the females of TS2 (Figures 1, Appendix 7; Figures 4 and 5, Appendix 9). Furthermore, when an average of 60% VO₂max. for males and 71% VO₂max. for females was matched with the 4 mmol.l⁻¹ lactate level, an interesting finding was that of a common RPE of 13.2 (Figures 6.2b, 7.5a and b) which identifies closely with the the guidelines of 13 - 15 of the above authors.

The combined data of the 30 min. test for the experimental groups of males from TS1 and TS2 (Figure 8.3) revealed that the pre-training test (T1) was completed at a relative work intensity of 62% VO₂max. and lactate concentration of 3.7 mmol.l⁻¹. The experimental female group (Figure 8.4) completed the same test at 68% VO₂max., producing a lactate
concentration of 3.4 mmol.l⁻¹. These relative work intensities and attendant lactate concentrations, were sufficiently close to the model provided by the Power Lactate Tests to be acceptable. The steady-state form of the 30 min. test, with greater emphasis on lactate clearance than its production (Stainsby and Brooks, 1990), would be more likely to produce lactate values below the 4 mmol.l⁻¹ level, identified graphically by the Power Lactate graded exercise test. The closely comparable lactates for both males and females suggest a common mechanism shared by both groups in completing a similar type of work. The 6% VO₂max. disparity between the males and females, pre-training, would appear to reflect more the initial choice of absolute power output by the females than differences in threshold maximal oxygen uptake values or smaller female numbers. This was emphasised by the skilful use of the testing protocol by the females during TS2, in progressively lowering the load after five minutes until the conclusion of the test (Figure 7.7). An accompanying, if delayed, oxygen uptake pattern followed that of the exercise intensities (Figure 7, Appendix 9). It would seem that the female group made an over-ambitious choice of power output from which they never quite fully recovered.

However, the influence of the powerful trinity of intensity, frequency and duration of the training regimen is apparent in the common relative intensities of the repeat test (TR) at 54% VO₂max. for both males and females and the re-rated test (T2) at 73% VO₂max. for the male, and 74% VO₂max. for the female, groups, respectively. Lactate profiles were sensitive to these relative exercise fluctuations but differed significantly between genders (TR: 3.0 mmol.l⁻¹ males, 2.0 mmol.l⁻¹ females; T2: 6.5 mmol.l⁻¹ males, 5.0 mmol.l⁻¹ females; Figures 7.10a and b). Yet such a wide range of concentrations is not unusual in either untrained or trained subjects exercising for at least 30 minutes (2 - 7 mmol.l⁻¹: McLellan and Jacobs, 1989; McLellan et al., 1991). This sensitivity was also true of pulmonary ventilation, formerly identified as an important precursor for RPE (Robertson et al., 1984). The implication would appear to be that fluctuations in RPE in completing these tests were primarily dependent on the relative work intensity which was closely mirrored by heart rates and pulmonary ventilation since these were common to both sexes.

8.4 Metabolic Responses as Factors in the Determination of RPE.

The relative work intensity profiles (Figures 8.3 and 8.4), consciously monitored by a two-point difference in RPE following training at the same absolute workrate (TR), were matched by similar profiles in lactates (Figures 7.10a and b), free fatty acids (Figures 7.12a and b) and glycerol (Figures 1a and b, Appendix 9A), and noradrenaline (Figure 7.11a). The sensitivity of these metabolic and hormonal patterns was made apparent by means of the inclusion of the repeat test, post-training, together with a test protocol which permitted subjects to readjust
Figure 8.3 Relative work intensity ($%\text{VO}_2\text{max}$) of the 40 middle-aged male experimental group in completing the 30 minute pre-training tests (62%), the repeat tests (54%) and the post-training tests (73%).
the power output every three minutes, if they so wished, depending on their sensation of the
mismatch between fatigue and RPE14.

No such advantages were available to the experimental subjects of TS1 (Chapter 6): consequently, the post-training test (T2) averaged an RPE of 14.8 (Table 6.5), a lactate concentration of 7.7 mmol.l⁻¹ and a relative exercise intensity of 77% VO₂max. As an untrained group they were more successful at arriving at an RPE closer to 14 (X 13.7), a lower concentration of lactate at 3.2 mmol.l⁻¹, and a relative work intensity of 62% VO₂max. This relative intensity was within 1% of that determined by the Power Lactate Test in establishing a lactate reference criterion at which long term endurance performance can be maintained (Davis, 1985). It is also the point at which lactate production, though increased many fold prior to the reference point being reached, is balanced, it is believed, by an almost equal increase in its rate of removal (Stainsby and Brooks, 1990). Despite these attributes of the untrained group, they were found to be working at a 50% lower absolute workload than in the trained state.

In seeking to determine the underlying causes which enabled the trained group of TS1 to maintain an average RPE close to the stipulated level of 14 at a significantly higher power output, a number of factors are worthy of consideration. Individuals endurance-trained over 12 weeks are likely to possess a greater proportion of mitochondria, an increased concentration of aerobic enzymes and a preponderance of Type I fibres which have a rich blood supply (Saltin, 1990). These adaptations allow for a greater potential for oxidative phosphorylation and an improved lactate clearance. Furthermore, endurance training decreases lactate dehydrogenase (LDH) activity (Saltin, 1990) and permits the mobilisation of fatty acids to occur (Brooks and Fahey, 1985). By virtue of the sampling techniques, taken before, and immediately following, each of the 30 min. ergometer tests, the high concentration of lactate following T2 (Figure 6.6) of TS1 (Chapter 6) may well reflect enhanced clearance rates though high R values (Table 6.5) tend to discount this assumption.

It would seem self-evident that FFA utilisation is desirable, not only in sparing glucose metabolism, but in alleviating the sense of stress and in explaining the maintenance of an RPE of 14 at an elevated power output. Unfortunately, FFA concentrations (Figure 6.7), although almost twice as high as those of the controls, were unchanged between the two tests. This was so even though noradrenaline concentration, recognised as the most powerful stimulant of FFA mobilization, was high for T2 (Figure 6.8; Isserksutz, 1964; Boyd et al., 1974). Insulin, probably the most important counter-regulatory hormone for the lipolytic process (Pruett, 1971; Bjorntorp, 1988), may also have been stimulated in the latter stages of T2 as witness the enhanced blood glucose levels. This, in turn, may have been mobilized by a combination of increased adrenaline concentration (Figure 6.8), the muscular contractions of
Figure 8.4 Relative work intensity (%VO2max) of the 14 middle-aged female experimental group in completing the 30 minute pre-training tests (68%), the repeat tests (54%) and the post-training tests (74%).
the continuing exercise (Cohen, 1981; Richter et al., 1982) and enhanced gluconeogenesis from the various glucose precursors such as the raised lactate and glycerol levels (Felig et al., 1975; Conlee et al., 1979; Issekutz, 1981; Saltin and Gollnick, 1983). All this could be supportive evidence for the sense of stress which was approaching an RPE of 16 by the conclusion of the test (Figure 6.5). With subjects working within the upper intensities of 60 - 90% VO2 max., fatigue is associated more with depleted stores of muscle glycogen (Saltin and Karlsson, 1972) rather than increased metabolism of FFA.

The lower mental stress of the tests of TS2 (Chapter 7) was reflected in the diminished adrenaline responses (Docktor and Sharkey, 1971; Frankenhaeuser et al., 1969) of the male experimental group and the low, sustained plateau level of that of the females (Figure 7.11b). When compared with TS1, these low concentrations and the conditions they reflect, would appear to be solely a factor of the improved test protocol where loads could be adjusted as the 30 min endurance test progressed in order to prevent changes in perceived exertion over time (Pandoff et al., 1972; Borg and Johansson, 1985). Rather than a two point increase in RPE during the test, as with TS1, a two point diminution was registered when the pre-training test was repeated (TR). This was invaluable in identifying the sensitivity of the associated metabolic parameters. Lactate (Figure 7.10, a and b) and noradrenaline (Figure 7.11, a) concentrations, together with relative work intensities (Figures 8.3 and 8.4) were lowered for this repeat test and these measures have been closely identified as probable indicators of changing RPEs. (Docktor and Sharkey, 1971; Young et al., 1982; Hill et al., 1987). The more sensitive and significant fluctuations in FFA and glycerol (Figure 7.12a and b, and Figure 1a and b, Appendix 9A) would suggest noradrenaline concentrations as reflecting as much a stimulation of FFA mobilization (Issekutz, 1964) as of sympathetic 'spillage' (Levick, 1991) and a function of the cardiovascular regulatory system (Docktor and Sharkey, 1971). Noradrenaline may therefore be serving a different role in each of the two studies.

Subjects in TS2, ingesting a near-normal diet (Figure 7.13) other than a slightly elevated protein intake, and engaged in progressively increasing aerobic training could be considered to be obtaining an increasing proportion of their energy from fat (Christensen and Hanson, 1939) as training continued. This would not necessarily be revealed during any of the comparatively short - intensity laboratory tests. Even during the pre- and post-training 30 min tests of T1 and T2 and the attendant relative work intensities, the high proportion of carbohydrate in the diets would tend to favour a higher contribution of carbohydrate in the energy metabolism (Bergstrom and Hultman, 1967). It is an attractive, even convenient, argument to suggest that, as a result of the manipulation of RPE which the test protocol provides, a greater contribution to metabolism is made available from lipolytic sources. However, these proposals are tinged with caution in view of R values which do not fall below
0.90 (Table 7.5) and RPE requirements which fall within the upper range of metabolic intensity (50 - 85% \( \text{VO}_{2\text{max}} \); Pollock et al., 1986b). From these arguments it should be clear that the hormonal regulation of fat and carbohydrate metabolism and their affects on RPE during exercise, depends on a very complicated balance from among many factors.

Finally, as a marker of the increased mobilization of fatty acid in the blood (Pruett, 1970) and of the possible prophylactic value (Miller and Miller, 1975; Kiens et al., 1980; Wood et al., 1983) of exercise and training, the cholesterol levels were measured together with their subfractions. The duration, length, frequency, intensity and dietary composition of a specific training regimen were examined as popular sources for such beneficent effects. Total cholesterol and its lipoprotein moieties were found to be unresponsive, however, following each of the present training stimuli. Nor does the past, or current, literature (Gulbrandsen et al., 1974; Hudson et al., 1987; Stensel et al., 1993) appear to resolve, conclusively, the controversy which exists regarding the value of training and its various close relatives in manipulating cholesterol and its constituents, favourably.

8.5 The 'Set Point' Concept.

The 30 min test protocol in its final form, as shown during TS2, has numerous advantages over other test methods in validating a 'Set Point' concept of RPE. Unlike GXTs, where spuriously high correlations may be found between heart rates and RPEs. (Borg, 1962a; Skinner et al., 1969; Bar-Or et al., 1972), the technique of reproducing a single RPE seemingly overcame this problem. It has been suggested (Skinner et al., 1973) that subjects based their RPE of a particular work load on the information obtained from previous workloads when completing a GXT. For instance, since the power output, and therefore heart rate, increased in a stepwise fashion, subjects might logically have rated in the same way. By confining subjects to a single RPE of 14, the statistical linearity normally associated with physiological responses such as those of Exp.1 and the Power Lactate Tests, could be removed. It was hypothesised that what is actually perceived and subjectively rated therefore, could be more easily identified. The factor of the duration of a 30 min. exercise test was also considered capable of influencing RPE cues more potently. After an initial, sharp rise with the onset of exercise, an asymptote could be anticipated from measures of functional capacity whilst less rapidly responding biochemical processes would have time to reveal their influences as mediators (Edwards et al., 1972; Astrand and Rodahl, 1977). These concepts were verified during TS1 from measures of oxygen uptake (Figure 2, Appendix 7), power output (Figure 6.3) and, arguably, the results of biochemical assays. Unfortunately, the key measure of the RPE which it was desirable to maintain at a constant level, was found to change (Figure 6.5) and this has been discovered in previous studies (Pandolf et al., 1972; Borg and Johansson, 1985).
This major weakness was overcome in TS2 by the simple expedient of permitting the subject to manipulate the power output during the test itself, in order to match the changing sense of stress as experienced by an increasing feeling of fatigue. Even so, a mismatch between power output and rating could still occur in the event of an early overshoot in load-setting as depicted by the females of Figure 7.7. Yet within the span of the six studies of this thesis, a test has been evolved capable of isolating a single RPE and differentiating between measures likely to have influenced such a choice of rating. The term 'Set Point' has been adopted in order to identify this choice since it embraces Borg's (1962a) own view that RPE is a 'gestalt' of many sensations, or a 'multiple integration of many factors' (Pandolf, 1982), but extends the concept to include a variability common to other set point theories. Both Keeseys (1986) set point theory of obesity and that of Hammel and coworkers' (1963) set point theory of thermoregulation, are believed to have their controlling mechanisms in the hypothalamus. 'Catch-up growth' (Marshall, 1977) can be regarded as a set point related to a child's growth and may also have its source in the nervous system (Tanner, 1963) adjacent to the anterior pituitary. Ulmer (1985) examined the 'innervation of the system' when athletes were asked to run, swim and cycle at three RPE levels. He demonstrated their skill in their ability to arrange performances according to these ratings and referred to 'set point' as the actual degree of exertion. The comparison between the actual value of exertion at any given moment and this set point degree ensured such an optimal arrangement of performances.

This 'mismatch' between two signals appears to be a common element in all concepts of set point. In the present context it is illustrated by the 30 min repeat test (TR) of TS2 by both males and females (Figures 8.3 and 8.4, respectively). Through the method of training, the RPE14 set point has been raised. In repeating the pre-training power output against a background of improved performance, the 'old' power output would appear easier and the set point consequently lower by an average of two points. Confirmation that set point is dependent on the relative work intensity and independent of the maximum oxygen uptake is provided by the set point of RPE12 at a common relative work intensity of 54% VO\(_2\)max compared with the RPE14 of T2 at a near identical value - 73% and 74% VO\(_2\)max, males to females, respectively. Thus, manipulation of the set point can be said to be mediated by training. Interestingly, this two point difference, induced by training adaptations, is similar to the findings of a GXT carried out before and after 8 weeks of training (Ekblom and Goldbarg, 1971). The RPE was 1.5 to 2 points lower (p<0.05) for 8 healthy male subjects after training (x age 24 yr.) Similarly, Linderholm (1967), in studying Swedish military conscripts before and after 4 months of training found that both heart rate and RPE were reduced by approximately 20% at the same submaximal power output following training. These comparative studies suggest that the older age groups of the present study are capable of similar physiological
and perceptual adaptations as their younger counterparts but may require more time to achieve them. It has also been proposed (Rockefeller and Burke, 1979) that the most conclusive point in favour of RPE as a measure of exercise intensity is that, following training, there is a significant decrease in RPE at a given power output. The RPE assists in adjusting the intensity to a level that is both suitable for eliciting a training effect and that is psychologically tolerable - the basis on which the present training studies were devised.

In considering the possible mechanisms underlying a new perception of the same set point following a period of training, a number of factors may be germane. Anthropometric changes, such as those of body weight and composition, can contribute to the increase in VO2max. in older men and women. Any reduction in body weight in response to such training will result in an increase in VO2max. expressed in ml.kg.min⁻¹, even if the absolute level of whole body oxygen consumption is unchanged (Ogawa et al., 1992). The subjects of both training studies following training which included non-weight bearing forms of exercise, revealed decreases in both total body mass - significantly for TS1 (p<0.05) - and in percentage body fat (p<0.01; Tables 6.1 and 7.1). Even more pertinent to the present discussion was the finding that training had increased thigh volumes (p<0.05) as determined by water displacement, anthropometric and ultrasonic measures of TS1. These changes were associated with a 48% improvement in leg extension forces (Figures 6.12 and 6.13). Thus, although increases in VO2max. have not been observed in response to resistance exercise training in middle-aged men (Hurley et al., 1984), the corollary that endurance training cannot improve muscular force does not hold true. The age-related decline in the capacity to generate force (Bassey et al., 1992; Stanley and Taylor, 1993) appears to have been delayed; a large proportion of the age-related decline in VO2max. in untrained individuals as a consequence of the age-associated loss of muscle mass (Fleg and Lakatta, 1988) would also appear to have been counteracted by the form of the endurance training and as revealed by a 20 second isometric test of the leg extensors. It is apparent that when training is of a sufficiently high intensity, the middle-aged and elderly can adapt in a manner comparable to younger age groups (Coggan et al., 1992). Regular physical activity would appear to possess the potential to attenuate the age-related deficits in skeletal muscle (Brown, 1987).

The increased capacity of muscle to produce force, combined with its facility to make better use of the oxygen delivered to it, would, it is speculated, provide an environment of diminished stress as portrayed by a lowered RPE when individuals work at the same absolute work intensity following training. Other measures of lowered functional capacity (Table 7.5) support this hypothesis. Above all, are the diminished lactate concentrations and their accompanying hydrogen ions (Figure 7.10, a and b) which may well orchestrate the 'gestalt' responsible for the 'set point' of the sensorium (Figure 8.5).
It has been proposed that, as muscles fatigue, the increased neuromotor drive necessary to maintain a constant power output is coded within the sensory cortex as a peripheral signal of exertion arising from the involved limbs (Cafarelli, 1977; Cafarelli, 1982; Kostka and Cafarelli, 1982). The lower the blood H+ concentration, the higher the pH and consequently, the lower the differentiated peripheral ratings. Experiments in acid-base shifts have emphasised that peripheral signals affecting sensations of exertion were only responsive at higher work intensities in the region of 80\% VO2max. associated with higher metabolic acidosis (Robertson et al., 1986). The mechanism probably involves a disruption of the contractile and energy properties of skeletal muscle (Paulus et al., 1974; Kostka and Cafarelli, 1982), interrupting the glycolytic production of ATP for myofibrillar contraction (Jones et al., 1977; Jones, 1980; Sutton et al., 1981). As muscles fatigue, the motor unit recruitment and firing frequency are increased, making a constant power output feel progressively harder (Cafarelli, 1977; Lollgen et al., 1980; Kostka and Cafarelli, 1982) and hence, raising the RPE as in TS1 (Figure 6.5).

Of the central signals known to be associated with perceived exertion (HR, VE, RR, VO2 and \%VO2max.), only VE is believed to be consciously monitored (Mihevic, 1981; Robertson, 1982), but only in a positive way at 80\% VO2max. when the conscious monitoring of central ventilatory signals is linked to isocapnic buffering of metabolic acidosis at exercise intensities above the lactate threshold (Robertson, 1982). Respiratory Rate (RR) appears to be the primary mechanism in signalling sensations of ventilatory exertion and discomfort by mediation of the ventilatory muscles: intercostals, diaphragm and abdominal complex. Since both central and peripheral signals of exertion are dependent upon alterations in contractile properties of skeletal muscle, both types of differentiated signals may share a common neuromotor pathway (Robertson et al., 1986) as represented by Figure 8.5.

The 80\% VO2max. is a useful criterion in the context of the present studies. Despite lactate levels being measured above the reference value of 4 mmol.l\(^{-1}\) for T2: 6.5 mmol.l\(^{-1}\) for males and 5 mmol.l\(^{-1}\) for females (Figures 7.10a and b), the relative work intensity did not rise above 74\% VO2max. and consequently did not reach the level of perception of 80\% VO2max. which would, a priori, require the RPE of 14 to be altered. In this study, the 4 mmol.l\(^{-1}\) lactate reference value has been found to occur at an average of 63\% VO2max. as determined by the Power Lactate Test. This is not in general agreement with the established literature where the relative work intensity varies from 72\% VO2max. (Belman and Gaesser, 1991), 78\% VO2max. (Yoshida, 1984) and 84\% VO2max. (Tanaka et al., 1983) at such a lactate concentration. Such discrepancies, however, may be accounted for by the lack of consistency in the protocols, the varied state of training and the ages which ranged from 20 to
Figure 8.5 Possible mediators of a sensory 'Set Point'.

Sedentary - Age - Gender - Training

CENTRAL

Cardiorespiratory

\[ \dot{V}E \]

\[ HR \rightarrow \dot{V}CO_2 \rightarrow \dot{V}O_2 \]

\[ H^+ + HCO_3^- \]

\[ pH \]

Baroreceptors \(\rightarrow\) Neural \(\rightarrow\) Chemoreceptors

SENSORIUM 'SET POINT' (RPE 14)

Extrapyramidal \(\rightarrow\) Neural \(\rightarrow\) Pyramidal

\[ pH \]

\[ H^+ + HCO_3^- \]

[ LACTIC ACID ]

Fibre Recruitment

Mechanical

PERIPHERAL

Metabolism \(\rightarrow\) Muscle \(\rightarrow\) Substrates
is lowered (Pilegaard et al., 1994) as for TR (Figure 7.10a and b) where subjects recorded the lowered set point of RPE 12.

It is an attractive hypothesis to suggest that the increased concentration in lactate as seen in T2 for both males and females provides the biochemical 'drive' to return set point to RPE14. The fact that subjects experienced no additional discomfort at this higher lactate concentration and higher relative work intensity compared with pre-training values could be explained by the more economical blood distribution which training provides. It could also prevent an imminent fall in pH by 'diluting' the acidaemia created by the muscle tissue. All this could be mediated by the known sensory mechanism of pulmonary ventilation which would alter appropriately, depending on the work intensity and level of training. This would overcome Cafarelli's (1982) reservation that there was no evidence to suggest a peripheral mechanism sensitive to pH since the role of monitoring pH would be taken over by the central ventilatory mechanism. In support of such a contention is the finding that infusion of NaHCO₃ for the correction of exercise-induced acidaemia (Paulus et al., 1974) had no effect on the subjective feelings of fatigue. This led Mihevic (1981) to the conclusion that the influence of elevated blood lactate concentration on perceived exertion is not mediated by a reduction in blood pH and that blood lactate may reflect a mediation pathway other than blood acidaemia. That pathway may well lead to ventilation. For maximal CO₂ production and pCO₂ did increase significantly as a result of the reaction between HCO₃⁻ and H⁺ during these manipulations (Paulus et al., 1974). It could be deduced that ventilation would be increased as a consequence and, by inference, support the role of pH. In passing, it should be noted that the test protocol was one of a GXT to exhaustion so there are limitations when comparing it with the findings of a 30 min steady-state endurance test.

The flow diagram of Figure 8.5 is consequently controversial in some of its suggested pathways. A resolution of the full influence of pH may only be possible from measurements of muscle pH followed by serial analysis of blood pH within the context of total blood volume. In turn, the total blood volume can only be validated with regard to the thermoregulatory conditions which it serves. Also relevant is the insulin sensitivity of ageing man. The fluctuations in insulin concentration could determine the interplay between anaerobic and aerobic metabolism and hence figure significantly in the greater or lesser production of blood lactate for a given absolute power output and, by inference, the FFA contribution, and dependent on the subject's training status. In the present context, and overlying all the parameters suspected of influencing RPE, is the difficulty in identifying which factors play the largest part. Overall is the inability, in many instances, to discriminate between the response of the body to external factors, such as reduced, or increased, activity and those that are
considered to be intrinsic changes associated only with the aging process (Cunningham et al., 1985).

CONCLUSION
The major aim of the studies presented in this thesis was to validate the findings of a 30 min endurance test using the single term of 'Just Tolerable Discomfort'. When both highly active and inactive subjects used this terminology in order to select an exercise intensity, there were no significant differences between the heart rates of the two groups but the active group chose an exercise intensity twice that of the inactive group. Two successive training studies, each carried out over 12 weeks, revealed increases of 50% and 40%, respectively, in exercise intensities. These differences had been established by means of an RPE of 14 which was designed to replicate the term, 'Just Tolerable Discomfort', used in the initial study. Such an RPE was found to be capable of monitoring adaptations in functional capacity over the training span and in revealing concomitant metabolic, hormonal and blood lipid concentrations. Such revelations were possible by means of an improved protocol enabling the RPE to be maintained at a constant level until its completion. Furthermore, by conducting a repeat test, following training, which mimicked the absolute work intensities of the pre-trained status, it was anticipated that those parameters which were most influential in determining RPE14, could be more closely identified. The most sensitive responses were those of heart rates, pulmonary ventilation, lactates and noradrenaline; they could be regarded as inter-related as proposed by a model of peripheral and central influences impinging on the sensorium of the brain. Equally sensitive, and dependant on the influence of training on the 'set point' of the RPE14, was the relative work intensity whose relativity could be more clearly understood from findings revealed by Power Lactate Tests. In addition, anthropometric analyses of changes in thigh volumes and thigh forces were invaluable in providing further data as to the possible causes of an alleviation in the sense of stress following training. For example, motor recruitment as relayed through proprioception, is known to be consciously monitored, as is pulmonary ventilation which may share similar pathways. A dietary study determined changes and contributions of macro-nutrients over the training period whilst experiments in the application of a variety of field tests were found to be valid and enabled safer methods of predicting VO₂ max to be administered. The battery of tests, developmental in nature, was able to substantiate an RPE14 as capable of monitoring age-related, and training-induced adaptations. This 'set-point' criterion of sensitivity suggested that some of the changes of the initial study had been achieved but that further progress along the continuum of training adaptation was only possible by adopting training as a lifestyle.
REFERENCES


Biomedical Data Package [BMDP] (1985) 2V Statistical Software Inc., 1440 Sepulveda Boulevard, Los Angeles, CA90025, U.S.A.

Biomedical Data Package [BMDP] (1985) 2V, Statistical Software Inc., 1440 Sepulveda Boulevard, Los Angeles, CA90025, USA.


Rowell, L. (1962) Factors affecting the prediction of maximal oxygen intake from measurements made during submaximal work with observations related to factors which may limit maximal oxygen intake (Thesis). Minneapolis: Univ. of Minnesota.


CALCULATIONS OF OXYGEN UPTAKE AND CARBON DIOXIDE PRODUCTION

WORKED EXAMPLE

AT REST: Readings - \( \text{VO}_2 = 17.5\% \) \( \text{VCO}_2 = 3.1\% \) \( \text{VE (STPD)} = 5.17 \text{l.min}^{-1} \)

- \( \%N_2 \) inspired = 100 - (20.93 + 0.03) = 79.04%
- \( \%N_2 \) expired = 100 - (17.5 + 3.1) = 79.4%

\[ \text{VI} = \text{FIN}_2\% \times \text{VE (STPD)} \]  
therefore \( \text{VI} = \frac{79.4 \times 5.17}{79.04} \text{l.min}^{-1} \)

**OXYGEN**

\( \text{VO}_2 \) inspired = \( 5.19 \times \frac{20.93}{100} = 1.086 \text{l.min}^{-1} \)

\( \text{VO}_2 \) expired = \( 5.17 \times \frac{17.5}{100} = 0.905 \text{l.min}^{-1} \)

\( \text{VO}_2 \) consumed = \( 1.086 - 0.905 = 0.181 \text{l.min}^{-1} \)

**CARBON DIOXIDE**

\( \text{VCO}_2 \) expired = \( 5.17 \times \frac{3.1}{100} = 0.1603 \text{l.min}^{-1} \)

\( \text{VCO}_2 \) inspired = \( 5.19 \times \frac{0.03}{100} = 0.00156 \text{l.min}^{-1} \)

\( \text{VCO}_2 \) produced = \( 0.1603 - 0.00156 = 0.1587 \text{l.min}^{-1} \)

**RESPIRATORY EXCHANGE RATIO (R)**

\[ R \text{ Value} = \frac{\text{VCO}_2}{\text{VO}_2} = \frac{0.1587}{0.181} = 0.877 \]

N.B. The Resting Metabolic Rate (RMR) of this subject is 0.88 indicating that a mixture of Fat and Carbohydrate is used for metabolism. A more ideal value is 0.85
APPENDIX 2

HAEMATOLOGY: Before, and immediately after, each 30 minute test, a 10 ml. venous blood sample was obtained from the antecubital vein of the subject's arm, usually the left. Samples were transferred to heparinised tubes; from these, aliquots were drawn off for the analysis of haemoglobin, haematocrit, glucose and lactate. The remainder of the sample was treated for analysis of blood lipid and catecholamine assays. Details of these methods can be found under the various assays. Lactates for the Power Tests were assayed from arterialised capillary blood from the thumb prick of the right hand at the conclusion of each of the four-minute stages of the test. 20ul of blood was taken and passed into 200ul of Perchloric Acid (2.5%), centrifuged and stored at -20°C for assaying once the study had been completed.

2a) HAEMOGLOBIN ASSAY

Preparation and procedure using the standard cyanomethoglobin method:-
1). Pipette 5 ml. of Drabkin's solution into Hb. glass tubes (Use White pipette, white tips).
2). Place spectrophotometer 546 nm. in photometer and switch on 20-30 min. prior to use.
3). Place 'blank' (Drabkin's soln. only) in cuvette and read optical density (OD).
4). Add 20ul (0.02 ml.) of blood to each of the 5 ml. tubes and mix thoroughly on the whirlmix.
5). Transfer each sample to the cuvette, determine OD and calculate delta values.
6). Convert to g/100 ml. by means of regression line evolved from known standards using Boehringer Mannheim pack. Regression equation for the present studies was:-
   \[ y = 37.2x + 0.06 \]
   where \( y = \text{g/100 ml.} \) and \( x = \text{Delta OD.} \)
7). Coefficient of variation for haemoglobin samples over 24 hours had been found to be no greater than 2.02% but it was possible, during the present studies, to analyse samples on the evening of the same day that they were taken.

2b). HAEMATOCRIT

Haematocrit percentages were determined from collections in heparinized tubes, spun for 5 minutes, and read from the Hawksley Microhaematocrit Scale.

2c). LACTATE ASSAY

Test Principle: Lactate \[ \xrightarrow{\text{NAD}} \xrightarrow{\text{LDH}} \text{Pyruvate} \]
Sequence of preparation and procedures:-

1). Take Standards and Samples out of freezer: let them warm up to room temperature for an hour. Alternatively use water bath - 10 minutes. N.B. Standards made up of: 20ul of standard in 200ul Perchloric Acid, as with samples.

2). Collect: Waterproof pen, plastic racks, fluorimeter tubes, tweezers, tissues, LDH and NAD (from fridge).

3). Number tubes: 3 x Blanks, 3 x Standard 1, 3 x S2, 3 x S2, 3 x S3, 3 x S4 and 2 for each sample tubes (sample + duplicate).

4). Break up samples using whirlimix.

5). Centrifuge samples for 3 minutes; they will separate into: supernatant and dark brown cell debris.

6). Set 200ul (yellow top) pipette to 20ul (020).

7). Pipette 20ul of each standard and of sample supernatant into glass fluorimeter tubes (2 samples from each plastic tube). USE NEW YELLOW PIPETTE TIPS FOR EVERY SAMPLE - Avoid touching insides of tubes with tips; avoid bubbles. Cover with tissue paper.

8). Make up reaction mixture (RMx) as follows:-

- will need 200ul (0.20ml.) RMx. per glass tube of Hydrazine Buffer.
- count number of glass tubes + 5 extra for total number.
- total number x 200ul = RMx. volume Xul = RMx. volume Yml.

9). Calculate the following per ml. of RMx (Hydrazine Buffer - pH 9.1):-

- 2mg. NAD
- 5ul LDH

10). Measure out NAD.

11). Add the Hydrazine Buffer.

12). Add the LDH.

13). Mix well and dispense into the glass tubes using the 1000ul pipette (Blue top) set at 200ul (020) or BCL8000 Dispenser set at No.2 with 6ml holder.

14). Mix contents of every tube on the whirlimix.

15). Cover with tissue and incubate for 30 minutes. SWITCH ON LOCARTE FLUORIMETER.

16). Add 1ml Lactate Diluent - 0.07M HCL ( Stops the reaction).

- Dispense 1ml into every tube.
- Mix well on whirlimix.

17). Read on fluorimeter after 6 displays. Calibrate fluorimeter by spanning the range: blank at c.2 and highest standard at c.160 - full range is up to 190 so permitting higher sample readings.

18). Calculate results on computer using BBC ('ASSANAL'); dilution factor 1.

N.B. Wash hands before assay: lactate on fingers may contaminate samples.
2d). **GLUCOSE ASSAY**

**Test principle:** Glucose + O$_2$ + H$_2$O $\rightarrow$ Gluconate + H$_2$O$_2$

**Sequence of preparation and procedures:**

1). Collect nitric washed tubes, plastic rack, waterproof pen and 20ul pipette.
2). Take Standard GOD (Glucose Oxidase) and samples out of fridge and freezer respectively: incubate at room temperature for 30-60 min. or use water-bath for 10 min.
3). Calculate number of tubes + 3 Standards and 2 Blanks; code the tubes.
4). Break-up samples with whirlimix and spin down for 5 min.
5). Pipetting: use 200ul pipette set at 20ul (020) and yellow tips.
6). Place 20ul of supernatant in every tube, excluding standards; 20ul of Standard in 3 tubes and 20ul GOD for blanks.
7). Turn on PHOTOMETER set at wavelength 436nm.
8). Carefully dispense 1 ml. of GOD in every tube. Incubate for 20 min. Mix on whirlimix.
10). Pour sample into photometer cuvette, read value, empty cuvette and drain on tissue.
11). Record and calculate results using formula:-

$$
\text{Concentration (c)} = 5.55 \times \frac{A_{\text{Sample}}}{A_{\text{Standard}}} \text{ (mmol.L}^{-1})
$$

12). BBC computer programme available: DLOAD "GLUCOSE" using "PHYSIOLOGY" disc.
APPENDIX 3

3a) FREE FATTY ACIDS: PHOTOMETRIC, COLORIMETRIC ASSAY FOR FFA
(Modified from Chromy et al., 1977).

Method and Procedure:-
1). Spin 10 ml. blood sample for 20 min. at 6000 rpm. in Koolspin.
2). Draw off 3 ml. sample of plasma with 1 ml. pipette.
3). Add 200 ul of anti-oxidant comprising 100 mmol.l⁻¹ of GSH (Reduced Glutathione) and 100 mmol⁻¹ of EGTA (Ethylenglycol tetra-acetic acid) when storing in freezer.

ASSAY:-
4). Use Oxford pipetter to deliver 3 ml. CHM into screw-capped Hb. glass tubes.
5). Add 100 ul of plasma or standard or 50 ul plasma for post-exercise sample and x2 results. (N.B. CHM only for blank: prepare 3 blanks, 3 of each standard but only 2 of each sample).
6). Add 1 ml. of stable copper reagent to everything, including blank. Reagent kept in refrigerator.
7). Shake vigorously in shaker for 10 min. set at 80; then centrifuge for 10 min. in Koolspin set at 4000 rpm.
8). Prepare appropriate number of glucose tubes containing 0.25 ml. TAC using dispenser and cover.
9). Transfer 1 ml. upper phase from Hb. tubes to glucose tubes with TAC. (N.B. Use special white tips: Ref. G23894 - C1000).
10). Mix well on low setting of whirlimix.
11). Read at 578 nm.

N.B. If carrying out glycerol assay also, start that first, then FFA. For best results, Hb tubes should be washed in clean HNO₃ if possible and kept specially for this assay.

SOLUTIONS.
1. Extraction Solvent (CHM):-
   280 ml. Chloroform. N.B. Amounts not absolutely critical but measure as close
   210 ml. N-heptane. as possible. Record amount of chloroform used.
   10 ml. Methanol. CHM very volatile.

2. Stable Copper Reagent:-
   For 500 ml.:-
   3.756g. Sodium Citrate.
   33.55g. Triethanolamine.
   16.25g. Copper Nitrate.
   125g. Sodium Chloride.
(N.B. Keep in fridge; stable for 6 months).

3. TAC (Noma et al., 1973):-
Dissolve 40 mg. 2-Thiozolylazo-p-Cresol (TAC) in 400 ml. ethanol. Must be accurate - the colorimetric factor.

Standards:-

<table>
<thead>
<tr>
<th>Stock soln.</th>
<th>CHM</th>
<th>mM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blank</td>
<td>0.00</td>
<td>5.00</td>
</tr>
<tr>
<td>Standard 1</td>
<td>0.25</td>
<td>4.75</td>
</tr>
<tr>
<td>Standard 2</td>
<td>0.50</td>
<td>4.50</td>
</tr>
<tr>
<td>Standard 3</td>
<td>1.00</td>
<td>4.00</td>
</tr>
<tr>
<td>Standard 4</td>
<td>1.25</td>
<td>3.75</td>
</tr>
</tbody>
</table>

Fat will not dissolve in H₂O so CHM used and proportions are made up from 4mM stock solution of palmitic acid as follows:-

N.B. Keep refrigerated in Hb tubes. Place in a beaker and seal with Nescofilm.

Coefficient of Variation:-
Fasting, rested values: \( \bar{x} = 0.217 \text{mM} \). SD = 0.0173mM. (8%) Date: 4.5.86
Post-exercise values: \( \bar{x} = 0.81 \text{mM} \). SD = 0.0348mM. (4.3%) Date: 18.12.87

References:-


3b) FLUORIMETRIC GLYCEROL ASSAY
(Modified from Laurell and Tibbling, 1966).

Method and Procedure:-
1). Place 0.25 ml. zinc sulphate into small centrifuge tubes.
2). Add 50ul of sample or standard. Modify as follows:

50ul of standards.

50ul of pre-exercise plasma sample.

25ul of post-exercise plasma sample + 25ul of 0.9% NaCl to make up volume; put saline in first and multiply results by 2.

3). Add 0.25 ml. barium hydroxide: avoid disturbing solute. Mix immediately.

4). Chill in freezer (-20°C) for five minutes.

5). Centrifuge for five minutes.

6). Pipette 200ul supernatant into long, acid-washed, lactate tubes.

7). Add 100ul of reaction mixture using BCL 8000 and mix.

8). Cover and incubate for 60 minutes.

9). Add 1 ml. of diluent with 1ml. adaptor and mix.

10). Read as for lactates on Fluorimeter or Perkin-Elmer.

SOLUTIONS.

a). Zinc sulphate: 0.087M i.e. 6.25g ZnSO₄·7H₂O (mw 287.54) in 250 ml. distilled water.

b). Barium hydroxide: 0.083M i.e. 6.85g Ba(OH)₂·8H₂O (mw 315.4) in 250 ml. DW. (Use only supernatant).

c). Cysteine (prepared daily): 0.2M i.e. 35mg cysteine in 1 ml. 0.4M NaOH (made from 6M NaOH - 1 part to 14 parts of DW). Cysteine will dissolve if mix in 'boat' on whirlimix). Be accurate.

d). Hydrazine - HCL buffer 1M (kept at 4°C).

1M Hydrazine i.e. 19 ml. of hydrazine hydrate (64% soln. of hydrazine: wt/ml of 1.03g in 250 ml. DW with 1.5mM MgCl₂ i.e. 76.2mg in 250 ml. DW. Adjust pH with HCL to 9.4). Prepare in beaker or conical flask.

e). Reaction mixture (100ul per tube).

Per ml. of reaction mixture: 12mg ATP, 20mg NAD (Be accurate).

: Add 100ul Cysteine.

900ul Hz-HCL buffer.

1ul Glycerokinase.

5ul Glycerin -3- phosphate dehydrogenase.

Diluent: 0.01 NaOH with 1mM EDTA (Diaminoethanetetra-acetic acid, disodium salt) i.e. 0.4g NaOH + 372.2mg EDTA up to 1 litre DW. Prepare in beaker using magnetic mixer.

STANDARDS.

a). Prepare approx. 4mM stock solution of glycerol i.e. 36.8mg or 29.2ul in 100 ml. DW. Calculate exact molarity from weight as follows:-

MW Glycerol: 92.10

1M glycerol is 92.10g. dissolved in 1 litre of DW.
Therefore 0.03684g. = 0.03684 - 1.261 = 0.0292149 ml. = 29.2 ul i.e. 29.2 ul dissolved in 100 ml. DW = 4mM Stock Solution.

b). Dilute x 10 to give approx. 0.4mM i.e.:-

10ul of 4mM glycerol in 90ul DW will give 0.4mM.

or 100ul " " 900ul DW " "

or 0.1 ml. " " 0.9 ml. DW " "

c). Take approx. 0.4mM as 100% then:-

\[
\begin{array}{ccc}
0.4\text{mM} & \text{DW} & \text{mM} \\
(\text{ml}) & (\text{ml}) & \\
20\% & 0.5 & 2.0 & 0.08 \\
40\% & 1.0 & 1.5 & 0.16 \\
60\% & 1.5 & 1.0 & 0.24 \\
80\% & 2.0 & 0.5 & 0.32 \\
\end{array}
\]

Blank is merely reaction mixture.

Coefficient of Variation:-

\[ n = 10 \bar{x} = 0.198 \quad \text{SD} = 0.00422 \ (2.1\%). \]

Date: 9.6.86

N.B. Keep in fridge when not assaying: Hydrazine Buffer, Glycerol Standards, Enzymes, Samples.

Reference:

3c) CHOLESTEROL + HDL- C ASSAYS

Test Principle: Chylomicrons, VLDL (very low density lipoproteins), and LDL (low density lipoproteins) are precipitated by adding phosphotungstic acid and magnesium ions to the sample. Centrifugation leaves only the HDL (high density lipoproteins) in the supernatant; their cholesterol content is determined enzymatically.

Method and Procedure:-
Carry out both assays together: 10ul plasma Cholesterol + 1 ml. Reaction mixture (Rx) + same for Standards.

: 20ul prepared plasma for HDL-C + 1 ml. Rx.
: Blanks - 1 ml. of reagent only.
Prepare HDL- C first as follows:-

1). Use centrifuge tubes i.e. small lactate tubes.

2). Pipette 100ul plasma into tubes: sample + duplicate.

3). Add 250ul precipitant. (Dilute 80 ml. bottle of precipitant 4:1 with DW.

   Make up, for safety: 4.25 ml. HDL- C reagent + 1.062 ml. DW:
   1 ml. + 62ul - will provide one spare).

4). As complete each tube, mix thoroughly.

5). Allow to stand for 10 minutes at room temperature.

6). Centrifuge for 10 min. @ 4000 rpm.

7). Place 20ul supernatant in glucose tubes and add 1 ml. reagent for CHOD-PAP method (20ul because so low a conc.). Set POSITIVE DISPLACEMENT PIPETTE at 20(0) - red (M25).

8). Now include Cholesterol: 10ul plasma plus 1 ml. reagent. Positive Displacement Pipette set at 10(0) - red. Take tip to bottom of glucose tube. Wipe tip end before ejecting.

Note on technique:- Pipette reagent SLOWLY to avoid bubbles: keep tip vertical: mix each one as complete : wipe tip end each time, avoiding the very end: no need to change tip if using same reagent. TAKE YOUR TIME. Assay

Assay no more than five subjects. Have available small tube of DW to wash M25.

9). Mix each sample as add reagent.

10). Turn on photometer at 546nm.

11). Incubate for 30 minutes.

Standards and Blanks:-

Do not shake standards. Blanks comprise reagents only - reagent bottle holds 30 ml.

HDL Standards: obtained from 50 mg.dl⁻¹ (1.29 mmol. l⁻¹) reagent as follows:-

<table>
<thead>
<tr>
<th>mmol. l⁻¹</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.323</td>
<td>1 part 1.29 mM + 3 parts DW.</td>
</tr>
<tr>
<td>0.645</td>
<td>2 parts 1.29 mM + 2 parts DW.</td>
</tr>
<tr>
<td>0.968</td>
<td>3 parts 1.29 mM + 1 part DW.</td>
</tr>
</tbody>
</table>

Total Cholesterol: PRECISET kit:-

<table>
<thead>
<tr>
<th>mg.dl⁻¹</th>
<th>mmol. l⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>1.29</td>
</tr>
<tr>
<td>100</td>
<td>2.59</td>
</tr>
<tr>
<td>150</td>
<td>3.88</td>
</tr>
<tr>
<td>200</td>
<td>5.17</td>
</tr>
<tr>
<td>300</td>
<td>7.76</td>
</tr>
</tbody>
</table>

Make up standard + duplicate.

Control: 5.21 mmol. l⁻¹ (Bought - expensive ).
N.B. To be more economical, standards can be reduced by a half together with reduced reagent of 0.5 ml.

References:

APPENDIX 4

PLASMA CATECHOLAMINE ASSAY BY MEANS OF LIQUID CHROMATOGRAPHY WITH ELECTROCHEMICAL DETECTION (LCEC)

Test Principle: The LCEC method relies on a simple liquid-solid extraction of the catecholamines onto alumina, followed by their elution with dilute acid. The alumina is selective for catechols and allows for their preconcentration prior to liquid chromatography.

General Method: The method described combines the beneficial aspects of previous LCEC assays with the advantageous use of Microfilter cartridges for low volume sample clean-up and Biophase ODS 5μm reverse phase columns for sufficient resolution. The catecholamines are adsorbed onto the alumina directly from plasma at pH 8.6. No deproteinization is necessary. After a suitable shaking period, the alumina is washed and aspirated twice with water, followed by elution with a small volume of acid in a Microfilter. This acid serves as the injectable extract. Acting as a dynamic ion exchanger, sodium actyl sulphate is used to adjust the reverse phase separation to the desired selectivity. Combined with the careful adjustment of mobile phase pH, the separation may be optimised for noradrenaline, dopa, adrenaline, dopamine, and an internal standard.

Method and Procedure:-

a). As for FFA assay, spin 10 ml. blood sample for 20 min. at 6000 rpm. in Koolspin.
b). Draw off 3 ml. sample of plasma with 1 ml. pipette.
c). Add 200ul of anti-oxidant comprising 100 mmol.⁻¹ GSH (Reduced Glutathione) and 100 mmol.⁻¹ EGTA (Ethylenglycol tetra-acetic acid) when storing in freezer.


Will need: 3 ml. + of plasma sample.

1 ml. Tris buffer (1.5M Tris, pH 8.6) to obtain correct pH.

200ul (20 picomol) Internal Sample.

ASSAY:-

1). Thaw plasma sample (c. 1 hr.): speed up with water bath (10 min.)

2). Spin in grey centrifuge for 5 min.

3). Place alumina in Hb tubes (washed in clean HNO₃).

4). Place 1 ml. Tris in each Hb tube.

5). Draw off as much plasma as available with special white tips.

    Keep a record of plasma drawn - needed for computer record.

    Place plasma in Hb tubes.
6). Add 100ul of Internal Standard (IS) to resting samples.
   Add 200ul of Internal Standard to post-exercise samples.
   Therefore IS on computer: 10 picomoles at rest.
   20 picomoles for post-exercise.

7). Spin in centrifuge: 5 min. @ 6000 rpm.
8). Place in Shaker for 10 min. set at 80.
9). During 7) and 8) prepare catecholamine tubes. Equipment needed:-
   i). 1 ml. pipette per Hg tube.
   ii). Check and assemble tubes and filters.
   iii). Check double distilled water + 1 ml. pipette.
10). Remove plasma with 1 ml. disposable pipettes.
11). Give two washes - add 2ml. doubled DW.
    - mix on whirlmix.
    Again, remove DW when alumina settled.
    Throw away ml. pipette after use.
12). Add a little DW and transfer to 'catecholamine tubes' using fresh ml. pipettes.
13). Remove alumina from Hb tubes with supernatant DW from 'cat. tubes'.
14). Remove excess DW with ml. pipettes.
15). Spin to dryness: two minutes in grey centrifuge.
16). Remove DW from lower tube to replace with fresh lactate tube.
17). Add 100ul 0.1 M HClO₄ (Perchloric Acid): will elute catecholamines.
18). Mix in whirlmix: squeeze diaphragm as mix to loosen alumina.
19). Spin two minutes in grey centrifuge.
20). Lactate tubes now contain 100ul eluted catecholamines.

To analyse:-
   a). Inject: - Resting sample - 100ul.
      Post-ex. sample - 2 x 50ul.
   b). Analyse samples from one subject together; if make an error it will be common to them all.
   c). For practice purposes, instead of plasma, can use: 3 ml. phosphate buffer + 200ul of H
      (Consists of 20ul of each of Noradrenaline, Adrenaline and IS made up to 2 ml. with 0.1 M
      HClO₄).
   d). 50 - 60% is normal recovery i.e. 50 - 60% of catecholamines are recovered from the alumina.

Analysing Equipment:-
   Spectra - Physics Integrator SP4290
   Gilson Pump, Model 302.
   Pressure provided by: Gilson Manometric Module, Model 802C.
To inject 50ul sample: use Hamilton Microlitre Syringe.
Figure 1  The relationship between heart rate and oxygen uptake during the test of progressive intensity.

Figure 2  The relationship between heart rate and VE in completing the modified PWC170 test.
Figure 3 The work related oxygen uptake parameters during the progressive, incremental load test.

Figure 4 R value responses during the progressive, incremental load test.
APPENDIX 6

CALCULATION OF ENERGY EXPENDITURE AND MECHANICAL EFFICIENCY DURING 30 MINUTE SUBMAXIMAL TEST (EXPERIMENTAL STUDY 2).

Worked Example: Active group at 30 min. during 'Just Tolerable Discomfort' Test.
Data:- \( \bar{x} \) weight of group = 76.7 kg.
At Rest: \( \text{VO}_2 = 0.32 \text{ l.min}^{-1} \)
\( \text{VCO}_2 = 0.29 \text{ l.min}^{-1} \) \( R = 0.91 \)
During Exercise (29 - 30 min.):
\( \text{VO}_2 = 1.87 \text{ l.min}^{-1} \)
\( \text{VCO}_2 = 1.85 \text{ l.min}^{-1} \) \( R = 0.99 \)
Ergometer Work (Output):
\( = 126.50 \text{ watts. 1 kcal} \approx 69.75 \text{ watts.} \)
\( = 1.813 \text{ kcal.} \)

Calculations
REST Let \( x = \text{g CHO oxidised. Let } y = \text{g Fat oxidised.} \)

\[
\text{VO}_2 0.32 = 0.828x + 1.989y
\]
\[
\text{VCO}_2 0.29 = 0.828x + 1.419y
\]
\[
0.03 = 0.57y
\]
Therefore \( y = 0.03 - 0.57 = 0.053 \text{ ie. in 1 minute, at rest, 0.053g. fat was oxidised. It is permissible to accept 'g' because the calculation is based on 1g of CHO and 1g FAT and their litre equivalent in O}_2 \text{ and CO}_2. \)
Substituting for \( y \)

\[
0.32 = 0.828x + (0.053 \times 1.989)
= 0.828x + 0.105
x = 0.32 - 0.105 - 0.828 = 0.259
\]
\ie 0.259g. of CHO was oxidised per min.
The amount of energy required to metabolise1g.CH0 = 4.1 kcal. 1g.FAT = 9.3 kcal.
So: 0.053g. FAT produced 0.053 \times 9.3 kcal. = 0.4929 kcal.
0.259g. CHO produced 0.259 \times 4.1 kcal. = 1.0619 kcal.
Therefore, Total Resting Metabolic Rate (RMR) = \(1,554.8\) kcal, or \(6,505\) kJ.

**EXERCISE**

\[
\begin{align*}
&\text{VO}_2 \quad 1.87 = 0.828x + 1.989y \\
&\text{VCO}_2 \quad 1.85 = 0.828x + 1.419y \\
&0.02 = 0.57y 
\end{align*}
\]

Therefore \(y = 0.02 \div 0.57 = 0.035\)g. FAT  
And substituting:-  
\[
1.87 = 0.828x + (0.035 \times 1.989)
\]
\[
= 0.828x + 0.0696
\]
\[
x = \frac{1.87 - 0.0696}{0.828} = 2.174\text{g. CHO}
\]

Thus total Exercising Metabolic Rate (EMR):-  
\[
0.035 \times 9.3 = 0.326\text{ kcal. FAT}
\]
\[
2.174 \times 4.1 = 8.913\text{ kcal. CHO}
\]

Total EMR = \(9.239\text{ kcal. min.}\), or \(38.655\) kJ.

**MECHANICAL EFFICIENCY (M.E.) = \(\frac{\text{OUTPUT}}{\text{INPUT}} \times 100\)**

Where 'Output' is the energy expended on the ergometer = \(1.813\) kcal.  
And 'Input' is the nett energy expended during exercise = \(9.239 - 1.555 = 7.684\) kcal.

Therefore M.E. = \(\frac{1.813 \times 100}{7.684} = 23.6\%\)

# TABLE 1

OXYGEN UPTAKE (litre.min⁻¹)

(Mean ± SD)

## PRE-SET LOAD

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.7 kp)</th>
<th>INACTIVE (2.2 kp)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5'</td>
</tr>
<tr>
<td>x</td>
<td></td>
<td>2.21</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.08</td>
</tr>
<tr>
<td>x</td>
<td></td>
<td>1.82</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>p</td>
<td></td>
<td>0.01</td>
</tr>
</tbody>
</table>

## 'JUST TOLERABLE DISCOMFORT'

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.1 kp)</th>
<th>INACTIVE (1.1 kp)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5'</td>
</tr>
<tr>
<td>x</td>
<td></td>
<td>1.85</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.68</td>
</tr>
<tr>
<td>x</td>
<td></td>
<td>1.13</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.27</td>
</tr>
<tr>
<td>p</td>
<td></td>
<td>0.05</td>
</tr>
</tbody>
</table>

p = Significant differences between Active and Inactive Groups.
### TABLE 2

**MINUTE VENTILATION (litre.min⁻¹)**

*(Mean ± SD)*

#### PRE-SET LOAD

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.7 kp)</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>INACTIVE (2.2 kp)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
</tr>
<tr>
<td>5'</td>
<td>58.8</td>
<td>13.1</td>
<td>50.0</td>
<td>7.56</td>
<td>60.2</td>
<td>20.4</td>
<td></td>
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<tr>
<td>10'</td>
<td>58.7</td>
<td>13.3</td>
<td>49.2</td>
<td>7.63</td>
<td>49.7</td>
<td>7.29</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15'</td>
<td>58.8</td>
<td>13.3</td>
<td>49.7</td>
<td>7.29</td>
<td>50.0</td>
<td>6.34</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20'</td>
<td>58.9</td>
<td>14.1</td>
<td>50.0</td>
<td>6.34</td>
<td>50.3</td>
<td>6.39</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25'</td>
<td>58.5</td>
<td>14.9</td>
<td>50.3</td>
<td>6.39</td>
<td>52.4</td>
<td>5.27</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30'</td>
<td>60.2</td>
<td>20.4</td>
<td>52.4</td>
<td>5.27</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

| p    | NS | NS | NS | NS | NS | NS |

---

### 'JUST TOLERABLE DISCOMFORT'

<table>
<thead>
<tr>
<th>Time</th>
<th>ACTIVE (2.1 kp)</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>INACTIVE (1.1 kp)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
<td>Xi</td>
<td>SD</td>
</tr>
<tr>
<td>5'</td>
<td>46.1</td>
<td>16.9</td>
<td>26.4</td>
<td>5.4</td>
<td>26.5</td>
<td>6.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10'</td>
<td>45.8</td>
<td>16.8</td>
<td>26.3</td>
<td>5.1</td>
<td>26.1</td>
<td>6.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15'</td>
<td>45.9</td>
<td>17.9</td>
<td>26.1</td>
<td>5.9</td>
<td>26.1</td>
<td>6.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20'</td>
<td>46.2</td>
<td>19.5</td>
<td>25.9</td>
<td>6.4</td>
<td>25.9</td>
<td>6.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25'</td>
<td>47.1</td>
<td>19.8</td>
<td>25.5</td>
<td>6.4</td>
<td>25.5</td>
<td>6.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30'</td>
<td>48.7</td>
<td>19.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| p    | 0.05 | 0.05 | 0.05 | 0.05 | 0.05 | 0.05 |

p = Significant differences between Active and Inactive Groups.
NS = No Significant differences between Groups.
Figures 1 and 2. Time-related oxygen costs for both active and inactive subjects in completing the two 30 min ergometer tests.
Figures 3 and 4. Relationships between heart rates and RPEs, following the two 30 min ergometer tests.
Figure 1. Blood lactate responses (Mean ± SEM) before, and immediately following, the 12 week training regimen.

Figure 2. Time-related oxygen costs for both subjects and controls during the 30 min ergometer test pre (T1) and post (T2) 12 weeks of training.
### APPENDIX 7A

#### TABLE 1

**Absolute plasma metabolite concentrations within and between pre (T1) and post (T2) training 30 min Tests (TS1).**

(Mean ± SEM)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>n = 24</th>
<th></th>
<th>Controls</th>
<th>n = 11</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lactates</strong> (mM)</td>
<td><strong>Glucose</strong> (mM)</td>
<td><strong>FFA</strong> (mM)</td>
<td><strong>Lactates</strong> (mM)</td>
<td><strong>Glucose</strong> (mM)</td>
</tr>
<tr>
<td>Pre</td>
<td>3.9 ± 0.4</td>
<td>3.6 ± 0.1</td>
<td>0.5 ± 0.05</td>
<td>Pre</td>
</tr>
<tr>
<td>Post</td>
<td>8.3 ± 0.6**</td>
<td>4.6 ± 0.2**</td>
<td>0.5 ± 0.04</td>
<td>Post</td>
</tr>
<tr>
<td><strong>Glycerol</strong> (mM)</td>
<td><strong>Adrenaline</strong> (nM)</td>
<td><strong>Noradrenaline</strong> (nM)</td>
<td><strong>Glycerol</strong> (mM)</td>
<td><strong>Adrenaline</strong> (nM)</td>
</tr>
<tr>
<td>Pre</td>
<td>0.2 ± 0.02</td>
<td>1.5 ± 0.2</td>
<td>6.1 ± 0.5</td>
<td>Pre</td>
</tr>
<tr>
<td>Post</td>
<td>0.2 ± 0.01</td>
<td>2.3 ± 0.3**</td>
<td>13.8 ± 1.4***</td>
<td>Post</td>
</tr>
</tbody>
</table>

**Significantly higher post-training (p<0.01)**

**Significantly higher post-training (p<0.005)**
APPENDIX 7A

TABLE 2
Changes in plasma lipoprotein pre and post-training.
(Mean ± SEM)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>n=24</th>
</tr>
</thead>
</table>

**Cholesterol**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>(mg.dl⁻¹)</td>
<td>197.2±8.2</td>
<td>205.6±4.8</td>
</tr>
<tr>
<td>(mM)</td>
<td>5.1±0.2</td>
<td>5.3±0.1</td>
</tr>
</tbody>
</table>

**HDL-C**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>43.1±2.1</td>
<td>46.2±2.0</td>
</tr>
<tr>
<td></td>
<td>1.1±0.1</td>
<td>1.2±0.1</td>
</tr>
</tbody>
</table>

**LDL-C**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>154.2±8.7</td>
<td>159.4±5.3</td>
</tr>
<tr>
<td></td>
<td>4.0±0.2</td>
<td>4.1±0.1</td>
</tr>
</tbody>
</table>

**Controls**

(n=11)

**Cholesterol**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>(mg.dl⁻¹)</td>
<td>181.5±11.3</td>
<td>185±8.1</td>
</tr>
<tr>
<td>(mM)</td>
<td>4.7±0.3</td>
<td>4.8±0.2</td>
</tr>
</tbody>
</table>

**HDL-C**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>39.7±2.9</td>
<td>44.5±2.9</td>
</tr>
<tr>
<td></td>
<td>1.0±0.1</td>
<td>1.2±0.1</td>
</tr>
</tbody>
</table>

**LDL-C**

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>141.7±11.5</td>
<td>140.5±8.4</td>
</tr>
<tr>
<td></td>
<td>3.7±0.3</td>
<td>3.6±0.2</td>
</tr>
</tbody>
</table>

* Significantly higher post-training (p<0.05)
** Significantly higher between subjects and control group (p<0.01)
APPENDIX 7A

TABLE 3

COMPARATIVE STUDIES OF BLOOD LIPIDS

<table>
<thead>
<tr>
<th>Authors</th>
<th>Training</th>
<th>N</th>
<th>Sex</th>
<th>Age</th>
<th>Lipoproteins</th>
<th>TG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Klens et al., 1980</td>
<td>12 weeks</td>
<td>24</td>
<td>M</td>
<td>40yr</td>
<td>TC ↓ HDL ↑ LDL ↓ TG ↓</td>
<td></td>
</tr>
<tr>
<td>Wood et al., 1983</td>
<td>1 year</td>
<td>48</td>
<td>M</td>
<td>45yr</td>
<td>TC ↓ HDL ↑ LDL ↓ TG ↓</td>
<td>-</td>
</tr>
<tr>
<td>Peltonen et al., 1981</td>
<td>15 weeks</td>
<td>20</td>
<td>M</td>
<td>40yr</td>
<td>TC ↓ HDL ↑ LDL ↓ TG ↓</td>
<td>*</td>
</tr>
<tr>
<td>Weltman et al., 1980</td>
<td>10 weeks</td>
<td>34</td>
<td>M</td>
<td>47yr</td>
<td>TC ↓ HDL ↓ LDL ↓ TG ↓</td>
<td>-</td>
</tr>
<tr>
<td>Seals et al., 1984</td>
<td>1 year</td>
<td>11</td>
<td>M&amp;F</td>
<td>63yr</td>
<td>TC * HDL ↓ LDL * TG ↓</td>
<td></td>
</tr>
<tr>
<td>Thomas et al., 1984</td>
<td>12 weeks</td>
<td>35</td>
<td>M&amp;F</td>
<td>25yr</td>
<td>TC * HDL * LDL * TG *</td>
<td></td>
</tr>
<tr>
<td>Lipson et al., 1980</td>
<td>6 weeks</td>
<td>10</td>
<td>M&amp;F</td>
<td>21yr</td>
<td>TC ↓ HDL ↓ LDL ↓ TG ↓</td>
<td>*</td>
</tr>
<tr>
<td>Allison et al., 1981</td>
<td>8 weeks</td>
<td>48</td>
<td>M&amp;F</td>
<td>22yr</td>
<td>TC * HDL ↓ LDL Δ TG *</td>
<td></td>
</tr>
</tbody>
</table>

Eight comparative studies showing total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL) and triglycerides (TG) following varied periods of training. Concentrations post-training reveal either a fall (↓), an increase (↑) or no change (⋆) in metabolites.

**Lipoproteins**

![Graph](image)

Figure 1  High density (HDL) and low density lipoprotein (LDL) cholesterol concentrations as revealed by both subjects and controls over the 12 week period.
APPENDIX 8

ANTHROPOMETRIC DETERMINATION OF LEG FAT AND MUSCLE PLUS BONE VOLUMES.

An anthropometric method has been derived for partitioning the volume of the leg into six segments which are similar to truncated cones.

Equipment.
- Holtain Digital Anthropometer.
- Holtain Fat Caliper.
- Metric Steel Tape (Flexible).
- Demographic Pencil.

Protocol.

Method.

With the subject standing erect and the feet slightly apart, the height

\[ \theta = 39.3 \]
\[ \theta = 37.4 \]
\[ \theta = 27.8 \]
\[ \theta = 27.3 \]
\[ \theta = 25.2 \]
\[ \theta = 28.1 \]
\[ \theta = 19.6 \]

above the floor and the circumferences are taken at the seven sites on the leg. The sites are illustrated above; they are: the gluteal furrow, one third subischial height measured up from the tibial-femoral joint space, the minimum circumference above the knee, the maximum circumference around the knee joint space, the minimum circumference below the knee, the maximum calf circumference and the minimum ankle circumference. The levels are marked with a dermatographic pencil; the circumferences are measured with a flexible steel metric tape, and the heights above the floor level are measured with a digital reading anthropometer.
(Tanner and Whitehouse, 1957). Skinfold thicknesses are measured with a Holtain fat caliper at four sites, viz. the anterior and posterior thigh in the mid-line at the one third subischial height level, and at the medial and lateral calf at the maximal calf circumference. Because the calipers pick up a double layer of skinfold tissue under pressure of 10 g/mm², \(10.0 \times 10^4\) N/m², the reading is converted to a true single measurement using a regression equation applicable to age, sex and fat site (Jones, 1970). This is based on a comparison between X-ray fat and caliper fat using the linear relationship which has been found to exist between the two (Jones and Pearson, 1969).

**Calculation.**

The formula to calculate the volume of a truncated cone is:

\[
\frac{1}{3}h(a + ab + b)
\]

where \(a\) and \(b\) are the areas of two parallel surfaces derived from circumference measurements. This can be expanded to give:

\[
\frac{1}{3}h\left(\frac{c^2}{4\pi} + \frac{c.d}{4\pi} + \frac{d^2}{4\pi}\right)
\]

where \(c\) and \(d\) are the circumferences of the two parallel surfaces. This enables actual anthropometric measurements to be applied directly to the formula which is then used to calculate the volume of the six truncated cones of the leg.

Using measurements from the diagram, an example has been worked as follows:-

**Segment 1.**

\[
4.0/3 \left(39.3^2 /4\pi + (39.3)(37.4)/4\pi + 37.4^2 /4\pi\right)
\]

\[
= 1.333 (122.9 + 116.96 + 111.31)
\]

\[
= 1.333 (351.17)
\]

\[
= 468.11 \text{ cm}^3
\]

**Segment 2.**

\[
13.6/3 \left(37.4^2 /4\pi + (37.4)(27.8)/4\pi + 27.8^2 /4\pi\right)
\]

\[
= 1158.49 \text{ cm}^3
\]

**Segment 3.**

\[
5.8/3 \left(27.8^2 /4\pi + (27.8)(27.3)/4\pi + 27.3^2 /4\pi\right)
\]

\[
= 350.34 \text{ cm}^3
\]

Therefore total volume of thigh:

\[
= 468.11 + 1158.49 + 350.34
\]

\[
= 1976.94 \text{ cm}^3
\]

**Segment 4.**

\[
2.7/3 \left(27.3^2 /4\pi + (27.3)(25.2)/4\pi + 25.2^2 /4\pi\right)
\]

\[
= 148.13 \text{ cm}^3
\]
Segment 5.
6.5/3 (25.2^2/4\pi + (25.2)(28.1)/4\pi + 28.1^2/4\pi) 
= 367.73 cm^3

Segment 6.
16.9/3 (28.1^2/4\pi + (28.1)(19.6)/4\pi + 19.6^2/4\pi) 
= 773.08 cm^3

Total volume of calf:-
= 148.13 + 367.73 + 773.08 
= 1288.94 cm^3

Volume of Total Leg = Volume of Thigh + Volume of Calf = 3265.88 cm^3

N.B. All results should be for two legs, therefore multiply the answer by two.

To estimate the muscle plus bone volume, the two corrected fat caliper readings for the thigh and calf are summed, the results are subtracted from their respective diameters and the inner cone volumes are calculated as previously indicated. An estimate of the subcutaneous fat volume is obtained by subtracting the muscle plus bone volume from the total leg volume (Jones and Pearson, 1969).
**APPENDIX 8A**

**TORQUE CREATED DURING THE FIRST SECOND OF A TWENTY SECOND ISOMETRIC CONTRACTION OF THE QUADRICEPS MUSCLES PRE- AND POST-TRAINING.**

(Torque in Newton/metres ±SD)

**SUBJECTS**

**RIGHT LEG**

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th></th>
<th>POST</th>
<th></th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torque</td>
<td>117.5 ± 27.2</td>
<td></td>
<td>114.8 ± 27.3</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Torque/Wt.</td>
<td>1.63 ± 0.33</td>
<td></td>
<td>1.60 ± 0.32</td>
<td></td>
<td>NS</td>
</tr>
</tbody>
</table>

**LEFT LEG**

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th></th>
<th>POST</th>
<th></th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torque</td>
<td>116.2 ± 29.2</td>
<td></td>
<td>115.8 ± 24.6</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Torque/Wt.</td>
<td>1.61 ± 0.35</td>
<td></td>
<td>1.62 ± 0.30</td>
<td></td>
<td>NS</td>
</tr>
</tbody>
</table>

Significant differences between means: * p<0.05; ** p<0.01; NS = Not Significant.
Figure 1  Prediction of VO₂ max by means 2 Mile run time (After Bland, 1982).
Figure 2 Estimated VO$_2$max, by means of 2 Mile run time.

Figure 3 Estimated VO$_2$max, by means of the Multistage Fitness Test.
Figures 4 and 5  Blood lactate concentrations following the Power Lactate Test before and after training for males and females.
Figures 6 and 7  Oxygen uptake following each of the three 30 minute tests for both males and females.
Figure 1 a) and b) Glycerol values for both males and females following the three 30 min. endurance tests.
Figure 2  Total dietary intake for both males and females pre- and post-training.
Figure 3  Lipoprotein values for both males and females, before and after training.