ENDURANCE TRAINING IN ELDERLY MALES

By

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"Of all the Methods proper for preserving Health, and for preventing, and even curing a great number of Diseases, there is none equal to moderate Exercise."

M. Andry, Orthopaedia
1743

ABSTRACT

Twelve 60-70 and ten 20-30 year old sedentary healthy males participated in this study to determine the effects of endurance training in improving the exercise capacity of elderly individuals and to examine the mechanisms related to oxygen transport that may contribute to any such change. Oxygen intake ($\dot{V}O_2$), CO_2 output ($\dot{V}CO_2$), ventilation ($\dot{V}E$), heart rate, blood pressure, cardiac output, and the ability of muscles to generate short-term power were measured during exercise before and after twelve weeks of high intensity endurance training.

Before training, the elderly had a 37% lower peak $\dot{V}O_2$ than the young subjects, associated with a lower peak cardiac output, and arterio-venous oxygen ($(a-\bar{v})O_2$) difference, heart rate and stroke volume and a lower vascular conductance. The capacity to generate short-term power was also about 30% lower in the elderly.

After training, peak $\dot{V}O_2$ (l/min) increased from 1.599 $\pm$ 0.073 to 2.212 $\pm$ 0.073 (38%) in the elderly and 2.536 $\pm$ 0.141 to 3.263 $\pm$ 0.181 (29%) in the young. Although the extent of the improvement was similar, different mechanisms contributed to the improvement in the two groups.

Firstly, in the elderly, peak cardiac output (l/min) increased from 12.7 $\pm$ 0.6 to 16.5 $\pm$ 0.4 (30%); the $(a-\bar{v})O_2$ difference (ml/l) increased only by 6% (126.5 $\pm$ 3.7 to 134.7 $\pm$ 2.7). In the young, there were equal

increases in cardiac output and $(a-\bar{v})O_2$ difference (14%). Secondly, the increase in peak cardiac output in the elderly was accompanied by a marked increase in peak stroke volume (78.9 $\pm$ 3.5 to 95.6 $\pm$ 2.5 ml, 21%), with a smaller but significant increase in peak heart rate (161 $\pm$ 3.8 to 173 $\pm$ 3.9 beats/min, 7%); in the young, stroke volume and heart rate increased to a similar extent (9% and 5% respectively). In addition, significant increases were observed after training in the elderly in submaximal stroke volume, cardiac output and vascular conductance; their systolic blood pressure during submaximal exercise was significantly lower. There were no changes in the young group. The changes in short-term power output were similar in both groups; total work increased by 13% in old and 8% in young, associated with decreases in plasma lactate and muscle fatigue. These changes together with decreases in the RER suggest improved aerobic metabolic processes in muscle.

In conclusion, elderly individuals respond to high intensity endurance training by increases in peak $\dot{V}O_2$ that are of the same magnitude as in young subjects. Marked training-associated increases in submaximal and peak stroke volume and cardiac output in the elderly are accompanied by decreases in heart rate, systolic blood pressure and increases in vascular conductance. These changes suggest improvement in the perfusion of exercising muscles which enabled these trained elderly to achieve higher levels of exercise and peak $\dot{V}O_2$.

DEDICATION

To my father Dr. Diomedes Issaias, who died before this work was completed, for teaching me that:

τά ἀγαθά κόποις κτῶνται

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1. HISTORICAL OVERVIEW

1.1 Introduction

Aging is an inevitable biological phenomenon. It is associated with a number of changes affecting virtually every physiological function and organ system. Many of the aging characteristics exhibited by old primates in different species are similar and in some respects are analogous to characteristics of aging humans; erosion of teeth, deepening creases in facial skin, changes in hair colour, loss of reproductive functioning and decreases in functional reserve capacity are examples of common aging characteristics among different species (Hrdy, 1981).

Aging is an event not many of us view positively. It is associated with physical deterioration or disease, with discrete events in the life cycle that signify the end of youth such as menopause, retirement or becoming a grandparent, and is also associated with death. It is a process somehow genetically programmed, with definable physiological limits representing a maximum of about 90 to 100 years of life.

The cliche that one "is as old as one feels" has a valid physiological basis, as the rate of aging is

different among individuals. Some individuals lead active, independent lives well into their 70s and 80s and may have the physical work capacity of individuals many years younger. Thus biological age does not go hand in hand with chronological age. It is biological aging, primarily of the ability to do physical work, that is the broad topic of this thesis.

There are undoubtedly structural changes inherent in the aging process that affect an individual's capacity to do work. The extent to which such structural changes impair function is dependent on a number of factors; freedom from disease, genetic make-up, healthy lifestyle and regular physical activity. The active centenarians living in the mountainous regions of the China-Afghanistan border and the foothills of the Caucasus mountains are able to continue working in heavy physical occupations well into their old age (figure 1). The 117 year old shown in figure 1 climbs a steep hill daily to pick potatoes for his lunch and continues farming as his daily occupation. The master athletes who remain active throughout life have higher capacities for physical work than sedentary individuals of the same age. Presumably improvements can be brought about by physical activity that may inhibit the deterioration in exercise capacity commonly associated with aging. Such improvements are likely to

Figure 1. An active centenarian approximately 117 years of age (Leaf 1973, reproduced with permission).

affect the functional rather than the structural age-associated changes.

A measure of the physical deterioration that occurs with aging is the decrease in maximal oxygen intake ($\dot{V}O_2max$). This is a highly integrated response dependent on the functional capacity of the cardiovascular, respiratory and musculoskeletal systems.

The work described in this thesis centres around the age-associated changes in the oxygen delivery mechanisms associated with the decline in the $\dot{V}O_2max$ and the physiological adaptations that may occur when the aging cardiovascular system is put under the stress of high intensity endurance training. This chapter will provide a review of previous work on this topic; it will outline the age-associated changes in exercise capacity and the training-associated adaptations and mechanisms that may contribute to any such adaptations.

1.2 The Maximal Oxygen Intake

1.2.1 Measurement of the Maximal Oxygen Intake

Maximal exercise capacity in healthy subjects is conventionally measured by the maximal oxygen intake ($\dot{V}O_2max$), defined as the highest oxygen intake an individual can attain during physical work (Astrand and

Rodahl, 1970). It increases linearly with physical work performed until the $\dot{V}O_2$max is reached at which point central oxygen delivery mechanisms are considered to have reached their uppermost limit.

Objective criteria for the definition of $\dot{V}O_2$max were first described by Astrand (1952), as the attainment of a plateau beyond which there is no further increase in oxygen intake despite further increases in workload; a blood lactate concentration greater than 8 mmoles/litre is another criterion of oxygen delivery having reached a maximum. Taylor et al (1955) defined the $\dot{V}O_2$max plateau during treadmill exercise as an increase in $\dot{V}O_2$ of less than 150 ml/min or 2.1 ml/kg/min associated with an increase in slope of 2.5% at a constant speed of 7 mph.

In the work described in this thesis, we determined the peak $\dot{V}O_2$ during cycle exercise, defined as ~~the $\dot{V}O_2$ attained~~ at the highest sustainable power output; the point at which the subjects were unable to maintain the required pedalling frequency despite maximal effort. As this value was not necessarily associated with a plateau in $\dot{V}O_2$, reference will be made to peak rather than maximal values to indicate the highest values reached by the subjects.

1.2.2 Age-Associated Changes in Peak $\dot{V}O_2$

It was in the middle of the 16th century that the relationship between muscular work and increased breathing was first discovered by Robert Boyle (Haldane, 1922). Boyle explained that the increased breathing which occurs during muscular exertion was a necessary accompaniment of the increased consumption of what he called a "nitro-aerial spirit" (oxygen). However, it was not until the middle of the 18th century that Joseph Black discovered that carbon dioxide is given off by the lungs; this was followed by Priestley's discovery that oxygen is used during respiration. Lavoisier continued Black's and Priestley's work with experiments on man and was the first to propose that the consumption of oxygen and output of carbon dioxide were increased during muscular work (Haldane, 1922). Almost 200 years later, in the early part of this century, the measurement of oxygen consumption during muscular work became possible.

The first study that examined the relationship between peak $\dot{V}O_2$ and age was that of Robinson in 1938. A 70 year old male in Robinson's study attained only 60% of the peak $\dot{V}O_2$ of a 25 year old. Both cross-sectional investigations, in which individuals of different ages are studied, and longitudinal investigations, where the same individuals are followed over a period of time, have been

employed to quantify the changes in peak $\dot{V}O_2$ with increasing age. These studies are summarized in tables 1 and 2.

The annual rate of decline reported in cross-sectional studies varies from 0.34 to 0.52 ml/kg/min (table 1). The data of Astrand et al (1958) fit the Robinson (1938) data well; from age 25 to 75 years the annual rate of decline in peak $\dot{V}O_2$ was 0.46 ml/kg/min (Robinson, 1938) and 0.52 ml/kg/min to age 65 in Astrand et al (1958). Shephard (1966) reviewed the world literature on the change in peak $\dot{V}O_2$ with age in about 4,000 males 25 to 75 years of age; the mean peak $\dot{V}O_2$ of a 25 year old was 3.11 l/min (45.0 ml/kg/min) and that of a 75 year old was 1.49 l/min (21.0 ml/kg/min), an average annual decline of 0.48 ml/kg/min, or a decline of about 52% over 50 years. Dehn and Bruce (1972) calculated a mean annual decline of 0.40 ml/kg/min. According to their equation, peak $\dot{V}O_2$ (ml/kg/min) = 57 - 0.40 (age), the predicted peak $\dot{V}O_2$ of a 25 year old was 46.0 ml/kg/min and that of a 65 year old was 31.0 ml/kg/min, a decline of about 33% over 40 years.

The population study of Chiang et al (1970) deserves special mention as it consisted of over 1,000 males 10 to 69 years of age, who represented approximately 50% of the total eligible population in the community of Tecumseh, Michigan. Continuous treadmill testing was

Table 1. Mean Rate of Decline in Peak $\dot{V}O_2$ with Age as Reported in Cross-Sectional Studies

Study	Number of Subjects	Age Range (years)	Activity Level	Exercise Protocol	Mean Rate of Decline ($ml.kg^{-1}.min^{-1}.year^{-1}$)
Vogel et al, 1986	1889 1514 male 375 female	17-55	U.S. Army Recruits & Soldiers	Treadmill	0.50
Convertino et al, 1986	32 15 male 17 female	53-57 male 54-56 female	Healthy non-athletic	Cycle	0.34
Astrand, 1958	81	50-64	Male truck drivers (moderately active)	Cycle	0.52
Binkhorst et al, 1966	52	22-60	Healthy clerical & light manual workers (non-athletic)	Cycle	0.39
Shephard, 1966	4000	25-75	Untrained, active and athletic	Treadmill & cycle	0.48
Dehn & Bruce, 1972	86	40-72	Healthy, non-athletic males	Treadmill	0.40
Taylor et al, 1973	385	40-59	General population of males	Treadmill	0.20
Chiang et al, 1970	1064	10-69	General population of males	Treadmill	0.50
Robinson, 1938	52	25-75	General population	Treadmill	0.46

employed at 3.0 mph starting at 0% grade and increasing every 3 mins by 3.0% to maximal effort. The mean annual decline was 0.50 ml/kg/min. At age 25 mean peak VO_2 was 40.0 ml/kg/min declining to 22.0 ml/kg/min at age 65, 45% over 40 years. However, for subjects over 40 years of age, the test was stopped at a heart rate of 160 beats/min which may have resulted in an overestimation of the decline in VO_2 with age.

The most recent population study is that of Vogel et al (1986) in 1514 U.S. army soldiers, 17 to 55 years of age. A discontinuous treadmill exercise test was used consisting of exercise bouts of 3 to 4 mins at either 6 or 7 mph with increasing grades of 2.5% after brief rest periods. A mean annual decline of 0.50 ml/kg/min was reported. Convertino et al (1986) compared the peak VO_2 in 15 middle-aged men (55 years) and 15 young men (21 years) using a supine cycle ergometer test starting at 0 W and increasing by 30 W every 3 minutes to fatigue. The mean peak VO_2 of the 21 year olds was 3.52 l/min (47.2 ml/kg/min) and that of the 55 year olds was 2.74 l/min, a decrease of 21% over 35 years or 0.34 ml/kg/min per year.

Results from longitudinal studies generally report a higher rate of decline compared to cross-sectional studies; between 0.23 and 1.04 ml/kg/min (table 2). The study with the lowest rate of decline was that of Dill

Table 2. Mean Rate of Decline in Peak VO_2 with Age as Reported in Longitudinal Studies

Study	Number of Subjects	Age Range (years)	Years of Follow-up	Activity Level	Exercise Protocol	Mean Rate of Decline ($ml.kg^{-1}.min^{-1}.year^{-1}$)
Dill et al, 1967	16	27-47	20	Champion runners	Treadmill	1.04
Dehn & Bruce, 1972	40	Middle-aged	2.3	Active and inactive males	Treadmill	0.94
Astrand et al, 1973	66 35 female 31 male	27-48	21	Physically active	Cycle	0.64
Kasch et al, 1985	13	45-63	18	Exercising professionals	Cycle & treadmill	0.04
MacKeen et al, 1985	315	50-63	13	Active and inactive	Treadmill	0.29 (inactive) 0.44 (active)
Dill et al, 1985	1	37-93	56	Not stated	Treadmill	0.23
Robinson et al, 1975	37	20-50	30	Healthy non-athletic	Treadmill	0.46

(1985) who followed his own peak $\dot{V}O_2$ from the age of 37 years to the age of 93 years. His peak $\dot{V}O_2$ declined from 3.3 l/min at age 37 to 2.8 l/min at age 66, 0.23 ml/kg/min per year. At 93 years of age, his peak $\dot{V}O_2$ was 0.760 l/min which amounts to a decline of about 77% over the 56 years of follow-up. This is the only study reporting a peak $\dot{V}O_2$ in a subject over 90 years of age and it is interesting for this reason alone as well as for the fact that the decline between 37 and 66 years was low, only about 15%, with a sharp decline over the last 30 years. It is thus likely that the decline in $\dot{V}O_2$ with increasing age is not linear, but accelerates at some point above the age of 60. Dill's (1985) activity level is not stated but a peak $\dot{V}O_2$ of 2.8 at age 65 is high compared to values reported in the literature. If we assume a body weight of 75 kg, this would amount to a peak $\dot{V}O_2$ of 37 ml/kg/min at age 65 which is about 19% higher compared to the predicted value by the Dehn and Bruce equation (1972) and about 30% higher compared to Shephard's (1966) values; 29 ml/kg/min and 25 ml/kg/min respectively.

Follow-up measurements of peak $\dot{V}O_2$ in 37 males tested in 1940 and retested in 1962 and 1971 by Robinson et al (1975) revealed a 28% decline over 31 years of follow-up, from 48.5 ml/kg/min at age 19 to 37.0 ml/kg/min at age 40 and 35.1 ml/kg/min at age 50. These values were lower

than those reported in the cross-sectional study by Robinson (1938); 41.0 ml/kg/min for 40 year olds and 38.0 ml/kg/min for 50 year olds. The interesting finding in this longitudinal study was that the subjects who were active and increased their participation in vigorous sports such as squash, tennis and jogging, maintained or even increased their peak $\dot{V}O_2$ over the years of follow-up, whereas the men who remained sedentary showed a decline in peak $\dot{V}O_2$.

Dill's et al (1967) 16 champion runners followed over 20 years had the highest rate of decline (1.04 ml/kg/min per year). However, when the subjects were divided into those who continued training and those who did not, the rate of decline paralleled closely their activity levels; those who stopped training and assumed a sedentary way of life smoked more, gained the most weight and had the highest rate of decline, 1.04 to 1.59 ml/kg/min. in contrast, those who continued training had the lowest rate of decline, 0.24 to 0.77 ml/kg/min. Astrand et al (1973) reported a decline of 0.64 ml/kg/min per year in male physical education teachers from 25 to 45 years of age. The subjects trained regularly in their youth and most continued training during the years of follow-up. Their mean peak $\dot{V}O_2$ at age 25 was 4.08 l/min (58.7 ml/kg/min)

declining to 3.28 l/min (45.3 ml/kg/min) at age 45, a decline of 20% over 20 years.

Results from longitudinal studies reveal higher rates of decline in sedentary subjects compared to active subjects which suggests that exercise may slow down the decline in physical capacity that is commonly associated with aging. The results of Dehn and Bruce (1972) over 2.3 years of follow-up concur with results from longer follow-up studies; higher rates of decline in peak $\dot{V}O_2$ in sedentary subjects (1.62 ml/kg/min) compared to 0.56 ml/kg/min in active subjects.

It would appear from the above studies that there are different rates of decline in peak $\dot{V}O_2$ with increasing age and that physical activity or inactivity is a major factor; either slowing down or accelerating the rate of decline. Classification of subjects by two levels of physical activity, active and inactive, may be an oversimplification; there are likely a number of different slopes of the relationship between peak $\dot{V}O_2$ and age that represent different physical activity levels and lifestyles. The higher rates of decline suggested by longitudinal studies may be related to the initial levels of fitness of the subjects; individuals with high initial peak $\dot{V}O_2$ values who become sedentary as they grow older may have a higher annual rate of decline, over 1.0 ml/kg/min as

reported by Dill et al (1967) and Dehn and Bruce (1972). However, individuals with average or above average peak $\dot{V}O_2$ in youth, who engage in some form of endurance training, may experience a moderate rate of decline, similar to that reported in cross-sectional studies; between 0.40 and 0.60 ml/kg/min per year. Based on the findings of Kasch et al (1985), subjects who participate in a regular, high intensity training program may have a much slower rate of decline, at least up to the age of 60 years, as also documented in the case study of D.B. Dill (Dill et al, 1985) and in aging master athletes (Houston and Green, 1981; Heath et al, 1981).

Apart from differences in the physical activity levels of the subjects, differences in testing procedures add to the difficulty in comparing results from different studies. Furthermore, there is a relative lack of subjects over the age of 60 years who are not ex-athletes. Only three cross-sectional and two longitudinal studies included over 60 year old untrained subjects; only eight out of a total of 81 subjects in Astrand's (1958) study, 14 out of 86 subjects in the Dehn and Bruce (1972) study and 30 out of 1,064 in Chiang et al (1970). Two of the 16 champion runners studied by Dill et al (1967) were over 60 years of age. Only in the study by Kasch et al (1985) was the majority of subjects over 60 years of age. However, in

this study, the subjects were training over the 18 years of follow-up. Thus the rate of decline in peak $\dot{V}O_2$ reported in the literature is based largely on peak $\dot{V}O_2$ values of subjects in their middle years and may not be representative of the elderly non-athletic population.

Another limitation in interpreting the results from different studies is a reluctance to test elderly, untrained subjects to maximal effort. In the study by Chiang et al (1970), subjects as young as 40 years of age were not allowed to exceed a heart rate of 160 beats/min, which amounts to about 85% of their predicted peak heart rate. Therefore, the likelihood exists that in at least some of their subjects the observed values were not representative of the subjects' maximal effort.

It is for these reasons that we undertook a cross-sectional study of males 15 to 70 years of age, as a background investigation to the training studies reported in this thesis; a baseline was needed for comparison with the young and elderly subjects who participated in the present study. Details of the cross-sectional study will be given in Chapter 2. Briefly, attention was paid to the number of subjects in each age group from 15 to 70 years, and to body height, weight and levels of physical activity. The testing protocol and personnel were the same in both the cross-sectional and training studies, and the subjects

who participated in both studies came from the same community. Thus we feel justified in using the results of our cross-sectional study as a baseline for comparison with the results in the present investigation. Based on the cross-sectional study, the predicted peak $\dot{V}O_2$ of a 25 year old was 44.0 ml/kg/min and that of a 65 year old was 26.4 ml/kg/min, a reduction of 40% over 40 years. The mean annual decline in peak $\dot{V}O_2$ was 0.44 ml/kg/min, which compares well with results reported in cross-sectional studies and further supports the earlier argument that the annual decline in peak $\dot{V}O_2$ with increasing age lies between 0.40 and 0.60 ml/kg/min.

1.3 Age-Associated Changes in Haemodynamic Function

The decline in peak $\dot{V}O_2$ with increasing age is associated with a number of age-related changes in cardiovascular and muscle function. Associated with these functional changes are changes in structure. The emphasis in this thesis is on age-associated changes in function and the role of endurance training in improving the functional capacity of elderly subjects; structural changes will be reviewed briefly only to clarify changes in function.

1.3.1 The Fick Principle

Cardiac output ($\dot{Q}$) may be related to oxygen intake and arterio-venous oxygen ($(a-\bar{v})O_2$) difference through the Fick principle:

$$\dot{Q} = \frac{\dot{V}O_2}{(a-\bar{v})O_2 \text{ diff.}} \qquad \text{Equation 1}$$

Also, cardiac output is the product of stroke volume (V_s) and cardiac frequency (f_c):

$$\dot{Q} = V_s \times f_c \qquad \text{Equation 2}$$

The peak $\dot{V}O_2$ is influenced by the maximum cardiac output and maximum arterio-venous oxygen difference:

$$\dot{V}O_2 = \dot{Q} \times (a-\bar{v})O_2 \text{ diff.} \qquad \text{Equation 3}$$

Changes in cardiac output and its determinants, heart rate and stroke volume, are capable of exerting major influences on peak $\dot{V}O_2$.

The cardiac output is a measure of the rate at which the heart pumps the blood through the circulatory system and has been the subject of speculation since the discovery of the circulation by William Harvey in 1628 (Leake, 1970). Harvey theorized that "...if the heart in a single beat in man, sheep, or ox, pumps one dram (8 drams = 1 ounce) and there are 1,000 beats in half an hour, the total amount pumped in that time would be ten pounds five

ounces" (Leake, 1970, p. 76). This was the first attempt at a quantitative estimate of cardiac output. Harvey also recognized that a number of factors influence cardiac output ..."I know and state to all that the blood is transmitted sometimes in a larger amount, other times in a smaller, and that the blood circulates sometimes rapidly, sometimes slowly, according to temperament, age, external or internal causes, normal or abnormal factors, sleep, rest, food, exercise, mental condition, and such like" (Leake, 1970, p. 77). Thus, Harvey was the first to recognize that exercise and age may affect cardiac output.

Almost three centuries passed before the cardiac output in man could be experimentally determined. This was initiated by Adolph Fick, who in 1870 described what has since been called the Fick Principle; that all the blood pumped by the heart passes through the lungs where the exchange of oxygen and carbon dioxide occurs. Therefore, Fick pointed out, that a knowledge of either the oxygen or carbon dioxide contents of the blood, before and after passage through the lungs together with a knowledge of the total oxygen or carbon dioxide exchange, gave the necessary data for the calculation of the cardiac output. Mixed venous blood was obtained by puncture of the right heart; arterial blood was obtained from the left heart or from any artery. By analysis of these bloods the amount of

oxygen taken up by the tissues per unit volume of blood and the amount of carbon dioxide taken up by the blood could be determined. Thus based on the Fick equation:

$$Q = \frac{\dot{V}O_2}{(a-\bar{v})O_2 \text{ diff.}}$$

The Fick principle was first applied by Grenhant and Quinquad in 1886 in dogs and by Zuntz and Hagemann in 1898 in two horses (Grollman, 1932). Zuntz and Hagemann were the first to demonstrate that cardiac output increases during exercise. The first application of the direct Fick procedure in man was performed by Grollman in 1930 (Grollman, 1932). The procedure involved cardiac puncture of the left and right heart through the first intercostal space which was both impractical and potentially hazardous. Nevertheless, these early studies were the first to demonstrate resting cardiac output values in man, reported to range between 3.0 and 5.0 l/min (Grollman, 1932).

Grollman's pioneer study of repeated determinations of cardiac output in one subject, at several day intervals during a period of two years, revealed that cardiac output under basal conditions is not subject to daily or seasonal variations. The values reported in this subject varied between 3.60 l/min and 4.16 l/min, mean of 3.90 $\pm$ 0.2 l/min. The experiments were carried out in the subject's home, shortly after the subject woke up in the

morning. The subject was reported to be "young, healthy, of good physique and complacement temperament." No other descriptive characteristics of the subject were given.

By the early 1940s, when cardiac catheterization was popularized, the Fick principle began to be widely used for the measurement of cardiac output in man (Guyton et al, 1973). Since then, a number of non-invasive procedures have been used for the determination of cardiac output at rest and during exercise. In this thesis, cardiac output was measured by the indirect Fick principle using the CO_2 rebreathing equilibration method (Jones et al, 1967; Jones and Rebuck, 1973). This method is based on the estimation of arterial and venous carbon dioxide concentrations from respired gases, and the measurement of the carbon dioxide output from which cardiac output is calculated by the Fick principle (Chapter 3):

$$\dot{Q}\ (l/min) = \frac{VCO_2}{(\bar{C}v - Ca)CO_2} \qquad \text{Equation 4}$$

1.3.2 Age-Associated Changes in Cardiac Output

Grollman (1932) pointed out that "our knowledge of the variation of the cardiac output with age is sadly deficient." This statement is to some extent true today. Relatively few studies have examined the changes in cardiac output during exercise in older individuals (60 years of

age). Thus much of what we know on this topic as well as on the regulation of cardiac output during exercise is based on studies that employed young subjects.

Grollman (1932) theorized that "the circulation, like many other physiological functions, becomes more sluggish with advancing age." He based this hypothesis on the "ruddy colour and bright red mucous membranes of youth" which "change to the dull anemia and cyanosis of age" (Grollman, 1932, p. 90). These statements do not paint a bright picture for older individuals and are a good example of the perceptions of not so many years ago on the physiology of aging. We know from master athletes that older individuals are capable of maintaining a high level of physical functioning and that they need not become anemic, cyanotic or sluggish. Nevertheless, changes do occur in the aging circulation and these are reflected by changes in the peak cardiac output with increasing age.

The work of Brandfonbrener et al in 1955 stimulated interest on the effect of age on cardiac output. Resting cardiac output was measured in 67 males free from signs of cardiovascular disease, between 19 and 86 years of age, using an indicator dilution technique. Resting cardiac output decreased with increasing age from about 6.5 l/min at age 25-35 years to 3.9 l/min at age 75-85 years,

an average decrease of about 1% per year; stroke volume decreased on the average about 0.7% per year.

Studies that included exercise cardiac output measurements in older individuals, over 60 years of age, are few. The first study on haemodynamic function in older individuals is the work of Strandell in 1964. Resting and exercise cardiac output were measured in 17 healthy males 61 to 83 years of age using the direct Fick method. Resting cardiac output (5.78 l/min) and stroke volume 86.1 ml) were 24% lower in the old compared to the young men (mean age 23 years). During exercise (cycle ergometer), cardiac output increased linearly with increasing $\dot{V}O_2$; the slope of this relationship was 6.0 l/min per litre increase in $\dot{V}O_2$ and was not significantly different from the slope observed in the young subjects. However, the intercept of this relationship was lower in the old (3.01 l/min) which resulted in a lower cardiac output (about 1.5 l/min) in the elderly at a given level of oxygen intake.

Similar findings were reported by Julius et al (1967) using the indicator dilution method during cycle ergometry; peak cardiac output was 12 l/min in 50-69 year old and 16 l/min in 18-34 year old males, a difference of about 25%. This was associated with a lower heart rate (15%) and stroke volume (11%). Faulkner et al (1977) examined the cardiac output-oxygen intake relationship in

men 17 to 71 years of age using a CO_2 rebreathing method and found no differences in the slope of this relationship for men of different ages. The intercept was significantly lower for inactive men, older than 40 years, with peak $\dot{V}O_2$ values less than 30 ml/kg/min. For these men, cardiac output could be estimated if an intercept of 49 ml/kg/min and a slope of 5.9 were used. For physically active men of all ages a slope of 5.2 and an intercept of 66 ml/kg/min was reported to give a good estimate of cardiac output at rest and during exercise.

Hossack et al (1980) estimated the cardiac output of 99 healthy men 20 to 73 years of age, based on the regression equation calculated from the results in 10 men 20 to 64 years, using the direct Fick method during treadmill exercise; $\dot{Q}$ (l/min) = 5.31 + 4.6 ($\dot{V}O_2$). Peak $\dot{V}O_2$ decreased from 20 to 73 years at a rate of 0.45 ml/kg/min per year, associated with a decrease of 0.17 l/min per year in estimated cardiac output. Stroke volume and heart rate decreased at a rate of 0.36 ml and 1.0 beat/min per year respectively. No major age-associated trends were observed by Hanson et al (1968) in cardiac output estimated by the dye-dilution method at rest and during treadmill exercise, in 75 healthy males 20 to 49 years of age. Fifteen out of their twenty-five 40 year olds completed the same peak power output (14% grade) as the 20 year olds, which

suggests similar exercise capacities in the two groups. The 10 men who did not complete this power output had a lower peak cardiac output (21%), 17.9 l/min compared to 22.6 l/min in the 20 year olds.

A recent study by Rodeheffer et al (1984) reported no decline in peak cardiac output in 61 males 25 to 79 years of age. The subjects were a subset of the subjects from the Baltimore Longitudinal Study on Aging who are healthy, physically active, community-dwelling individuals tested every 1 1/2 to 2 years. Freedom from cardiac or hypertensive disease was established by history, examination, exercise electrocardiography and stress thallium scintigraphy. Cardiac output, stroke volume and cardiac volumes were determined using gated radionuclide scintigraphy during upright cycle exercise. There was no age effect on resting or peak cardiac output ($\dot{Q}$ (l/min) = 20.4 - 0.05 age). Since peak heart rate decreased with increasing age (heart rate = 208 - 0.95 age), cardiac output was maintained by a higher stroke volume, which was associated with a higher end-diastolic volume. Peak VO_2 was not measured in this study.

These findings differ from studies reviewed earlier which showed a fall in peak cardiac output with increasing age associated with a fall in peak heart rate and stroke volume. This may be explained by differences in the subjects used. The subjects in the Baltimore

Longitudinal Study on Aging were a select group of physically active, highly motivated individuals with above average exercise abilities; the decline with age in the peak workload (watts) in this group was only 0.54 W per year (maximum workload (watts) = 157 - 0.54 age) which would give a predicted maximum workload for an 80 year old of 114 W (about 690 kpm/min) and 146 W (860 kpm/min) for a 20 year old, a decrease of only 22% over 60 years. However, in most studies of 20 year old males, maximum power output is about twice this value. Furthermore, the young age group in this study was too broad, 20 to 44 years; the remainder of the subjects were divided into groups ranging between 45 and 64 and 65 and 80 years, which tends to put a greater emphasis on the over 45 year olds.

In summary, there appears to be a decline in peak cardiac output of about 30 to 35% between 25 and 65 years, associated with a fall in peak heart rate and stroke volume of about 12 to 14%. In the following section, relevant structural changes that may contribute to these age-related changes will be reviewed.

1.3.3 Age-Associated Structural Changes

Studies of isolated hearts and cardiac muscle from aging animals suggest significant decreases in overall cardiac function resulting from a number of age-related

changes. The early studies of Wilens and Sproul in 1938 documented the presence of an age-related increase in myocardial fibrosis in the aged rat heart with hypertrophied bundles intermingled among the fibrous tissue. The increase in the amount of connective tissue within the myocardium is believed to result in diminished myocardial compliance (Tomanek et al, 1972).

Progressive obliteration of the myocardial capillaries was reported by Rakusan and Poupa (1964) with a decrease in the capillary-fibre ratio in the hearts of senile rats. These findings were confirmed by Tomanek (1969) in non-exercised rats. However, exercised old animals in the same study (Tomanek, 1969) had a capillary-fibre ratio equal to that of non-exercised young adult animals. The increase in the capillary-fibre ratio was associated with an increased capillary density.

Autopsy data in about 7,000 human hearts examined by Linzbach and Akuamoa-Boateng (1973) revealed that between 30 and 90 years of age the heart increases in mass on an average of 1 to 1.5 grams/year (cited in Lakatta, 1979). Left ventricular wall thickness determined by echocardiography appears to increase from 20 to 80 years in healthy men and women distributed evenly among sedentary and manual occupations (Sjogren, 1971). The subjects in this study were free of cardiovascular disease based on

repeated clinical evaluation and treadmill exercise testing, with blood pressure values less than 140/90 mmHg.

Thus it appears that the aging heart enlarges with increasing age and the myocardium becomes more fibrotic and less vascular. These changes in structure will likely affect function, the extent to which may be modified by physical activity.

1.4 Skeletal Muscle Blood Flow

1.4.1 Integration between Cardiac Output, Arterial Pressure and Vascular Conductance

William Harvey recognized in 1628 that "the arteries are the vessels carrying blood from the heart to the body, the veins returning blood from the body to the heart, the one away from the heart, the other toward the heart, the latter carrying imperfect blood unfit for nourishment, the former perfected, nutritious blood" (Leake, 1970, p. 72). Harvey theorized that the arteries which are high pressure vessels are equipped with strong vascular walls; "because they must withstand the pressure of the pumping heart and rushing blood" (Leake, 1970, p. 130). The veins on the other hand, transport the blood back to the heart and have flaccid, thin walls compared to

the arteries. These descriptions outlined over 400 years ago, hold true today.

The arterioles are the terminal branches of the arterial tree; their muscular walls can constrict or dilate altering the diameter of the vessels and controlling organ blood flow. For this reason, they are referred to as the resistance vessels or the vasomotor system (Rowell, 1986). The amount of blood that flows into the veins is therefore regulated by the arterioles.

The veins are also equipped with a muscular wall but are highly distensible vessels capable of storing large amounts of blood as the pressure increases. For this reason, they are referred to as capacitance vessels or the venomotor system (Rowell, 1986).

Blood flow (Q) through a vessel is determined by the pressure difference tending to push blood through the vessels and the vascular conductance (g). Therefore:

$$Q = (Pa - P\bar{v}) \times g \qquad \text{Equation 5}$$

where Pa and $P\bar{v}$ are the arterial and venous pressures.

The mean arterial pressure is the average pressure tending to push blood through the systemic

circulation. Since the right atrial pressure in healthy subjects is nearly 0 mmHg, the amount of blood that flows into the heart each minute (venous return) and that is pumped each minute (cardiac output) is determined by the arterial pressure and the vascular conductance:

$$\dot{Q} = Pa \times g \qquad \text{Equation 6}$$

Therefore, assuming a constant or nearly constant arterial pressure, venous return and cardiac output are proportional to the vascular conductance. In other words, when the peripheral arterioles dilate, venous return and cardiac output increase. However, this will only occur if arterial pressure can be maintained.

From equation 6, arterial pressure is related to cardiac output and vascular conductance as follows:

$$Pa = \frac{\dot{Q}}{g} \qquad \text{Equation 7}$$

If all arterioles were to become fully vasodilated, cardiac output would have to increase by a proportional amount to maintain arterial pressure. Therefore, the peripheral arterioles control blood flow through the various organs by vasodilation or vasoconstriction, which need to be balanced between the metabolic needs of the tissues and the capacity

to raise cardiac output so that arterial pressure is maintained. From equation 7, vascular conductance is expressed as the ratio of cardiac output to mean arterial pressure:

$$g\ (l/min/mmHg) = \dot{Q}/Pa \qquad \text{Equation 8}$$

The above discussion outlines the delicate balance between the peripheral and central circulation in maintaining the arterial pressure necessary for adequate perfusion.

1.4.2 Regulation of Skeletal Muscle Blood Flow

The ability of arterioles to alter their diameter was first recognized by William Harvey in the 16th century when he wrote: "So the terminal arteries appear like veins, not only in structure, but also in function, for they rarely show a perceptible pulse unless when the heart beats violently, or the arteriole dilates or is more open at the particular point" (Leake, 1970, p. 130). The arterioles are under the influence of an inherent constrictor tone, known as basal tone (Barcroft, 1963). Neural and humoral influences modify this tone either increasing or decreasing it and thus regulating the amount of blood flow to the tissues. An increase in basal tone will result in vasoconstriction which will decrease

conductance, while the opposite is true if the basal tone is decreased. Skeletal muscle vessels have a high basal tone and are also under the influence of a tonic sympathetic vasoconstrictor outflow that provides a resting sympathetic tone. A number of factors alter the vasoconstrictor tone in skeletal muscle arterioles.

1.4.2.1 Neural Control

Sympathetic postganglionic nerve endings to the smooth muscle of the arterioles release norepinephrine which binds with alpha receptors on the cell membrane to produce the vasoconstrictor effect. The binding of norepinephrine to the alpha receptors causes an increase in the permeability to calcium in the cell membrane and sarcoplasmic reticulum which initiates the contraction of vascular smooth muscle. The interaction between the alpha adrenergic receptors and norepinephrine is inhibited by alpha adrenolytic agents such as phentolamine (Shepherd and Vanhouette, 1975).

The presence of vasoconstrictor sympathetic nerves in skeletal muscle vessels was shown by Barcroft and Swan (1953). After release of vasoconstrictor tone by blocking of the radial, median and ulnar nerves above the elbow, which were considered to carry the sympathetic nerves to the muscles, blood flow almost doubled on the side of the nerve blocks. The significance of arteriolar

vasoconstrictor tone is evident from experiments where sympathetic outflow to the periphery was blocked by total spinal anaesthesia; this resulted in a fall in arterial pressure from 100 to 50 mmHg (Guyton et al, 1973). Therefore, if all tonic constrictor influences were removed and vascular smooth muscle were fully relaxed, cardiac output would need to increase beyond its capacity to maintain a constant arterial pressure (Rowell, 1986). Thus alterations in arteriolar vasoconstrictor tone have a profound effect on arterial pressure and cardiac output.

The presence of sympathetic vasodilator fibres in human skeletal muscle vessels is less clear; the first demonstration of a vasodilator effect was reported by Barcroft and Swan (1953) during an experiment in which the subject fainted; during the faint, the subject's blood pressure fell but forearm blood flow increased. The neural mechanism was believed to be mediated via sympathetic nerve fibres, since vasodilation was absent in sympathectomized forearms (Barcroft, 1963). These fibres are believed to be cholinergic and to travel together with vasoconstrictor sympathetic fibres in peripheral motor nerves (Burnstock, 1980).

Mental and emotional stress and reduction in circulating blood volume were shown to result in marked vasodilation in the forearm (Greenfield, 1966). Support for cholinergic vasodilation in human muscle was reported

by Blair et al (1959) after emotional stress (subjects were frightened because they believed that they were suffering from severe blood loss). The vasodilator responses were reduced by atropine but were unaffected by anaesthetic blockade of cutaneous nerves in the forearm (Blair et al, 1959). Holling (1964) on the other hand, found that sympathetic nerve block did not abolish the vasodilator response to mental arithmetic, and Allwood et al (1959) reported a reduced response after adrenalectomy. Thus there is no consensus as to whether this response is neural or humoral.

1.4.2.ii Metabolic Control

Blood flow is closely related to the metabolic needs of the tissues. Gaskell proposed in 1877 that the close link between metabolism and blood flow may be mediated via sympathetic nerve stimulation which results in skeletal muscle vasodilation. However, three years later, he reported that vasodilation could be induced in denervated muscles by oxygen lack, lactic acid and increased CO_2 (Gaskell, 1880; Balis, 1901); these were among the first reports implicating locally mediated metabolic factors in skeletal muscle vasodilation. Hilton (1953) showed that the muscular contractions and vasodilation elicited by motor nerve stimulation are abolished by curare. Since curare does not affect

vasodilator nerve endings, it was concluded that the vasodilation must be related to the contractile process (Hilton, 1953; Honig and Frierson, 1976).

The basic concept of a metabolic control is that the high basal tone which characterizes skeletal muscle vessels can be inhibited by local accumulation of metabolic by products. This in turn will regulate vascular conductance as a close function of the metabolic needs of the tissues. The general approach taken in investigations of the relationship between metabolism and vasodilation has been to increase the amount of work or to lower the availability of oxygen. On this basis, some of the suggested vasodilators are lower PO_2 and elevated CO_2 H^+ concentration, potassium and adenosine.

Vasodilation occurs in skeletal muscle immediately before muscle contraction (pre-exercise vasodilation) as well as during and after muscle contraction (exercise and post-exercise vasodilation). The pre-exercise response is believed to be mediated by sympathetic cholinergic nerves as part of an anticipatory reaction to muscular exercise, which serves to direct the increase in cardiac output to the muscles who are about to exercise (Shepherd and Vanhouette, 1975; Burnstock, 1980). The vasodilator response during and after contraction does not appear to involve cholinergic nerves,

as the response persists in the presence of atropine (a cholinergic nerve antagonist) and after sympathectomy (Barcroft, 1963; Jones and Berne, 1964).

Stowe et al (1975) observed that when venous blood draining exercising dog skeletal muscle (with low PO_2, pH and high PCO_2) was pumped into a resting control muscle, vasodilation resulted. Raising the PO_2 of the venous blood from the exercising muscle removed most of the vasodilator effect. This led to the implication of a fall in arteriolar wall PO_2 as a primary vasoactive mediator. However, changes in venous PO_2 and blood flow do not follow each other closely and arterial PO_2 must fall below 40 to 50 mmHg before flow is affected (Rowell, 1986).

The evidence for adenosine as a mediator of skeletal muscle vasodilation is also weak; venous plasma adenosine concentrations in blood-perfused dog hind limb at constant flow did not increase during muscle contraction in sufficient amount to suggest a role in muscle vasodilation (Bockman et al, 1975). Furthermore, theophylline infused in blood-perfused isolated dog gracilis muscle did not affect blood flow during muscle contraction (Tabaie et al, 1977). Honig and Frierson (1976) verified and extended the results with theophylline by showing that dipyridamole (an adenosine potentiating agent) did not increase blood flow during exercise. However, in about half of the muscles tested, dipyridamole prolonged the recovery in blood flow

after muscle contraction had ceased. Thus adenosine may have a limited role during exercise, but may contribute to the post-exercise hyperaemia.

Forrester (1981) proposed that adenosine triphosphate (ATP) may be a more potent vasodilator than adenosine; venous blood draining active human forearm muscles (Forrester, 1972) and exercising dog hindlimb (Chen et al, 1972) had ATP concentrations in amounts paralleling the level of metabolism. Since cell membranes are considered to be impermeable to ATP, the mechanism of ATP release into venous blood may be facilitated by hypoxic conditions within the smooth muscle cell which are believed to make the cell membrane more permeable to ATP. However, Dobson et al (1971) could not confirm the release of ATP in contracting frog sartorius muscle.

Perhaps the strongest evidence for local vasoactive substances in exercising skeletal muscle is from cross-perfusion studies when venous blood draining active muscle infused into resting muscle resulted in vasodilation. However, there is no concensus as to the nature of the vasoactive substance or the mechanism of its release. A major difficulty relates to the measurement of tissue metabolites in the interstitial spaces where their action on blood vessels is likely to take place, as

concentrations from venous effluent may not reflect interstitial concentrations.

1.4.2.iii Interaction between Neurogenic and Metabolic Factors

An important aspect in blood flow regulation during exercise is the ability of skeletal muscle to vasodilate even in the presence of intense vasoconstriction induced by sympathetic stimulation, as for example during maximal exercise. The term functional vasodilation is often used to refer to the net result of a competition between central vasoconstrictor and local vasodilator influences. Thus during exercise, sympathetic discharge to the resistance vessels of non-exercising muscles is believed to result in vasoconstriction, while local metabolic factors prevent vasoconstriction in active muscles which would then vasodilate. This may explain the increase in vascular conductance observed during exercise.

However, Donald (1970) and Rowell (1974) have shown that vasoconstriction can occur in active skeletal muscles even during heavy exercise; they determined leg blood flow in dogs during treadmill exercise before and after stimulation of the lumbar sympathetic chains. Sympathetic stimulation resulted in a decrease in leg blood

flow, that was more during mild exercise (60%) compared to 45% during heavy exercise. These findings suggest that as the exercise stimulus increases, the vasoconstrictor sympathetic influence is progressively diminished, but it is not abolished. Thus exercising muscle can be vasoconstricted by increased sympathetic activity. As discussed earlier in this chapter, the vasoconstriction-vasodilation interaction during exercise is important in the regulation of arterial pressure.

Rowell (1986) argues that the capacity of the peripheral vasculature to dilate and thus to increase vascular conductance exceeds the capacity to increase cardiac output. Thus if all skeletal muscle arterioles are maximally vasodilated, as would be expected to occur during whole-body heavy exercise, cardiac output would be unable to sustain an adequate level of arterial pressure. Therefore, the interaction between neurogenic vasoconstriction and metabolic vasodilation in active skeletal muscle is balanced against the capacity to raise cardiac output so that arterial pressure does not fall.

1.4.2.iv Age-Associated Changes in Skeletal Muscle Blood Flow

There is a lack of studies on skeletal muscle blood flow during exercise in elderly subjects. Wahren et

al (1974) reported the first comparative study on changes in blood flow during exercise in seven middle-aged (50-59 years) and eight young (25-30 years) highly trained males. Resting leg blood flow measured by an intra-arterial indicator infusion technique was similar in the two groups. During exercise, blood flow increased in a curvilinear fashion in the older subjects; from a resting value of 0.53 l/min the increase was linear up to a $\dot{V}O_2$ of 2.5 l/min (about 60% of peak $\dot{V}O_2$; during exercise at heavy, near maximal work intensities, the blood flow tended to level off. The rise in leg blood flow relative to $\dot{V}O_2$ was significantly lower in the middle-aged subjects associated with a higher leg arerio-venous oxygen difference, so that the leg $\dot{V}O_2$ was similar in the two groups.

The finding of a lower leg blood flow in these middle-aged, well trained males may indicate a hypokinetic circulation with increasing age; this is in agreement with the conclusions of Granath et al (1964) who reported a lower cardiac output (about 2 l/min) during exercise in elderly compared to young subjects. Amery et al (1969) observed a decrease in maximum blood flow (Xenon clearance method) after flexion and extension exercise at the ankle joint in both healthy and hypertensive patients 17 to 75 years of age.

Van Brummelen et al (1981) recently reported a decreased vascular response to intra-arterial infusion of isoprenaline (a beta-adrenoreceptor agonist) in elderly subjects. Vascular beta-adrenoreceptor sensitivity was examined in seven 53-70 years (mean 59 years) and eight 12-24 years (mean 21 years) healthy subjects by measuring the change in forearm blood flow (venous occlusion plethysmography) in response to intra-arterial infusion of isoprenaline. Resting forearm blood flow was similar in the two groups; however, the increase in blood flow with increasing doses of isoprenaline was significantly lower in the older group. The chronotropic response to incremental doses of intravenous isoprenaline was also significantly lower in the elderly. These findings suggest a blunted beta-adrenoreceptor sensitivity with increasing age.

Fleisch et al (1970) examined the activity of beta receptors in aortic strips of young and old animals by determining the responses to isoproterenol after blockade of alpha receptors. The aortic strips from the old animals failed to relax in response to isoproterenol in contrast to the relaxation observed in specimens from young animals. Similar results in animal studies were also reported by Fleisch and Hooker (1976) and Ericsson and Lundholm (1975).

An interesting finding reported by Van Brummelen et al (1981) was that blood flow related

significantly with plasma noradrenaline ($r = -0.77$) in the older subjects, which suggests a predominance of alpha adrenergic influences in this group. In the young subjects, blood flow related significantly with plasma adrenaline ($r = -.78$), which in low doses is considered to have a vasodilator effect via its beta-adrenoreceptor agonist action. A higher alpha-adrenergic predominance in older subjects may at least partly explain the greater increase in blood pressure during exercise with increasing age, which is a consistent finding in a number of studies (Robinson, 1938; Granath et al, 1964; Julius et al, 1967; and others).

1.4.3 Age-Associated Structural Changes

A decrease in aortic compliance with increasing age was first reported by Roy over 100 years ago (1880-1882). Aged aortae as well as small arteries were found to exhibit a greater resistance to deformation (Roach and Burton, 1959). Stiffness of a vessel was quantitated in terms of its elastic moduli, the higher the elastic modulus the greater the stiffness (Learoyd and Taylor, 1966; Bader, 1967). At a pressure of 100 mmHg, the modulus of elasticity in aortae from persons over 35 years of age was found to be twice as high as that from persons younger than

35 years, so that by the age of 85 years, the aorta behaved like a rigid tube (Bader, 1967).

An arterial rigidity index, defined as the change in pulse pressure relative to the change in diastolic pressure following inhalation of amyl nitrite was also found to increase with age (Abboud and Huston, 1961). The increased rigidity of the arterial wall is believed to be at least partly explained by a relative loss of elastin and an increase of collagen in the aging arterial wall (Roach and Burton, 1959). Urschel et al (1968) substituted a stiff tube for the aorta in dogs while heart rate was maintained at a constant rate. Imposition of the rigid tube resulted in a high systolic left ventricular pressure and a higher diastolic left ventricular volume with a delay in ventricular ejection. Furthermore, the less compliant old aorta, tends to impose a heavier workload on the left ventricle, as there is less diastolic recoil and therefore a decreased aortic contribution to forward flow (Bader, 1967). The increase in impedance to left ventricular ejection by structural changes in the vascular wall may contribute to the greater systolic arterial pressure and decreased capacity to raise cardiac output during exercise in the elderly.

1.5 Age-Associated Changes in Respiratory Function

1.5.1 Introduction

With increasing age, the lung undergoes a number of structural changes that influence its ability to exchange oxygen and carbon dioxide between air and blood. The extent to which such changes influence function is variable and is often difficult to isolate the effect of aging from that resulting from exposure to pollutants such as smoking.

Main structural changes in the aging lung are a gradual loss of alveolar surface area and surrounding pulmonary capillaries (Thurlbeck, 1976), loss of elastic recoil with stiffening of the overlying thoracic cage and weakness of the respiratory muscles (Niewomer et al, 1975; Turner et al, 1965; Black and Hyatt, 1969). These changes contribute to a number of age-related changes in overall respiratory function.

1.5.2 Lung Volumes

The loss in elastic recoil leads to a change in the pressure-volume relationship in the aging lung so that lung compliance increases; thus at a given transpulmonary pressure, lung volume is higher in elderly subjects (Begin et al, 1975; Knudson et al, 1977). These changes are

reflected in a higher functional residual capacity (FRC) and residual volume (RV). Grimby and Soderholm (1963) observed an increase, in FRC and RV, of 15 and 22 ml per year respectively in 152 healthy males 20 to 65 years of age. During expiration, premature airway closure (at higher lung volumes) resulting from a loss in elastic recoil and lack of support in the small airways, resulted in a fall in vital capacity at a rate of 25 ml/year; about 17% from the age of 20 to 60 years (Morris et al, 1971).

1.5.3 Maximum Voluntary Ventilation

The loss in elastic recoil and premature airway closure together with the reduction in respiratory muscle strength contribute to a fall in maximal voluntary ventilation from about 182 to 125 l/min, a 31% fall between 20 and 60 years (Grimby and Soderholm, 1963). The decline reported by Ericsson and Irnell (1969) in 67 healthy males 57 to 71 years of age was 24%, 140.2 l at age 59 and 106.4 l at age 69 which suggests a greater rate of decline over 60 years of age.

1.5.4 Pulmonary Blood Flow

With increasing age, there is a gradual loss of the small vessels in the pulmonary vascular bed which is associated with an increased resistance to flow. These

changes contribute to a higher resting mean pulmonary artery pressure, from about 12 mmHg at age 20 to 15 mmHg at age 60; during exercise, the increase in the same age range is from 18 to 30 mmHg (Ehrsam et al, 1983).

1.5.5 Arterial Blood Gases

Arterial PO_2 declines progressively with increasing age, from about 99 mmHg at age 20 to 88 mmHg at age 60, a fall of about 3 mmHg per decade (Sorbini et al 1968; Raine and Bishop, 1963; Mellemgaard, 1966; Conway et al, 1965). The factors that may contribute to this decline are alveolar hypoventilation, diffusion impairment, shunt and increased ventilation-prefusion ($\dot{V}/\dot{Q}$) mismatch. The latter three factors contribute to the alveolar-arterial PO_2 difference which increases with age (Mellemgaard, 1966; Harris et al, 1976). Alveolar hypoventilation and shunt are not considered to change significantly with increasing age (Filley et al, 1954; Sorbini et al, 1968). However, there is some impairment in the diffusing capacity of the aging lung likely due to the reduction in alveolar surface area (Van Kessel, 1982).

More uneven $\dot{V}/\dot{Q}$ ratios have been reported by Read (1959) in 34 healthy subjects 20 to 69 years of age. Intrapulmonary inert gas distribution has also been shown to become less uniform with increasing age, especially

after the age of 40 years (Greifenstein et al, 1960; Sandqvist and Kjellmer, 1963). The poor matching of ventilation and perfusion with increasing age may be the result of changes in the alveoli and blood vessels, which may lead to lower $\dot{V}/\dot{Q}$ ratios in the bases of the lung with resultant wasted blood flow and higher V/Q ratios at the apices of the lungs, with resultant wasted ventilation. These changes likely contribute to the fall in PaO_2 with increasing age. However, despite this, oxygen saturation is not affected, as will be discussed in the following section (Brischetto et al, 1984); thus in terms of oxygen delivery to the active muscles during exercise, the fall in PaO_2 is not likely to be a significant limiting factor. Arterial PCO_2 during exercise shows little change with age (Brischetto et al, 1984).

1.5.6 Ventilation during Exercise

The ventilatory response to exercise is greater in elderly compared to young subjects (Robinson et al, 1976; DeVries and Adams, 1972; Montoye, 1982). It is well kown that during exercise, ventilation is closely related to CO_2 output. Brischetto et al (1984) observed that for a given increase in $\dot{V}CO_2$, ventilation increased by 29.7 l/min in 10 67-79 year old compared to 25.3 l/min in 22-37 year old healthy subjects. In addition, the minute ventilation at a single level of work (50 W) during steady-state

cycling was significantly greater in the elderly (32.4 ± 1.5 1/min). This occurred even though there were no significant differences in the $\dot{V}CO_2$ between the two groups at this workoad (0.971 ± 0.063 1/min in the elderly and 0.860 ± 0.047 1/min in the young); oxygen saturation was also similar in the two groups (97.3 ± 0.2% in the elderly and 96.5 ± 0.1% in the young). When the change in ventilation relative to $\dot{V}O_2$ was examined the higher ventilatory response in the elderly was evident (30.1 ± 2.2 1/min compared to 24.6 ± 2.1 1/min in the young); no significant differences were observed in the respiratory exchange ratio between the two groups at the common workload (0.95 ± 0.03 in the elderly and 0.93 ± 0.03 in the young). Arterial PCO_2 measurements before and during steady-state cycling at 50 W revealed that despite the higher ventilation during exercise in the elderly, there was no fall in $PaCO_2$ and that exercise remained essentially isocapnic (Brischetto et al, 1984).

These findings suggest that ventilation is less efficient in the elderly. This may be explained by an increased physiological dead space due to areas in the lung with a high $\dot{V}/\dot{Q}$ ratio, as discussed in the preceding sections, which results in an increase in wasted ventilation in the elderly. Increased physiological dead space with increasing age has been reported in a number of studies (Raine and Bishop, 1963; Bradley, 1976);

thus the increase in ventilation during exercise in the elderly appears to be a compensatory mechanism for their wasted ventilation so that exercise remains isocapnic. This was supported by Brischetto's et al (1984) finding of no significant change in $PaCO_2$ before and during steady-state exercise in two elderly subjects selected among those with the highest ventilatory response to exercise. Thus in the elderly, ventilation is less efficient, so a higher ventilatory response during exercise is necessary to maintain appropriate levels of $PaCO_2$.

1.6 Summary of Age-Associated Changes in Peak $\dot{V}O_2$, Haemodynamic and Respiratory Function

A measure of the physical deterioration that occurs with aging is exemplified by a decrease in the peak oxygen intake of about 30-40% between 25 and 65 years. This is associated with a decrease in peak cardiac output (30-35%) and arterio-venous oxygen difference (5-10%). The lower peak cardiac output in the elderly is associated with a lower heart rate (12-14%) and stroke volume (12-14%). Blood flow measurements in elderly subjects are lacking, but available data suggest a lower blood flow in exercising muscles in the elderly associated with a decreased vascular conductance and an increased mean arterial pressure. These

changes are accompanied by declines in breathing capacity and pulmonary gas exchange.

Structural changes in the aging cardiovascular and respiratory systems undoubtedly affect function, but the extent to which function is affected may be modified by physical activity. Structural changes include decreased myocardial and peripheral vascular compliance, loss of alveolar surface area and surrounding pulmonary capillaries and reduction in elastic recoil in the aging lung.

In the following section, age-associated changes in skeletal muscle structure and function will be reviewed.

1.7 Age-Associated Changes in Skeletal Muscle Function

1.7.1 Lean Muscle Mass

Loss of lean muscle mass is a common characteristic of aging skeletal muscle. A decline of 12% in upper leg lean muscle mass determined by computed tomography was reported in 44 males 45 to 69 year of age (Borkan et al, 1983). Tzankoff and Norris (1977) observed a 45% decline in lean muscle mass based on 24-hour creatine excretion in 959 healthy males 20 to 90 years of age who were participants in the Baltimore Longitudinal Study on

Aging. The rate of decline in muscle mass appears to accelerate over the age of 70 years (Grimby et al, 1982); a 15% loss in muscle mass estimated from total body potassium was observed in 24 men and women in their 70s and 80s. The changes in lean muscle mass may be the result of reductions in the number and size of muscle fibres.

1.7.2 Muscle Fibre Number and Size

Recent findings from autopsied whole vastus lateralis muscles in 12 previously healthy males 19 to 37 and 70 to 73 years of age revealed that the size of the muscles of the older individuals was 18% smaller; this could be accounted by a 25% lower total number of muscle fibres (Lexell et al, 1983). No significant difference was found in mean fibre size in the two groups. The reduction in the number of muscle fibres in the elderly was largely due to preferential reduction in type II fibre number. Similar findings were reported by Sato et al (1984) in the pectoralis minor muscle of 200 females 26 to 80 years of age who had surgical resection of chest muscles for breast carcinoma. The reduction in muscle fibre number was significant after the age of 60 years (Sato et al, 1984). The size of the type I muscle fibres increased after the age of 60 years, whereas after the age of 40 years, there was a progressive reduction in the size of type II fibres.

There seems to be general agreement that type I muscle fibres are relatively insensitive to age-associated changes, at least up to the age of 60 years (Lexell et al, 1983; Aniansson et al, 1980). Larsson et al (1978) found no significant change in type I fibre area in muscle biopsies from the vastus lateralis of 55 heathy males 22 to 65 years of age. However, a 33% decrease in type II fibre area was observed in the same group. The findings of Sato et al (1984) in the pectoralis minor muscle are also in agreement with the stability in type I and the reduction in type II fibre area, reported to occur after the age of 40 years.

Beyond 70 years, reductions in fibre size appear to occur in all fibre types but these appear to be most striking in the type II fibres. Tomonaga (1977) obtained biopsies from muscles in the upper and lower limbs and trunk of 79 subjects 60 to 90 years of age. In the 60 to 79 year old group, neuropathic changes such as group atrophy, small dark angulated fibres and target fibres were most prevalent (30%) followed by type II fibre atrophy (28%); over the age of 80 years, the most prevalent change (53%) was type II fibre atrophy (Tomonaga, 1977). The increase in size in the type I fibres reported by Sato et al (1984) in the pectoralis minor was not supported in

studies on the vastus lateralis (Aniansson et al, 1980; Grimby et al, 1982).

The age-associated changes in muscle fibres may be related to some abnormality in the innervating process. Campbell et al (1973) reported a progressive fall in the number of functioning motor units in muscles of the foot of elderly subjects (60-95 years) as compared to controls (3-58 years). Considerable individual variation was found in the denervating process which did not usually commence before the age of 60. Similar findings were reported by Brown (1972) in subjects 13 to 89 years of age. These findings resulted in the proposition that at least part of the reduction in fibre number is due to loss of functioning motor neurons (Campbell et al, 1973; Brown, 1972).

Motor neurons innervating a certain fibre type may be more vulnerable to degeneration, which may at least partly explain the loss of fast twitch neurons (Campbell et al, 1973) and increased proportion of type I fibres (Larsson, 1978). Denervation of fibres may also occur due to disturbances in the micro-circulation of peripheral nerves; a significant correlation was reported between the proportion of type II muscle fibres and the degree of tissue injury in acute ischaemia (Sjostrom et al, 1982) and ischaemic pain during exercise in individuals with peripheral arterial insufficiency (Sjostrom et al, 1980).

These findings suggest that type II fibres may be more susceptible to ischaemia and may explain the preferential loss of these fibres with increasing age.

1.7.3 Capillarization

The changes in capillarization of skeletal muscle with increasing age are not as extensively studied as changes in fibre number and size. Parizkova et al (1971) observed no differences in the number of capillaries per square millimeter or the diffusion distances in the quadriceps muscle of young men (mean 20.7) years) and two subgroups of older men; a physically active group (mean age 72.4 years) and inactive controls (mean age 73.9 years). Comparison between the two groups of active and inactive older subjects revealed similar findings. Aniansson et al (1980) found decreases in the number of capillaries per fibre in males but not in females. Other studies reported similar number of capillaries per muscle fibre between old (Grimby et al, 1982) and young males and females (Anderson and Henriksson, 1977; Nygaard, 1981. Based on these limited experimental data, it would appear that capillarization may be unaffected by aging.

1.7.4 Muscle Function

Muscle function studies have focused primarily on determining the maximal force capabilities and endurance of muscles from individuals of different ages. Larsson (1978) examined the maximum isometric and dynamic strength and endurance in the quadriceps muscles of subjects 10 to 76 years of age using an isokinetic dynamometer. Both isometric and dynamic strength reached a peak by the age of 20 years, remained relatively unchanged up to the age of 49 years and progressively declined from then on (Larsson, 1978). Over the age range of 20 to 65 years, the loss in strength was about 32%, but most of this loss occurred after the age of 50 years (Larsson et al, 1979). In contrast, Vandervoort (1984) reported that 80 to 90% of maximal voluntary static strength of dorsi and plantar flexors is retained up to the age of 79 years with an accelerated decline after the age of 80 years. The reduction in muscle strength with increasing age may be the result of a number of factors; reduced muscle mass and number of muscle fibres (Aniansson et al, 1980); atrophy of type II muscle fibres (Larsson, 1978); loss of functioning motor units (Campbell et al, 1973) and lack of physical activity (Larsson, 1978).

Contrary to the age-associated decline in strength, there is agreement among studies that muscle

endurance, both isometric (ability to sustain a contraction at a given percent of maximum isometric strength) and dynamic (percent decline in peak torque over repeated maximal contractions) do not change significantly with increasing age (Johnson, 1982; Larsson and Karlsson, 1978; Larsson, 1978; Petrofsky et al, 1975). The relative stability of muscle endurance and type I muscle fibres with increasing age has led to the proposition that the two may be causally related (Larsson, 1978).

1.7.5 Summary

Muscle strength appears to be maintained at least up to the fifth decade in life, with moderate declines occurring in the sixth and seventh decades and sharp declines after the age of 80 years. These changes are associated with structural changes in the form of loss of lean muscle mass, reduction in the number and size of muscle fibres, reduction in functioning motor units and a preferential loss of type II fibres. Ability to sustain maximal isometric and dynamic contractions (muscle endurance) does not appear to be affected by aging and may be associated with the relative stability of type I muscle fibres with increasing age.

Thus the changes in muscle function are not as striking as the changes in haemodynamic function, although

both are likely to contribute to the decline in physical work capacity with increasing age.

In the remainder sections in this chapter, previous work on the effects of endurance training on haemodynamic, respiratory and muscle function in elderly subjects will be reviewed.

1.8 Effects of Endurance Training on Peak Oxygen Intake

The capacity of older individuals to respond to endurance training began to be questioned in the 1960s. Hollman (1964) claimed that endurance training after the age of 40, in previously sedentary subjects, resulted in little adaptive changes while after the age of 60 it produced practically no observable effect. This conclusion was reached after studying over 3,000 trained and untrained males and females 6 to 80 years of age. The distinction made by Hollman (1964) was that, in order for endurance training to have any observable effect on the physical work capacity of elderly individuals, it had to begin in youth and continue into the middle and old years. Hollman's (1964) conclusions were suported by Benestad (1965) who found no change in peak oxygen intake in males 70 to 80 years of age after 5 to 6 weeks of endurance training. Over the age of 50 years, the improvement was

less than in younger individuals (Wilmore, 1970; Roskamm, 1967; Kilbom, 1971).

However, the studies of Hartley and Saltin in 1969 challenged these findings (Hartley et al, 1969; Saltin et al, 1969). In these studies, a mean increase of 18% was reported in sedentary healthy males 34 to 63 years after 8 weeks of training.

The longitudinal studies reported by Kasch and associates (Kasch et al, 1973; Kasch and Wallace, 1976; Kasch, 1976) provided further evidence on the trainability of older subjects and challenged the concept that the decline in physical work capacity is an unavoidable occurrence of the aging process.

In these studies, healthy middle-aged men in their 40s and 50s were trained for a variable length of time, from 2 to 10 years, at an intensity corresponding to 60-90% of maximal heart rate. In the 2-year training study, a control group of similar age was included. The major findings from these studies were a 17% increase in peak $\dot{V}O_2$ in the 2-year training group (most of this increase occurring within the first year) with no significant change in the controls; no change in peak $\dot{V}O_2$ was observed in the men who trained over the 10-year period. The expected decline is in the order of 9% (Heath et al, 1981).

An improvement in peak $\dot{V}O_2$ of about 17% was

reported by Tzankoff et al (1972) in untrained men 44 to 66 years of age (mean 54 years) after 25 weeks of a training program consisting of vigorous recreational activities (racket ball games). Maximal ventilation and blood lactate also increased, 17% and 13% respectively with no major change in maximal heart rate. Pollock et al (1971) trained sedentary males in their 40s and 50s using a 20-week walking program at an intensity designed to elicit heart rates in the 135 to 145 beats/min range; this resulted in increases of 28% in peak $\dot{V}O_2$ and 18% in ventilation with no change in peak heart rate. The values at submaximal exercise were not given except for heart rate which was significantly lower. There were no changes in the controls (Pollock et al, 1971).

Training studies that employed previously sedentary subjects over 60 years of age are few. Stamford (1972) found a significant decrease in submaximal heart rate and systolic blood pressure after 12 weeks of training in elderly males in their 70s with no change in the controls; no measurements of peak $\dot{V}O_2$ were obtained. In a controlled study of males and females (mean 70 years) a 38% increase in peak $\dot{V}O_2$ was found in the training group (Barry et al, 1961). Peak heart rate and ventilation increased 11% and 50% respectively with significant decreases at submaximal exercise. No changes were observed in the controls.

De Vries (1969) trained (walk/jog program) 52 to 88 year old sedentary males for 6 to 42 weeks. Heart rate was not allowed to exceed 145 beats/min during training or exercise testing. Modest increases in peak $\dot{V}O_2$ were reported after 6 and 42 weeks of training (about 5% and 8% respectively). Improvement in maximal aerobic capacity of 25-30% was reported in a recent investigation after 12 months of training in subjects in their late 60s (Seals et al, 1984). At maximal exercise, ventilation increased significantly with no change in mean arterial pressure and systemic vascular resistance. The submaximal values in these variables were lower. There were no changes in the controls. Thomas et al (1985) reported a 12% increase in peak $\dot{V}O_2$ in 63 year old males after one year of a walking/jogging program, three times a week, at 70% of heart rate reserve.

Comparing athletes who engage in lifelong intensive training with sedentary subjects of the same age has consistently demonstrated higher peak $\dot{V}O_2$ values in the athletes, in the order of 20 to 30% (Saltin and Grimby, 1968; Astrand and Rodahl, 1970; Andersen and Hermansen, 1965; Grimby and Saltin, 1966). Three case reports of older endurance athletes provide additional evidence that the decline in peak $\dot{V}O_2$ with increasing age may be reduced in individuals who continue to train (Faria et al, 1977;

Maud et al, 1981; Pollock et al, 1974). These individuals had peak VO_2 values of 61 ml/kg/min (60 year old distance runner); 60 ml/kg/min (70 year old marathon runner) and 50 ml/kg/min (70 year old cyclist). These peak VO_2 values are over 50% higher than the predicted value of about 24 to 29 ml/kg/min (Jones et al, 1985) in untrained subjects of the same age.

Pollock et al (1987) recently reported the results of a 10-year follow-up in 24 runners who continued to train over the period of follow-up. Their average age at the time of re-study was 62 years. An interesting finding in this study was that the athletes who had maintained their training mileage and speed had also maintained their peak $\dot{V}O_2$, with a decrease of only 2% over the 10 years. In contrast, the athletes who maintained their training mileage but decreased their speed, experienced a 13% decline in peak $\dot{V}O_2$ over the 10 years of follow-up. Thus an important consideration in training studies in older individuals is not only the frequency and duration of training, but also the intensity of training.

In summary, it would appear that elderly individuals in their 60s can increase their peak $\dot{V}O_2$ in response to endurance training. The reported increases are overall modest, with the exception of the 38% improvement in peak $\dot{V}O_2$ reported by Barry et al (1961). However, as

discussed earlier, part of this change may have been the result of low pre-training values, as in some of their subjects the pre-training test had to be stopped prematurely because of electrocardiographic abnormalities.

The question of whether previously sedentary elderly subjects (over 60 years) can tolerate high intensity training and can respond by similar adaptations in peak $\dot{V}O_2$ is as yet unanswered. Hagberg (1987) concluded that a lower training intensity is necessary in older men and women than in a younger population. He based this conclusion on the findings of improvements in peak $\dot{V}O_2$ in elderly subjects at heart rates as low as 40% of peak $\dot{V}O_2$ (Seals et al, 1984); 57% of peak $\dot{V}O_2$ (Badenhop et al, 1983) and 40 to 50% of peak $\dot{V}O_2$ (DeVries, 1971). However, the observed improvements are modest and as will be discussed in Chapter 2, they are likely the result of the low pre-training fitness levels of the elderly subjects. No study reported training previously sedentary young (20 year old) and elderly (60 year old) subjects at the same period of time, the same frequency and duration and at the same high intensity.

1.9 Effects of Endurance Training on Haemodynamic Function in the Elderly

There are few investigations on the effect of endurance training on haemodynamic function in elderly subjects over 60 years. The majority of the studies have used middle-aged athletes who either continue in competitive sports or remain physically active. A consistent finding is that endurance training in middle-aged athletes, as in young subjects, results in increases in peak cardiac output, which are associated with increases in stroke volume, as peak heart rate changes little. The increase in stroke volume may be at least partly attributable to decreases in afterload, reflected by decreases in systolic blood pressure and vascular resistance, and increases in pre-load, reflected by higher end-diastolic volumes. The extent to which these training adaptations are possible in elderly subjects who had been sedentary for a number of years is not known.

Eight weeks of training in 38 to 55 year old males, which increased their peak $\dot{V}O_2$ by about 14%, resulted in a significant increase in stroke volume and cardiac output at peak exercise with no major change in arterio-venous oxygen difference (Hartley et al, 1969).

These findings were compared to results in younger subjects (mean 23 years) reported in the literature (Saltin et al, 1968; Ekblom et al, 1968). In these studies, the increase in peak oxygen intake could be accounted for to a similar degree by a widening of the arterio-venous oxygen difference and an increase in cardiac output. Thus a major diffference observed between young and old, was in the ability to increase peak arterio-venous oxygen difference which was reported to be diminished in older individuals (Hartley et al, 1969). Saltin et al (1968), reported that training in young, relative to older subjects, resulted in a greater proportion of the total cardiac output to be directed to the working muscles at peak exercise in agreement with Hartley et al (1969).

However, in a recent investigation, Seals et al (1984) found no significant change in peak cardiac output after 12 months of training in 61 to 67 year old subjects. The increase in peak $\dot{V}O_2$ was associated with a significant increase in arterio-venous oxygen difference. Peak cardiac output was estimated by multiplying peak stroke volume (estimated by averaging the stroke volumes during three submaximal work rates) by the heart rate at the time that peak $\dot{V}O_2$ was attained. The estimation of peak stroke volume in this study was based on the assumption that stroke volume tends to increase up to about 40% of peak $\dot{V}O_2$

and levels off thereafter. This assumption is questionable as there is some degree of variability in stroke volume behaviour, which may progressively increase up to 60% of peak VO_2 or higher and either level off or even decrease somewhat from then on. Thus a source or error may have been introduced in their estimation of peak cardiac output.

Niinima and Shephard (1978) determined cardiac output during treadmill exercise using a CO_2 rebreathing method in 8 males 60 to 76 years of age, before and after 11 weeks of endurance training; heart rate varied between 120 and 145 beats/min with some subjects training between 135 and 155 beats/min. A 5.2% increase in peak VO_2 was reported, attributed to a decrease in resting and submaximal heart rate. Training had no effect in this study on peak cardiac output, stroke volume or arteriovenous oxygen difference. The lack of change may relate to the intensity of training, which is not stated, other than the range of training heart rates. It may be that the majority of their subjects trained at the lower intensities, which as will be discussed in chapter 2, will likely limit the extent of the training-induced adaptations.

Comparisons between athletes and sedentary controls reveal higher maximal cardiac outputs and stroke

volumes in the athletes. Young world calibre athletes with a peak $\dot{V}O_2$ over 70 ml/kg/min (71-80 ml/kg/min) had a mean maximal cardiac output of 36.0 l/min as compared to 28.4 l/min in athletes of similar age with a lower peak $\dot{V}O_2$ (64-69 ml/kg/min). Since mean heart rate was similar between the two groups, the difference in maximal cardiac output was due to a higher stroke volume in the top athletes, 189 mls compared to 149 mls (Ekblom and Hermansen, 1968).

1.10 Effects of Endurance Training on Muscle Function in the Elderly

Despite the age-associated changes in skeletal muscle structure reviewed earlier in this chapter, endurance training appears to be associated with a number of improvements in the function of aging skeletal muscle.

1.10.1 Prevention of Muscle Atrophy

Treadmill exercise continued for 1 year resulted in the prevention of atrophy in both type I (soleus) and II fibres (extensor digitorum longus) in aging rats compared to non-exercising controls (Brown, 1985). Similar findings, but largely related to the type II fibres (gastrocnemius), were reported after 5 months of treadmill exercise in female aging rats. The non-exercising controls had a higher percentage of atrophied type II fibres with no

change in the type I fibres between the two groups (Stebbins et al, 1985). A 9% fibre loss was reported in the rat diaphragm compared to a 30% loss in the soleus, although both are slow-twitch muscles. The lower fibre loss in the diaphragm was considered to be due to the continuous use of this muscle.

Grimby et al (1982) reported a decrease in the size of type II fibres in the vastus lateralis muscle of subjects in their 70s and 80s, with no reduction in the type II fibres of the biceps brachii. It was suggested that this may be related to the more physically active biceps brachii during activities of daily living. The effect of endurance training on skeletal muscle in subjects of different ages (up to 58 years) and states of physical training was investigated by Gollnick et al (1972). Both the area and size of type I and II muscle fibres were significantly larger in the trained subjects. These findings point to the beneficial effects of physical exercise in counteracting the age-associated decrease in muscle size.

1.10.2 Improvement in Oxidative Capacity

Suominen et al (1977) reported a significant increase in enzymes associated with aerobic metabolism in sedentary males 56 to 70 years of age after 8 weeks of

physical training. Similar results have been reported by Kiessling et al (1974) after 13 weeks of training in sedentary males 46 to 62 years. The pre-training enzyme levels of these subjects were about half the level found in well-trained athletes of the same age and increased to about 80% after training. Results from other studies confirm these findings (Saltin et al, 1976; Suominen et al, 1977; Orlander and Aniansson, 1981).

Comparisons of succinate dehydrogenase activity (SDH, enzyme involved in aerobic muscle metabolism) fn biopsy samples of the vastus lateralis and deltoid muscles in 74 sedentary males and highly trained athletes (17 to 58 years) revealed higher SDH activity in the athletes (20 to 150%, Gollnick et al, 1972). For both the trained and sedentary subjects, the SDH activity was highest in the most active muscles; this was 2.5-fold greater in the leg muscles of the cyclists compared to that of the sedentary subjects. Similarly, the SDH activity of the deltoid muscle of the canoeists and swimmers was 2.2- and 2.4-fold higher respectively than that of the untrained groups. Muscle glycogen concentrations were also higher in the athletes than those of the sedentary subjects; the muscle that was used most extensively in the sport activity had the highest glycogen content (Gollnick et al, 1972). These findings suggest a localized training effect and an

increase in the aerobic potential of skeletal muscles of trained individuals, at least into the middle years.

Suominen's et al (1977) findings of significant increases after training in the activities of enzymes representing aerobic muscle metabolism in 56 to 70 year old males extend Gollnick's et al (1972) findings, and suggest that training leads to an enhanced capacity for aerobic metabolism at least up to 70 years of age. An interesting finding in the study by Gollnick et al (1972) was that the 2.2-to 3.5-fold increase in the aerobic potential of the muscles of the athletes was not associated with an increase of the same magnitude in peak $\dot{V}O_2$; their peak $\dot{V}O_2$ was only 37% higher compared to the untrained subjects. The findings of the studies reviewed above suggest a large aerobic potential in trained muscles, even after short term training.

In summary, endurance training may lead to significant increases in the oxidative capacity of aging skeletal muscle, which appears to lead to an increased capacity to utilize glycogen aerobically and to oxidize fatty acids; these adaptations may delay the onset of fatigue and result in an increased capacity for physical work. The large aerobic potential of trained muscles speaks against the oxidative capacity of muscle as being a

major limiting factor in the capacity to utilize oxygen during maximal work.

1.11 Summary

The capacity to exercise, expressed as the peak power output or peak $\dot{V}O_2$, depends on a complex interplay between many factors related to the cardiovascular, respiratory and musculoskeletal systems. The function of these systems declines with increasing age, either because of structural changes or reductions in functional capacity.

Structural changes include an age-related increase in myocardial fibrosis and a decrease in the capillary-fibre ratio within the myocardium; the aging arterial wall becomes progressively less compliant associated with a reduction in elastin and an increase in colagen; elastic recoil in the aging lung declines and there may be loss of alveolar surface area; skeletal muscle mass declines and there is selective atrophy of type II fibres.

Observable functional changes that may parallel these structural changes in aging individuals, include a decrease in peak cardiac output and heart rate, and an increase in blood pressure; vascular conductance decreases and there may be a reduction in blood flow to exercsing muscles; ventilation/perfusion mismatching becomes

exaggerated, with impairment in gas exchange; muscle strength and the capacity to generate power decreases progressively in aging skeletal muscle. Most of these changes are similar in extent, with declines in function of about 1% per year.

Despite these changes, physical activity appears to have a favourable effect on maximal exercise capacity as shown in the higher peak $\dot{V}O_2$ values of master athletes and older individuals engaged in heavy physical work as part of their daily lives. Presumably, improvements are brought about by adaptations in the functional, rather than the structural changes associated with aging. Thus by comparing the training-related responses in old and young subjects, we hoped to identify the functional changes that may be amenable to improvement with short-term endurance training.

The following chapter will outline the background studies that led to the work described in this thesis, some relevant considerations related to training studies in elderly individuals and the objectives of this investigation.

2. SCOPE OF THE PROBLEM AND STUDY OBJECTIVES

2.1 Introduction

In light of previous work discussed in the Historical Overview chapter, the extent of the decline in maximal exercise is variable, ranging between 0.20 and 1.04 ml/kg/min (tables 1 and 2). The variability in these findings is to a large extent dependent on whether the study was cross-sectional or longitudinal; whether the subjects were well-trained, physically active or sedentary, whether elderly subjects (over 60 years of age) were included; and if included, whether they were allowed to exercise to maximal symptom-limited levels. These issues together with differences in testing procedures make comparisons between the studies difficult.

Therefore, to examine the decline in exercise capacity with increasing age, we undertook a cross-sectional study of 100 healthy males and females 15 to 70 years of age. The primary purpose of this study was to establish normal values in exercise capacity for subjects in the same population as those who took part in the training study using the same testing procedures. This provided the baseline for comparison with the subjects in the training study, thus avoiding the difficulties of

cross-comparisons with work reported in the literature. In the following sections, the cross-sectional study will be reviewed as it forms the background on which the work described in this thesis was based.

2.2 Review of Cross-Sectional Study

Fifty male and 50 female healthy volunteers were recruited from the local community to provide as wide a distribution of body height and level of habitual physical activity as possible in each of 5 age groups, 15-24, 25-34, 35-44, 45-54 and 55+ (Appendix 1). Past and present competitive athletes were excluded as were subjects on medication, with known cardiac or respiratory disease and systemic hypertension (resting blood pressure > 150/90 mmHg). Habitual physical activity was graded on the basis of a questionnaire into category 1 (exercising less than 1 hr/wk), category 2 (exercising 1-3 hrs/wk), category 3 (exercising 3-6 hrs/wk) and category 4 (exercising more than 6 hrs/wk). Over half of the subjects (64%) exercised only 3 hrs or less per week (category 2) with 54% reporting sedentary occupations. Nine percent reported exercising more than 6 hrs/week (category 4) and 11% were in occupations requiring mostly standing and walking. Submaximal and maximal measurements of $\dot{V}O_2$, $\dot{V}CO_2$, respiratory exchange ratio, ventilation, heart rate

and blood pressure were obtained during a progressive incremental cycle ergometer test beginning at 100 kpm/min (16.3 w) and increasing by 100 kpm/min at the end of each minute. Since the subjects were not accustomed to heavy physical exertion, a $\dot{V}O_2$ plateau was not a requirement. Thus the mean of the highest three measurements of oxygen intake at maximum effort - indicated by the subject's inability to maintain the required pedalling frequency - was considered to be that subject's peak $\dot{V}O_2$. The test procedure and methods are described in Appendix 1.

On a different day, measurements of maximal peak power, total work and fatigue (percent decline in average power over the duration of the test) were obtained during a 30 second maximal cycling exercise on an isokinetic ergometer. The velocity of pedalling was controlled by a 3 hp motor and an electrical speed controller at 60 rev/min (same as the velocity used during the incremental test). The subject was required to pedal with maximal effort, under constant encouragement, for a total of 30 seconds. The torque exerted on the pedals was recorded via strain gauges bonded to the pedal cranks and sampled by a laboratory computer at 10 ms intervals (Appendix 2).

The decline in peak $\dot{V}O_2$ with increasing age observed in this study was in agreement with results

reported in the literature, (Heath et al, 1981; Dehn and Bruce, 1972; Drinkwater et al, 1975). As shown in figure 2, the highest peak $\dot{V}O_2$ values were achieved between 20 and 30 years with a gradual decline from then on. The relationship in the male subjects between peak $\dot{V}O_2$ and age (years) was expressed in the following equation:

$$\text{Peak } \dot{V}O_2 \text{ (ml/kg/min)} = 55 - 0.44 \text{ (age)}$$

According to this equation, the predicted peak $\dot{V}O_2$ of a 25 year-old male is 44.0 ml/kg/min and that of a 65 year-old is 26.4 ml/kg/min (40% difference over 40 years). A finding of interest was the close relationship ($r = 0.89$ $p < 0.001$, figure 3) between total work achieved in 30 seconds and peak $\dot{V}O_2$. Results of multiple regression analysis revealed that total work (a composite function of maximal power and the fatigue index) was the variable most closely related to peak $\dot{V}O_2$ ($p < 0.001$). Furthermore, the decline in total work (Wtot) with increasing age was similar in extent to that observed in peak $\dot{V}O_2$ (figure 4). The equation expressing this relationship in the male subjects was:

$$\text{Wtot (kJ)} = 20.8 - 0.14 \text{ (age)}$$

According to this equation, the predicted value for the total work achieved over the 30 second test is 17.4 kJ for a 25 year-old male and 11.9 kJ for a 65 year-old male (32% difference over 40 years).

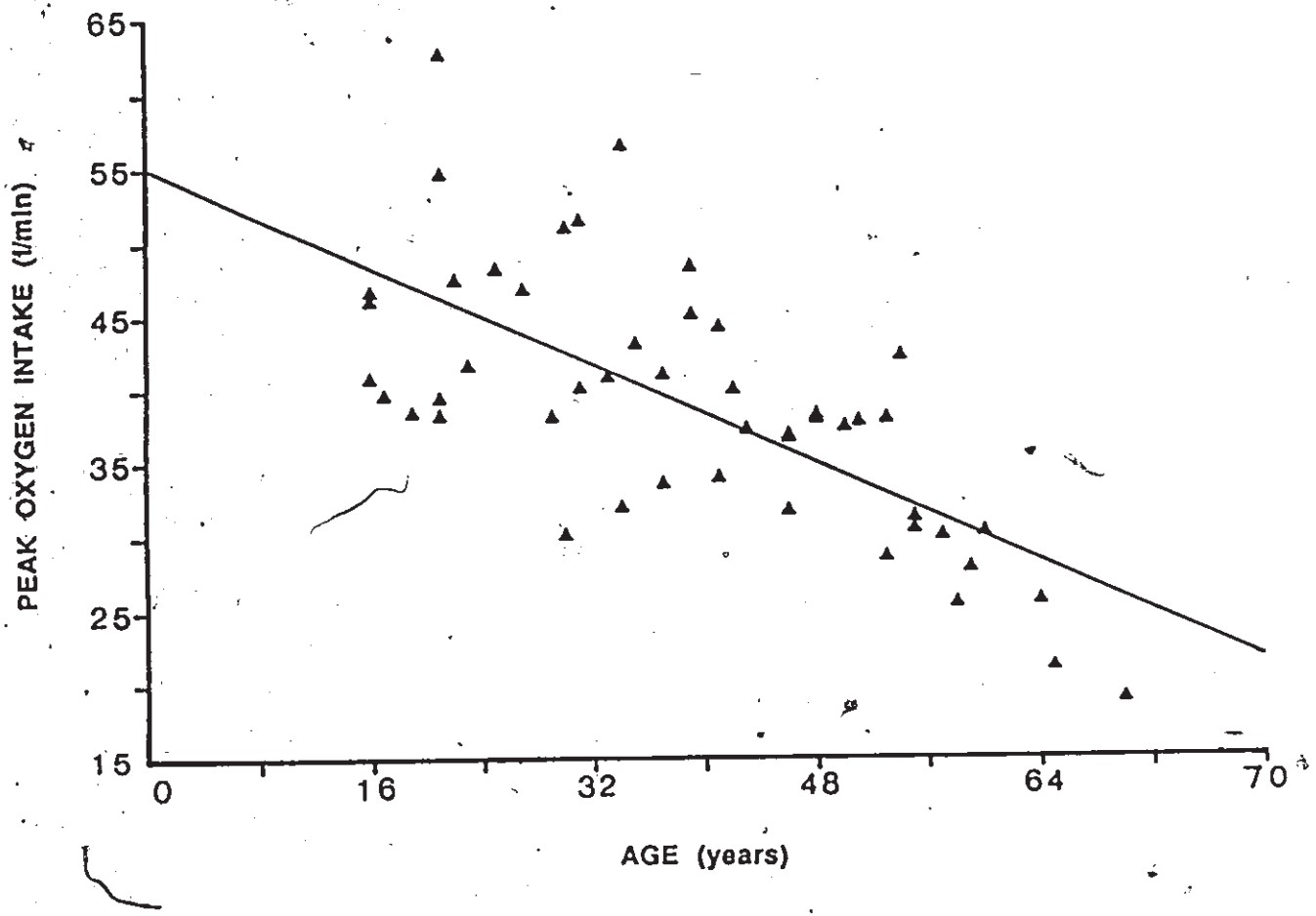


Figure 2. Peak oxygen intake during progressive incremental exercise related to age in males 15 to 70 years of age.

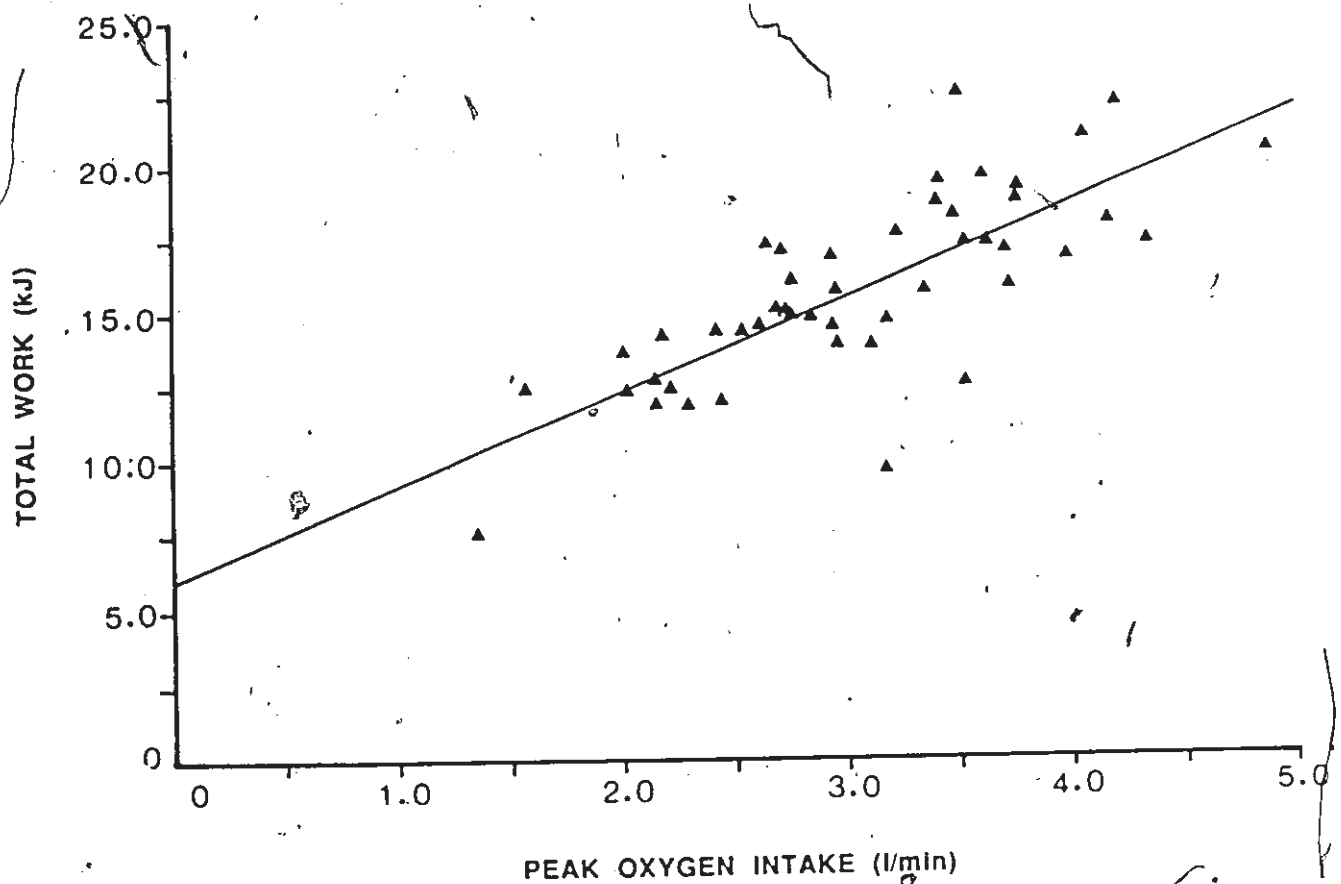


Figure 3. Total work in 30 seconds of maximal isokinetic cycling related to peak oxygen intake in males 15 to 70 years of age.

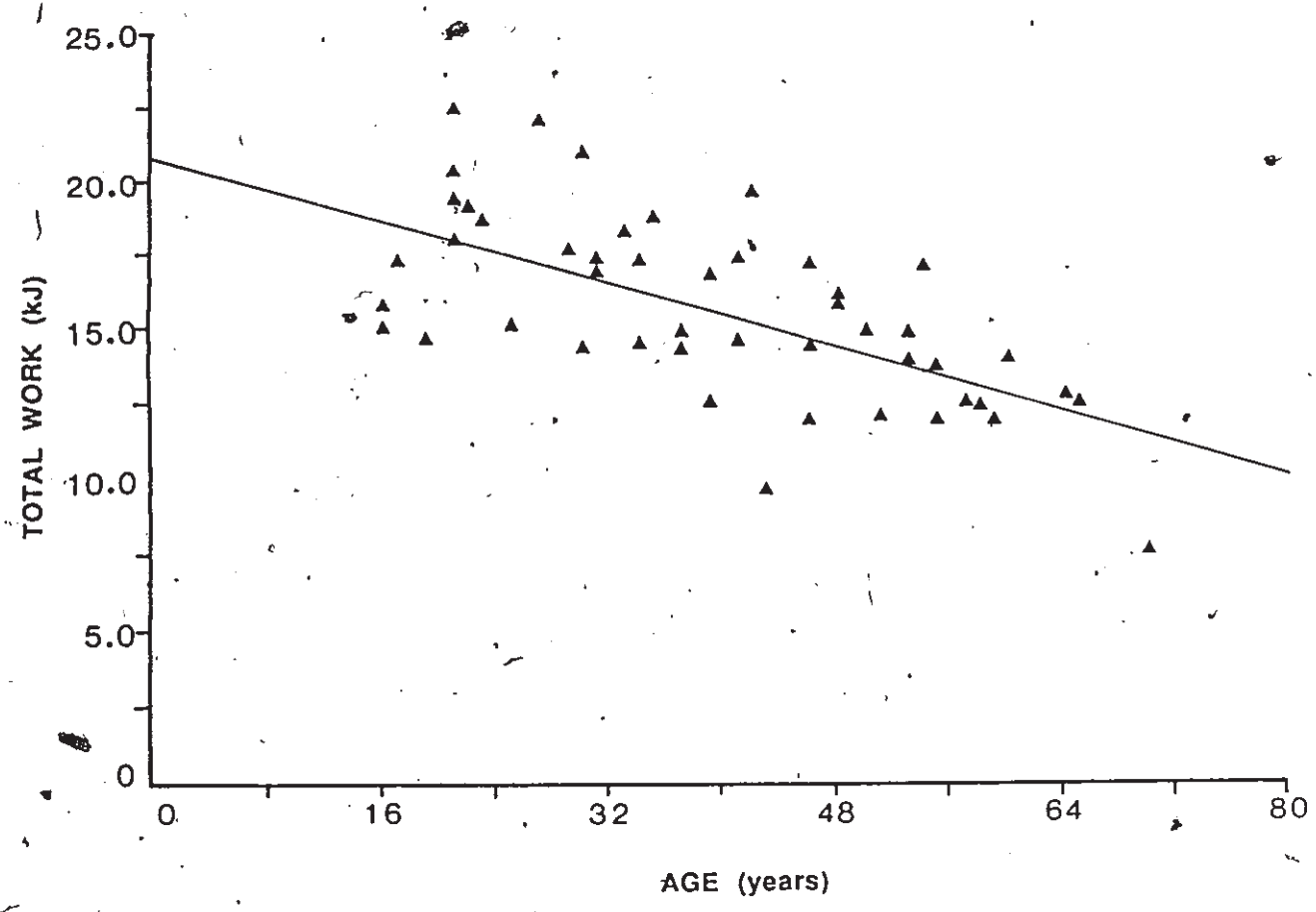


Figure 4. Total work in 30 seconds of maximal isokinetic cycling at 60 rpm related to age in males 15 to 70 years of age.

It was also of interest that the general classification of subjects into the four broad categories of leisure activity was sufficient to differentiate the subjects into 4 categories of "fitness"; those with the highest peak $\dot{V}O_2$ values (mean 3.10 $\pm$ 1.22 l/min) reported exercising more than 6 hrs/wk, followed by those who exercised 3-6 hrs/wk (mean peak $\dot{V}O_2$ 2.84 $\pm$ 0.85), 1-3 hrs/wk (mean peak $\dot{V}O_2$ 2.15 $\pm$ 0.76 l/min) and those who were totally sedentary and had the lowest peak $\dot{V}O_2$ values (mean 1.97 $\pm$ 0.80 l/min).

These findings raised two general questions: Can endurance training increase maximal exercise capacity in sedentary elderly subjects, and to what extent do cardiorespiratory and peripheral muscle measures contribute to any training-associated change in peak $\dot{V}O_2$?

In light of previous work discussed in Chapter 1, two general experimental models have been used to examine the role of endurance training in counteracting the decline in maximal exercise capacity with aging; comparative studies of athletes or individuals involved in life-long physical activity and sedentary subjects who undertake a physical activity program for a specified period of time.

2.3 The Aging Athlete or Physically Active Subject

Comparing athletes with sedentary subjects of the same age (Saltin and Grimby, 1968; Astrand and Rodahl, 1970; Andersen and Hermansen, 1965; Grimby and Saltin, 1966) offers an attractive experimental model as it provides two clearly distinguishable populations of well-trained and sedentary individuals. However, aging athletes are not representative of the general aging population. Their high exercise capacity may be a result of intensive training, general endowment, lean body build, a healthy lifestyle or a combination of these factors and it is difficult to isolate the role of endurance training alone. These considerations may apply to a lesser extent in individuals who engage in life-long physical activity, although not at a competitive level.

2.4 The Aging Untrained Subject

The majority of studies on sedentary elderly individuals have employed subjects in their middle years (Hartley et al, 1969; Saltin et al, 1969; Kasch et al, 1973; Kasch and Wallace, 1976; Kasch, 1976; Tzankoff et al, 1972). Training studies in subjects over 60 years of age are few, results are conflicting and direct comparisons with young subjects who undertake the same training program and testing procedures are lacking. Another major

limitation in these studies is the intensity of the training program which is generally low to moderate.

2.5 Intensity and Duration of Training

The magnitude of change in peak $\dot{V}O_2$ is determined by the intensity, frequency and duration of training (Hickson et al, 1981; Pollock et al, 1975; Davies and Knibbs, 1971; De Vries, 1971; Kilbom, 1971; Shephard, 1968). However, in training studies of older subjects (over 60 years of age) there is a reluctance to use high intensity training programs, over 75% of peak $\dot{V}O_2$, or training heart rates over 135 beats/min. Furthermore, the duration of training is relatively brief (6-8 weeks, Benestad, 1965; DeVries, 1971; Suominen et al, 1977; Suominen et al, 1977).

Seals et al (1984) trained male and female subjects in their sixties for six months at low intensity (heart rate <120 beats/min), and six months at high intensity (75-85% of maximal heart rate) reaching a mean training heart rate of 156 beats/min during the final two months of the program. High intensity training resulted in a significantly greater increase in peak $\dot{V}O_2$ compared to the low intensity program (18% versus 12%).

2.5.1 Safety of Exercise Training and Testing

The primary reason for the reluctance to exercise older subjects at high intensities relates to issues of safety. Some of these fears are not unfounded as the risk that exercise may provoke a musculoskeletal injury increases in older individuals, particularly if they have been sedentary over a number of years (Kilbom et al, 1969; Mann et al, 1969). A high prevalence of musculoskeletal injuries (sore muscles, joints and tendons) was reported by Tzankoff et al (1972) in their 44-66 year old subjects, largely as a result of jogging. After the jogging requirement was withdrawn and training consisted mainly of racket ball games, the injuries were eliminated. However, such high prevalence of injuries is an exception rather than the rule in studies of this nature.

With regard to the safety of maximal exercise tests, Sidney and Shephard (1977) reported no ill effects in 55 maximal tests in subjects aged 60-83 years. There was one episode of ventricular tachycardia which subsided spontaneously within a few minutes. The risk of a fatal event during exercise testing in subjects of all ages, including some with coronary heart disease, was reported to be one in 10,000 (Cumming, 1976). A recent survey of 1,375 centres (600,000 exercise tests) reported the risk for a fatal event to be 0.5 deaths per 10,000 exercise tests, 70%

of which were maximum symptom-limited tests. The total of all complications (arrhythmias, infarctions and deaths) was 8.86 per 10,000 exercise tests (Stuart and Ellestad, 1980). No difference of risk was found between maximal and submaximal effort in over 170,000 tests (Rochmis and Blackburn, 1971; Stuart and Ellestad, 1980). In view of these findings, it appears that the risk of appropriately monitored exercise and exercise testing is very small.

2.6 Initial Level of Fitness

The adaptive changes that occur as a result of training are inversely related to the pre-training fitness of the subjects (Shephard, 1968; Kilbom, 1971; Stamford, 1972; De Vries, 1970; Sidney and Shephard, 1978; Tzankoff et al, 1972; Saltin, 1969). Subjects with high initial peak VO_2 values (over 45 ml/kg/min) training for an average period of sixteen weeks improved their peak VO_2 by 4% to 12%. In contrast, subjects of the same age with low initial peak VO_2 values (less than 35 ml/kg/min) training at the same intensity and frequency, showed a greater improvement varying between 22% and 43% (Pollock, 1973). Thus the initial level of fitness of the subjects is an important confounding variable that needs to be considered in comparative studies of young and elderly subjects.

For reasons of safety, Sidney and Shephard (1978) did not perform measurements of peak $\dot{V}O_2$ until their elderly subjects (60-83 years) had participated in a seven week conditioning program. The increase with training from week 7 to the final week 21 was only 5%. They speculated that the largest gain in peak $\dot{V}O_2$ may have occurred during the initial seven week period of conditioning, which was not included in their overall evaluation of the program. On the other hand, Barry et al (1961) found a 38% increase in peak $\dot{V}O_2$ (1.20 to 1.66 l/min) in their 55 to 78 year old subjects after twelve weeks of training starting at low (<120 beats/min) and progressing to high intensity (approximately 130 beats/min). However, their subjects reached low pre-training peak heart rate values (126 beats/min) with a $\dot{V}O_2$ max of only 1.20 l/min. Furthermore, in some subjects, the exercise test had to be stopped because of electrocardiographic abnormalities, and maximal values may not have been reached. Therefore, high initial fitness, combined with a brief duration of training, may result in no gains in peak $\dot{V}O_2$, which may be the case in the study reported by Benestad (1965). The opposite would be true if the initial level of fitness is very low (1.20 l/min), as in the case of Barry et al (1961).

On the basis of the above considerations, we recruited two groups of young and old healthy males who were sedentary and willing to undergo intensive endurance training. Results from the cross-sectional study reviewed earlier in this chapter enabled us to compare the initial level of fitness in the young and elderly subjects with a larger sample from the same population, who underwent the same testing procedures. On the basis of this, we were able to show that the two groups of young and elderly had a comparable initial level of fitness, approximately 80% of the predicted average for their age. We also attempted to eliminate as many as possible of the confounding variables related to body size, smoking history, level of leisure and occupational activity and motivation to exercise, by careful selection of the subjects on the basis of a pre-recruitment interview.

Many factors contribute to the increase in exercise capacity with endurance training; changes in heart rate, stroke volume, cardiac output, arterio-venous oxygen difference and changes in the peripheral circulation and muscle. These were reviewed in the previous chapter. Although there is some information in the literature on training-associated changes in older individuals, the conflicting results, lack of direct comparisons between elderly and young subjects who have comparable initial

levels of fitness and trained at the same intensity, confound the results. Furthermore, we do not know whether over 60 year-old subjects can tolerate high intensity training and whether the adaptations after training are similar in quantity or quality with those expected in young subjects.

The present study was an attempt to provide some answers to the above questions by examining the effect of high intensity endurance training in two groups of elderly and young subjects.

2.7 Purpose of the Study

The purpose of this study was to examine the effects of aging on the training-associated changes in the cardiorespiratory responses to progressive incremental exercise and in the capacity to generate power in short-term dynamic exercise.

2.8 Hypothesis

It is hypothesized that:

High intensity endurance training will result in a significant improvement in exercise capacity, determined by increases in peak $\dot{V}O_2$, in 60-70 and 20-30 year old healthy, sedentary males. However, the magnitude of the improvement

in the elderly will be less than that in the young subjects.

2.9 Objectives of the Study

The study objectives were to:

(i) determine the extent to which healthy older subjects (over 60 years) who had been sedentary over a number of years are capable of increasing their maximal exercise capacity.

(ii) determine the feasibility of using high intensity endurance training in previously sedentary over 60 year old males.

(iii) compare the magnitude of change in short-term power output and maximal exercise capacity (peak $\dot{V}O_2$) and related cardiorespiratory variables in young and old subjects who had undergone high intensity endurance training of the same duration, frequency and intensity.

(iv) examine the relative role of different functional characteristics in any training-induced change in peak $\dot{V}O_2$ in young and old subjects. These include cardiac output, heart rate, stroke volume, blood pressure, vascular conductance, tissue oxygen extraction and the muscle's ability to generate

and maintain power during short-term dynamic exercise.

In the following chapter the methods and procedures of the study will be described.

3. METHODS AND PROCEDURES

3.1 Introduction

A group of young (20-30 years) and a group of older (60-70 years) healthy, sedentary males participated in this study, which employed a pre- to post-test repeated measures design. The main reasons for selecting these age groups were:

(i) Physical work capacity reaches a peak by the age of 20-30 years. This was based on results reported in the literature and our own cross-sectional study.

(ii) We wanted to obtain an age difference of at least forty years between the young and old.

(iii) The likelihood of recruiting subjects in their sixties who were not on medication and were willing to undergo intensive endurance training was greater than in subjects in their seventies.

In the following sections, the subjects, testing procedures, training program, and statistical analysis will be described.

3.2 Subjects

Twenty-four previously sedentary, healthy males, twelve aged 20-30 years, and twelve aged 60-70 years, entered the study. Two of the young subjects withdrew from the study for job-related reasons (transfer to another city). A total of ten young and twelve older subjects completed the study. Descriptive characteristics of the subjects are given in Appendices 3 and 4. The subjects were recruited by advertising in the local newspaper. A total of 217 volunteers responded to the call for subjects. Each volunteer was contacted by telephone to determine suitability for the study and approximate height and weight.

The admission criteria to the study were freedom from known cardiovascular, respiratory and other diseases, no medications known to influence exercise capacity, blood pressure below 150/90 mmHg, normal spirometry and resting electrocardiogram, sedentary lifestyle and motivation to complete the study.

The subjects were similar in body weight, the young subjects ranged in weight between 57.0 and 85.0 kg (mean = 71.0 kg); the older subjects ranged in weight between 61.0 and 89.0 kg (mean = 73.0 kg). The young subjects were taller; range 163.0 - 184.0 cm (mean =

176.0cm) compared to 162.0 - 178.0 cm (mean = 171.0) in the older group.

The occupational status of the subjects was similar, the majority being in sales, managerial or professional occupations. Six of the older subjects were retired and two of the young subjects were university students.

Upon admission to the study, each subject completed two questionnaires to determine general health status and level of physical activity and smoking history (Appendices 5 and 6). The older subjects were required to have a medical examination by their own physician (Appendix 7) which was accompanied by a brief description of the study (Appendix 8). Some subjects had minor musculoskeletal problems, mostly knee and back problems, but these did not inhibit physical activity. The majority were non-smokers, although seven of the older subjects had smoked when they were younger and stopped smoking 10-24 years ago. No subject was involved in any physical activity such as walking or sports for more than 1-3 hrs/week (leisure activity level 1, Appendix 3).

The study objectives, procedures and possible risks were described in detail and signed informed consent was obtained (Appendix 9). The study was approved by the university ethics committee. No remuneration was offered.

3.3 Experimental Protocol

Two tests were performed by each subject: a multistage progressive exercise test on a cycle ergometer and a 30 second maximal power output test on an isokinetic cycle ergometer. The multistage progressive exercise test was always performed first to determine the suitability of the subjects to undertake vigorous exercise training. The subjects returned to the laboratory on two different days to perform brief maximal bouts of cycling on an isokinetic cycle ergometer at two pedal velocities. The order with which the maximal power output tests were performed was determined at random. The testing procedures were repeated at the end of the twelve week training program.

3.3.1 Progressive Incremental Exercise Test

The subjects came to the laboratory at least two hours after their last meal. The progressive incremental exercise test was performed on a calibrated electrically braked cycle ergometer (Siemens Elema, model EM 370). The saddle and valve mouthpiece were placed in comfortable positions. The initial power setting for the younger subjects was 200 kpm/min (33 W) with successive increases of 200 kpm/min every 4 minutes. For the older subjects, the initial power setting was 150 kpm/min (25 W) with increases of 150 kpm/min every 4 minutes. The

subjects exercised to a symptom-limited maximal power output, defined as the point at which they were unable to maintain the required pedalling frequency (60 rpm) despite maximal effort. The criteria used for a test to be stopped by the observers are listed in Appendix 10. No test was stopped because of these criteria.

During the last minute of each power output, measurements of oxygen intake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$), ventilation ($\dot{V}E$), respiratory exchange ratio (RER), blood pressure and heart rate were obtained, followed by cardiac output measurements during the last 15-20 seconds of each power output.

Oxygen intake was measured by the open circuit method using a calibrated automated exercise metabolic system (Sensor Medics Horizon MMC, Anaheim, CA). The MMC Horizon System is a microprocessor-controlled system (Norton, 1982). Oxygen is measured with a temperature-controlled, fast-response paramagnetic oxygen analyzer (Beckman OM-11). Carbon dioxide is measured with an infrared CO_2 analyzer (Beckman LB_2). The system is calibrated with a zero gas (100% N_2) and a precision calibration gas (4% CO_2 and 16% O_2). It checks for noise and drift by sampling room air and prints a warning when gain, zero or room air analyses are outside normal limits ($\pm$ 0.05%). Volume was calibrated by delivering a fixed volume (1 litre) at varying flow rates. The accuracy of

the gas analyses is within $\pm$ 0.05% for O_2 and $\pm$ 0.03% for CO_2. The equations used to calculate oxygen intake, carbon dioxide output and ventilation are given in Appendix 11.

Heart rate was measured from five consecutive R to R intervals on the electrocardiogram (lead V5). In the older subjects, a 12-lead electrocardiogram was also obtained at intervals throughout the exercise test for monitoring purposes. Blood pressure was measured by auscultation.

3.3.2 Cardiac Output Determination

Cardiac output was measured by the indirect Fick principle using the CO_2 rebreathing equilibration method (Jones et al, 1967; Jones and Rebuck, 1973). Suitable mixtures of CO_2 in O_2 were rebreathed from a 5-litre anaesthesia bag to obtain equilibration of PCO_2 in the lung-bag system. During the last minute of each power output, after measurements of VO_2, heart rate and blood pressure were obtained, the subject was switched to the rebreathing bag at the end of an expiration and was encouraged to take several rapid deep breaths to ensure good mixing between the gas in the bag and that in the lungs. The volume of the rebreathing mixture was about 1.5 to 2 times the subject's tidal volume. The CO_2 concentration was typically between 9% and 16%, depending

on the subject's carbon dioxide output, with higher concentrations used during heavy exercise when mixed venous CO_2 ($P\bar{v}CO_2$) is high. Rebreathing was maintained up to about 15 seconds. The equilibrium PCO_2 was accepted if it remained at a stable level ($\pm$ 1 mmHg) for several breaths before recirculation (12 seconds).

The $P\bar{v}CO_2$ was derived from the equilibrium PCO_2 ($P_{EQ}CO_2$; Appendix 12). In cases where an equilibrium was not attained, $P\bar{v}CO_2$ was estimated by extrapolation of a line joining the points for expired PCO_2 at 8 and 12 seconds of rebreathing to the point at 20 seconds, as described by Jones and Rebuck (1973). This value was found to lie within $\pm$ 2 mmHg of the equilibrium value (Jones and Rebuck, 1973). Arterial PCO_2 ($PaCO_2$) was estimated from end-tidal PCO_2 ($P_{ET}CO_2$; Appendix 12).

Cardiac output ($\dot{Q}$) was calculated by applying the Fick principle to CO_2 as follows:

$$\dot{Q}\ (l/min) = \dot{V}CO_2 \,/\, (C\bar{v}CO_2 - CaCO_2)$$

where $C\bar{v}CO_2$ and $CaCO_2$ are derived from the corresponding PCO_2 values through the CO_2 dissociation curve for oxygenated blood (Appendix 12).

The peak cardiac output was estimated, as the subjects could not tolerate high CO_2 concentrations during rebreathing at peak exercise. This was done by calculating the regression equation expressing the $\dot{Q}/\dot{V}O_2$ relationship

during submaximal exercise for each individual subject and extrapolating the cardiac output value at the maximal power output. The extrapolation extended over one power output commonly and in only two cases did it extend over more than two power outputs; in terms of $\dot{V}O_2$, the extrapolation extended for a mean of 325 ml/min in the old and 505 ml/min in the young subjects. The mean number of cardiac output measurements available for each subject was 4.6 in the old and 5.3 in the young group.

Stroke volume (V_S), arterio-venous oxygen $(a-\bar{v})O_2$ difference and vascular conductance (g) were calculated as follows:

$$V_S \text{ (ml)} = \dot{Q} \; / \; fc$$

$$(a-\bar{v})\ O_2 \text{ diff. (ml/min)} = \dot{V}O_2/\dot{Q}$$

$$g \text{ (l/mmHg)} = \dot{Q}/Pa$$

where Pa is the mean arterial pressure (Appendix 12).

3.3.3 Maximal 30 Second Isokinetic Test

The 30 second maximal cycling test was carried out on an isokinetic ergometer at pedalling frequencies of 60 and 110 rpm. The isokinetic ergometer was extensively tested by McCartney (1983). Continuous measurement of the maximum torque applied to the cranks of the ergometer can be obtained at a pre-determined constant velocity, maintained by a 3 hp electric constant torque motor (Boston

Gear 18300C) and a speed controller (SC1154B). A wide range of pedalling speeds is provided, 13 to 166 rpm, by a sprocket assembly containing a unidirectional clutch, which prevents the pedals from turning until the subject begins to cycle.

The forces applied to the pedal cranks are detected by matched pairs of strain gauges (Micro-measurements CEA-06-250 VW - 350) attached to each pedal crank. The signals from the strain gauges are amplified and transmitted to a Hewlett Packard chart recorder (7700 series) through a slip ring and a Wheatstone bridge circuit. On-line calculation of the results is performed by a laboratory computer (Digital Equipment Corporation), which samples torque at 10 millisecond intervals and performs integration with respect to time to get the average power for each pedal revolution. Calibration of the system by suspending weights from each pedal up to 136 kg (1334.7 N) over several weeks revealed a mean coefficient of variation of 1.5% (McCartney, 1983). Calibration was performed before each test by applying a known weight on each pedal locked in the 90° position.

A standard procedure was followed for each test. The optimal saddle height was selected for each subject (slight knee flexion with pedal vertically down) and the feet were secured to the pedals by toe clips and

straps. A restraining harness around the hips prevented lateral and forward movement and ensured that the major contribution to power output was from the quadriceps muscles. The test was explained to the subject and a brief practice run was allowed. When the motor was activated, the subject was allowed a brief time (2-3 seconds) to catch up to the speed setting before exerting maximal force on the pedals. Constant encouragement was given to the subject during the test to exert maximal effort. This procedure was performed at a pedal velocity of 60 and 110 rpm on different days.

Venous blood was sampled from the ante-cubital vein 5 minutes after completing the exercise for measurement of plasma lactate concentration. This time was found to be adequate for plasma lactate concentration to reach a steady-state level (McCartney, 1983). Samples for lactate were centrifuged within 5 minutes and the plasma was stored at -20^{o} C. Plasma lactate was determined by a fluoroscopic enzymatic technique (Hohorst, 1965).

Measurements of average power and work for each pedal revolution, the cumulative work and the fatigue index over the 30 second test were calculated on-line by a laboratory computer. Peak torque (Nm) is the product of the mass (kg) multiplied by the gravitational constant (9.807 m. s^{-2}) and the length of the lever arm (m). Work

(J) is equal to the product of the constant angular velocity (rad. s^{-1}) and the integral of torque with respect to time. Peak power (W) was calculated from peak torque multiplied by the angular velocity of the pedal cranks. Average power is equal to the work divided by the time taken for the pedal crank to rotate 360°. The decline in power during the 30 seconds of the test (fatigue index) was calculated as follows:

$$\text{fatigue index} = \frac{\text{initial mean power} - \text{final mean power} \times 100}{\text{initial mean power}}$$

where initial power was the mean of the three pedal strokes representing the highest observed power output and final power was the mean of the last three pedal strokes of the test.

The repeatability of the measurements was examined by duplicate tests within 2-4 days (at pre-test). Each subject repeated the test at one pedal velocity chosen at random. No significant differences were found between the initial (day 1) and repeat measurements (day 2) in either group; young group: mean maximal average power (W) 768.7 ± 166.3 on day 1, 746.0 ± 133.8 on day 2 (difference of 3%); mean total work (kJ) 15.9 ± 2.0 on day 1, 16.2 ± 1.7 on day 2 (difference of 2%); mean fatigue index (%) 46.2 ± 20.3 on day 1, 45.7 ± 20.7 on day 2 (difference of 1%); older group: mean maximal average power (W) 536.1 ±

102.1 on day 1, 560.6 ± 139.8 on day 2 (difference of 4%); mean total work (kJ) 11.4 ± 1.4 on day 1, 11.7 ± 1.2 on day 2 (difference of 3%); mean fatigue index (%) 44.4 ± 18.3 on day 1, 45.4 ± 19.0 on day 2 (difference of 2%). The values of the repeated measurements for each subject are given in Appendices 13 and 14. Similar findings were reported by McCartney (1983) in healthy young males performing the test over a wide range of pedal velocities.

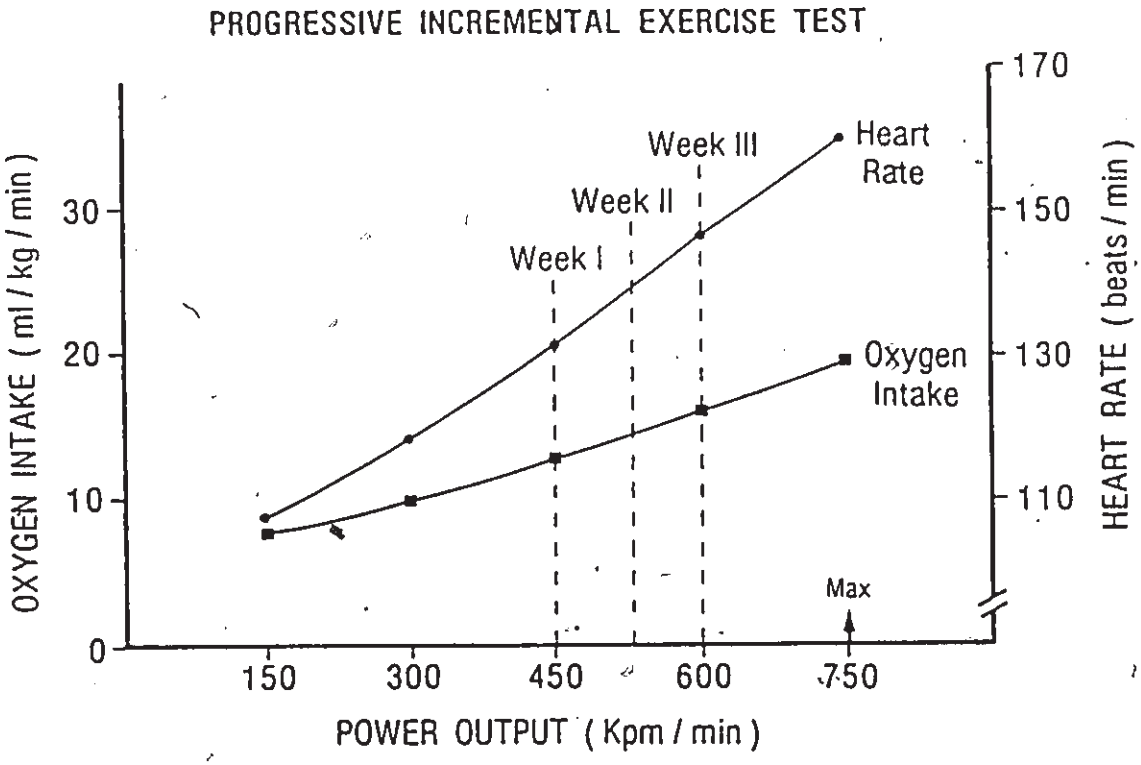
3.4 The Training Program

Upon completion of the tests, the subjects were brought together for an orientation session. The training procedures were explained and instruction on the monitoring of the radial and/or carotid pulse was given. The subjects were required to exercise for approximately one hour excluding warm-up and cool-down sessions, 3 days/week for twelve weeks. An interval training regime was used consisting of exercise bouts of 5-minute duration on a cycle ergometer adjusted to elicit a heart rate corresponding to about 65% of peak $\dot{V}O_2$, followed by recovery periods at a lower intensity (45% of peak $\dot{V}O_2$) during the first week. The intensity was gradually increased so that by the third week, the subjects were training at a heart rate corresponding to 85% of their peak

$\dot{V}O_2$, followed by recovery periods at a heart rate corresponding to 65% of peak $\dot{V}O_2$ (Figure 5).

The training protocol used was similar to the one described by Hickson et al (1977). The 5-minute exercise bouts were separated initially by 5-minute active recovery periods which decreased gradually to 3 minutes in duration. This was repeated initially five times, progressing to seven times by the third week of training. Heart rate at the end of each exercise period was recorded in the subjects' log book. Mean training heart rates were 166 beats/min in the young and 140 beats/min in the older group.

In the elderly subjects, the mean initial training power output was about 400 kpm/min progressing to 600 kpm/min by the third week of training when the prescribed training heart rate was reached. During the remaining nine weeks of training, the heart rate remained the same while the power output required to maintain the prescribed heart rate increased progressively to 900 kpm/min by the final week of training (an increase of 50%; Appendix 15). In the young subjects, the mean initial training power output was about 600 kpm/min progressing to 900 kpm/min by the third week of training when the prescribed training heart rate was reached. During the remaining nine weeks of training, the heart rate remained



EXERCISE PRESCRIPTION

	Intensity (%)	Time (mins)	Repetitions	VO_2 (ml/kg/min)	Power Output (kpm/min)	Training Heart Rate (beats/min)	(beats/10s)
Week I	65	5	5	12.9	450	125-135	21-23
	45	5	5		200	105-115	17-19
Week II	75	5	6	14.6	550	135-145	22-24
	55	4	6		250	110-120	18-20
Week III	85	5	7	16.6	600	140-150	23-25
	65	3	7		300	115-125	19-21

Figure 5. Example of exercise prescription based on a progressive incremental exercise test (60 year old male).

the same while the power output required to maintain the prescribed heart rate increased progressively to 1200 kpm/min by the final week of training (an increase of 33%; Appendix 15).

The maximal power output attained by the two groups during the pre-training progressive incremental exercise test was 725 kpm/min in the old and 1080 kpm/min in the young group. This value was exceeded on the average by the sixth week of training in the elderly and the ninth week of training in the young subjects (Appendix 15). By the end of the training program, the subjects were training at a mean power output that was 24% higher in the elderly and 11% higher in the young than the maximal power output attained during the initial incremental exercise test.

Each subject completed all required training sessions; if a subject was unable to attend at the scheduled time, alternative arrangements were made so that the required number of training sessions was attained with minimal interruption. Attendance and performance at the sessions were closely monitored and reviewed with each subject. The subjects tolerated the training program well and there were no injuries or other adverse effects.

3.5 Statistical Analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS) on a CYBER 6600 computer. Mean values and standard error of the mean for peak $\dot{V}O_2$, $\dot{V}E$, $\dot{V}CO_2$, RER, cardiac output, heart rate, systolic blood pressure, conductance, stroke volume and arterio-venous oxygen difference were calculated for each group before and after training. Mean submaximal values ($\pm$ S,E.M.) were also calculated for each group before and after training; these represent the mean value for each exercise level completed by all subjects. Means and standard error of the mean were calculated for the 30 second maximal isokinetic test at both pedalling frequencies; average power, total work, fatigue index and plasma lactate concentration. Analysis of variance was used to test for differences in the following comparisons:

(i) before training: old compared to young

(ii) old: before compared to after training

(iii) young: before compared to after training

(iv) after training: old compared to young.

These analyses permitted examination of both aging and training-induced differences in the capacity to perform maximal physical work and to generate short-term maximal power output. The relationships between peak $\dot{V}O_2$ and peripheral muscle measures (average power, total work,

fatigue index and plasma lactate) were tested with Pearson correlations.

Linear regression analysis by the method of least squares was used to examine the relationship between changes in cardiac output, heart rate and systolic blood pressure with increasing $\dot{V}O_2$ during the incremental exercise test.

To test for differences between regression equations, a three-part process was employed. In the first part, the relationship is modelled as a single straight line which assumes that no differences exist between the two groups or between pre- and post-training. The second model assumes that the two regression lines run parallel to each other (same slope). The third model assumes that there are two regression lines with different slopes. We tested which model best fitted the data by using the standard F statistic during each of these steps. To test if the model was appropriate, residual plots were used as described by Draper and Smith (1966). This analysis permits examination of the differences between the actual value and the predicted value from the regression line. No residuals fell outside $\pm$ 3 SDs.

4. RESULTS

4.1 Introduction

This study examined the effects of aging and exercise and the interaction between them during both progressive incremental and short-term maximal isokinetic exercise chosen to quantify changes in oxygen transport mechanisms and muscle function. The pre-training data served as the baseline for comparison with the post-training data. This permitted the examination of training-associated changes within and between the two groups. We were able to examine the extent of the training-associated change in the young and older subjects, to quantify the differences between them before and after training, and to identify mechanisms likely to contribute to the pre-training differences between the young and old, and to any training-associated adaptations.

Initially, the pre-training changes during exercise in the elderly and young subjects and comparisons between them will be presented to identify the age-related differences between the two groups. The post-training changes during exercise in each group and comparisons between them will be presented next, to indicate the

training-associated adaptations and differences in these between the elderly and young subjects. The individual values both pre- and post-training in the two groups during the progressive incremental exercise and short-term maximal power output tests are given in Appendices 16 and 17.

4.2 Before Training

4.2.1 Response of Elderly Subjects to Progressive Incremental and Maximal Isokinetic Exercise

The older subjects achieved a maximal power output during the progressive exercise test of 725 ± 31.2 kpm/min (Figure 6). Their oxygen intake increased linearly throughout the incremental exercise to reach a peak VO_2 of 1.599 ± 0.073 l/min. This was accompanied by a linear increase in cardiac output to a peak value of 12.7 ± 0.61 l/min. The cardiac output-oxygen intake relationship was expressed by the following equation (Figure 7):

$$\text{Cardiac output (l/min)} = 4.5 + 5.4\ (\dot{V}O_2)$$

$$r = 0.95 \qquad \text{S.E.M.} = 1.21$$

The arterio-venous oxygen difference increased during exercise from 80.6 ± 3.58 ml/l at the initial power output to 126.5 ± 3.70 ml/l at peak VO_2.

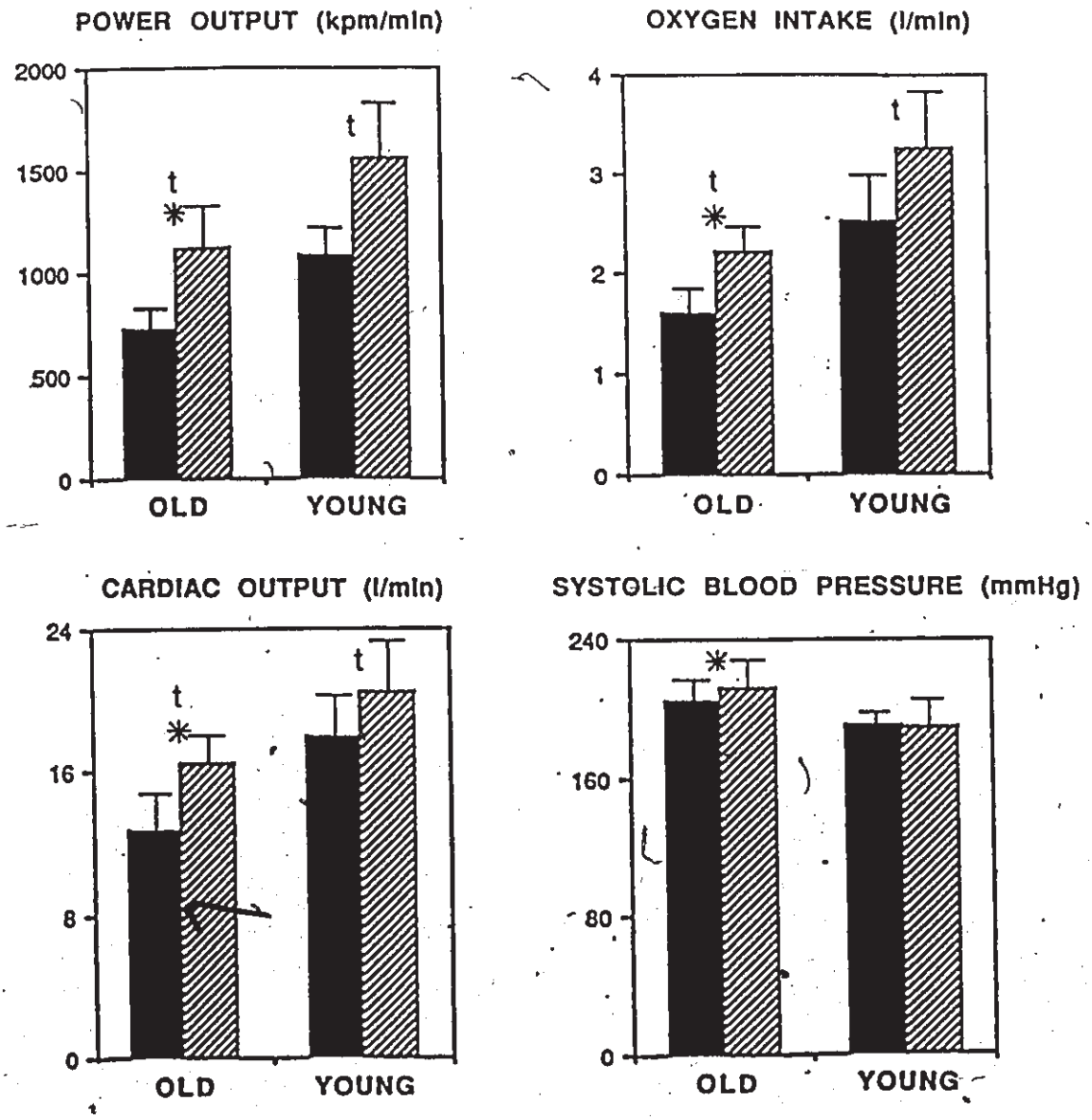


Figure 6. Mean values + SD at peak exercise before (▬) and after (▨) training in 60-70 and 20-30 year old males.

* Old significantly different compared to young at both pre- and post-training.
t Significant differences between pre- and post-training.

Peak cardiac output was estimated for each subject from individual regression equations during submaximal exercise.

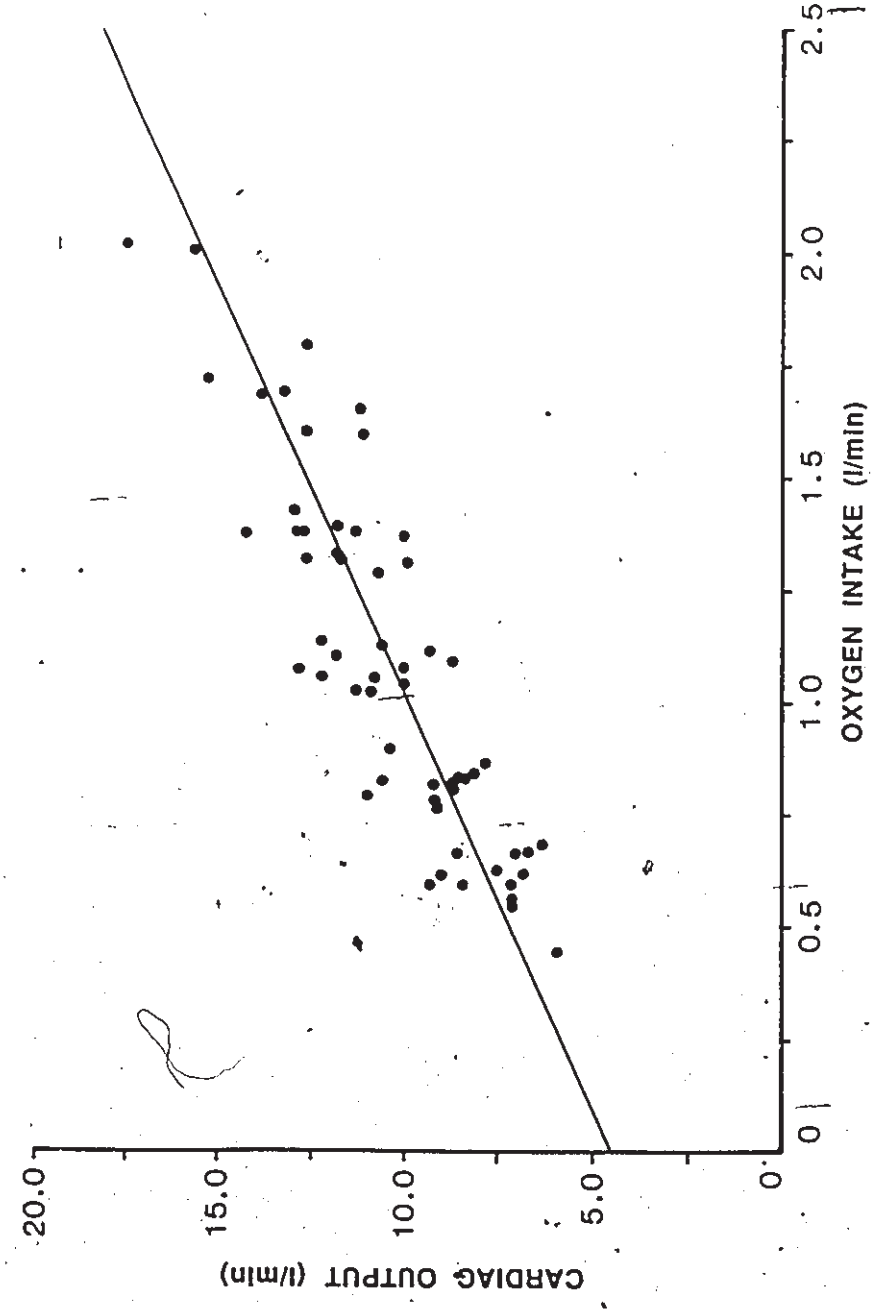


Figure 7. Before Training: Cardiac output related to oxygen intake during progressive incremental exercise in 60-70 year old males.

Heart rate increased linearly with increasing $\dot{V}O_2$ to a maximal value of 161 $\pm$ 3.79 beats/min (Figure 8):

$$\text{Heart rate (beats/min)} = 57.2 + 62.7\ (\dot{V}O_2)$$

$$r = 0.95 \qquad \text{S.E.M.} = 13.5$$

There was a gradual increase in stroke volume during exercise up to about 65% of peak $\dot{V}O_2$ (88.2 $\pm$ 4.68 ml) with a progressive decrease as exercise approached maximal values. Systolic blood pressure increased linearly to a maximal value of 205 $\pm$ 3.58 mmHg (Figure 6). This relationship is expressed in the following equation (Figure 9):

$$\text{Systolic blood pressure (mmHg/l)} = 131.6 + 44.1\ (\dot{V}O_2)$$

$$r = 0.90 \qquad \text{S.E.M.} = 18.9$$

Ventilation and carbon dioxide output increased during exercise to maximal values of 65.4 $\pm$ 3.96 l/min and 1.801 $\pm$ 0.096 l/min respectively. At peak exercise the respiratory exchange ratio was 1.12 $\pm$ 0.02.

During the 30 second maximal isokinetic exercise at 60 rpm, the older subjects achieved an initial maximal average power output of 468.8 $\pm$ 15.43 W and total work of 11.4 $\pm$ 0.40 kJ. The post-exercise plasma lactate concentration was 9.0 $\pm$ 0.69 mmoles/l (Figure 10). The power generated by the leg muscles declined progressively

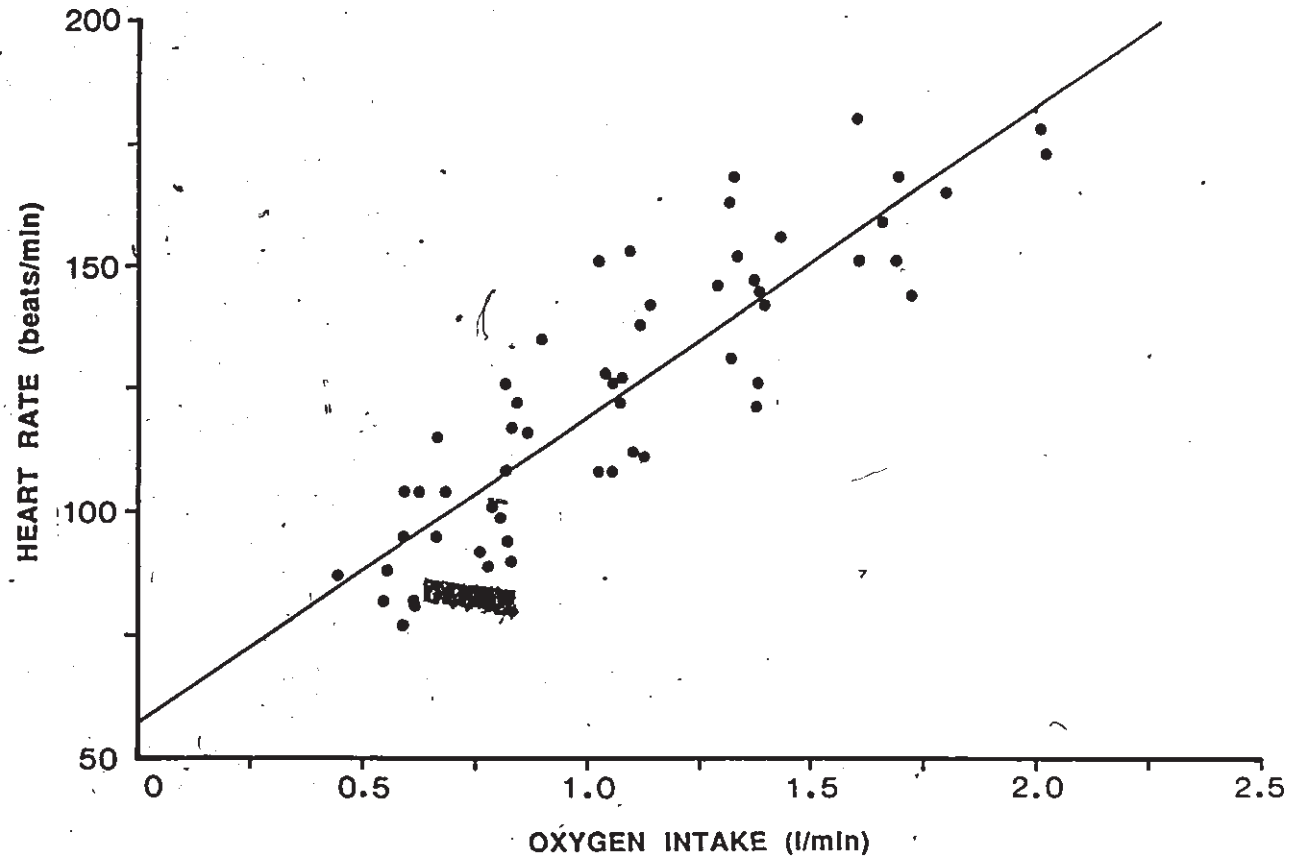


Figure 8. Before Training: Heart rate related to oxygen intake during progressive incremental exercise in 60-70 year old males.

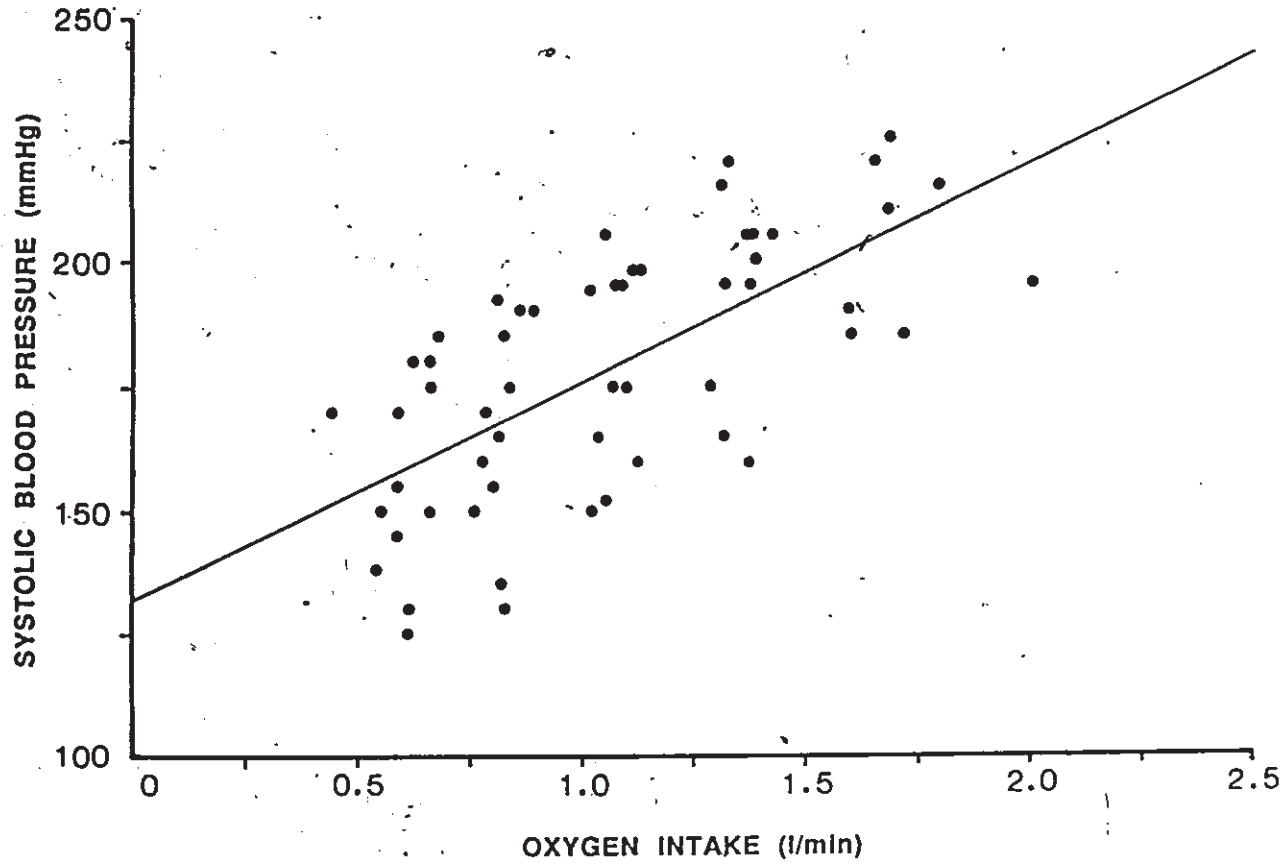


Figure 9. Before Training: Systolic blood pressure related to oxygen intake during progressive incremental exercise in 60-70 year old males.

during the test so that the percent decline from the beginning to the end of the test (fatigue index) was 34.3 ± 10.1%.

At the higher pedalling frequency (110 rpm), maximal average power was 625 ± 33.38 W (Figure 11). Total work (11.2 ± 0.40 kJ) was similar to the value attained at 60 rpm. The plasma lactate concentration was similar (10.5 ± 0.61 mmoles/l), but the fatigue index was almost twice the value observed at 60 rpm (59.0 ± 2.40%).

A significant relationship was found between the ability to generate and maintain power during 30 seconds of maximal cycling and the peak $\dot{V}O_2$ attained during progressive incremental exercise (Figures 12 and 13). This relationship was greater at the pedalling speed of 60 rpm, which was the same speed maintained for both the short-term power output and incremental exercise tests; for maximal average power the correlation coefficient was $r = 0.84$ and for total work $r = 0.87$. The corresponding values at 110 rpm were $r = 0.65$ and $r = 0.78$. The fatigue index related negatively with peak $\dot{V}O_2$, but the relationship was not significant; $r = -0.34$ at 60 rpm and $r = -0.24$ at 110 rpm.

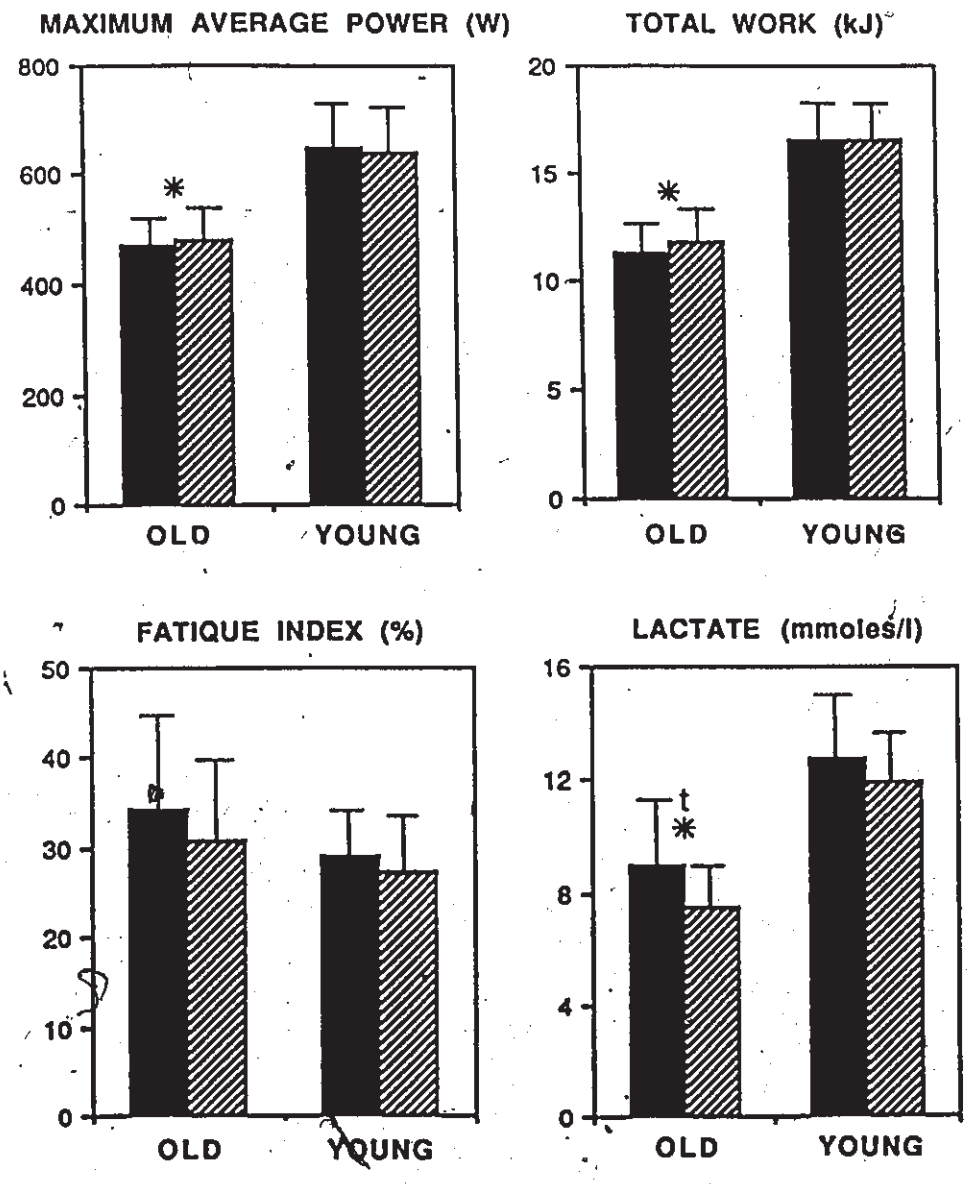


Figure 10. Mean values + SD during 30 seconds of maximal isokinetic cycling at 60 rpm before (■) and after (▨) training in 60-70 and 20-30 year old males.

* Old significantly different compared to young at both pre- and post-training.
t Significant differences between pre- and post-training.

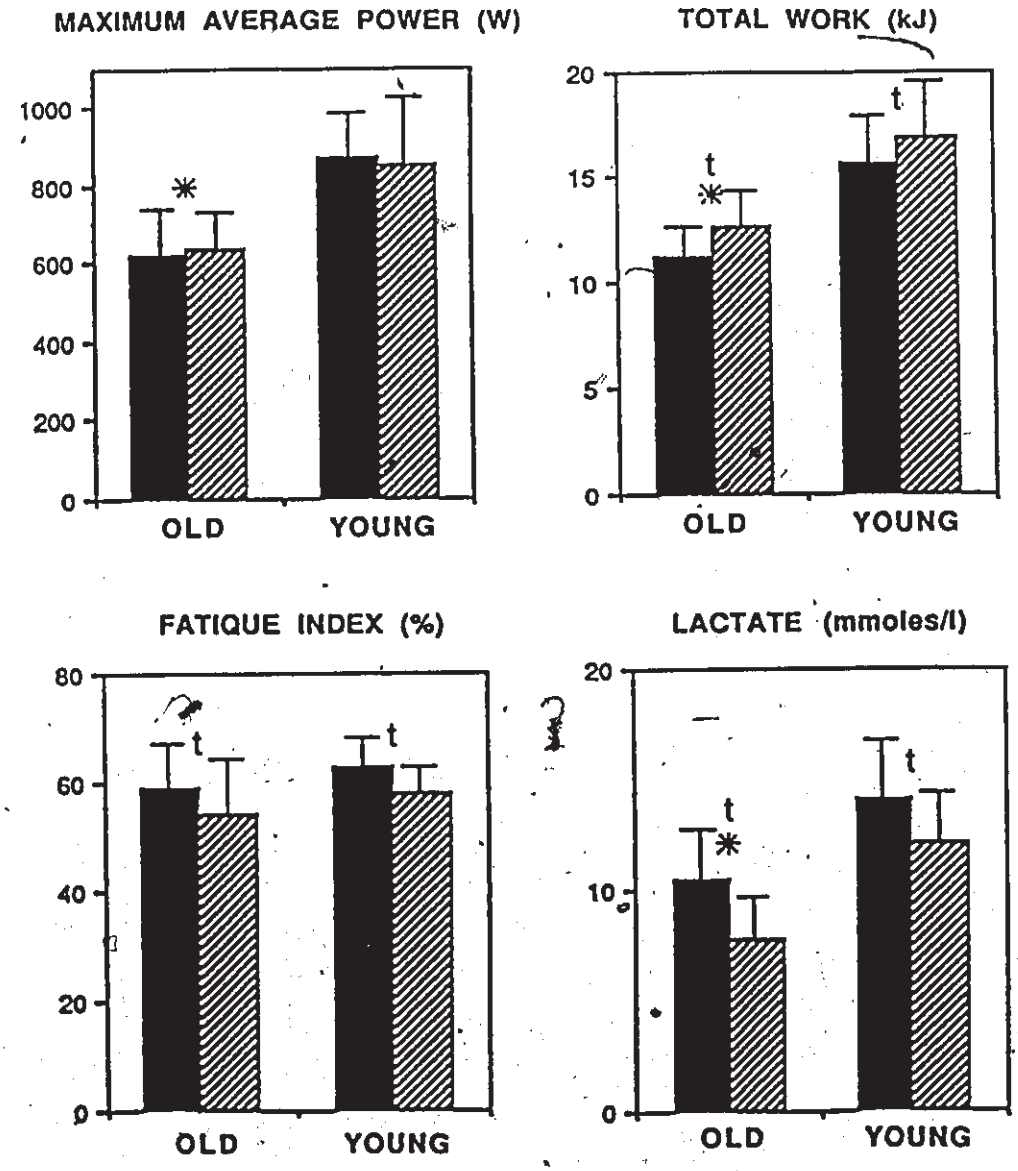


Figure 11. Mean values + SD during 30 seconds of maximal isokinetic cycling at 110 rpm before (■) and after (▨) training in 60–70 and 20–30 year old males.

* Old significantly different compared to young at both pre- and post-training.
t Significant differences between pre- and post-training.

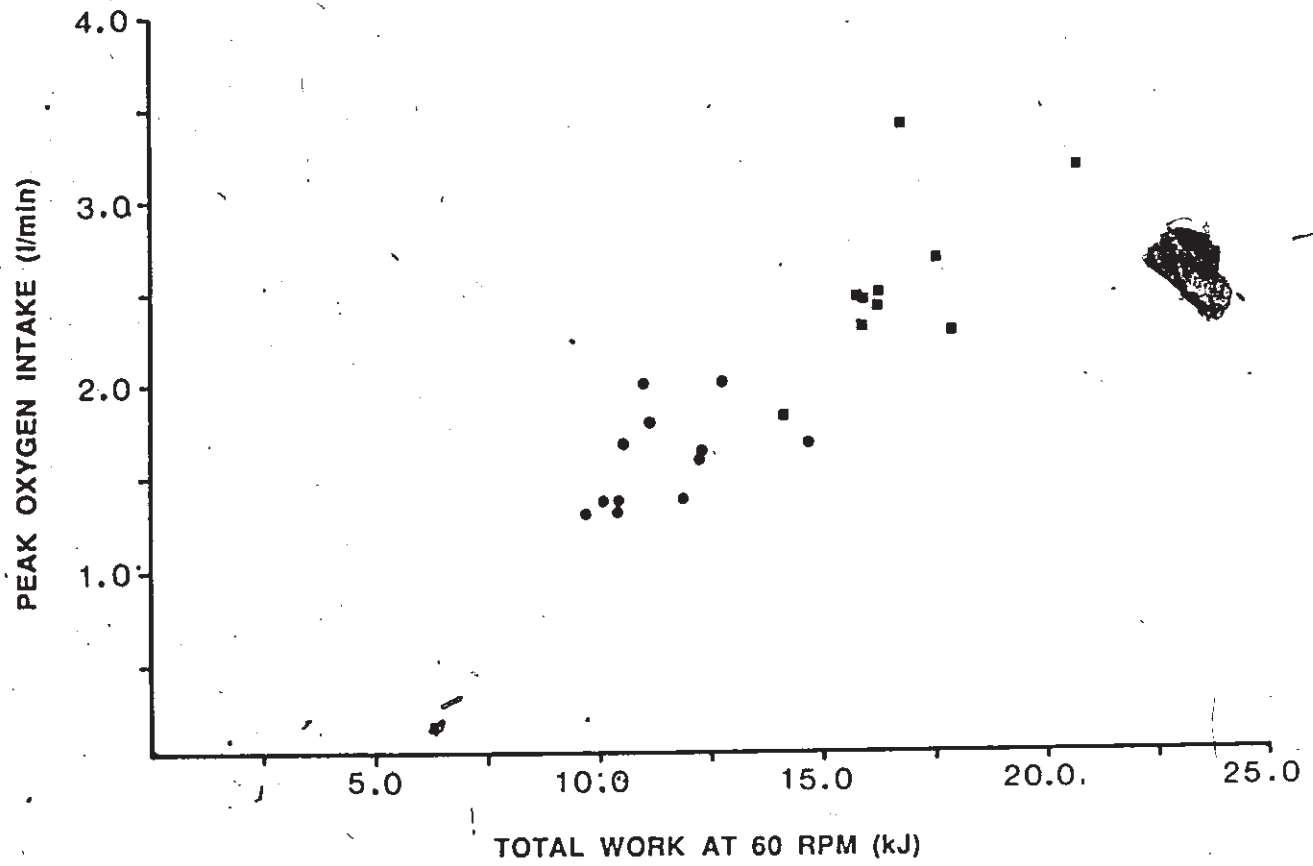


Figure 12. Before Training: Total work in 30 seconds (60 rpm) related to peak oxygen intake during progressive incremental exercise in 60-70 (•) and 20-30 (■) year old males.

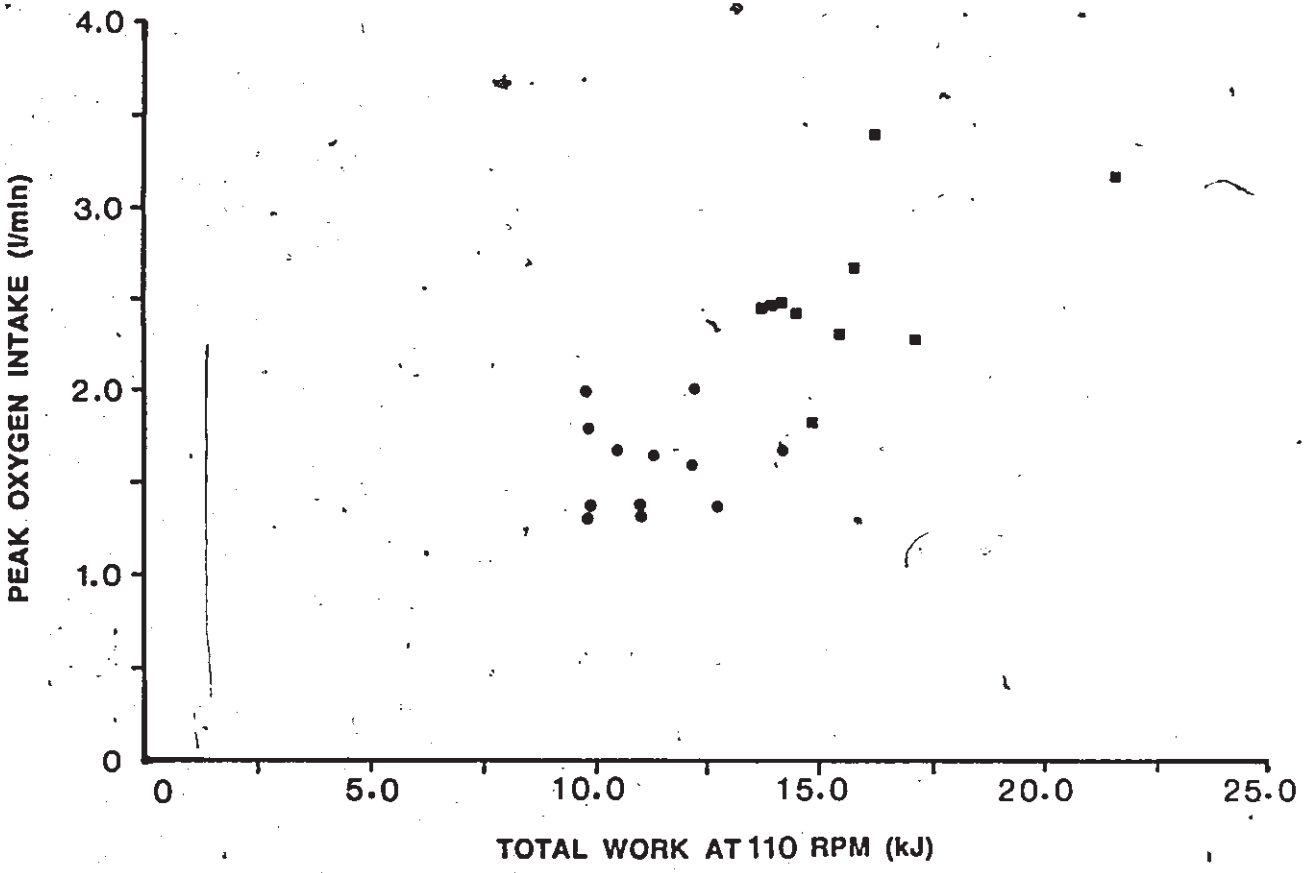


Figure 13. Before Training: Total work in 30 seconds (110 rpm) related to peak oxygen intake during progressive incremental exercise in 60-70 (•) and 20-30 (■) year old males.

4.2.2 Response of Young Subjects to Progressive Incremental and Maximal Isokinetic Exercise

The young subjects achieved a maximal power output of 1080 ± 61.08 kpm/min (Figure 6). Their oxygen intake increased linearly throughout the progressive incremental exercise to reach a peak $\dot{V}O_2$ of 2.536 ± 0.141 l/min. This was accompanied by a linear increase in cardiac output to a peak value of 18.0 ± 0.73 l/min. The cardiac output-oxygen intake relationship was expressed by the following equation (Figure 14):

$$\text{Cardiac output (l/min)} = 6.6 + 4.7\ (\dot{V}O_2)$$

$$r = 0.93 \qquad \text{S.E.M.} = 1.34$$

The arterio-venous oxygen difference increased during exercise from 82.3 ± 2.53 ml/l at the initial power output to 140.4 ± 3.96 ml/l at peak $\dot{V}O_2$.

Heart rate increased linearly with increasing $\dot{V}O_2$ to a maximal value of 192.2 ± 2.28 beats/min (Figure 15):

$$\text{Heart rate (beats/min)} = 74.2 + 44.8\ (\dot{V}O_2)$$

$$r = 0.95 \qquad \text{S.E.M.} = 14.0$$

There was a gradual increase in stroke volume during exercise up to about 60% of peak $\dot{V}O_2$ (99.6 ± 4.34 ml) with a progressive decline as exercise approached maximal values.

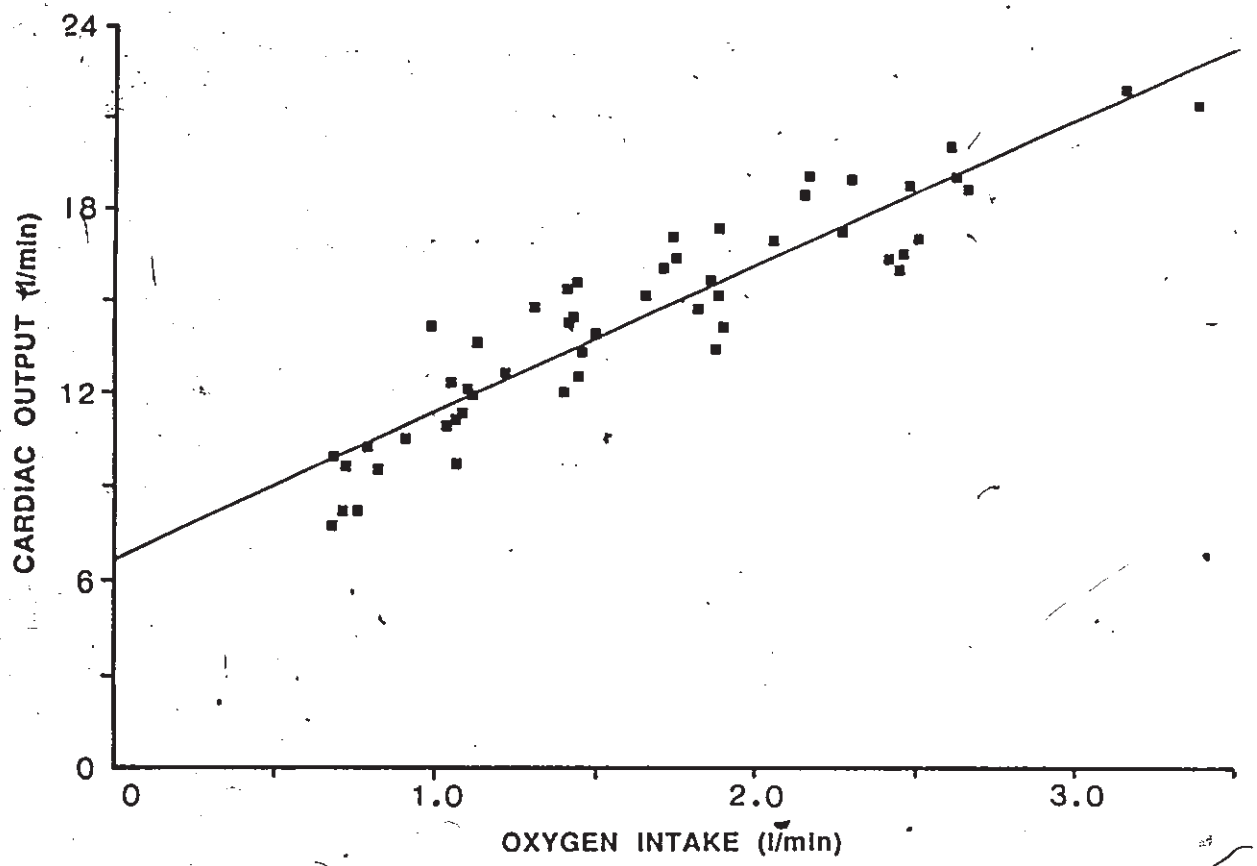


Figure 14. Before Training: Cardiac output related to oxygen intake during progressive incremental exercise in 20-30 year old males.

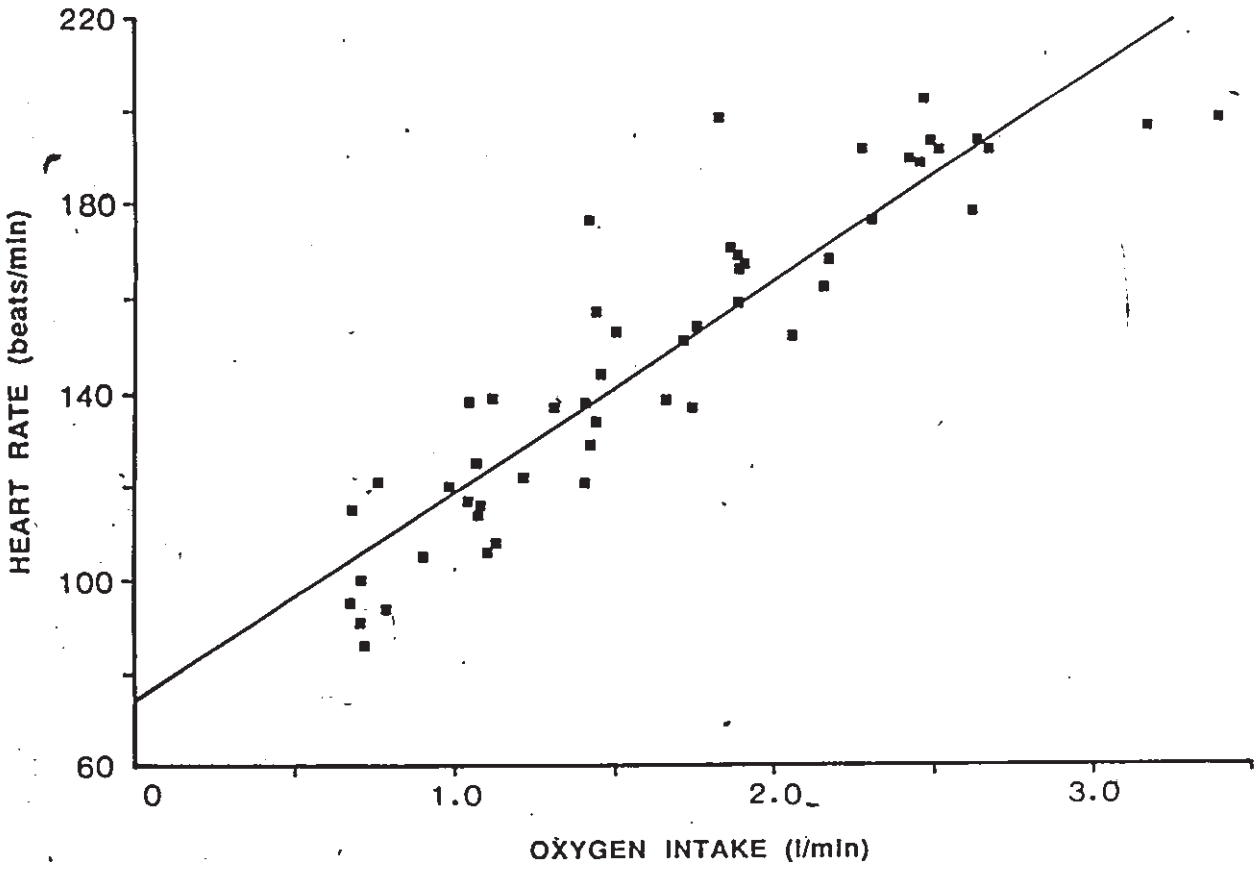


Figure 15. Before Training: Heart rate related to oxygen intake during progressive incremental exercise in 20-30 year old males.

Systolic blood pressure increased linearly to a maximal value of 190.2 ± 2.31 mmHg. This relationship was expressed by the following equation (Figure 16):

$$\text{Systolic blood pressure (mmHg)} = 112.7 + 31.2\ (\dot{V}O_2)$$

$$r = 0.88 \qquad \text{S.E.M.} = 12.0$$

Ventilation and carbon dioxide output increased with increasing $\dot{V}O_2$ to maximal values of 86.4 ± 5.76 l/min and 2.939 ± 0.018 l/min respectively. The respiratory exchange ratio increased from 0.91 ± 0.03 to 1.16 ± 0.02 at peak exercise.

During the 30 second maximal isokinetic exercise at 60 rpm, the young subjects achieved a maximal average power of 650.3 ± 25.2 W and total work of 16.6 ± 0.54 kJ. The post-exercise plasma lactate concentration was 12.8 ± 0.73 mmoles/l (Figure 10). The power output generated by the leg muscles declined progressively during the test yielding a fatigue index of 29.5 ± 1.58%.

At the higher pedalling frequency (110 rpm) maximal average power output was 868.6 ± 35.3 W (Figure 11). Total work (15.7 ± 0.73 kJ) was similar to the value attained at 60 rpm. The plasma lactate concentration was similar (14.1 ± 0.85 mmoles/l), but the fatigue index was twice the value attained at 60 rpm (62.8 ± 1.84%).

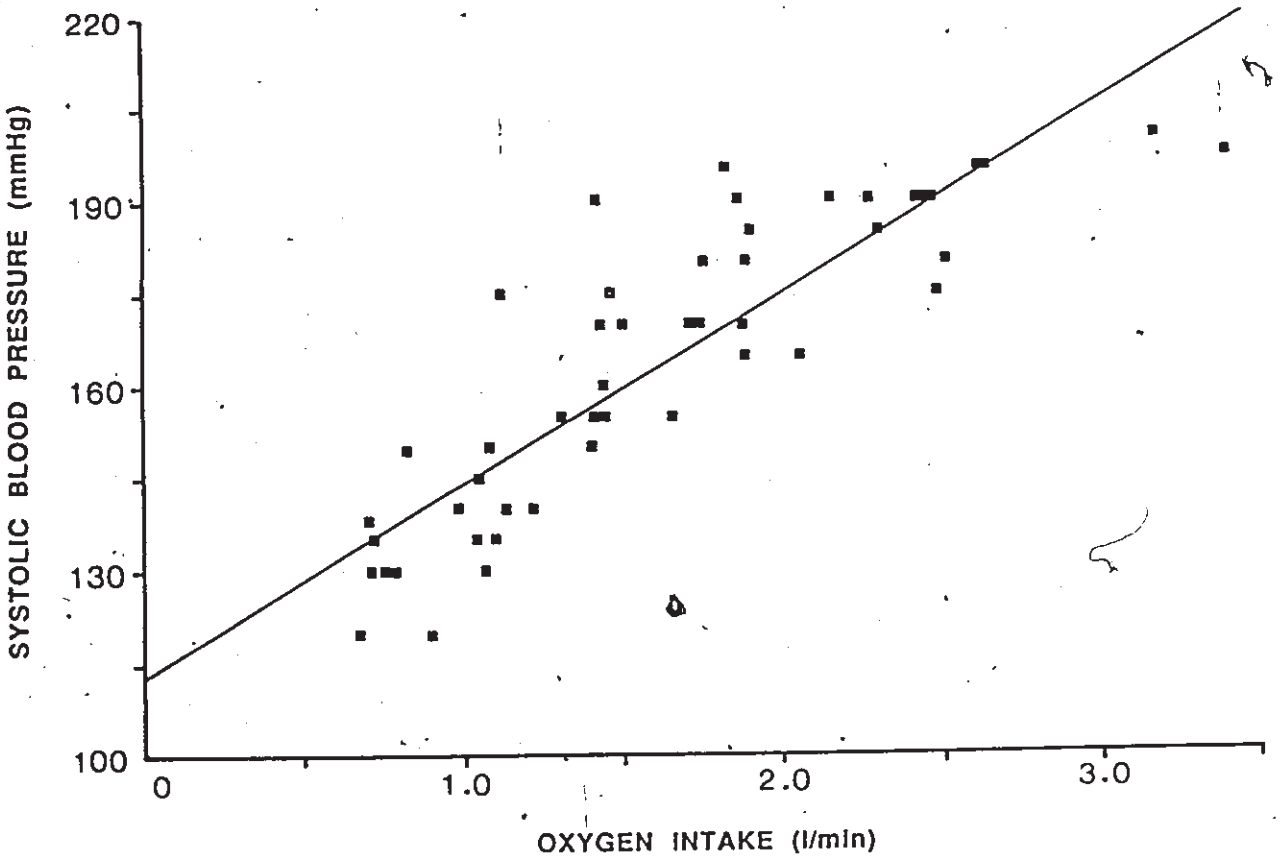


Figure 16. Before Training: Systolic blood pressure related to oxygen intake during progressive incremental exercise in 20-30 year old males.

4.2.3 Comparisons of Pre-Training Responses between the Elderly and Young Subjects

The peak power output of the older subjects was 67% of that attained by the young subjects (Figure 6). Compared to the young subjects, the lower peak $\dot{V}O_2$ (37%) of the elderly was associated with a lower peak cardiac output (30%) and arterio-venous oxygen difference (10%). The lower cardiac output of the elderly was associated with a lower maximal heart rate (16%) and stroke volume (16%).

During exercise, the rise in cardiac output with increasing $\dot{V}O_2$ was similar in the old and young subjects (5.4 l/min in the elderly and 4.7 l/min in the young per litre/min increase in $\dot{V}O_2$). However, the intercept of this relationship was significantly lower in the older subjects ($p<0.05$). The rise in heart rate for a litre/min increase in $\dot{V}O_2$ was significantly greater ($p<0.001$) in the elderly (63 beats/min compared to 45 beats/min in the young) associated with a lower stroke volume.

With increasing exercise, systolic blood pressure rose significantly more in the elderly ($p<0.001$), 44 mmHg compared to 31 mmHg in the young). This was associated with a lower vascular conductance compared to the young subjects (Figure 17).

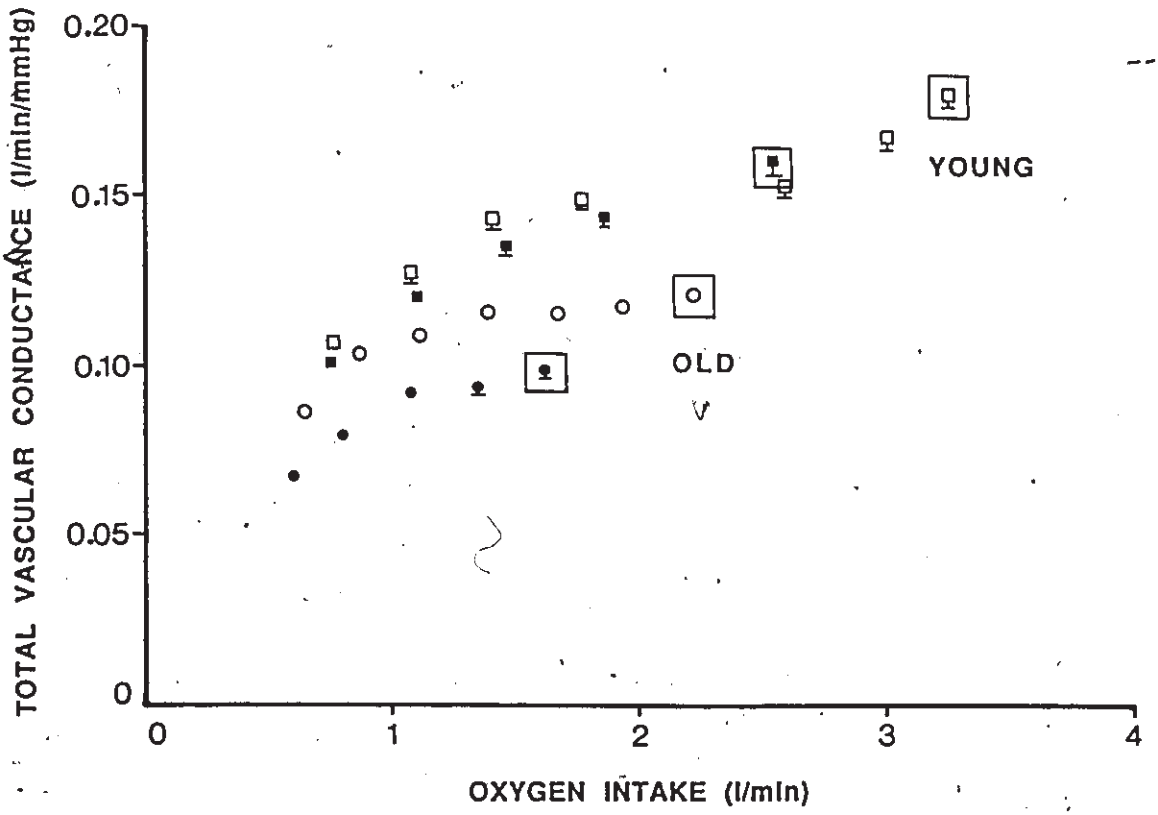


Figure 17. Total vascular conductance ± S.E.M. during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

The ventilatory response to exercise was higher in the elderly (Figure 18). Comparisons between increments in ventilation relative to increments in $\dot{V}O_2$ up to exercise levels where the $\dot{V}E/\dot{V}O_2$ relationship was linear (450 kpm/min in the old and 600 kpm/min in the young), revealed significantly higher ventilation in the elderly ($\Delta \dot{V}E/\Delta \dot{V}O_2$ old = 36.3 $\pm$ 2.34 l/min and young = 24.2 $\pm$ 3.09 $p<0.001$). At peak exercise, both ventilation and $\dot{V}CO_2$ were lower in the elderly ($p<0.001$, Table 3).

During the 30 second maximal isokinetic exercise the elderly subjects achieved about 70% of the power and total work attained by the young subjects; 60 rpm, 468 $\pm$ 15.4 W and 11.4 $\pm$ 0.40 kJ for maximal average power and total work respectively, compared to 650.3 $\pm$ 25.2 W and 16.6 $\pm$ 0.54 kJ attained by the young subjects (Figure 10). The corresponding values at 110 rpm were 625.2 $\pm$ 33.4 W and 11.2 $\pm$ 0.40 kJ in the elderly and 868.6 $\pm$ 35.3 W and 15.7 $\pm$ 0.73 kJ in the young (Figure 11).

The mean fatigue index (percent decline in average power over the 30 second test) was similar in the two groups. At 60 rpm this was 34.3 $\pm$ 2.9% in the elderly and 29.2 $\pm$ 1.6% in the young subjects. At the pedalling frequency of 110 rpm the fatigue index almost doubled in

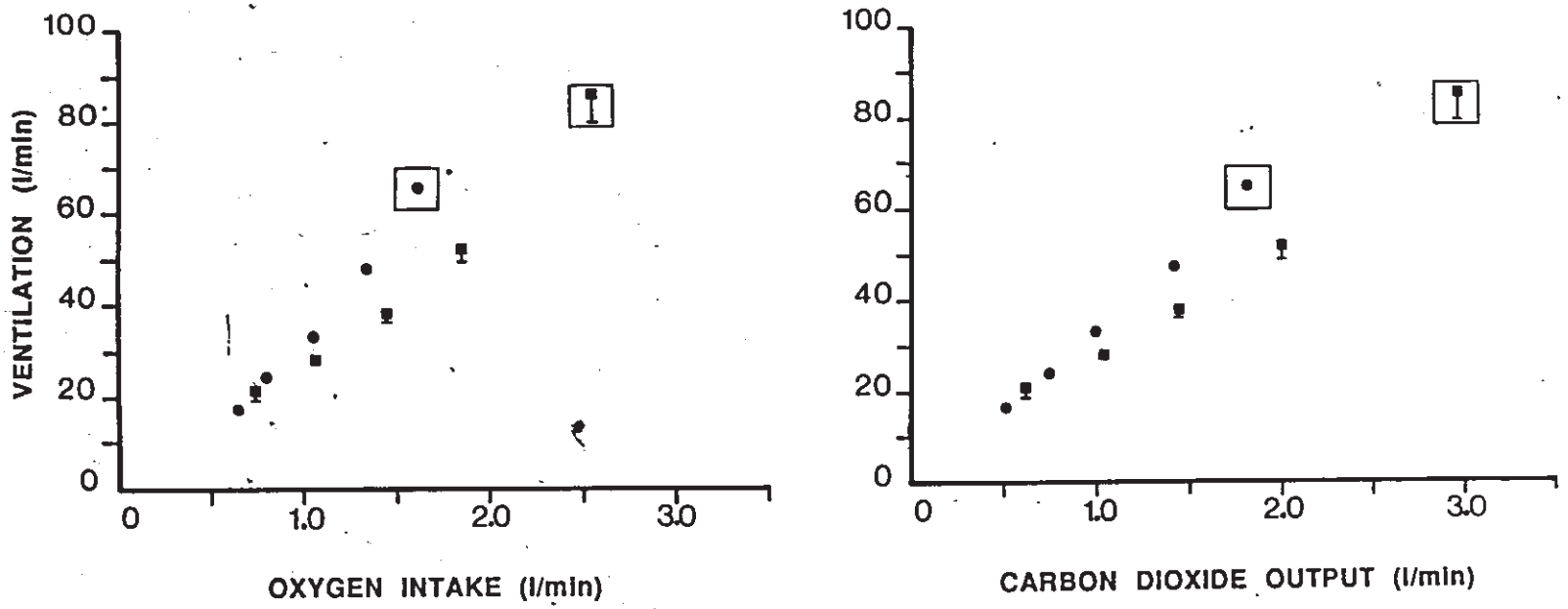


Figure 18. Before Training: Mean ventilation ± S.E.M. related to oxygen intake and carbon dioxide output during progressive incremental exercise in 60-70 (•) and 20-30 (■) year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12), young group: n = 10).

Table 3. Within and Between Group Comparisons (Mean + S.E.M.) at Peak Exercise in 20–30 and 60–70 Year Old Males

Group		Power kpm/min	Oxygen Intake (l/min)	Carbon dioxide Output (l/min)
20–30 yrs	Pre-test	1080 61.1	2.536 0.141	2.939 0.178
	Post-test	1560[t] 83.2	3.263[t] 0.181	3.784[t] 0.207
	% Change	44.4	28.7	28.8
60–70 yrs	Pre-test	725 31.2	1.599 0.073	1.801 0.096
	Post-test	1125[t] 43.4	2.212[t] 0.073	2.510[t] 0.066
	% Change	55.2	38.3	39.4

---

[t] Significant differences between pre- and post-test ($p<0.05$).

[t] Peak cardiac output was estimated for each subject from individual regression equations during submaximal incremental exercise.

Ventilation (l/min)	RER	Cardiac Output (l/min)	Heart Rate beats/min	Stroke Volume (ml)	$(a-v)O_2$ Diff. (ml/l)	Systolic B.P. (mmHg)
86.4	1.16	18.0	192	93.9	140.4	190.2
6.8	0.02	0.7	2.3	3.9	3.9	2.3
116.4[t]	1.16	20.5[t]	201[t]	102.2[t]	158.7[t]	189.5
6.4	0.02	0.9	3.0	3.4	4.9	4.6
34.7	0	13.9	4.7	8.8	13.0	-0.4
65.4	1.12	12.7	161	78.9	126.5	205.0
3.9	0.02	0.6	3.8	3.5	3.7	3.6
87.0[t]	1.14	16.5[t]	173[t]	95.6[t]	134.7[t]	211.7
4.3	0.01	0.4	3.9	2.5	2.7	4.7
33.0	1.8	29.9	7.4	21.2	6.5	3.3

The pre-test differences between the old and young subjects in all measurements except the RER were significant ($p<0.001$).

The post-test differences between the old and young in all measurements except the RER and stroke volume were significant ($p<0.001$).

both groups; 59.0 ± 2.4% in the elderly and 62.8 ± 1.7% in the young.

4.3 After Training

4.3.1 Response of Elderly Subjects to Progressive Incremental and Maximal Isokinetic Exercise

After twelve weeks of high intensity endurance training, the elderly subjects achieved a maximal power output during the progressive exercise test of 1125 ± 43.4 kpm/min. Peak $\dot{V}O_2$ increased by 38% from 1.599 ± 0.073 l/min before to 2.212 ± 0.073 l/min after training (Table 3 and Figure 6). Cardiac output increased linearly during exercise to a peak value of 16.5 ± 0.4 l/min. This relationship was expressed in the following equation (Figure 19):

$$\text{Cardiac output (l/min)} = 6.3 + 4.6\ (\dot{V}O_2)$$

$$r = 0.95 \qquad \text{S.E.M.} = 0.96$$

The increase in peak $\dot{V}O_2$ was accompanied by an increase in peak cardiac output (30%) and to a lesser extent an increase in peak arterio-venous oxygen difference (6%). There was a tendency for a decrease in the post-training arterio-venous oxygen difference during submaximal exercise but this was not statistically significant (Figure 20).

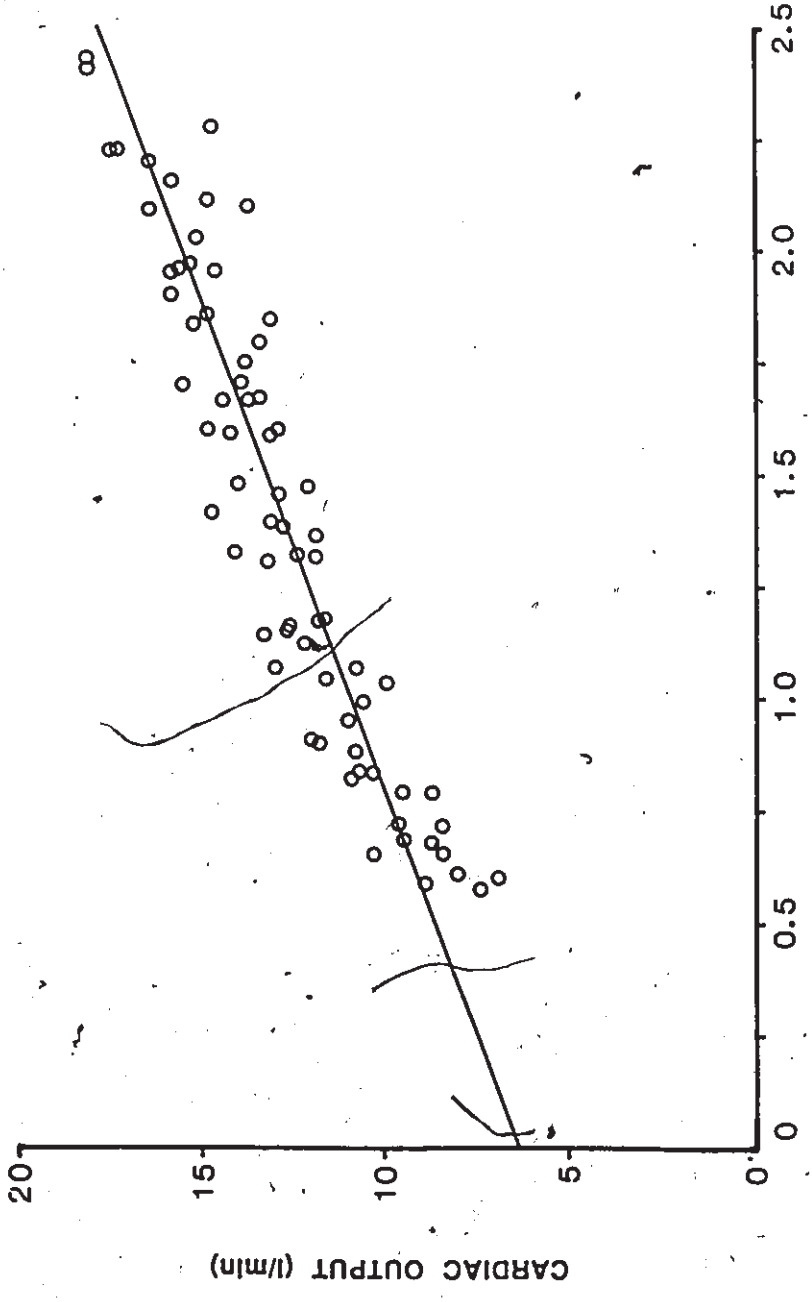


Figure 19. After Training: Cardiac output related to oxygen intake during progressive incremental exercise in 60-70 year old males.

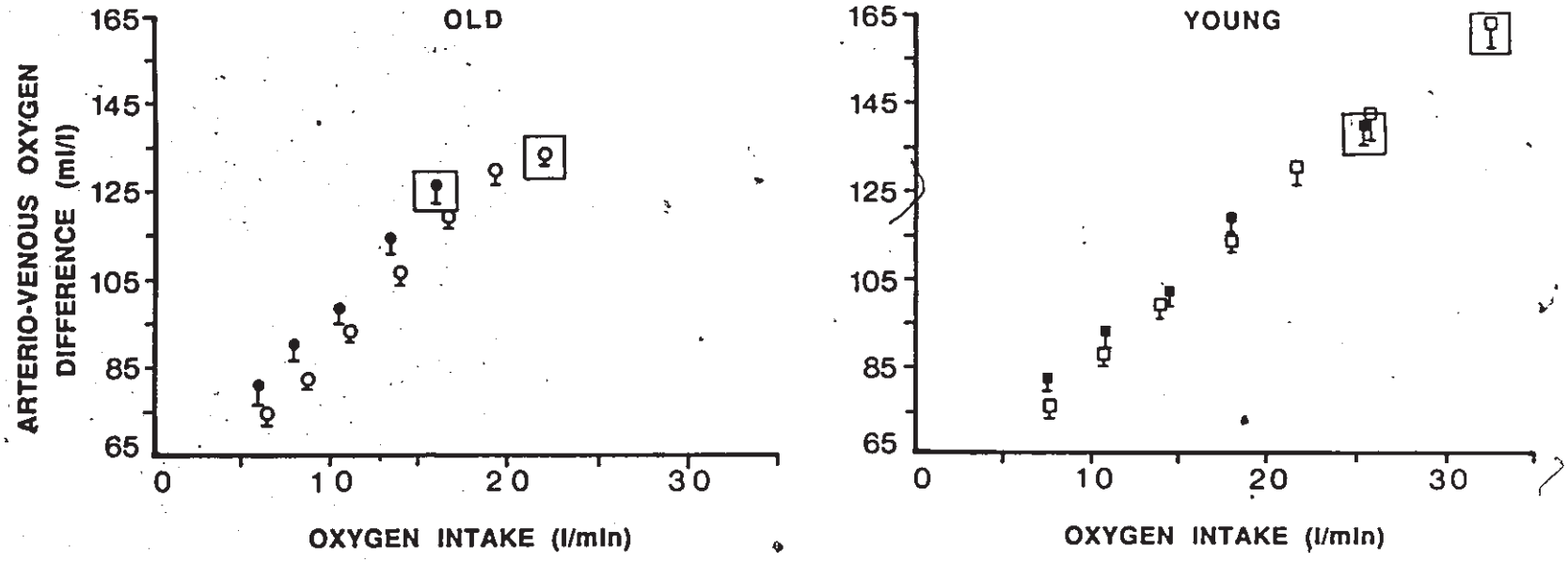


Figure 20. Mean arterio-venous oxygen difference ± S.E.M. related to oxygen intake before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

The post-training increase in peak cardiac output was associated with a large increase in stroke volume (21%) and a small increase in peak heart rate (7%, Table 3). Heart rate rose linearly with increasing $\dot{V}O_2$ to a maximal value of 172.7 ± 3.9 beats/min. This relationship was expressed by the following equation:

$$\text{Heart rate (beats/min)} = 52.2 + 51.7\ (\dot{V}O_2)$$

$$r = 0.95 \qquad \text{S.E.M.} = 11.0$$

The rise for a litre/min increase in $\dot{V}O_2$ was 51.7 beats/min, significantly lower than the corresponding value observed before training ($p<0.001$) (Figure 21). While submaximal heart rate decreased after training, stroke volume during exercise increased markedly ($p<0.01$) after training (Figure 22).

At peak exercise systolic blood pressure (212 ± 4.7 mmHg) was not significantly different compared to the pre-training value (205 ± 3.6 mmHg), despite a 30% increase in peak cardiac output (Figure 6). At a given $\dot{V}O_2$, systolic blood pressure was significantly lower ($p<0.001$) after training, but the rate of change for a l/min increase in $\dot{V}O_2$ was similar; 44.1 mmHg before and 48.8 after training (Figure 23). The relationship (post- training) was expressed by the following equation:

$$\text{Systolic blood pressure (mmHg)} = 112.1 + 48.8\ (\dot{V}O_2)$$

$$r = 0.91 \qquad \text{S.E.M.} = 16.9$$

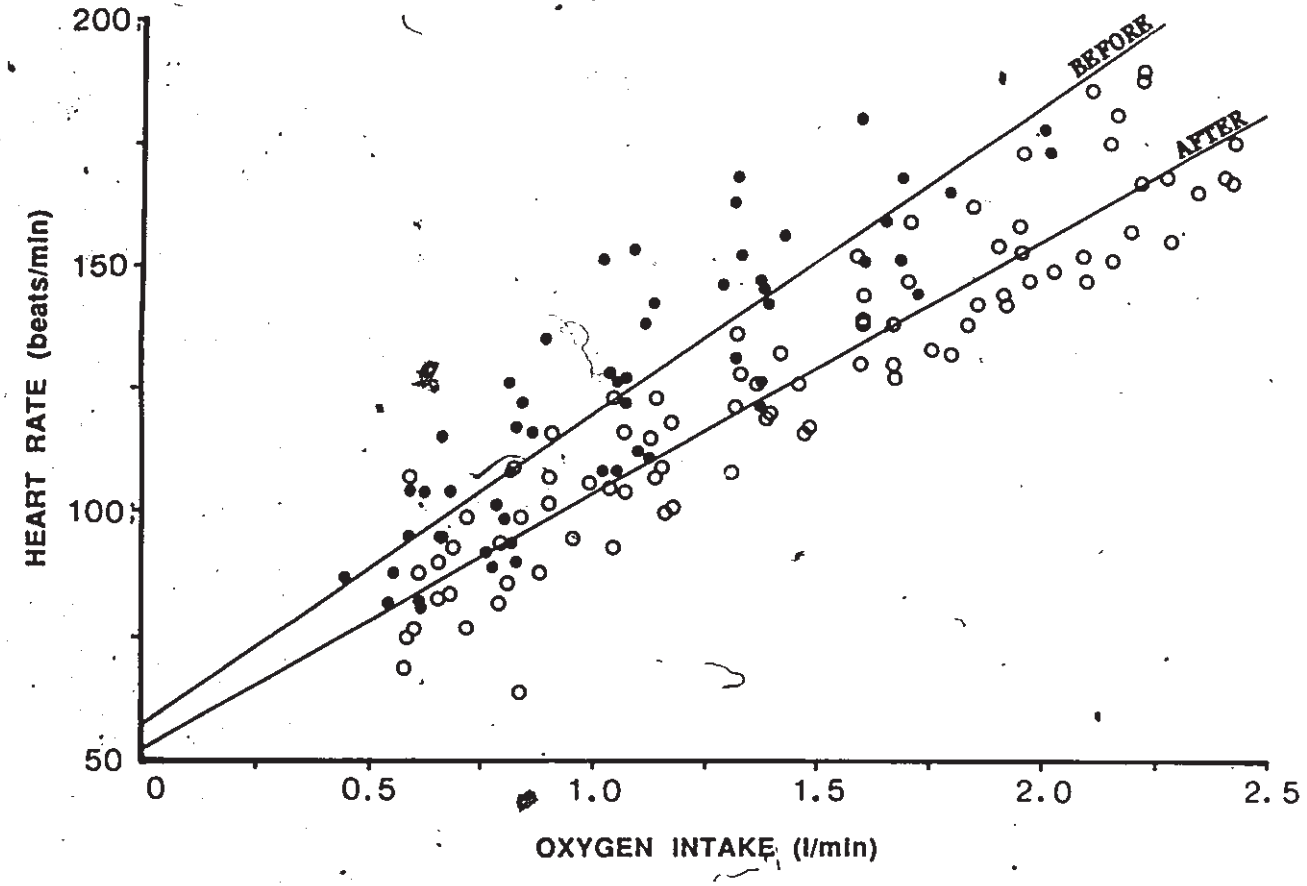


Figure 21. Heart rate related to oxygen intake during progressive incremental exercise before (•) and after (o) training in 60-70 year old males.

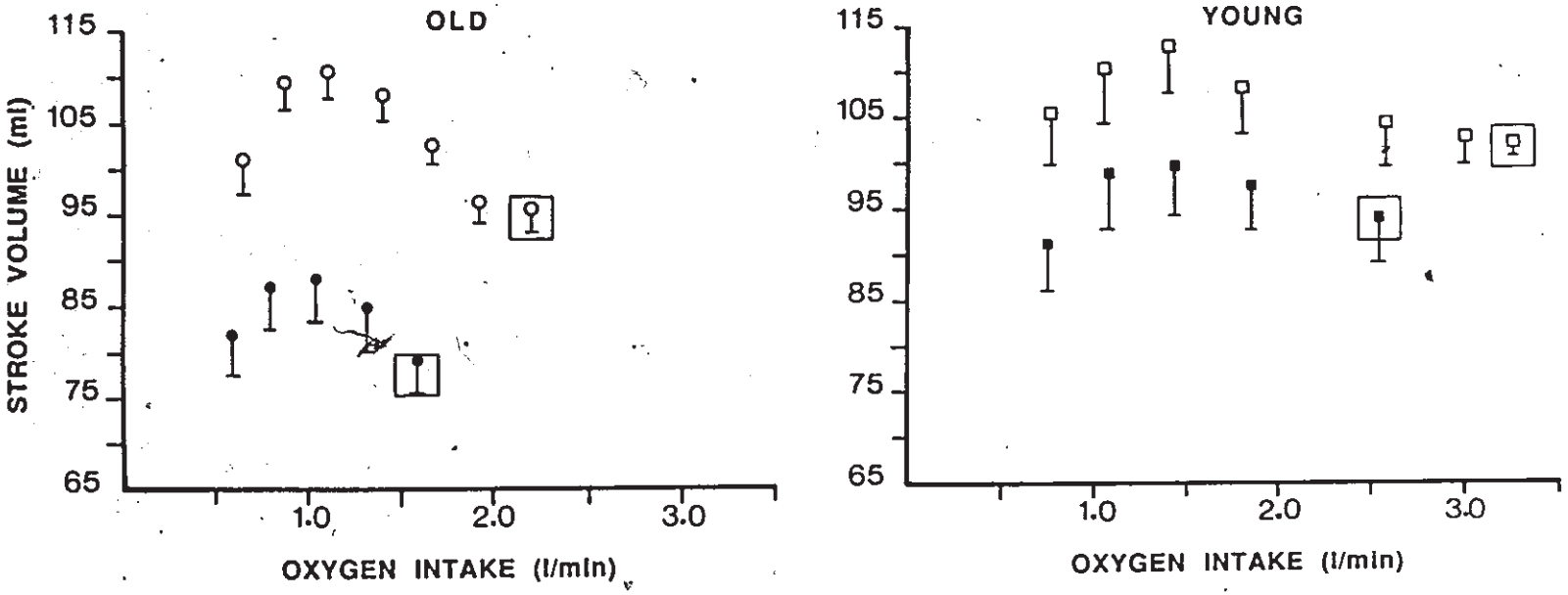


Figure 22. Mean stroke volume ± S.E.M. during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

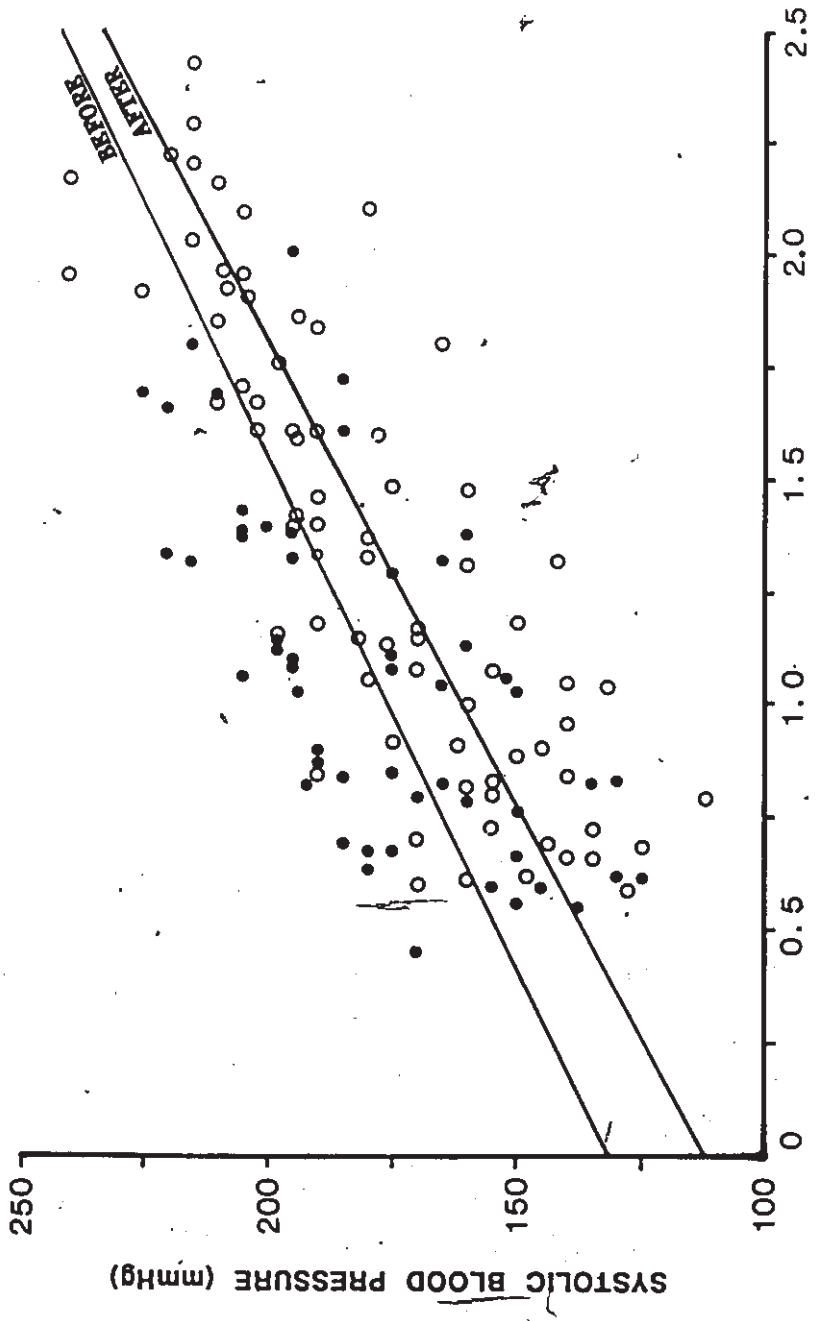


Figure 23. Systolic blood pressure related to oxygen intake during progressive incremental exercise before (●) and after (○) training in 60-70 year old males.

Ventilation at low exercise levels (up to 300 kpm/min) was similar before and after training (Figure 24). However, as exercise progressed, the ventilatory response to increasing exercise was significantly lower after training ($p<0.01$). This was associated with a similar decrease in the respiratory exchange ratio ($p<0.01$, Figure 25).

In contrast to the haemodynamic and ventilatory measurements, small changes were observed after training in the peripheral muscle measures. At 60 rpm, both power and total work were not significantly different from the values attained before training (Figure 10, Table 4). The only exception was a fall (17%) in plasma lactate after training (9.0 ± 0.69 mmoles/l to 7.5 ± 0.43 mmoles/l, $p<0.05$).

However, at the higher pedalling frequency (110 rpm), the elderly subjects achieved a significantly higher total work (13%, 11.2 ± 0.40 kJ to 12.6 ± 0.49 kJ). This was associated with a marked fall (26%, $p<0.001$) in plasma lactate (10.5 ± 0.61 mmoles/l to 7.8 ± 0.52 mmoles/l, Figure 11). Furthermore, the ability to maintain power over the duration of the test increased, as indicated by a fall in the post-training fatigue index (8%, $59.0 \pm 2.4\%$ to $54.2 \pm 2.86\%$). Maximal average power was unchanged after training at either pedalling frequency (Table 4).

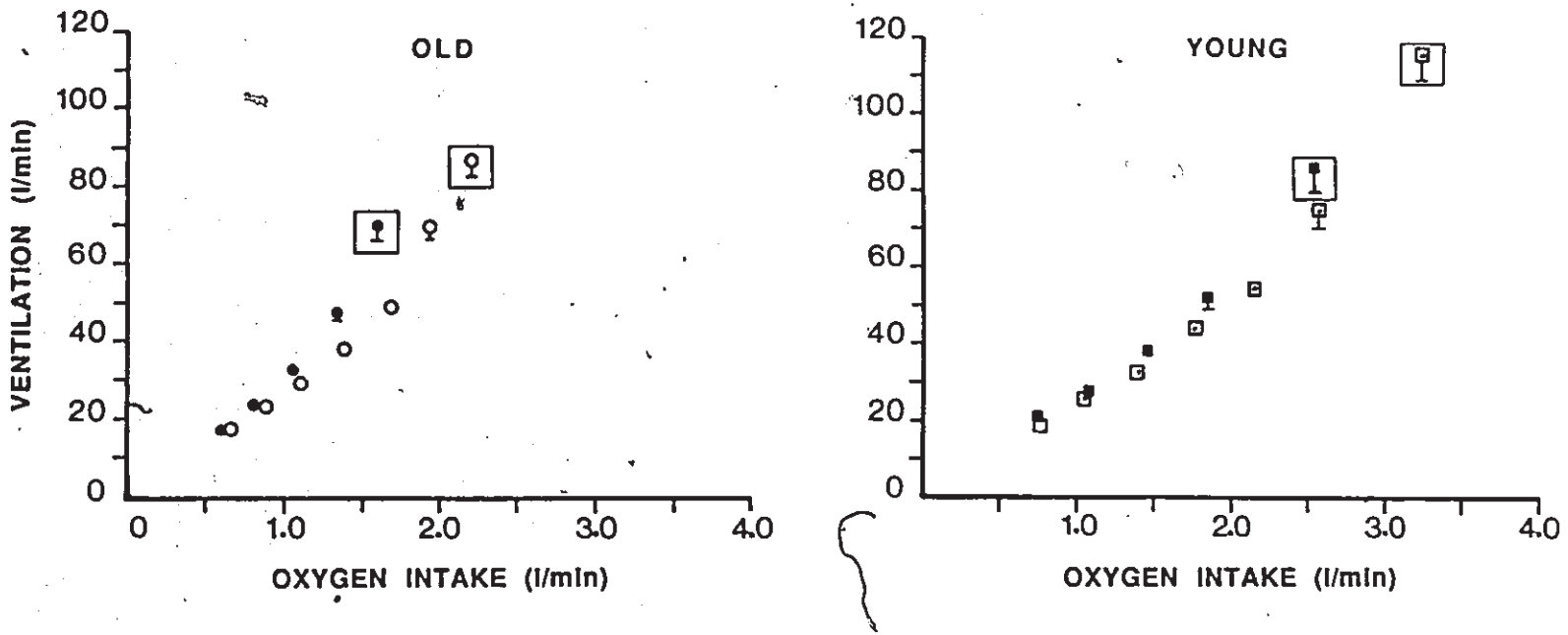


Figure 24. Mean ventilation ± S.E.M. (S.E.M. too small to show in some points) during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

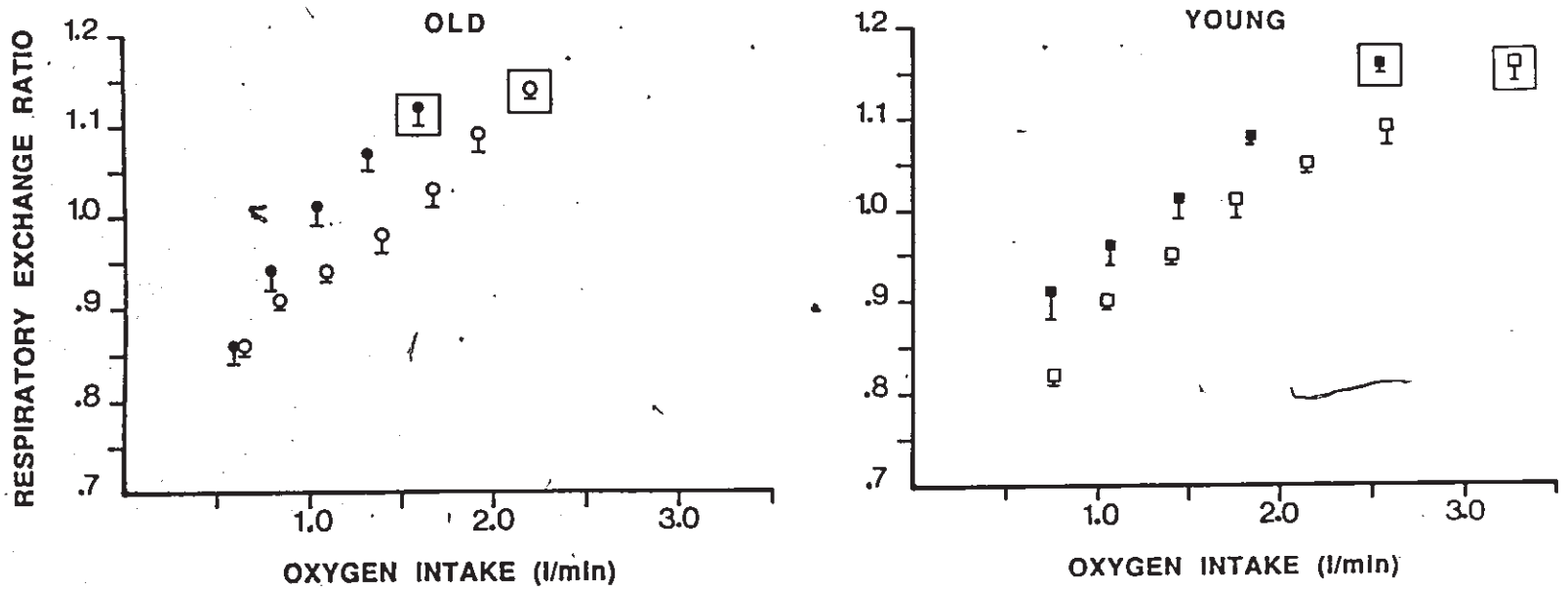


Figure 25. Mean respiratory exchange ratio ± S.E.M. during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

Table 4. Within and Between Group Comparisons (Mean ± S.E.M.) of Peripheral Muscle Function Measures During 30 seconds of Maximal Isokinetic Cycling at 60 rpm and 110 rpm

Group		Maximal Average Power* (W) 60 rpm	110 rpm	Total Work* (kJ) 60 rpm
20-30 yrs	Pre-test	650.3	868.8	16.6
		25.2	35.3	0.54
	Post-test	640.4	854.2	16.6
		26.2	52.2	0.57
	% Change	-1.5	-1.7	0
60-70 yrs	Pre-test	468.8	625.2	11.4
		15.4	33.4	0.40
	Post-test	478.2	636.9	11.7
		17.6	26.8	0.46
	% Change	2.0	1.9	2.6

* Significant differences between pre- and post-test ($p<0.05$).

Total Work* (kJ) 110 rpm	Fatigue Index (%) 60 rpm	Fatigue Index (%) 110 rpm	Lactate* (mmol/l) 60 rpm	Lactate* (mmol/l) 110 rpm
15.7 0.7	29.2 1.6	62.8 1.7	12.8 0.7	14.1 0.9
16.9 t 0.9	27.4 2.2	58.1 t 1.6	11.9 0.6	12.1 t 0.7
7.6	-6.2	-7.5	-7.0	-14.2
11.2 0.4	34.3 2.9	59.0 2.4	9.0 0.7	10.5 0.6
12.6 t 0.5	30.7 2.7	54.2 t 2.9	7.5 t 0.4	7.8 t 0.5
12.5	-10.5	-8.1	-16.7	-25.7

* Significant differences between old and young both at pre- and post-test ($p<0.001$).

4.3.2 Response of Young Subjects to Progressive Incremental and Maximal Isokinetic Exercise

After training, the young subjects achieved a maximal power output of 1560 ± 83.2 kpm/min. Peak $\dot{V}O_2$ increased 29%; 2.536 ± 0.141 l/min before to 3.263 ± 0.181 l/min after training (Table 3 and Figure 6). Cardiac output increased linearly during exercise to a peak value of 20.5 ± 0.89 l/min. This relationship was expressed by the following equation (Figure 26):

$$\text{Cardiac output (l/min)} = 8.3 + 3.8\ (\dot{V}O_2)$$

$$r = 0.94 \qquad \text{S.E.M.} = 1.34$$

The increase in peak $\dot{V}O_2$ was accompanied by similar increases in cardiac output (14%) and arterio-venous oxygen difference (13%). The submaximal arterio-venous oxygen difference was not significantly different compared to the pre-training value (Figure 20).

The post-training increase in peak cardiac output was associated with similar increases in peak heart rate (5%) and stroke volume (9%, Table 3). Heart rate rose linearly with increasing $\dot{V}O_2$ to a peak value of 200.6 ± 3.0 beats/min. the increase was 40.2 beats/min for a litre/min increase in VO_2, significantly lower ($p<0.001$) compared to the pre-training value of 45 beats/min. This relationship was expressed by the following equation (Figure 27):

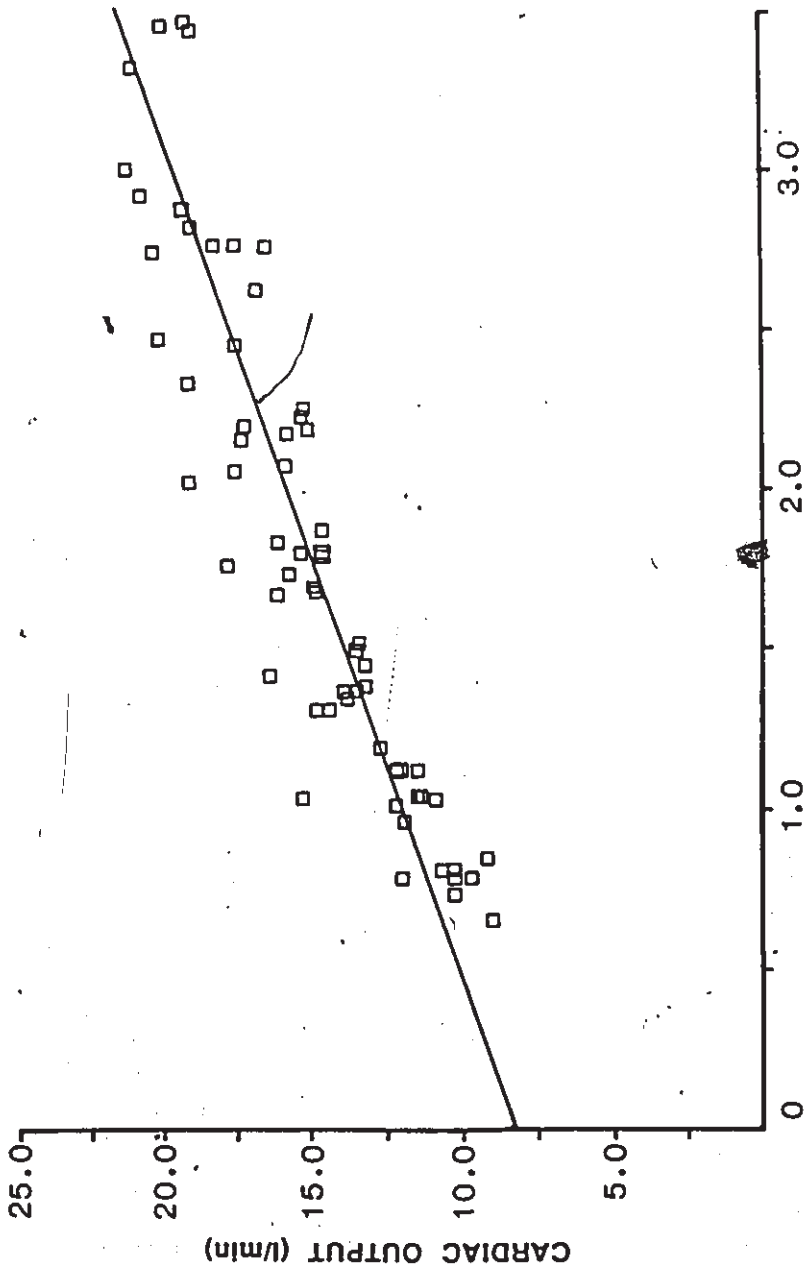


Figure 26. After Training: Cardiac output related to oxygen intake during progressive incremental exercise in 20-30 year old males.

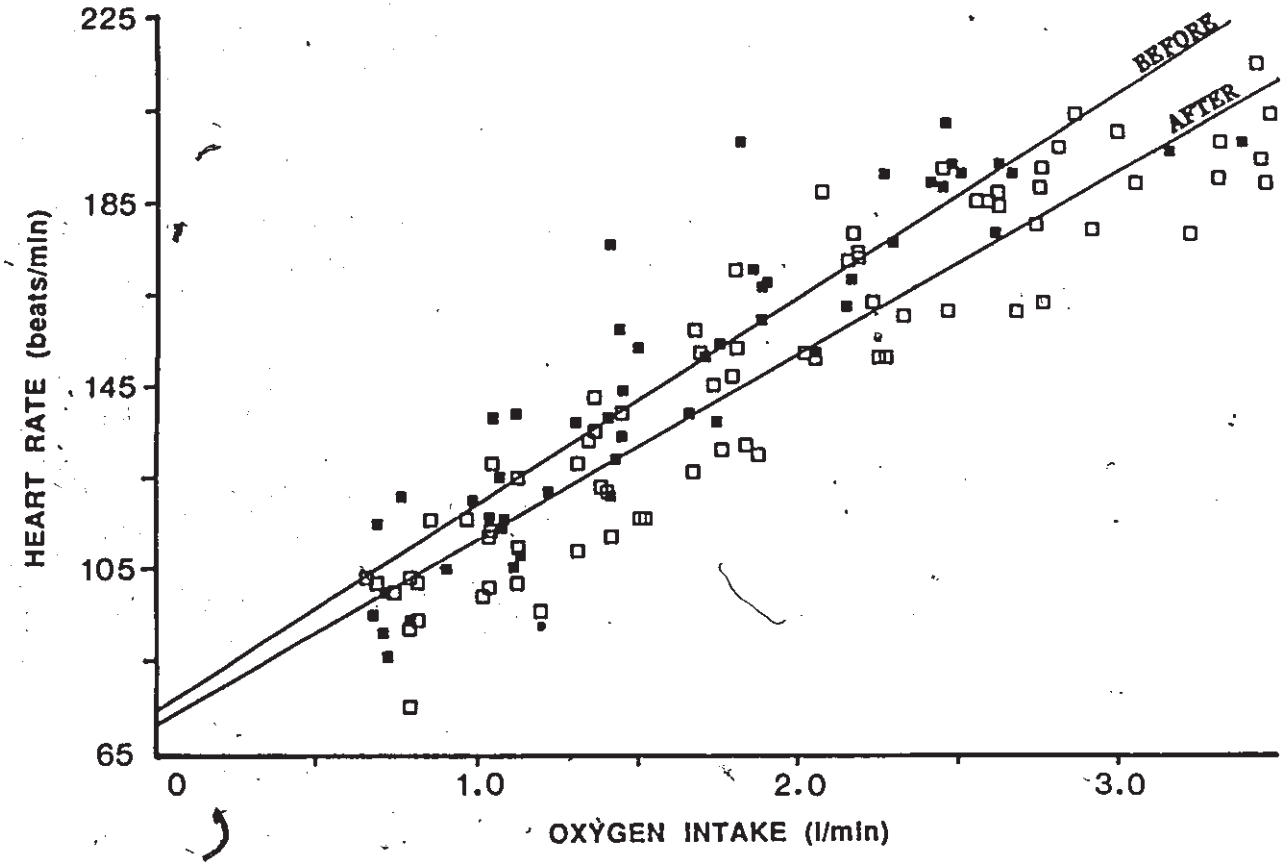


Figure 27. Heart rate related to oxygen intake during progressive incremental exercise before (■) and after (□) training in 20-30 year old males.

Heart rate (beats/min) = 71.1 + 40.2 ($\dot{V}O_2$)

r = 0.96 S.E.M. = 13.2

There was no change after training in the rise in systolic blood pressure with increasing $\dot{V}O_2$ in the young subjects. This was 31.2 mmHg before training and 31.1 mmHg (Figure 28). Similar values were also found at a given submaximal $\dot{V}O_2$ and at peak exercise; 190.2 ± 2.3 mmHg before and 189.5 ± 4.6 mmHg after training (Figure 6).

Ventilation at low exercise levels (up to 400 kpm/min) was similar before and after training (Figure 24). However, as exercise progressed, the ventilatory response to increasing exercise was significantly lower after training ($p<0.05$). This was associated with a significant decrease in the respiratory exchange ratio ($p<0.05$, Figure 25).

In contrast to the haemodynamic and ventilatory measurements, small changes were observed after training in the peripheral muscle measures. At 60 rpm power output, total work, plasma lactate and the fatigue index were not significantly different compared to the pre-training values (Figure 10 and Table 4).

At the higher pedalling frequency (110 rpm) the young subjects achieved a significantly higher total work (8%, 15.7 ± 0.73 kJ to 16.9 ± 0.85 kJ). This was associated with a lower plasma lactate (14%, $p<0.05$, 14.1 ±

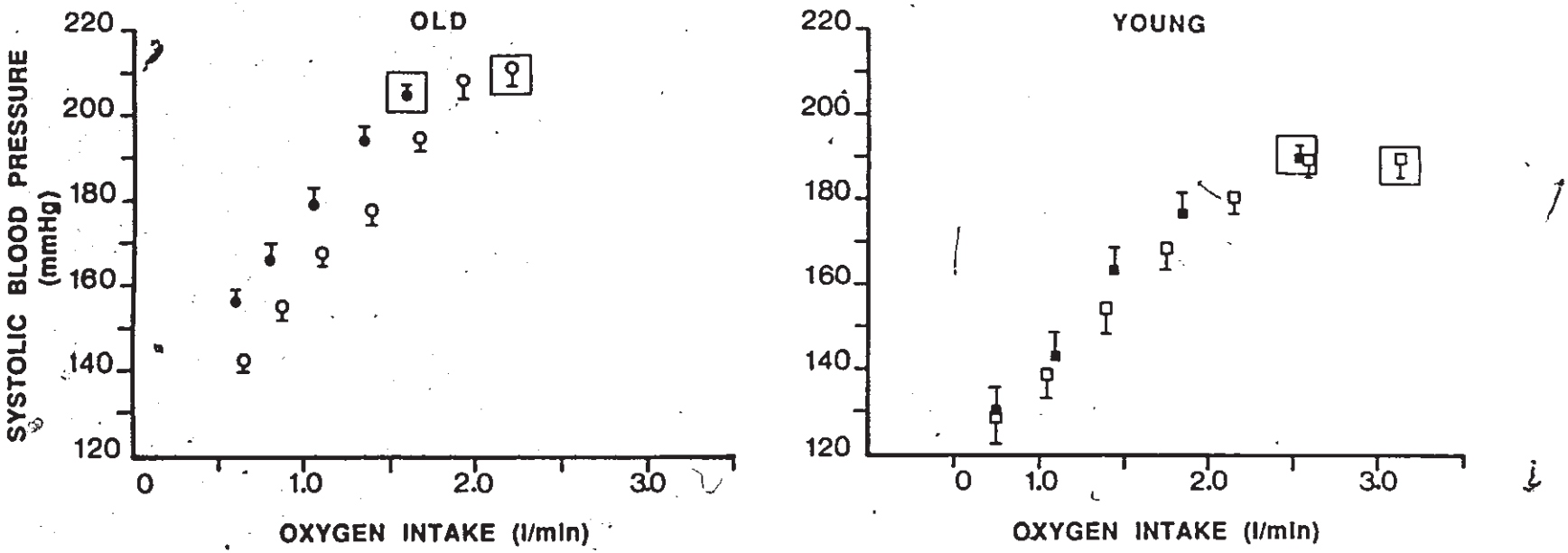


Figure 28. Systolic blood pressure ± S.E.M. related to oxygen intake during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

0.85 mmoles/l to 12.1 ± 0.70 mmoles/l, Figure 11 and Table 4). Maximal average power was unchanged after training. However, the fatigue index decreased after training (7%, 62.8 ± 1.68% to 58.1 ± 1.55%, $p<0.05$).

4.3.3 Comparisons of Post-Training Responses between the Elderly and Young Subjects

Peak power output in the progressive exercise test increased significantly in both groups after training amounting to 55% in the elderly and 44% in the young subjects (Table 3). Peak $\dot{V}O_2$ also increased in both groups (38% in the elderly and 29% in the young) with no significant difference between the two groups in the relative change. The increase in peak $\dot{V}O_2$ was accompanied by an increase in peak cardiac output in both groups. This was particularly striking in the older group in whom the peak cardiac output increased from 12.7 l/min pre-training to 16.5 l/min after training, a 30% increase, compared to a 14% increase in the young subjects (18.0 l/min pre-training to 20.5 l/min after training). However, the arterio-venous oxygen difference at maximal exercise increased only 6% in the elderly and 13% in the young subjects. Thus, the elderly subjects increased their peak $\dot{V}O_2$ largely by an increase in peak cardiac output with less of an increase in arterio-venous oxygen difference. In contrast, the

increase in peak $\dot{V}O_2$ in the young subjects was associated with increases in both peak cardiac output and arterio-venous oxygen difference.

Heart rate and stroke volume at peak exercise increased after training in both groups contributing to the increase in peak cardiac output. The change in heart rate was small, 7% in the elderly and 5% in the young with no significant differences between the groups in the relative change. However, stroke volume increased considerably more in the elderly amounting to 21% compared to a 9% increase in the young subjects. Therefore, the increase in peak cardiac output in the elderly was accompanied by an increase in stroke volume with less of a change in heart rate. In contrast, in the young subjects, increases in peak heart rate and stroke volume contributed to a similar extent to the rise in peak cardiac output after training.

During exercise, systolic blood pressure was lower after training in the elderly with no change in the young subjects (Figure 28). This was associated with a significant increase in vascular conductance in the elderly both at peak ($p<0.001$) and at submaximal exercise ($p<0.01$, Figure 17); there was no change in the young subjects.

Submaximal heart rate decreased significantly ($p<0.001$) in both groups after training, associated with an increase in submaximal stroke volume; this was

significant only in the old group ($p<0.01$, Figure 22). The increase in submaximal stroke volume after training in the elderly resulted in a higher ($p<0.05$) submaximal cardiac output (Figure 29). In contrast, in the young, submaximal stroke volume and cardiac output after training were not significantly different compared to the pre-training value. No significant changes were observed in the submaximal arterio-venous oxygen difference after training in either group (Figure 20).

The behaviour of the ventilatory response to exercise was similar in both the elderly and young subjects; no change at low exercise levels (up to 300 kpm/min in old and 450 kpm/min in young), with a significant decrease as exercise progressed ($p<0.05$, Figure 24). This was associated with a fall ($p<0.05$) in the respiratory exchange ratio in both groups (Figure 25).

During the 30-second maximal isokinetic exercise, the older subjects achieved 75% of the power output and total work attained by the young subjects (Figures 10 and 11, Table 4); 60 rpm: 478.2 $\pm$ 17.6 W and 11.7 $\pm$ 0.46 kJ in the old, 640.4 $\pm$ 26.2 W and 16.6 $\pm$ 0.57 kJ in the young respectively. The corresponding values at 110 rpm were 636.9 $\pm$ 26.8 and 12.6 $\pm$ 0.49 kJ in the old, 854.2 $\pm$ 52.2 W and 16.9 $\pm$ 0.85 kJ in the young subjects. Plasma lactate concentration decreased at 110 rpm after

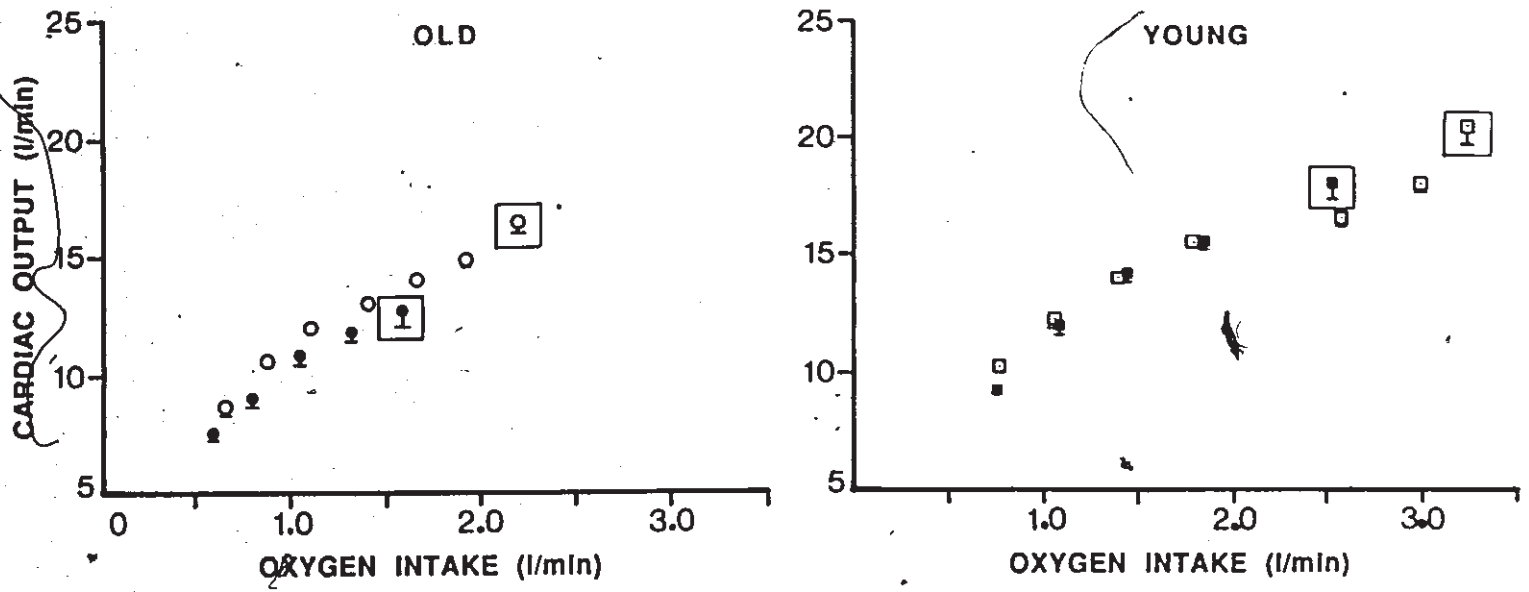


Figure 29. Cardiac output ± S.E.M. related to oxygen intake during progressive incremental exercise before (filled symbols) and after (open symbols) training in 60-70 and 20-30 year old males. Values at peak exercise indicated by large square.

Points refer to exercise levels completed by all subjects (old group: n = 12, young group: n = 10).

training to a similar extent in both groups. However, at 60 rpm the fall in lactate was significant only in the older group (Figure 10); 9.0 ± 0.69 mmoles/l before and 7.5 ± 0.43 mmoles/l after training. The corresponding values in the young subjects were 12.8 ± 0.73 mmoles/l before and 11.9 ± 0.57 after training. The fatigue index was not significantly different in the two groups as was the case at pre-test (Figures 10 and 11).

4.4 Summary of Results

Before training, the peak $\dot{V}O_2$ attained by the older subjects was about 37% lower compared to the young subjects. This was associated with a lower peak cardiac output (29%) and arterio-venous oxygen difference (10%). The lower cardiac output in the elderly was associated with a lower peak heart rate (16%) and stroke volume (16%).

The differences between the young and old in peripheral muscle function were similar in magnitude to the differences observed in the haemodynamic measures. Both power and total work were about 30% lower in the elderly compared to the young with similar decreases in the post-exercise plasma lactate concentration. However, the decline in power over 30 seconds of maximal cycling was similar in the two groups. Thus, no differences were

observed in muscle fatigue between the young and older group.

After the twelve week high intensity training program, physical work capacity increased markedly in both groups; peak $\dot{V}O_2$ increased about 38% in the elderly and 29% in the young with no differences in the relative change. However, differences were observed in the mechanisms contributing to the increase in peak $\dot{V}O_2$. In the elderly, this was accompanied by an increase in cardiac output (30%) with less of an increase in arterio-venous oxygen difference (6%). In contrast, both cardiac output and arterio-venous oxygen difference contributed equally to the increase in peak $\dot{V}O_2$ in the young subjects (approximately 14%). The mechanism for the increase in peak cardiac output after training was also different in the two groups. In the elderly, this was associated with a marked increase in stroke volume (21%) with a relatively small change in peak heart rate (7%). In contrast, in the young subjects, both stroke volume and heart rate contributed about equally to the increase in peak cardiac output after training (9% and 5% respectively).

Major differences were observed between the two groups in the submaximal response to exercise after training; stroke volume and cardiac output were significantly higher in the elderly, associated with a

marked increase in vascular conductance and fall in systolic blood pressure. There were no significant changes after training in these measurements in the young group.

In contrast, similar training-induced adaptations occurred at submaximal exercise, in both groups; arterio-venous oxygen difference (no significant change after training), heart rate, and the respiratory exchange ratio (significant decreases after training) and ventilation (significant decrease beyond the initial two exercise levels).

When the post-training results in the old were compared to the pre-training results in the young, many of the differences between the groups were no longer evident, with the exception of the consistently lower peak heart rate and higher arterial pressure in the old.

In contrast to the changes in peak $\dot{V}O_2$ and haemodynamic measures, the changes in peripheral muscle function after training were small and of a similar magnitude in both groups. The only changes observed were an increase in total work, 13% in the old and 8% in the young, with decreases in plasma lactate and muscle fatigue in both groups.

5. DISCUSSION

5.1 Introduction

The work described in this thesis examined the training-associated changes in oxygen transport mechanisms and muscle function in healthy, sedentary 60 to 70 year old males and compared these changes with those in a similar group of 20 to 30 year olds. Many studies in the literature have documented the structural and functional changes associated with aging, but few studies have examined the effects of high intensity endurance training in improving the exercise capacity of elderly individuals.

The few studies available on sedentary, elderly subjects have reported conflicting findings particularly related to the extent of expected improvement following training. The disparity in findings may relate to differences in testing protocols, intensity, format and length of training and the lack of a direct comparison group of young subjects with a similar level of fitness who undertook the same training program. The latter issue is particularly important both in terms of comparisons on the trainability of elderly individuals and in comparisons of

differences in the mechanisms likely to contribute to any training-associated changes.

A common practice in the literature is to use cross comparisons of results in young subjects reported in other studies. Such comparisons have many limitations as often the testing procedures, intensity of training and the initial level of fitness of the subjects are different among the young and elderly. We have approached these problems in two ways. Firstly, we examined the decline in exercise capacity in a cross-sectional study of 100 healthy males and females 15 to 70 years of age. This study served as the background for the training study that is the subject of this thesis; it allowed us to examine whether the two groups of elderly and young subjects were representative of a larger population and whether they were comparable in terms of their initial fitness level. Secondly, we compared the exercise responses of two groups of elderly and young subjects before and after the same training program at the same relative intensity.

Before discussing the physiological implications of our findings, some methodological considerations relating to the study design, the representativeness of the subjects and the methods of measurement will be discussed.

5.2 Methodological Considerations

5.2.1. Design of Study

In comparative studies of exercise and aging, a number of factors need to be considered in the study design. Firstly, it is important that the older individuals are free of cardiovascular or other disease so differences in exercise capacity can be attributed to aging and not to pathology. The subjects who participated in this study were on no medication and they were free of known cardiovascular or other diseases, except for minor arthritic changes in their joints.

Secondly, the issue of cross-sectional versus longitudinal data needs to be addressed. Cross-sectional investigations have one main limitation: the assumption that the older subjects were similar in youth to the young subjects studied. Longitudinal studies are preferable as the same subjects are followed over a period of time. However, in studies of aging, the period of follow-up needs to be extended over many years, which imposes both methodological and financial limitations. The representativeness of the study population is another concern, particularly in longitudinal investigations, as usually it is the more fit and physically active subjects who volunteer for long-term studies of this nature.

The present investigation employed a cross-sectional design. Therefore, it is subject to the limitations imposed by this type of investigation; the older healthy group of subjects were recruited from a total of approximately 110 older males who responded to the call for subjects and they may be a select subset of the population, as the prevalence of coronary heart disease and other pathologies increases in older populations. As reported by the Canada Fitness Survey (Fitness and Aging 1982) 51% of the 55-64 year old participants and 67% of participants 65 years and over were ineligible for the survey for health reasons. Thus, not only in our study, but in other studies of exercise and aging, older subjects who fulfill the health criterion (all of our subjects) comprise only about 35 to 45% of the general elderly population. Furthermore, it is impossible to rule out genetic and environmental differences between the two groups; we have no way of knowing how similar the older subjects were in their youth to the young subjects studied.

5.2.2 Representativeness of Study Samples

In this section, comparisons of the representativeness of the samples used in both the cross-sectional and training studies will be discussed. Firstly, the decline in exercise capacity with aging

observed in the background cross-sectional study will be compared with similar studies reported in the literature. This will establish similarities or differences in our findings with those reported in the literature. Secondly, relevant pre-training findings in exercise capacity and subject characteristics in the training study will be compared to relevant findings in the background cross-sectional study. Other comparisons in subject characteristics will also be outlined where appropriate.

5.2.2.1 **Findings of the Background Cross-Sectional Study**

Based on the results of this study, the predicted peak $\dot{V}O_2$ of a 65 year old is 26.4 ml/kg/min and that of a 25 year old is 44.0 ml/kg/min (Jones et al, 1985, Appendix 1). These values are comparable to values reported in the literature using larger samples; Dehn and Bruce (1972) reviewed 17 cross-sectional studies in treadmill exercise (about 700 observations) to derive the following equation: $\dot{V}O_2max$ (ml/kg/min) = 57 - 0.40 (year). Based on this equation, the predicted peak $\dot{V}O_2$ of a 65-year-old male is 31.0 ml/kg/min. Considering the fact that treadmill exercise yields peak $\dot{V}O_2$ values about 10% higher compared to cycling exercise, our results are comparable with those of Dehn and Bruce (1972).

In the population study of Chiang et al (1970) comprising 1[illegible]4 healthy males (about 50% of the total eligible population in Tecumseh, Michigan), the mean peak VO_2 (treadmill exercise) of a 65-year-old male was 21.8 ml/kg/min, about 10% to 15% lower compared to our values. This may be due at least in part to their exercise protocol which did not allow the subjects to exercise to their symptom-limited maximum; an upper limit in heart rate of 160 beats/min was imposed in subjects 40 years and older.

The most recent cross-sectional population study was that of Vogel et al (1986) in 514 U.S. army soldiers, 17 to 55 years of age. Mean peak $\dot{V}O_2$ values of about 50 ml/kg/min were reported (treadmill exercise) in males in their 20s, about 13% higher compared to the mean value of the 20-year-olds in our cross-sectional study. No subjects over 55 years were included in the Vogel et al (1986) study. However, a comparison with our older subjects can be made if the expected decrease in peak $\dot{V}O_2$ (9% per decade, Heath et al, 1981) is applied to their 40-year-old subjects (mean peak VO_2 of 39 ml/kg/min). This yields an estimated peak VO_2 of about 31 ml/kg/min in a 60-year-old male; 17% higher compared to the mean value in our 60-year-olds. However, our findings are comparable if one considers differences in testing protocols (treadmill

versus cycle) and differences in subjects (soldiers versus community-dwelling individuals).

Furthermore, the mean annual rate of decline in peak $\dot{V}O_2$ based on our study was 0.44 ml/kg/min, which is similar to the annual rate of decline reported in a number of cross-sectional studies (Table 1, Chapter 1). For these reasons, we feel justified in using the results of our cross-sectional study as a baseline for comparison with the initial findings of the present sample of young and elderly subjects.

5.2.2.ii Initial Findings of the Training Study

The subjects who participated in the present study were recruited from the same general population as the subjects in the larger cross-sectional study, and were tested using the same experimental protocol (Appendices 1 and 2). The values attained by the sedentary males in the present study were 22.0 ml/kg/min in the elderly, 83% of the predicted mean peak $\dot{V}O_2$, and 36.0 ml/kg/min in the young, 82% of the predicted mean peak $\dot{V}O_2$. Therefore, in terms of the initial level of fitness, the young and elderly subjects were comparable, with peak $\dot{V}O_2$ values in the low range of normal consistent with their sedentary occupations and lifestyles.

The measurements of muscle function obtained in the present study were not significantly different compared to the values obtained in the cross-sectional study; total work during 30 seconds of maximal cycling was 17.4 kJ at age 25 and 11.9 kJ at age 65. In the present study, the mean values for the 20-30 and 60-70 year old groups were 16.6 kJ and 11.9 kJ respectively. These findings indicate that both groups of elderly and young subjects were comparable in terms of muscle function and were representative of the larger population tested in the cross-sectional study.

Compared to the general population, the subjects' body weight in relation to their height was within 5% in the old (73.3 kg compared to 77.4 kg) and 4% in the young (71 kg compared to 73.9 kg, Fitness and Lifestyle in Canada, 1983). Their vital capacity, relative to height and age, was similar to predicted normal values reported in the literature; 4.032 $\pm$ 0.464 (old), 5.335 $\pm$ 0.585 (young), compared to predicted normal values of 3.568 and 5.173 respectively (Goldman and Becklake, 1959). Thus both groups had similar body size characteristics to the general population.

Another issue that needs to be addressed in this section is the use of peak $\dot{V}O_2$ as representative of the subject's maximal exercise capacity. The conventional

definition of $\dot{V}O_2max$ is the attainment of a plateau beyond which there is no further increase in oxygen intake, despite further increase in workload (Chapter 1). As discussed in Chapter 3, a plateau in $\dot{V}O_2$ was not a requirement in the present study; our values represent the peak $\dot{V}O_2$ attained at maximal exercise, defined as the inability to maintain the required pedalling frequency, despite maximal effort. Although this criterion may be criticized as being too subjective, the pre-training peak $\dot{V}O_2$ values attained by the 60-70 year old subjects in this study were similar to the pre-training values reported by Seals et al (1984) in an age-comparable population; 22.0 $\pm$ 3.8 ml/kg/min in our elderly subjects compared to 25.4 $\pm$ 4.6 ml/kg/min in the above study.

The Seals et al (1984) study is particularly relevant for comparison with the present study as the age of the subjects was comparable, but particularly because in the Seals et al study, strict criteria were used for the definition of $\dot{V}O_2max$; attainment of plateau in $\dot{V}O_2$, blood lactate of at least 8 mmoles/l, respiratory exchange ratio greater than 1.15 and attainment of age-predicted maximal heart rate. At least three of these criteria had to be present for the definition of $\dot{V}O_2max$ in the Seals et al study. Our subjects were sedentary for a number of years prior to admission to the study and their low peak $\dot{V}O_2$

values reflect this. There is no information on levels of habitual physical activity for the subjects in the study by Seals et al (1984). A mean peak $\dot{V}O_2$ of 22.0 ml/kg/min was reported by Chiang et al (1970) in sedentary 65 year olds and 31.0 ml/kg/min in a mixed sample of active and inactive subjects of the same age (Dehn and Bruce, 1972). Thus differences in the level of habitual physical activity may contribute to the differences between the Seals et al (1984) and the present study.

Furthermore, our elderly subjects achieved a mean peak heart rate of 161 beats/min, which is well within the predicted mean maximal heart rate of 168 beats/min based on the commonly used equation of 210 - 0.65 (age). In our young subjects, mean peak heart rate was 192 beats/min, which compares favourably with the predicted mean maximal heart rate of 194 beats/min based on the above equation. The peak heart rate values achieved by our subjects were also well within values reported by others; Seals et al (1984) and Saltin et al (1969) reported maximal values of about 174 b/min in 61-67 year old (mean 63) and 50-63 year old males (mean 55). These values are within 10% of the peak values attained by our 60-70 year old subjects. In both the Seals and Saltin studies, maximal exercise was defined according to well established criteria, such as the levelling-off criterion in $\dot{V}O_2max$

(Saltin et al, 1969) and criteria as outlined above for the Seals et al (1984) study. For these reasons, we feel that although attainment of the conventional criteria for a VO_2max plateau were not required in the present study, the VO_2 values attained by our subjects at peak exercise were the subjects' maximal values.

5.2.3 Cardiac Output

We determined cardiac output indirectly using the CO_2 rebreathing method for the measurement of $P\bar{v}CO_2$ from which cardiac output was derived by the indirect Fick principle, as described in the Methods and Procedures chapter. Cardiac output values calculated using this method are comparable to those of the dye dilution technique (Jones et al, 1967) and direct Fick method (Reybrouck et al, 1978).

The equation expressing the cardiac output-oxygen intake relationship in our young subjects (pre-training) was:

$$\dot{Q}\ (l/min = 6.6 + 4.7\ \dot{V}O_2)$$

The parameters of this equation are comparable with those reported in the literature: Donald et al, 1955 (direct Fick method): $\dot{Q}$ (l/min) = 5.04 + 5.3 ($\dot{V}O_2$), mean age 31 years; Faulkner et al, 1977 (CO_2 rebreathing method): $\dot{Q}$ (l/min) = 5.21 + 5.2 ($\dot{V}O_2$), mean age 36 years. The higher

intercept in the present study (6.6 l/min) is likely due to the fact that only exercise cardiac output values were included. In the above studies, both resting and exercise values were included in the calculation of the regression equations. If we use a theoretical $\dot{V}O_2$ of 1.5 and 2.5 l/min, the predicted cardiac output according to our equation is 13.7 and 18.4 l/min respectively. The corresponding values based on the above equations are 13.0 and 18.3 l/min (Donald et al, 1955) and 13.0 and 18.2 l/min (Faulkner et al, 1977). Thus our results are comparable to those reported in the literature.

The equation expressing the cardiac output-oxygen intake relationship in our elderly subjects (pre-training) was:

$$\dot{Q}\ (l/min) = 4.5 + 5.4\ (\dot{V}O_2)$$

The parameters of this equation are comparable with those reported in the literature; Granath et al, 1964 (direct Fick method): $\dot{Q}$ (l/min) = 3.21 + 5.2 $(\dot{V}O_2)$, mean age 71 years. At a theoretical $\dot{V}O_2$ of 1.0 and 2.0 l/min, the predicted cardiac output according to our equation is 9.9 and 15.3 l/min respectively. The corresponding values based on the above equation are 8.4 and 13.6 l/min (Granath et al, 1964). These differences may be explained by the fact that Granath et al (1964) included resting cardiac output values, whereas only exercise values were included

in the present study. Furthermore, differences in the age of the subjects would contribute to differences in the results; we tested subjects 60 to 70 years of age, mean age 65 years. The subjects of Granath et al (1964) were 61 to 83 years (mean age 76 years). Since age has been shown to affect the intercept of the cardiac output-oxygen intake relationship (Faulkner et al, 1977), the cardiac output values obtained by Granath et al (1964) would be expected to be lower compared to our values, as their subjects were on the average 10 years older.

The peak cardiac output, both at pre- and post-training, was estimated for each subject from submaximal values during incremental progressive exercise. Based on the residual plots analysis, as described in Chapter 3, a linear relationship was found between cardiac output and oxygen intake, therefore we felt justified to extrapolate the cardiac output value at peak $\dot{V}O_2$. The extrapolation extended commonly over only one exercise level and in only two cases did it extend over more than two exercise levels. The mean number of cardiac output measurements available for each subject was 4.6 in the old and 5.3 in the young group.

In the following sections in this chapter, the main pre-training differences in the exercise responses of the old and young subjects, and the mechanisms that may

contribute to any such differences will be discussed. This will be followed by a discussion of the training-associated adaptations in the exercise responses of the two groups to identify any differences in the mechanisms that may contribute to any such adaptations.

5.3 Pre-Training Exercise Responses in Old and Young

5.3.1 The Maximal Oxygen Intake

The maximal oxygen intake in healthy subjects is used to define the functional limits of the cardiovascular system and is influenced by the maximal cardiac output ($\dot{Q}max$) and the maximal arterio-venous oxygen ($(a-\bar{v})\ O_2$) difference as related in the Fick equation (Chapter 1):

$$\dot{V}O_2max = \dot{Q}max \times (a-\bar{v})\ O_2\ diff. \qquad \text{Equation 3}$$

The maximal cardiac output ($\dot{Q}max$) is influenced by the maximal heart rate (f_cmax) and stroke volume (V_smax) as expressed by the following equation:

$$\dot{Q}max = f_cmax \times V_smax \qquad \text{Equation 2}$$

Therefore, equation 3 can be expressed as:

$$\dot{V}O_2max = f_cmax \times V_smax \times (a-\bar{v})O_2 \text{ diff} \qquad \text{Equation 9}$$

The reciprocal of resistance expresses the vascular conductance (g) in the peripheral circulation and can be expressed as the ratio of cardiac output to mean arterial pressure (P_a) (Chapter 3):

$$g\ (l/min/mmHg) = \dot{Q}/P_a \qquad \text{Equation 8}$$

The above equations illustrate two points: that maximal exercise capacity as defined by the $\dot{V}O_2max$ is influenced by a number of central and peripheral factors; and that it is the interplay and integration between them that may ultimately define the physical capabilities of an individual. Each of these factors is affected by aging changes, both structural and functional. The discussion of the functional changes will be the subject of the subsequent sections. The structural changes will be discussed briefly, when needed to clarify differences in the mechanisms affecting exercise performance.

The equations given above will be used throughout this chapter to examine the relative contributions and interrelationships between a number of factors affecting maximal exercise capacity as defined by the peak oxygen intake.

5.3.2 Pre-Training Cardiac Output, Heart Rate and Stroke Volume

Applying equation 3 to the data in the two groups at peak exercise we have:

for the old: 1.60 (l/min) = 12.7 (l/min) x 126.5 (ml/l)

for the young: 2.54 (l/min) = 18.0 (l/min) x 140.4 (ml/l)

This comparison indicates that the 37% lower peak $\dot{V}O_2$ in the elderly was associated with a lower peak cardiac output (29%) and a lower arterio-venous oxygen difference (10%). To analyze the differences in the determinants of the maximal cardiac output, the data applied to equation 2 show:

for the old: 12.7 (l/min) = 161 (beats/min) x 78.9 (ml)

for the young: 18.0 (l/min) = 192 (beats/min) x 93.9 (ml)

This indicates that the lower peak cardiac output in the elderly was associated with a lower peak heart rate (16%) and stroke volume (16%).

5.3.2.1 Pre-Training Differences in Cardiac Output

The rise in cardiac output with increasing $\dot{V}O_2$ was similar in the old and young subjects (5.4 l/min in the elderly and 4.7 l/min in the young). However, the intercept of this relationship was lower in the elderly

($p<0.05$). These findings are in agreement with Strandell (1964) and Faulkner et al (1977). Thus at a given $\dot{V}O_2$, cardiac output was lower ($p<0.05$) in the elderly, as was the case at peak exercise. Faulkner et al (1977) examined the cardiac output-oxygen intake relationship in males 17 to 71 years of age and found no significant differences in the slope of this relationship. However, the intercept was lower ($p<0.05$) in 50 to 71 year old compared to 20 to 30 year old subjects (Faulkner et al, 1977). Strandell (1964) reported that the lower intercept in the elderly resulted in a lower cardiac output (about 1.5 l/min) at a given level of oxygen intake. Since heart rate at a given $\dot{V}O_2$ was higher ($p<0.001$) in the elderly, their lower cardiac output may be attributable to decreases in stroke volume, as will be discussed later in this section.

Our findings are in general agreement with others, as reviewed in Chapter 1, which showed a 25% to 30% decrease in peak cardiac output between 25 and 65 years. However, they are not in agreement with the findings of Rodeheffer et al (1984) and Gerstenblith et al (1987) who reported no age-related differences in resting, submaximal or peak cardiac output in participants of the Baltimore Longitudinal Study on Aging (B.L.S.A.). In this study, both submaximal and peak cardiac output were maintained at levels not significantly different from

those of younger subjects. This was achieved largely by increases in stroke volume, associated with a greater increase in end-diastolic volume.

The differences between the B.L.S.A. and our findings may be explained by differences in subject selection and assignment to young and older groups. The B.L.S.A. subjects were healthy, highly motivated and physically active males who have been tested repeatedly over a number of years. Our subjects were also healthy and highly motivated, but were sedentary for a number of years. In fact the latter was an admission requirement to the study. Furthermore, in the B.L.S.A., the young group consisted of subjects 25 to 44 years; the middle-aged group was 45 to 64 years, and the old group was 65 to 80 years (mean age for these groups was not given). Therefore there is a tendency in the B.L.S.A. towards a bias in favour of over 45 year old subjects which may mask differences between young and elderly subjects. In contrast, we had a much clearer distinction between the two groups; young subjects in their 20s and older subjects in their 60s.

The pre-training differences in the determinants of cardiac output, heart rate and stroke volume will be the subject of the following sections.

5.3.2.ii Pre-Training Differences in Heart Rate

Since the early work of Robinson in 1938, a number of investigators have documented the decline in peak heart rate with increasing age. The decrease is in the order of 15 to 20% and is a consistent finding in studies of both sedentary individuals (Van Brummelen et al, 1981; Jones et al, 1985; Julius et al, 1967; Hossack et al, 1980; Rodeheffer et al, 1984) and master athletes (Hagberg et al, 1985; Ekelund and Holmgren, 1967; Ekblom and Hermansen, 1968). The fact that peak heart rate decreases with increasing age is an undisputed phenomenon observed in many studies, despite differences in subjects and testing protocols. What is disputed are the mechanisms that may contribute to this age-related decrease which will be discussed below.

5.3.2.iii Decline in Intrinsic Sinoatrial Frequency

Blockade of cardiac autonomic nervous activity by intravenous injection of propranolol (used to block sympathetic stimuli) and atropine (used to block parasympathetic stimuli) showed that the intrinsic heart rate (I.H.R.) declines with increasing age (Jose et al, 1970). In healthy young adults, 20-30 years of age, the average I.H.R. was 104.4 $\pm$ 7.4 beats/min, with a progressive decline to 92.4 $\pm$ 9.0 beats/min by the age of 45 to 55 years (Jose, 1966). Intrinsic heart rate was

reported to decrease by 0.53 beats/min per year as expressed by the following equation (Jose, 1966):

$$I.H.R. = 117.2 - 0.53 \text{ (age)}$$

According to this equation, the I.H.R. of a 70 year old is predicted to be 80 beats/min, a decrease of about 23% from the age of 25 years, which is similar to the decrease in peak heart rate with increasing age. Application of the above equation to the elderly and young subjects in the present study reveals an average I.H.R. of about 83 beats/min and 104 beats/min respectively; a difference of about 20%, which is similar to the 16% difference observed in the two groups at peak exercise.

5.3.2.iv **Response of Beta Adrenergic Receptors**

The maximal heart rate response to isoproterenol (a sympathomimetic drug) was found to be about 15% lower in old (10-12 years) compared to young (1-4 years) dogs (Yin et al, 1979). Furthermore, both young and old animals responded equally to electrically-induced atrial pacing at a rate twice the maximal evoked by isoproterenol. Thus a decrease in the response of pacemaker cells to electrical stimulation was ruled out (Yin et al, 1979). Van Brummelen et al (1981) reported similar results in males over 50 years and under 25 years

of age. An attenuated heart rate response was found with progressively higher doses of intravenous isoproterenol in the older compared to the young subjects. In this study, the dose of isoproterenol required to produce an increase in heart rate of 25 beats/min was significantly higher in the older compared to the young subjects. These findings suggest that a diminished beta-adrenergic response to catecholamines may contribute towards the fall in peak heart rate with increasing age.

5.3.2.v Other Age-Related Changes

Inhalation of gas with a low PO_2 (40mmHg) resulted in an increase in heart rate of 34% in the young and only 11% in older subjects (Kronenberg and Drage, 1973). In the same study, an increase in $PaCO_2$ to 55 mmHg was associated with a 15% increase in heart rate in the young with no heart rate changes in the older group, suggesting a blunted chemoreceptor activity with advancing age.

A progressive increase in fat and connective tissue in aging cardiac pacemaker cells has been reported (Sell and Scully, 1965; Lev, 1954). The duration of cardiac muscle contraction and relaxation is 15-20% longer in old (24-31 months) compared to young rats 6-13 months (Lakatta et al, 1975; Weisfeldt et al, 1971). This was

associated with a prolongation of calcium activation of the myofibrils and its slower uptake by the sarcoplasmic reticulum. These changes may limit the capacity of aging pacemaker cells to increase their rate of firing so that an upper limit may be reached earlier, contributing to a lower peak heart rate.

In summary, it is clear that there is not one single mechanism contributing to the decline in peak heart rate with increasing age, but rather a multitude of factors. The available evidence weighs towards a diminished beta-adrenergic response as a major contributing factor to the age-related changes in peak heart rate. Other changes, as discussed above, will likely contribute, but perhaps to a lesser extent.

5.3.2.vi Pre-Training Differences in Stroke Volume

Peak stroke volume, in both groups, was reached at about 55-60% of peak $\dot{V}O_2$ and was 88.2 $\pm$ 4.7 ml in the elderly and 99.6 $\pm$ 4.3 ml in the young subjects, a difference of about 11%. At peak exercise, the calculated stroke volume in the elderly was 78.9 $\pm$ 3.5 ml compared to 93.9 $\pm$ 3.9 ml in the young subjects, a difference of about 16% ($p<0.001$, Table 3).

Our findings are in agreement with others (Granath et al, 1964; Astrand, 1967; Julius et al, 1967;

Hossack and Bruce, 1982). A decline in peak stroke volume of 0.36 ml/year was observed by Hossack et al (1980); about a 13% decline over the age range 25-65 years, which is similar to the 16% difference observed in the present study. A number of age-related changes in the mechanisms that regulate stroke volume may contribute to the decrease in stroke volume observed in the elderly subjects.

5.3.2.vii Myocardial Contractility

An attenuated inotropic response to catecholamines was observed in isolated hearts of senescent rats (25 months) compared to middle-aged (12 months) and young adult rats (6 months, Lakatta et al, 1975). The maximal rate of tension development (dT/dt) to both single and cumulative doses of norepinephrine was significantly less in the aged myocardium than in the myocardium of the middle-aged and young groups. Peak active isometric tension increased in the young and middle-aged groups but did not change significantly from the baseline value in the old group at any norepinephrine concentration. A similar pattern was observed in response to isoproterenol; dT/dt increased more and active tension was greater in the young compared to the old group. These findings suggest a decrease in myocardial contractility with increasing age.

A likely mechanism for this is a decreased

ability of catecholamines to increase the intracellular calcium available for contraction. This hypothesis was examined by Lakatta et al (1975) by observing the inotropic response to increasing concentrations of calcium; there was no age difference in the increase in developed tension or maximal rate of tension development, which suggests that the ability of the aged myocardium to respond to increased calcium is intact.

The diminished inotropic response of the aged myocardium appears to occur despite augmented plasma norepinephrine levels with increasing age (Fleg et al, 1985; Palmer et al, 1978). Plasma norepinephrine, epinephrine, and $\dot{V}O_2$ were measured at rest and during progressive incremental treadmill exercise in three groups of young (22-37 years), middle-aged (44-55 years) and old (68-77 years) healthy males (Fleg et al, 1985). Plasma norepinephrine was not age-related at rest, but epinephrine was significantly higher in the old compared to the young and middle-aged groups. At peak exercise, both plasma norepinephrine and epinephrine were significantly higher in the elderly compared to the younger subjects. This was also observed during submaximal exercise and at a given percent of peak $\dot{V}O_2$ (Fleg et al, 1985).

Plasma catecholamines may not necessarily reflect the magnitude of catecholamine release at the nerve

terminals but may also reflect their clearance rate, which has been reported to decrease with increasing age (Esler et al, 1981). Whatever the cause, plasma catecholamines appear to be higher in the elderly associated with a decrease in peak heart rate and myocardial contractility. Thus a diminished responsiveness to catecholamines may be a likely mechanism.

If this is the case, a diminished responsiveness to catecholamines in elderly subjects may result in a decrease in myocardial contractility, an increase in end-systolic volume, a decrease in ejection fraction, and a decrease in stroke volume. Gerstenblith et al (1987) assessed ventricular function via gated radionuclide angiography during exercise (cycle ergometry) in young and elderly healthy, physically active subjects (25-44 years, 45-64 years and 65-80 years) who were participants in the Baltimore Longitudinal Study on Aging. Neither stroke volume, nor cardiac output were found to be age-related. However, they observed a failure of the end-systolic volume to decrease during exercise in the elderly to the same extent as in younger subjects, which resulted in less of an increase in the ejection fraction of the elderly. Furthermore, the increase in exercise stroke volume in their elderly subjects was achieved by a greater increase in end-diastolic volume

rather than a greater decrease in end-systolic volume as was the case in the younger subjects. These findings suggest a greater reliance on the Starling mechanism in the elderly as a compensatory mechanism for diminished myocardial contractility.

5.3.2.viii Cytological Changes in the Myocardium

There appears to be an age-related increase in the amount of connective tissue within the myocardium associated with diminished myocardial compliance (Wilens and Sproul, 1938; Tomanek et al, 1972; Gerstenblith et al, 1976). Pathological examination of the hearts of 487 rats maintained on adequate diets and under constant laboratory conditions over their entire life span revealed myocardial fibrosis in 60% of the animals. The fibrotic lesion was evident at about the 400th day of life (about 32 years of human life) and became more marked with advancing age (Wilens and Sproul, 1938). Tomanek et al (1972) reported increases in connective tissue in the hearts of old (22 months) compared to young (6 months) rats. This was particularly evident at the attachments of the tricuspid and mitral valves and in the region of the atrioventricular bundle. These changes were similar in extent in both exercised (12 weeks treadmill running) and non-exercised young and old rats (Tomanek et al, 1972).

Echocardiographic examination at rest in 105 healthy males 25 to 84 years of age revealed decreased left ventricular filling rate with increasing age (Gerstenblith et al, 1977). This was based on the decreased rate of closure of the mitral valve which is considered to be proportional to the rate of left ventricular filling in early diastole (Zaky et al, 1968). The decreased rate of closure of the mitral valve may result from age-associated sclerosis and thickening of valve leaflets as reported by Sell and Scully (1965). Diminished early diastolic filling rate may also result from increased left ventricular stiffness (Templeton et al, 1975). Greater ventricular wall thickness as well as age-associated alterations in the amount of collagen and fibrous tissue in aged myocardium, as discussed previously, may also contribute to increased ventricular stiffness and decreased ventricular compliance. Thus diminished left ventricular filling rate may be the result of age-associated changes in the mitral valve and/or changes associated with myocardial compliance. These changes suggest increased resistance to filling of the heart.

However, despite these changes, end-diastolic volume does not appear to be compromised as shown by Rodeheffer et al (1984) and Gerstenblith et al (1987) in healthy, physically active males.

In summary, the lower peak heart rate in the elderly subjects together with their lower stroke volume result in a fall in cardiac output at peak exercise which will affect the capacity of these elderly subjects to do maximal physical work. The available evidence weighs toward diminished beta adrenoreceptor responsiveness as a major contributing factor to the decrease in peak heart rate and stroke volume. Structural changes in the myocardium will also contribute by decreasing myocardial compliance and increasing the resistance to filling of the heart. However, it would appear that in healthy, physically active elderly, stroke volume can be maintained, by increases in diastolic filling time and end-diastolic volume. Thus increased diastolic filling time may be a necessary compensatory mechanism for decreased ventricular compliance.

5.3.3 Pre-Training Differences in Mean Arterial Pressure

A major difference between the two groups was in mean arterial pressure, which was higher in the elderly at all levels of exercise ($p<0.01$). At peak exercise, this was 131.1 $\pm$ 3.4 mmHg in the elderly and 113.8 $\pm$ 2.2 mmHg in the young ($p<0.001$). The rise in systolic blood pressure for a given increase in $\dot{V}O_2$ (Figure 29) was higher in the elderly ($p<0.001$); 44 mmHg compared to 31 mmHg in the

young, associated with a diminished ability to increase vascular conductance during exercise (Figure 17). The higher systolic blood pressure in the elderly is a consistent finding in studies of both sedentary and trained individuals (Strandell, 1964; Hanson et al, 1965; Granath et al, 1964; Julius et al, 1967; Hagberg et al, 1985; Jones et al, 1985).

Major mechanisms likely to contribute to the increased arterial pressure in the elderly will be discussed below.

5.3.3.1 Structural Changes in the Arterial Wall

A decrease in aortic compliance with increasing age was reported by a number of investigators (Roy, 1952; Roach and Burton, 1959; Learoyd and Taylor, 1966; Bader, 1967) and is believed to be at least partly explained by a relative loss of elastin and an increase in collagen in the aging arterial wall. Urschell et al (1968) substituted a stiff tube for the aorta in dogs, while heart rate was maintained at a constant rate; they observed higher aortic systolic pressure and end-diastolic left ventricular pressure and volume, and lower ejection fraction and cardiac index. Furthermore, the increase in left ventricular end-diastolic pressure resulted in an increase in left ventricular wall tension. These findings suggest

that decreased aortic compliance increases the impedance to ejection and the tension load on the myocardium.

Compliance refers to the slope of the volume/pressure ($\Delta V/\Delta P$) relationship; i.e. the change in volume relative to the change in pressure. If aging vessels are less compliant, then a change in volume will accompany a relatively larger change in pressure compared to vessels that are more compliant. An indirect measure of vascular compliance can be estimated by applying the $\Delta V/\Delta P$ relationship to stroke volume and pulse pressure. At peak exercise:

Pre-training old:
Compliance (ml/mmHg) = 78.9/111.2 = 0.71

Pre-training young:
Compliance (ml/mmHg) = 93.9/115.2 = 0.82

Thus estimated vascular compliance in the elderly was about 13% lower compared to that in the young subjects. The reciprocal of compliance gives an index of stiffness in a vessel. Applying this to the above data, the elderly exhibit a higher estimated stiffness in their vessels; 1.41 mmHg/ml compared to 1.22 mmHg/ml in the young. Although such comparisons need to be interpreted with caution, as they are based on only indirect estimates, they are useful in providing some indication on the

volume/pressure relationship as blood is ejected into the arterial tree.

5.3.3.ii **Changes in Arteriolar Vasoconstrictor Tone**

As discussed in Chapter 1, the arterioles are under the influence of an inherent constrictor tone, known as basal tone (Barcroft, 1963). Neural and metabolic influences modify this tone and regulate the amount of blood flow to the tissues. Norepinephrine released by postganglionic sympathetic nerve endings binds with alpha receptors in the vascular wall to produce vasoconstriction. Stimulation of beta-adrenergic receptors, on the other hand, results in vasodilation. The close relationship between metabolism and vasodilation has led to the search for a metabolic by-product as the mediator of arteriolar vasodilation. Some of the suggested vasodilators are lower PO_2 and elevated CO_2, H^+ concentration, potassium and adenosine (Watts and Johnson, 1981; Stowe et al, 1975).

What is known about age-associated changes in vasoconstrictor tone is based largely on pharmacologic studies in animals that used specimens from large arteries. These studies demonstrate that at least the large arteries undergo functional age-associated changes that result in an impaired ability to relax in response to drugs (Fleisch et al, 1970; Fleisch, 1971; Ericsson and Lundholm, 1975).

Fleisch and Hooker (1976) reported that the degree of relaxation in response to isoproterenol, after alpha-receptor blockade, in the aorta and pulmonary artery of rats and rabbits decreased markedly with increasing age. Aortas from 2-year old rabbits relaxed to only 17% of maximum, whereas specimens from 2-month old rabbits relaxed to 85% of maximum. Pulmonary artery specimens responded in a similar fashion. Overall, only about 20% of the maximal response to isoproterenol remained in the large vessels from 9-month old rats and 2-year old rabbits compared to young animals.

Ericsson and Lundholm (1975) reported similar results in young and old rats; the relaxing action of isoproterenol on the aortic strips decreased with increasing age to the point that there was no relaxing effect in animals 6 months of age. The age at which isoproterenol begins to lose its ability to relax the aortic strips was 10 weeks, which coincides with the time that blood pressure begins to rise in normal rats (Okamoto and Aoti, 1963). These findings suggest an age-related decrease in vascular beta-adrenergic responsiveness in the large arteries of animals. We do not know if this also applies to the smaller vascular beds, which are largely responsible for the regulation of vascular conductance and mean arterial pressure.

A pioneer study on vascular beta-adrenoreceptor sensitivity in man was recently reported by Van Brummelen et al (1981). This was determined by measuring the change in forearm blood flow (venous occlusion plethysmography) in response to intra-arterial infusion of isoprenaline (isoproterenol) in two groups of old (53-70 years) and young (12-24 years) healthy subjects. Resting forearm blood flow and vascular resistance were of a similar magnitude in the two groups. However, the change in forearm blood flow with incremental doses of intra-arterial isoprenaline was significantly lower in the older group; the mean change in blood flow over all doses of isoprenaline was 48% lower in the elderly.

The relevance of animal studies for the interpretation of human data may be limited by the fact that in the animal studies isolated aortae or larger arteries were used. Nevertheless, the results demonstrate diminished beta-adrenoreceptor function with increasing age. Indirect evidence (Van Brummelen et al, 1981) that the observed differences in the vascular response to isoprenaline in the young and old were related to the beta-adrenoreceptor was provided by the parallel decrease in the heart rate response to isoprenaline; another beta-adrenoreceptor mediated function.

In summary, it would appear that structural changes in the arterial wall contribute to an age-related decrease in vascular compliance, so that a given volume will accompany a relatively larger increase in pressure compared to younger, more compliant vessels. These changes together with a diminished vascular beta-adrenoreceptor responsiveness likely contribute to the increase in arterial pressure and decrease in vascular conductance in the elderly subjects.

5.3.4 Short-Term Dynamic Muscle Function

Parallel to the decline in oxygen delivery mechanisms is an age-associated decrease in skeletal muscle structure and function. As discussed in Chapter 2, we examined the relationship between maximal power output (during 30 secs of isokinetic cycling) and peak $\dot{V}O_2$ (during progressive incremental cycling), to examine the extent to which integrity in muscle function may contribute to the maintenance of peak aerobic capacity. A finding of interest in that study was the close relationship between total work achieved in 30 secs of maximal isokinetic cycling and peak $\dot{V}O_2$, in 100 healthy males and females 15 to 70 years of age ($r = 0.89$, $p<0.001$, Appendix 2, Figures 3 and 4). We wanted to investigate this relationship further in this study to determine whether similar

training-associated adaptations may occur in both muscle function and aerobic capacity.

We tested the subjects in the present study at pedalling frequencies of 60 and 110 rpm. The 60 rpm velocity was used because it was the same frequency of pedalling used in the progressive incremental exercise test. We wanted to examine whether there was a muscle component to the aerobic capacity measurements. The higher velocity was used as it is the velocity at which peak power is developed (McCartney, 1983) and because we wanted to examine whether any training-induced adaptations may occur in the type II fibres, which were considered to contribute more to the cycling exercise at the higher speed.

Average power output and total work during 30 secs of maximal cycling were significantly lower ($p<0.001$, Table 4) in the elderly compared to the young subjects (28% and 30% respectively). Post-exercise plasma lactate was also lower (27%) in the elderly ($p<0.001$). However, no significant differences were observed in the fatigue index between the two groups; 34% in the elderly and 29% in the young at 60 rpm, and 59% and 63% respectively at 110 rpm (Table 4).

5.3.4.1 **Changes in Aging Skeletal Muscle**

As reviewed earlier (Chapter 1), major age-associated changes that may contribute to the observed differences between the elderly and young muscles are decreases in lean muscle mass, muscle fibre number and size and reduction in strength. The decrease in lean muscle mass is a common characteristic of aging muscle. A large epidemiologic study of 959 healthy males in the Baltimore Longitudinal Study on Aging revealed a 45% decrease in lean muscle mass (measured by 24-hour creatine excretion) from 20 to 90 years of age (Tzankoff and Norris, 1977). The changes in muscle mass may be the result of reductions in the number and size of muscle fibres.

A common change reported in the literature is a selective atrophy of the high force and fast contracting type II muscle fibres. This was found to occur particularly over the age of 50-60 years (Larsson, 1978; Grimby et al, 1982; Aniansson et al, 1978; Grimby and Saltin, 1982). The ratio of type II to type I fibres tends to decrease, from about 60% in 20-29 year olds to 40% in 60-69 year olds. This appears to be associated with an age-related decrease in the type II fibres and a relative stability in the type I fibres (Orlander et al, 1978; Kiessling et al, 1973).

From these and other studies reviewed in Chapter 1, it would appear that type I muscle fibres are relatively insensitive to age-associated changes at least up to the sixth decade of life. In contrast, a preferential atrophy of type II fibres appears to occur after the fourth decade of life. Beyond 70 years, reductions in fibre size tend to occur in all fibre types, although they continue to be more striking in the type II fibres (Tomonaga, 1977).

The decreases in muscle mass and fibre number and size are associated with decreases in strength in elderly individuals. Studies of isometric and dynamic strength from childhood to the seventh decade in life revealed a progressive increase in strength up to the age of 20 years, a relative stability up to the age of 49 years with a progressive decline from then on (Larsson, 1978). It appears that strength is reasonably well maintained at least up to the fifth decade in life.

Vadervoort (1984) recently reported maximal voluntary isometric strength data in subjects up to the age of 100 years; he found that significant decreases in muscle strength did not occur until after the age of 79 years. Isometric and dynamic muscle endurance on the other hand, show a relative stability with increasing age (Johnson, 1982; Larsson and Karlsson, 1978; Larsson, 1978; Petrofsky

et al, 1975). This is in agreement with our own findings of no differences between the elderly and young subjects in the fatigue index and may be associated with the relative chronologic stability of type I muscle fibres.

In summary, age-related decreases in skeletal muscle mass, fibre number and size and strength contribute to the decrease in the ability to produce short-term power output. However, the decline in average power during short-term maximal exercise is well maintained in these elderly subjects and appears to be associated with the relative stability of type I muscle fibres, in contrast to a preferential age-related atrophy of type II fibres.

5.4 Post-Training Exercise Responses in Old and Young

5.4.1 The Change in Peak Oxygen Intake

After the 12-week endurance training program, peak $\dot{V}O_2$ increased ($p<0.001$) in both groups with no differences in the magnitude of change in the two groups. The increase amounted to 38% in the elderly (1.599 $\pm$ 0.073 to 2.212 $\pm$ 0.073 l/min) and 29% in the young (2.536 $\pm$ 0.141 to 3.263 $\pm$ 0.181 l/min).

In Table 5, a summary of the present and previous training studies that employed middle aged and elderly subjects is given.

The 38% increase in peak $\dot{V}O_2$ is higher than previously reported in over 60 year old sedentary males, with the exception of the Barry et al (1961) study. However, as discussed in Chapter 1, part of the change in peak $\dot{V}O_2$ in the latter study may have been due to the fact that in some subjects, the pre-training exercise test had to be stopped prematurely because of ECG abnormalities, and peak initial values may not have been reached. Therefore, the reported pre-training peak $\dot{V}O_2$ of 16.1 ml/kg/min in the Barry study may not be the subjects' true peak $\dot{V}O_2$ value.

In other studies (Table 5), the percent increase in peak $\dot{V}O_2$ varies from 0% to 30%. Differences among the various studies in subjects and training protocols make comparisons difficult and undoubtedly contribute to the differences in findings. Major differences include the initial level of fitness of the subjects and differences in length, format and intensity of training.

Table 5. Summary of Present Study and Previous Training Studies in Older Subjects

Study	Number of Subjects	Age Range (years)	Activity Level	Type of Training	Length of Training (weeks)	Training Heart Rate (b/min)	Pre-Training Peak VO_2 (ml/kg/min)	% Change in Peak VO_2	% Change in Peak Q	% Change in Peak H.R.	% Change in Peak (a-v) O_2 diff.
Barry et al (1961)	8 5 males 3 females	55-78 mean 70	Sedentary	40 mins x 3/week Interval, cycling, balance, flexibility	12	at least 130 b/min	16.1 (1.20 ± 0.30 l/min)	38	-	14	-
Benestad (1965)	13 males	70-81 mean 75.5	Active	30 mins x 3/week Interval, treadmill walking	5-6	50-80% of max. aerobic capacity	27 ± 2.5	0	-	1.3	-
Hanson et al (1968)	7 males	40-49	Sedentary	60-90 mins x 3/week running, calisthenics, flexibility, volleyball, basketball, etc.	29	Not prescribed	35.1	17.6	Not determined at peak exercise	0	-
De Vries (1969)	112 males	51-87 mean 69.5	Not stated	60 mins x 3/week Interval walk, jog, calisthenics, swimming	42	<145	33.4 ± 7 (estimated by Astrand Test at heart rate of 145 b/min)	8.3	-	Peak heart permitted 145 b/min	-
Hartley et al[b] (1969)	15	38-55 mean 47	Sedentary	60 mins x 2-3/week Interval/Continuous walk, jog, calisthenics	8-10	Near maximal level	2.68 ± 0.44 (l/min)	14	13	3.2	0.7
Saltin et al[b] (1969)	42 males 8	34-50 mean 40 50-63 mean 55.3	Sedentary	60 mins x 2-3/week Interval/Continuous walk, jog, calisthenics	8-10	Near maximal level	37.5 ± 0.89 28.0	18 19	- -	-3.6 -2.8	- -
Willmore et al (1970)	7 males	45-59 mean 52.9	Sedentary	Jogging, 12-24 mins x 3/week	10	Not prescribed	40.3 ± 4.9	3.6	-	-5.5	-
Kilbom and Astrand (1971)	13 females 9 4	21-61 21-48 (Group A) mean 34.5 53-61 (Group B) mean 55	Sedentary	30 mins x 3/week Interval, cycling	7	70% of max. aerobic power	Group A: 33.2 ± 1.3 Group B: 25.6 ± 1.3	A: 10 B: 7	A: 10 B: 8	A: 0.5 B: 2.4	A: 0.7 B: -5

Study	Number of Subjects	Age Range (years)	Activity Level	Type of Training	Length of Training (weeks)	Training Heart Rate (b/min)	Pre-Training Peak VO_2 (ml/kg/min)	% Change in Peak VO_2	% Change in Peak Q	% Change in Peak H.R.	% Change in Peak (a-v) O_2 diff.
Pollock et al[b] (1971)	6 males	40-56 mean 48.9	Sedentary	40 mins x 4/week walking	20	132 progressing to 146	29.6	28	-	-0.3	-
Trankoff et al (1972)	8 males	54-66 mean 58.4	Sedentary	50-60 mins x 2-3/ week, handball, squash, tennis	25	Not stated	29.4	15.5	-	1.8	-
Kasch et al (1973)	7 males	39-60 mean 46.3	Sedentary	60 mins x 3/week 30 mins calisthenics and 30 mins walk/jog	104	70% of max. heart rate progressing to 86-87% by 52 weeks	32.6 ± 3.3	17	-	-3.2	-
Hiinima and Shephard (1978)	19 9 males 10 females	60-76 mean 65.4	Active	60 mins x 4/week Interval, walk, jog, balance, calisthenics	11	125-155	33.4 males 24.6 females	5.2	reported as slight	-2.2	slight change
Seals et al[a] (1984)	11 7 males 4 females	61-67 mean 63.6	Not stated	Low Intensity (L.I.): 20-30 mins 3/week, walking. High Intensity (H.I.): 30-45 mins 3-4/week, walk, jog, cycling.	52 26 L.I. 26 H.I.	L.I. mean 107 H.I. mean 156 (over final 8 weeks)	25.4 ± 4.6	30	6	0	14
Present Study	12 males	60-70 mean 65	Sedentary	60 mins x 3/week Interval cycling (excluding warm-up and cool-down)	12	140 (85% of peak VO_2)	22.0 ± 3.8	38	29.9	7.4	6.5

a VO_2max defined as presence of at least three of the following criteria: 1) no further increase in VO_2 with increase in work rate (levelling off criterion), 2) attainment of age-predicted maximal heart rate, 3) RER greater than 1.15, 4) blood lactate 4 mins. post-exercise of at least 8 mmoles.

b VO_2max defined as attainment of levelling-off criterion.

5.4.2 Comparison of Present with Previous Training Protocols

The training protocol in the present study required that the subjects train at a heavy intensity, mean training heart rate 140 beats/min. This was attained by the third week of training and was maintained without exception and with no major interruptions for the remaining 9 weeks (Figure 5); a missed session in one week was rescheduled in the same week. We were fortunate not to have any major musculoskeletal injuries other than minor knee and back problems. These were handled largely by adjusting the handle bars or seat on the cycle ergometers. The subjects were sufficiently motivated and committed to the study so their full cooperation and compliance with the program was secured. These are important considerations in training studies and are often not addressed sufficiently in published reports.

The format of training was interval cycling, with 5-minute bouts of heavy exercise (85% of peak $\dot{V}O_2$) interrupted only by a brief pause to allow the subjects to record their heart rate, and then continuing with 3 minutes at a lower intensity (65% of peak $\dot{V}O_2$, Figure 5). All subjects reached this intensity of training within two weeks of the start of the training program.

In previous training studies, the intensity of training is often not clearly stated and in some cases, no specific training heart rate is prescribed (Table 5); statements such as near maximal levels, less than 145 beats/min and other generalizations are often used to indicate intensity of training. Furthermore, of the stated number of weeks of training, there is no clear indication as to when the prescribed intensity was reached, or whether the required number of training sessions were attained with or without interruptions.

The length of training (Table 5) varies from 5 to 104 weeks. Since this is also an important consideration, some trends towards greater improvement with longer training could be expected. However, there is no clear trend evident as indicated in Table 5. The longest study, that of Kasch et al (1973) with 104 weeks of training reported a 17% increase in peak VO_2; DeVries (1969) with 42 weeks of training reported only an 8.3% increase; Hanson et al (1968) reported a 17.6% increase after 29 weeks of training. What is evident from Table 5 is that the initial level of fitness of the subjects and the intensity of training are likely the most important variables in the amount of improvement expected after training. The former was convincingly shown by Pollock (1973); subjects with high initial peak VO_2 (over 45

ml/kg/min) training for an average period of 16 weeks improved their peak $\dot{V}O_2$ by 4% to 12%. In contrast, subjects of the same age, with low initial peak $\dot{V}O_2$ values (less than 35 ml/kg/min) training at the same intensity and frequency, showed a greater improvement, 22% to 43%. There is less agreement with regards to the question of whether high intensity training should be advocated in elderly, previously sedentary individuals.

5.4.2.1 Safety of High Intensity Training in the Elderly

As recently as a few months ago, Ehsani (1987) and Hagberg (1987) proposed that endurance training can increase the exercise capacity of elderly subjects, but advised that training be of low intensity, and that intensity, duration and frequency be increased gradually over a period of months. Such statements are a common occurrence in reports dealing with exercise and aging and are based on the belief that high intensity training is unsafe for older individuals.

We have shown that high intensity training can be both safe and well tolerated by older subjects. Our over 60 year old subjects were sedentary for a number of years with peak $\dot{V}O_2$ values approximately 83% of predicted values for their age. They were not ex-athletes and had

never participated in endurance training programs other than regular sports in their youth. Yet they tolerated a high intensity training program of twelve weeks duration with no cardiac or other complications, and no interruptions in training. The training power output by the sixth week in the program exceeded the mean maximal power output achieved during the initial incremental exercise test.

It ~~is~~ recognized that these findings are based on only a small sample of older individuals. Consequently, generalizations to other subjects of comparable age need to be made with caution. However, we feel that our subjects were representative of the older sedentary, healthy population. Thus, assuming that older subjects are appropriately screened for cardiovascular and other diseases, that the exercise intensity is prescribed on the basis of an incremental exercise test and that the subjects are closely monitored during training, high intensity exercise may be as safe for 60-year-old as it is for 20-year-old subjects.

5.4.3 Effects of Training on Cardiac Output, Heart Rate and Stroke Volume

Applying equation 1 to the pre- and post-training data in the elderly we have:

pre-training: 1.599 (l/min) = 12.7 (l/min) x 126.5 (ml/l)

post-training: 2.212 (l/min) = 16.5 (l/min) x 134.7 (ml/l)

The comparison indicates that the 38% increase in peak $\dot{V}O_2$ in the elderly was associated with a 30% increase in peak cardiac output and only a 6% increase in the peak arterio-venous oxygen difference. In the young subjects, peak cardiac output and arterio-venous oxygen difference contributed to the same extent (14%) to the increase in peak $\dot{V}O_2$.

Haemodynamic changes after intensive endurance training in previously sedentary healthy males over 60 years of age and direct comparisons with subjects 40 years younger have not been reported before. There is little information on training-associated haemodynamic adaptations in elderly subjects in the literature with which we can compare our results. There are only four studies that reported haemodynamic changes before and after training in older, sedentary individuals (Table 5). Hartley et al (1969) reported a 14% increase in peak $\dot{V}O_2$, associated with a 13% increase in cardiac output; no significant change was found in the arterio-venous oxygen difference at peak exercise. The 7% increase in peak $\dot{V}O_2$ reported by Kilbom and Astrand (1971) was associated with an 8% increase in peak cardiac output, and a decrease in peak arterio-venous oxygen difference (-5%). Both of these

studies employed subjects in their middle years (38 to 61 years).

Recent studies (Niinima and Shephard, 1978; Seals et al, 1984) on endurance training in the elderly revealed small, not significant, changes in peak $\dot{V}O_2$ (5.2%) with no significant change in cardiac output or arterio-venous oxygen difference (Niinima and Shephard, 1978). Seals et al (1984) found a large increase in peak $\dot{V}O_2$ (30%) after twelve months of endurance training in elderly subjects, accompanied by increases in arterio-venous oxygen difference (14%) and only a small increase in cardiac output (not significant). The studies of Niinima and Shephard (1978) and Seals et al (1984) are particularly relevant to the present study as they are relatively recent studies that recruited subjects of comparable age. The differences in findings between Niinima and Shephard (1978) and the present study are likely due to differences in training intensity, as discussed earlier; essentially no major training-associated adaptations occurred in this study. However, in the Seals et al (1984) study, a large increase in peak $\dot{V}O_2$ was reported with no change in peak cardiac output. This may be at least partly explained by differences in the method of estimating peak cardiac output.

The method used in the present study was based on extrapolation of cardiac output from submaximal values to peak $\dot{V}O_2$, after calculation of the regression equation expressing the cardiac output-oxygen intake relationship for each subject (Chapter 3). In the study by Seals et al (1984), peak cardiac output was estimated by multiplying the average stroke volume during three submaximal work rates with the peak heart rate. This was based on the assumption that stroke volume reaches a peak value at about 40% of peak $\dot{V}O_2$ and levels off thereafter. This assumption is questionable as there is some degree of variability in stroke volume behaviour which may progressively increase up to 60% of peak $\dot{V}O_2$ of higher and either level off or decrease somewhat from then on. This may contribute to the differences in the results between the Seals et al (1984) and the present study.

It would appear from the limited studies available that peak cardiac output can increase in elderly subjects in response to endurance training. Our finding of almost a 30% increase has not been reported before in this population. Changes in the determinants of cardiac output and mechanisms likely to produce these changes will be discussed below.

5.4.3.1 **Changes in Heart Rate and Stroke Volume**

The increase in peak cardiac output in the elderly subjects was associated with marked increases in peak stroke volume (21%); heart rate at peak exercise increased by 7% (Table 3). These changes are illustrated by applying equation 2 to the data observed in the elderly which show:

pre-training: 12.7 (l/min) = 161 (beats/min) x 78.9 (ml)

post-training: 16.5 (l/min) = 173 (beats/min) x 95.6 (ml)

During submaximal exercise, the change in heart rate for a given increase in $\dot{V}O_2$ decreased significantly after training in both groups ($p<0.001$); from 63 beats/min to 52 beats/min in the elderly and from 45 beats/min to 40 beats/min in the young (Figures 22 and 27). This was accompanied by increases in stroke volume (Figure 22) that were statistically significant only in the older group ($p<0.001$). The increased stroke volume resulted in a significant increase in submaximal cardiac output ($p<0.05$) in the elderly with no change in the young subjects.

A number of mechanisms may contribute towards the training-associated changes in stroke volume and heart rate.

5.4.3.11 **Changes in Autonomic Nervous System Activity**

A well established adaptation of endurance training is a lower heart rate during submaximal exercise. Training-associated reductions in submaximal heart rate were found to be accompanied by lower plasma norepinephrine concentrations (Cousineau et al, 1977; Peronnet et al, 1981), suggesting a reduced sympathetic outflow to the heart. Clausen et al (1973) found that the reduction in heart rate after training was closely related with an increase in hepatic dye (indocyanine green) clearance ($r = -0.82$). Similar high negative correlations were also reported by Rowell et al (1964 and 1965) for heart rate and splanchnic-hepatic blood flow. In agreement with splanchnic blood flow studies, Grimby (1965) observed higher renal blood flow values in well-conditioned athletes at any given absolute VO_2 and a high inverse relationship between heart rate and renal blood flow ($r = -0.89$).

These findings suggest that the sympathetic outflow that accompanies increases in heart rate and vasoconstriction in abdominal viscera may be reduced after training. Further support for this is the finding of a greater decrease in heart rate after beta blockade (intra-venous injection of propranolol) in sedentary

subjects compared to well-trained athletes (Brundin and Cernigliaro, 1975).

Ekblom et al (1973) examined the heart rate response during cycle exercise before and after a 5-week endurance training program under conditions of parasympathetic (atropine) and sympathetic blockade (propranolol) in 15 healthy sedentary males 23 to 32 years of age. Parasympathetic blockade resulted in a higher heart rate at a given $\dot{V}O_2$ after training compared to the control value, which suggests that the training bradycardia may be partly explained by increased parasympathetic activity. However, this cannot be the only factor, as the heart rate at a given submaximal $\dot{V}O_2$ after the injection of atropine did not increase to the same level as before training. Administration of propranolol resulted in less of a reduction in heart rate at a given $\dot{V}O_2$ after training compared to the control value, which demonstrates a less pronounced effect of beta-adrenergic receptor activity after training. These changes were more pronounced in subjects with the greatest increase in peak $\dot{V}O_2$. Thus training-associated increases in parasympathetic activity and reduction in sympathetic activity may contribute towards the bradycardia observed after endurance training.

There is some evidence to suggest that the intrinsic heart rate may be affected by endurance training.

Untrained subjects were found to have a higher mean resting intrinsic heart rate than athletes after double blockade (Shephard, 1968). In longitudinal studies using double blockade before and after training, intrinsic heart rate was found to decrease the most in those subjects who exhibited the greatest increase in $\dot{V}O_2$max (Sutton et al, 1967). However, with short periods of training, no significant differences in intrinsic heart rate were reported. Studies in isolated preparations from trained and untrained animals report either no differences between the trained and untrained states (Tipton, 1968; Penpargkul and Scheuer, 1970), or a reduction in intrinsic heart rate after training (Bolter et al, 1973). Ekblom et al (1973) found an increase in the post-training heart rate, at rest, after double blockade compared to the pre-training value (young males). At a given percent of $\dot{V}O_2$, heart rate after training remained slightly higher, up to about 60% of peak $\dot{V}O_2$. As exercise progressed, the higher heart rate after training became progressively more pronounced compared to the pre-training value.

It is evident from the above that there is no consensus of opinion as to the effect of endurance training on intrinsic heart rate. Whether this is likely to be a factor in the present study is not known. Given the conflicting findings, it is doubtful that adaptations in

intrinsic heart rate played a major role in the training-associated changes in heart rate observed in this study.

5.4.3.iii Changes in Preload

The decrease in heart rate was associated with significant increases in stroke volume. Higher stroke volumes associated with higher end-diastolic volumes determined by radionuclide imaging techniques and echocardiography, have been reported in young athletes compared to sedentary controls (Morganroth et al, 1975; Roeske et al, 1975). Morganroth et al (1975) reported significantly higher (18%) left ventricular internal dimensions at end diastole in young endurance athletes (swimmers) compared to age-matched inactive controls.

Comparisons between competitive long-distance runners and wrestlers revealed greater ($p<0.001$) cardiac dimensions in the runners; 16% in left ventricular internal dimension (end diastole) and 31% in end diastolic volume. These changes were associated with a 35% higher stroke volume in the runners (116.5 $\pm$ 13.1 ml compared to 75.2 $\pm$ 8.1 ml in the wrestlers). Large increases in end-diastolic volume were also reported by Rerych (1980) in young athletes after six months of endurance training. Therefore, larger diastolic filling may result in an

elevated stroke volume via the Frank-Starling mechanism.

Although these findings are based on studies that employed young trained subjects, there is evidence to support that increases in end-diastolic volume may also occur in older subjects. An increase in stroke volume associated with a greater increase in end-diastolic volume and thus a greater reliance on the Frank-Starling mechanism was shown by Rodeheffer et al (1984) and Gerstenblith et al (1987) in physically active healthy elderly. This is further supported by an increase in diastolic filling time with increasing age (Gerstenblith et al, 1977). The marked decrease in submaximal heart rate observed in the present study together with an increase in diastolic filling time may contribute to an increase in end-diastolic volume which may in turn increase stroke volume via the Frank-Starling mechanism in these elderly subjects.

5.4.3.iv Changes in Myocardial Contractility

The capacity of the heart to pump against an increased afterload has been shown to increase after training (Clausen et al, 1973); young subjects who had undergone endurance training of the legs were able to increase cardiac output and stroke volume more during work with the untrained arms than was possible before leg training. These findings may suggest a training-associated

improvement in cardiac pumping capabilities in young subjects, but it is not likely to be a major factor in elderly subjects after short-term endurance training.

Myocardial contractility, determined by changes in ejection fraction and end-systolic volume, were found to change little after endurance training (Blomquist and Saltin, 1983). In the physically active subjects of the Baltimore Longitudinal Study on Aging, end-systolic volume was found to decrease less in elderly compared to young subjects, which resulted in less of an increase in ejection fraction during exercise (Gerstenblith et al, 1987). Thus changes in myocardial contractility are not likely to play a significant role in the large increase in stroke volume observed in the elderly subjects.

In summary, post-training submaximal and peak cardiac output increased in the elderly associated with marked increases in submaximal and peak stroke volume, marked decreases in submaximal heart rate and small but significant increases in peak heart rate. Based on the available evidence, it would appear that increased parasympathetic and decreased sympathetic activity may contribute to the decrease in the heart rate response to exercise. This, together with increased diastolic filling time may result in an increase in end-diastolic volume

which will in turn increase stroke volume via the Frank-Starling mechanism. There is no convincing argument that myocardial contractility is in fact increased to any great extent in elderly subjects and is not likely to be a major factor particularly after short term endurance training.

5.4.4 Effects of Training on Vascular Conductance and Arterial Pressure

A major training-associated adaptation in the elderly was a 30% increase in peak cardiac output accompanied by a 20% increase in vascular conductance (Figure 17) with no change in the mean arterial pressure at peak exercise. Similar training-associated changes were observed in the young, but the magnitude of the change was not as pronounced; a 14% increase in peak cardiac output accompanied by a 13% increase in vascular conductance and a small increase (4%) in mean arterial pressure at peak exercise.

Applying equation 8 on vascular conductance to the pre- and post-training data in the elderly subjects we have:

pre-training: 0.097 (l/min/mmHg) =12.7(l/min)/131.2 (mmHg)

post-training: 0.124 (l/min/mmHg) =16.5(l/min)/133.1 (mmHg)

The increase in vascular conductance at all levels of exercise was significant ($p<0.01$) in the elderly (Figure 17); there was no significant change in the young subjects. Also, submaximal systolic blood pressure was significantly lower ($p<0.001$) in the elderly after training with no change in the young subjects. These findings suggest training-associated adaptations in the peripheral circulation of these elderly subjects that resulted in an increase in vascular conductance and a decrease in the impedance to ejection.

The fall in blood pressure observed during exercise in these elderly subjects is likely the result of the marked increase in vascular conductance. Since blood pressure is directly related to cardiac output, and cardiac output increased both at submaximal and maximal exercise, the increase in vascular conductance was proportionally greater compared to the increase in cardiac output. In other words, if cardiac output remained unchanged, an increase in vascular conductance may result in a fall in arterial pressure. During heavy exercise with large muscle groups, when a large portion of the skeletal muscle vascular bed may be dilated, the extent of the vasodilation has to be balanced between the metabolic demands of the muscles to increase blood flow and the capacity to increase cardiac output so that arterial

pressure is maintained. Training-associated changes in sympathetic outflow are likely to play a major role in producing this close balance between peripheral and central circulatory changes.

5.4.4.1 Changes in Sympathetic Outflow

Decreased sympathetic vasoconstrictor outflow to the exercising muscles is a key mechanism for the increase in vascular conductance after training. Plasma norepinephrine concentrations are reduced after endurance training and are closely related to the training-associated reduction in heart rate (Peronett et al, 1981; Cousineau et al, 1977) and increase in vascular conductance (Rowell, 1984). However, as illustrated in Figure 17, vascular conductance in the elderly subjects tends to plateau as maximal exercise is approached. This suggests that as the heart's capacity to increase cardiac output is approached, arteriolar vasodilation tends to decrease to maintain arterial pressure.

There is a competition in active muscles between the metabolic factors that dilate the muscle vessels and the increased nervous activity that tends to constrict them (Mellander and Johansson, 1968; Kjellmer, 1965; Strandell and Shepherd, 1967). It was previously believed that the sympathetic vasoconstrictor influence was

unable to inhibit blood flow in muscles exercising at or near the maximal level. However, as demonstrated by Donald (1970) and Rowell (1974), vasoconstriction can occur in active skeletal muscles during heavy exercise; sympathetic stimulation in exercising dogs resulted in a decrease in leg blood flow during maximal exercise that was less compared to the decrease in flow during mild exercise. These findings suggest that as the exercise stimulus increases, the vasoconstrictor influence to the working muscles is progressively diminished but it is not abolished. The purpose of this vasoconstriction appears to be to adjust the maximal vasodilation in the working muscles to the central circulatory capacity.

5.4.5 Summary of Changes in Haemodynamic Function

Training resulted in significant increases in peak $\dot{V}O_2$ in these elderly subjects, associated with a relatively greater increase in peak cardiac output (30%) compared to peak arterio-venous oxygen difference (6%). During submaximal exercise cardiac output was higher ($p<0.05$) after training and arterio-venous oxygen difference was lower, although statistical significance was reached only at one exercise level (300 kpm/min). These findings suggest that a major mechanism for the increase in peak $\dot{V}O_2$ in the elderly is an increase in

blood flow and thus oxygen delivery to the working muscles. This is further supported by the marked increase in vascular conductance observed in the trained elderly. Neither submaximal cardiac output nor vascular conductance were significantly changed after training in the young subjects.

There are no experimental data on blood flow measurements before and after high intensity training in previously sedentary elderly subjects. As reviewed in Chapter 1, blood flow during exercise was lower in middle-aged compared to young athletes (Wahren et al, 1974). A decrease in maximal blood flow was also reported in subjects 17 to 75 years of age (Amery et al, 1969). The changes in blood flow with increasing age appear to be associated with a diminished beta-adrenoreceptor sensitivity (Van Brummelen et al, 1981). Our findings suggest that blood flow to exercising muscles can increase in elderly individuals as a response to endurance training.

The improved oxygen delivery to the working muscles was associated with marked decreases in heart rate and systolic blood pressure, consistent with decreases in the work of the heart at a given exercise level. The fall in submaximal heart rate would increase diastolic filling time and end-diastolic volume, as shown by Rodeheffer et al (1984) and Gerstenblith et al (1987) in physically

active elderly and thus increase stroke volume via the Frank-Starling mechanism. These adaptations allowed the elderly trained subjects to achieve higher levels of exercise and peak $\dot{V}O_2$.

5.4.6 Effects of Training on Ventilation

Training resulted in a lower CO_2 output at a given $\dot{V}O_2$ resulting in a fall in ventilation and the RER in both groups (Figures 24 and 25). There was no change in ventilation for a given $\dot{V}O_2$ at low exercise levels (Figure 24). However, as exercise progressed, the post-training $\dot{V}E/\dot{V}O_2$ curve shifted more to the right, resulting in a lower submaximal and a higher maximal ventilation. The level of ventilation is primarily determined by the CO_2 production, which at low levels of exercise, is related to O_2 utilization. However, as exercise progresses, blood lactate increases resulting in a relatively steeper increase in ventilation.

A major metabolic adaptation after training is a lower plasma lactate concentration at a given power output (Taylor and Jones, 1979; Issekutz et al., 1965), which results in a reduction in CO_2 output, a fall in the respiratory drive and a reduction in submaximal ventilation. Blood lactate was not measured during progressive incremental exercise in our subjects. However,

the fall in CO_2 output at a given $\dot{V}O_2$ after training, indicated by the fall in the RER, would suggest that blood lactate also decreased after training. A post-training decrease in plasma lactate concentration was evident following short-term cycling, which provides indirect evidence of a decrease in blood lactate during the incremental cycling test, associated with an increase in oxygen delivery to the exercising muscles. The higher ventilation at maximal exercise is likely due to the increased power output, leading to an increased CO_2 output and blood lactate.

These findings suggest significant training-associated improvements in the ventilatory efficiency of these elderly subjects. As discussed in Chapter 1, the higher ventilatory pattern of elderly subjects is likely the result of an increased physiological dead space due to areas in the lung with a high $\dot{V}/\dot{Q}$ ratio. The significant fall in submaximal ventilation at a given exercise level, together with a marked fall in the RER indicate improved oxygen delivery to the exercising muscles, consistent with the cardiovascular adaptations discussed earlier. These changes together with a more even matching of ventilation and perfusion in the trained state contribute towards a more efficient ventilatory pattern and

an increased capacity for higher levels of exercise and peak $\dot{V}O_2$.

5.4.7 Effects of Training on Short-Term Dynamic Muscle Function

No changes were observed after training in the maximal power output measures at 60 rpm with the exception of a fall in plasma lactate in the elderly (9.0 mmole/l) to 7.5 mmole/l, $p<0.005$, Figure 11). At the higher pedalling frequency, total work increased and the fatigue index and plasma lactate decreased (Table 4, Figure 11); total work increased by 13% in the elderly (11.2 kJ to 12.6 kJ) and 8% in the young (15.7 kJ to 16.9 kJ). The fatigue index decreased by 8% in the elderly (59.0% to 54.2%) and 7.0% in the young (62.8% to 58.1%). Plasma lactate concentration decreased 26% in the old (10.5 mmole/l to 7.8 mmole/l) and 14% in the young (14.1 mmole/l to 12.1 mmole/l). There was no change in maximal power output in either group. These findings indicate similar adaptations in peripheral muscle function in elderly and young subjects.

The lack of improvement after training in maximum muscle power, despite a marked increase in peak $\dot{V}O_2$, may suggest that changes in muscle fibre size are not likely to play a major role in the increased capacity to perform endurance exercise. However, the reduction in the

fatigue index and the increase in total work during 30 seconds of pedalling at 110 rpm, together with a reduction in the post-exercise plasma lactate concentration point to an increase in aerobic metabolic processes in muscle. This is consistent with the findings that during submaximal exercise of the same intensity (at the same percentage of the subject's peak $\dot{V}O_2$), trained individuals deplete their muscle glycogen stores more slowly than untrained individuals and derive a greater percentage of their energy from oxidation of fatty acids and relatively less from carbohydrate (Hermansen et al, 1967; Saltin and Karlsson, 1971). Furthermore, animal studies have shown that endurance training is associated with significant increases in the amount of mitochondria and the oxidative capacity of trained skeletal muscle (Barnard et al, 1970; Holloszy, 1967).

The training program used in the present study was aimed at increasing endurance. Consequently, it is not surprising that we found no changes in maximal muscle power after training. It is well known that the nature of the exercise stimulus determines the type of adaptation expected; hypertrophied muscles of weight lifters showed increases in strength with no change in aerobic potential (Gollnick et al, 1972). On the other hand, muscles of

trained rodents (daily running) showed increases in aerobic capacity with no change in muscle size or strength (Barnard et al, 1970; Holloszy, 1967; Pattengale et al, 1967). Our findings are consistent with improved aerobic capacity in peripheral muscle after endurance training. The extent of improvement was similar in the elderly and young subjects. As expected, power output during short-term exercise was not changed significantly, likely as a result of the type of the training stimulus; endurance type of exercise rather than strength training.

At the higher pedalling speed (110 rpm), the fatigue index was twice that at 60 rpm in both groups ($p<0.001$) as was the case at pre-test, in agreement with the findings of McCartney (1983) in young subjects. This suggests a greater relative contribution of type II motor units at the higher pedalling speed, which are capable of generating greater levels of power, but are more prone to fatigue (Burke et al, 1973; Thornstensson et al, 1976; Thorstensson and Karlsson, 1976). Comparisons between sprinters and long-distance runners revealed that sprinters are considerably more powerful than long-distance runners at higher pedalling speeds (over 100 rpm) consistent with the higher proportion of type II muscle fibres in the sprinters (McCartney, 1983; Thornstensson, 1976; Gregor et al, 1979).

There were no differences in the fatigue index between the elderly and young subjects, despite significant differences in power output and total work ($p<0.001$). The magnitude of the difference ranged between 25% and 30%, as was the case at pre-test (Figures 10 and 11). The similarities in fatigue behaviour in the two groups was a consistent finding both before and after training, and is in agreement with the relative stability of type I muscle fibres with increasing age (Larsson et al, 1978; Tomonaga, 1977; Orlander et al, 1978; Kiessling et al, 1973). Similar findings in the fatigue characteristics of 15 to 70 year old males were also observed in our cross-sectional study using the same experimental protocol (Makrides et al, 1985).

The improvement in total work at the higher pedalling speed suggests that the training-associated adaptations are specific to the type II muscle fibres. These adaptations are consistent with significant improvement in the ability of muscle to utilize substrates oxidatively; thus more work could be performed with less fatigue and blood lactate accumulation. Although the training program was primarily aimed at increasing cardiovascular endurance, the nature of the program was such that heavy exercise was performed over brief bouts of exercise; the subjects were required to pedal at a high

intensity for 5 minutes separated only by brief bouts (3 minutes) at a lower intensity, the pattern repeating itself 7 times. Thus the intermittent nature of the training program may explain the selective improvement in the fast-contracting type II fibres compared to the slow-contracting type I fibres.

In the following chapter, the major findings and conclusions of the thesis will be summarized.

6. GENERAL SUMMARY

6.1 Introduction

The physical deterioration that occurs with aging is exemplified by a decrease in the peak oxygen intake of about 30-40% between 25 and 65 years. This is associated with a decrease in peak cardiac output (30-35%) and arterio-venous oxygen difference (5-10%). The lower cardiac output in the elderly is associated with decreases in peak heart rate and stroke volume of about 12-14%. Blood flow measurements are lacking but available data suggest a lower vascular conductance and a higher mean arterial pressure in the elderly during exercise. These changes are accompanied by declines in muscle mass and strength, breathing capacity and ventilatory efficiency.

Many studies in the literature have argued in favour of increased physical activity in counteracting the age-associated decline in the capacity to do work. However, few studies have attempted to examine the role of endurance training in improving the work capacity of elderly, sedentary subjects. The majority of these studies reported modest increases in peak $\dot{V}O_2$ in response to endurance training. Major limitations in these studies

relate to the intensity of training and the lack of a comparison group of young subjects who had undergone the same training program and had a similar pre-training fitness level. Furthermore, only two studies (Seals et al, 1984 and Niinima and Shephard, 1978) included measures of haemodynamic function in sedentary elderly subjects (over 60 years of age) before and after endurance training; Seals et al (1984) reported a 25-30% increase in peak VO_2 with no change in peak cardiac output. Niinima and Shephard (1978) on the other hand, reported no significant change after endurance training in either peak VO_2 or cardiac output. Neither of these studies included a young group for comparison. Thus, although there appears to be a general consensus that exercise is beneficial in elderly individuals, evidence to substantiate this belief and to quantify the extent of improvement, in non-athletic elderly relative to young subjects, is lacking. Furthermore, there are conflicting views as to the mechanisms that may contribute to any such improvement in the elderly.

6.2 Summary of Major Contributions of the Thesis

The major aim of the thesis was to provide direct comparisons of training-induced adaptations in exercise capacity and related variables in over 60 year-old previously sedentary males with a similar group of subjects

40 years younger. The training program was of the same type, frequency, duration, and relative intensity in both the young and old. In the few published reports on this topic cross-comparisons are commonly carried out with younger populations reported in the literature who have undergone different training regimens making comparisons difficult and inferences drawn questionable.

The studies were carried out on the background of a large descriptive study of healthy males and females aged 15-70 years, that had quantified the reduction in exercise capacity with age, and suggested that the level of leisure activities was an important moderating factor.

The study provided data on training-induced adaptations in over 60 year-old males who underwent a high intensity training program 3 sessions/week for a total of 12 weeks at 85% of peak $\dot{V}O_2$, mean training heart rate 140 beats/min. The few published reports on the topic have either used less than 12 weeks of training, the intensity was low to moderate, or the subjects were not sedentary prior to entry to the study. Furthermore, data were obtained on the cardiovascular and muscular adaptations to high intensity endurance training in over 60 year-old previously sedentary males for comparison with a similar group of younger subjects. Previously, no study had examined the training-induced changes in both

cardiovascular and peripheral muscle function in the same population of young and elderly subjects who underwent the same training program and few had examined the component responses in cardiac output and other important variables.

6.3 Major Findings of the Thesis

6.3.1 Before Training

The subjects in both groups showed values for peak $\dot{V}O_2$ that were in the low range of normal, established in the cross-sectional study, consistent with their sedentary lifestyle. In the over 60 year-old males, peak oxygen intake (l/min) was 37% less (1.599 $\pm$ 0.073 old, 2.536 $\pm$ 0.141 young), peak cardiac output 30% less (12.7 $\pm$ 0.6 l/min old, 18.0 $\pm$ 0.7 l/min young), peak heart rate (161 $\pm$ 3.8 beats/min old, 192 $\pm$ 2.3 beats/min young) and stroke volume (78.9 $\pm$ 3.5 ml old, 93.9 $\pm$ 3.9 ml young) 16% less, and peak arterio-venous oxygen difference (126.5 $\pm$ 3.7 ml/l old, 140.4 $\pm$ 3.9 ml/l young) 10% less than in the younger group. A striking difference between the groups was the calculated vascular conductance which was 40% lower in the older subjects at peak exercise associated with a higher mean arterial pressure.

The decrease (30%) in peripheral muscle function (power and total work) in the over 60 year-olds paralleled the decrease in peak $\dot{V}O_2$. However, the fatigue characteristics of peripheral muscles during a brief bout

of maximal cycling were similar between the young and older subjects.

6.3.2 After Training

The subjects tolerated the 12 weeks of high intensity training well, compliance was excellent and no interruptions in training occurred. Peak oxygen intake increased in the older subjects by 38% (1.599 ± 0.073 l/min to 2.212 ± 0.073 l/min) with increases in peak cardiac output (12.7 ± 0.6 l/min to 16.5 ± 0.4 l/min, 30%), heart rate (161 ± 3.8 beats/min to 173 ± 3.9 beats/min, 7%), stroke volume (78.9 ± 3.5 ml to 95.6 ± 2.5 ml, 21%) and arterio-venous oxygen difference (126.5 ± 3.7 ml/l to 134.7 ± 2.7 ml/l, 6%). The magnitude of change in these variables was similar to that found in the young subjects. Thus, the older subjects came to be physiologically closer to the young subjects; their post-training peak oxygen intake, cardiac output, stroke volume, arterio-venous oxygen difference and ventilation were not significantly different from the pre-training values in the young.

A major training-associated adaptation in the elderly was a 30% increase in peak cardiac output, largely brought about by an increase in stroke volume, accompanied by a 20% increase in vascular conductance with no change in mean arterial pressure at peak exercise. Similar changes were observed in the young, but the magnitude of change was not as pronounced. These findings suggest

training-associated adaptations in the peripheral circulation that result in an increase in vascular conductance and improved perfusion of exercising muscles. The increase in vascular conductance during submaximal exercise was significant only in the elderly, contributing to a training-associated decrease in systolic blood pressure at a given VO_2; there was no change in the young group.

However, despite the marked training-associated circulatory adaptations in the elderly, their vascular conductance and cardiac output remained lower at peak exercise, which suggest that the increase in conductance has to be balanced against the heart's ability to increase cardiac output so that arterial pressure is maintained. Structural changes in the aged peripheral vessels would also influence the extent of circulatory changes, but it is evident that substantial improvements can occur in aging peripheral vessels even after short term endurance training.

Similar training-induced adaptations occurred at submaximal exercise (both groups); arterio-venous oxygen difference (no significant change after training), heart rate, ventilation and the respiratory exchange ratio (significant decreases after training). These changes are consistent with decreases in sympathetic and increases in parasympathetic outflow, a more efficient ventilatory

pattern after training and improved aerobic metabolic processes in exercising muscle.

The changes in short-term power output were relatively modest in both groups and of a similar magnitude. The only significant changes were a decrease in the fatigue index associated with an increase in total work and a decrease in the post-exercise plasma lactate concentration, which further support a training-induced improvement in aerobic metabolic processes in exercising muscle.

6.4 Conclusions of the Thesis

Based on the findings presented and within the limitations of this study, it is concluded that:

1. Over 60 year-old previously sedentary males may tolerate high intensity endurance training as well as a similar population of subjects in their 20's, and may respond to endurance training by quantitatively similar increases in peak $\dot{V}O_2$.

2. Short term high intensity endurance training may elicit adaptations in peak exercise capacity and related haemodynamic function so that an active 60 year-old may have values comparable to those of an inactive 20 year-old.

3. Training-associated adaptations may occur in the peripheral circulation and autonomic nervous system outflow of elderly subjects that result in significant decreases in submaximal heart rate and systolic blood pressure, and significant increases in stroke volume, cardiac output and vascular conductance.

4. Significant qualitative changes may occur in the peripheral muscle of over 60 year-old previously sedentary males as a result of high intensity endurance training, indicated by increases in total work and decreases in muscle fatigue, plasma lactate and the respiratory exchange ratio. The magnitude of change is similar to that found in young subjects.

5. Dynamic muscle endurance (ability to sustain dynamic exercise of brief duration) is maintained at least up to the age of 70 years.

6. Despite training-induced adaptations in peak VO_2 and related variables, an active 60 year-old will likely remain physiologically different from an active 20 year-old. Thus structural changes in the aging heart, lung, vascular and muscular systems may impose a biological limit on the elderly subject's capacity for maximal physical work.

6.5 Concluding Remarks

Sixty to 70 year-old previously sedentary males responded to high intensity endurance training by quantitatively similar adaptations in peak VO_2 and related variables. Many of the functional processes that contribute to the maintenance of peak exercise capacity may improve to an extent that active elderly become similar to inactive subjects several years younger. However, the physiological gap between young and old is only partially remedied by endurance training. Aging structural changes undoubtedly affect function, and if pathology is excluded, such changes will probably contribute to the decreased exercise capacity with increasing age. Two functions particularly affected are peak heart rate and peripheral vasodilatory capacity. Although both are amenable to improvement with endurance training, the extent of improvement is limited contributing to the decrease in exercise capacity with increasing age. However, the endurance training program used in this study was only 12 weeks in duration. It is not known whether further improvements may have occurred if training of the same intensity had continued over a longer period of time or whether the type and extent of the improvement would have been different.

The haemodynamic changes observed both before and after the training program in the elderly point towards changes in blood flow as a significant factor in the augmentation of peak exercise capacity. A major difference between the young and old before training was that at a given $\dot{V}O_2$, cardiac output was lower, and arterial pressure was higher in the elderly associated with a decreased stroke volume and vascular conductance. Major adaptations after training in the elderly were an increase in submaximal stroke volume contributing to increases in cardiac output and an increase in vascular conductance contributing to decreases in arterial pressure. These changes were significant only in the elderly and suggest marked training-associated improvement in the peripheral circulation. Blood flow measurements during exercise in young and elderly subjects are needed to determine changes in blood flow during exercise with increasing age, the extent of improvement that may be expected after endurance training and the mechanisms that may contribute to any such improvement.

The results of this study suggest that the deterioration in functional capacity normally associated with aging may be at least decreased by regular exercise.

APPENDIX I

Normal Standards for an Incremental Progressive Cycle Ergometer Test[1-3]

NORMAN L. JONES, LYDIA MAKRIDES, CHRISTINE HITCHCOCK, TANYA CHYPCHAR, and NEIL MCCARTNEY

SUMMARY One hundred healthy subjects (50 male and 50 female), selected to provide an even distribution of age (15 to 71 yr) and height (163 to 194 cm in males and 152 to 176 cm in females), underwent a progressively incremental (100 kpm/min each min) exercise test to a symptom-limited maximum. Measurements were made of O_2 intake and CO_2 output, ventilation and breathing pattern, heart rate and blood pressure, and rating of perceived exertion. The ventilatory anaerobic threshold was identified. Predictive data were derived for measurements at maximal and submaximal exercise. Maximal power output (Wmax) and oxygen intake ($\dot{V}O_2max$) varied with sex (0, male; 1, female), age (yr), and height (Ht, cm): Wmax = 20.4 (Ht) − 6.74 (Age) − 288 (Sex) − 1,909 kpm/min (SEE, 216; r, 0.858); $\dot{V}O_2max$ = 0.046 (Ht) − 0.021 (Age) − 0.62 (Sex) − 4.31 L/min (SEE, 0.458; r, 0.869). The extent of leisure time activity exerted a positive influence on $\dot{V}O_2max$ (r, 0.47; $p < 0.001$); $\dot{V}O_2max$ was also related to lean thigh volume (r, 0.79). Maximal heart rate (HR) declined as a function of age: HRmax = 202 − 0.72 (Age) beats/min (SEE, 10.3; r, 0.72). Maximal O_2 pulse (O_2Pmax) was related to height and was systematically higher in males than in females: O_2Pmax = 0.28 (Ht) − 3.3 (Sex) − 26.7 ml/beat (SEE, 2.8; r, 0.86). Ventilation was closely related to CO_2 output, and the maximal tidal volume was related to vital capacity. The $\dot{V}O_2$ increased linearly with power throughout the test; in an individual subject, the intercept of this relationship was positively influenced by weight and height. The slope of HR to O_2 intake was related to height, was steeper in females than in males, and lower in active than in inactive subjects. Ventilation increased as a function of CO_2 output, and tidal volume increased as a function of height and vital capacity. Systolic blood pressure increased linearly with power output and as a function of age. The ventilatory "anaerobic threshold" (Vat) was related to $\dot{V}O_2max$ (r, 0.79); leisure activity showed a positive effect on Vat, but not after the effects of height and age were accounted for: Vat = 0.024 (Ht) − 0.0074 (Age) − 2.43 L/min (SEE, 0.316; r, 0.651). Results obtained at standard power outputs of 300 and 600 kpm/min indicated that age and height accounted for most of the variation between subjects, but HR was systematically higher in females than in males of comparable height. The results provide a guide for the interpretation of measurements obtained in this type of test.

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Introduction

Exercise tests are being more widely applied in the investigation of cardiac and pulmonary disorders. A number of exercise protocols have been developed, most of which employ a number of increasing power outputs on a cycle ergometer or treadmill to the subject's maximum. In the interpretation of results, there is reasonable agreement with regard to normal standards for maximal O_2 intake ($\dot{V}O_2max$) per kilogram body weight and for the decline in maximal heart rate (HRmax) with age (1-3). However, it has been argued that weight is not the most reliable predictor of maximal exercise capacity (4), in that it leads to overestimates of $\dot{V}O_2max$ in overweight subjects (5, 6); a suggested alternative is the use of a prediction based on the average weight estimated for the subject's height (5, 6). Thus, although $\dot{V}O_2max$ and HRmax have been measured in large populations in which age and body size are likely to have been normally distributed, there are difficulties in applying suitable correction factors to allow for the effects of gender, age, height, and weight in predicting normal values for these and other variables used in clinical exercise studies, such as ventilation, blood pressure, the oxygen pulse, and the ventilatory anaerobic threshold (7, 8).

The present investigation employed a standardized progressive incremental exercise protocol on a cycle ergometer (5) in normal subjects selected to provide a range of age and height in the two sexes. Although the results largely confirm previous studies, they may allow a greater precision to be applied to predictive standards than was previously possible.

Methods

Subjects were obtained from the local university and the general population by advertising, and they were recruited by stratifying into 5 age groups for both sexes until a total sample of 50 subjects had been obtained for each sex (table 1). We tried also to obtain equal numbers in 5 groups on the basis of weight; although we were reasonably successful in males, the distribution of height in females was narrower (figure 1). Information regarding previous health, leisure habits, occupation, exercise-related symptoms, and smoking history was obtained by questionnaire. Competitive athletes were excluded, as were subjects with serious illness in the past or any chronic disorder; resting systolic and diastolic blood pressures were required to be below 150 and 90 mmHg. The procedures, including the known risks, were described in detail, and informed consent was obtained. Remuneration was not offered.

The instructions given to the subjects were similar to the routine instructions sent to patients coming to the laboratory for clinical exercise testing (5). Subjects came to the laboratory at least 2 h after their last meal. Spirometry was performed with a calibrated dry spirometer (Vitalograph); measurements of vital capacity (VC) and forced expiratory volume in one second (FEV_1) were recorded at BTPS. An estimate of lean thigh volume (muscle and bone) was obtained from anthropometric and skin-fold measurements (9).

Subjects exercised on a cycle ergometer (Siemens Elema 370) calibrated by torsion balance. Care was taken to place the saddle and valve mouthpiece in comfortable positions. The initial power setting was 100 kpm/min (16.3 W), increasing by 100 kpm/min at the

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TABLE I

ANTHROPOMETRIC VARIABLES FOR MALE AND FEMALE SUBJECTS*

	Males						Females					
Age (yr)	n	Height (cm)	Weight (kg)	FEV$_1$ (L)	VC (L)	Lean Thigh Volume (L)	n	Height (cm)	Weight (kg)	FEV$_1$ (L)	VC (L)	Lean Thigh Volume (L)
15–24	11	183	78	5.0	5.9	10.3	10	166	60	3.8	4.1	6.9
SD		6.6	10.7	0.48	0.73	0.65		4.8	5.9	0.16	0.31	1.29
25–34	10	178	79	4.7	5.7	9.3	10	167	59	3.8	4.4	6.2
SD		6.0	10.8	0.64	0.75	1.74		4.3	4.9	0.35	0.49	1.31
35–44	9	179	79	4.6	5.7	9.1	10	162	60	3.1	3.6	5.1
SD		7.4	8.8	0.50	0.66	1.57		5.0	7.2	0.44	0.53	0.88
45–54	10	176	78	4.0	4.7	9.3	10	164	65	2.9	3.8	4.6
SD		7.2	10.9	0.69	0.89	11.46		5.3	8.6	0.51	0.56	1.45
55–71	10	177	78	3.6	4.7	8.6	10	164	62	2.8	3.4	5.4
SD		6.6	9.3	0.61	0.70	0.83		5.0	3.8	0.47	0.51	0.87

* Values are mean ± SD

end of each min of the test. Subjects exercised to a symptom-limited, maximal power output; objective criteria were established for a test to be stopped by the observers (5), but no test was stopped because of these reasons. A modified lead V5 electrocardiograph was used to measure heart rate and for monitoring purposes. Blood pressure was measured by the cuff method by auscultation. The rating of perceived exertion was obtained using the Borg category scale (5, 10).

Subjects breathed through a low resistance, high velocity Hans Rudolph valve (Hans Rudolph, Kansas City, MO) connected on the inspired side to a dry gas meter (Parkinson Cowan CD4) and on the expired side to a 10-L mixing chamber containing a fan. Expired gas was sampled at 50 ml/min and analyzed for O_2 and CO_2 by a respiratory mass spectrometer (MGA 1100; Perkin-Elmer Medical Instruments, Oakbrook, IL) calibrated by standard Lloyd Haldane analyzed calibrating gases. Data were processed on-line by a microcomputer (PDP 11; Digital Electronics Corp., Maynard, MA) programmed to correct for delays between signals for volume and gas concentration, perform standard calculations (5), and present average values for every 15 s for the following variables: ventilation ($\dot{V}_E$, BTPS), O_2 intake ($\dot{V}O_2$, STPD), CO_2 output ($\dot{V}CO_2$), respiratory exchange ratio (R), tidal volume (V_T, BTPS), frequency of breathing (f), and heart rate (HR). The system was validated by independent measurements using the Douglas bag method, which indicated the 95% confidence limits to be 45 ml/min for $\dot{V}O_2$, 40 ml/min for $\dot{V}CO_2$, and 1.6 L/min for $\dot{V}_E$. The fourth 15-s average for each power output (45 to 60 s) was taken as the collection period for the given power output. Blood pressure (cuff and auscultation) and rating of perceived exertion (Borg scale indicated manually) were recorded at alternate power outputs.

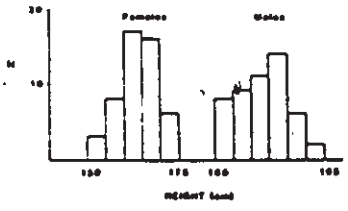

Fig 1. Histogram to show distribution of height in males and in females in intervals of 5 cm.

The calculated data were entered into a Hewlett-Packard 3000 computer (Hewlett-Packard, Cupertino, CA) for statistical analysis, using the Statistical Package for the Social Sciences (11). Means and standard deviations were obtained for values in subjects grouped according to sex, age, height, and weight; stepwise multiple linear regression analysis was performed to identify weighting of different variables. For most variables the correlation coefficient (r) is reported with the standard error of the estimate (SEE). Relationships were examined between the variables measured during exercise and also to test the statistical effects of independent variables using analysis of variance.

Results

Subject Characteristics

The selection criteria ensured equal numbers of subjects in age decades (table 1) and a range of heights (figure 1). The extent of physical activity in leisure time in the past year and of occupation were graded according to the responses to a questionnaire similar to that described by Saltin and Grimby (12). The range of active exercise in leisure time was similar in males and females, and there was no significant effect of age ($p > 0.1$); there were 26 subjects in Grade 1 (exercising less than 1 h/wk), 42 in Grade 2 (1 to 3 h/wk), 23 in grade 3 (3 to 6 h/wk), and 9 in Grade 4 (more than 6 h/wk). Occupational activity was also similar in the two sexes; 54 had sedentary occupations, 30 had occupations requiring some walking, and 10 had occupations associated with constant standing or walking; no subject had an occupation that required heavy manual labor; in 6 subjects, occupational information was unavailable. Smoking history was similar in males and females: 50 subjects had never smoked, 31 were previous smokers, 13 were smoking less than 15 cigarettes per day, and 6 more than 15 per day.

Variables at Maximal Exercise

The highest power output completed (Wmax) showed significant correlations to size-related variables and to age and sex (table 2). In males, weight did not exert a significant effect once age and height had been taken into account in stepwise regression, but a minor effect was found in females and when data from males and females were combined (table 2). Leisure activity was associated with an increase in maximal power output (Wmax), as indicated in the following equation: Wmax = 12.7 (Ht) − 9.1 (Age) − 275 (Sex) + 77 (Lei) + 5.6 (Wt) − 1,133 (SEE, 204; r, 0.877), where Wmax is expressed in kpm/min, Ht in cm, age in yr, and Wt in kg; sex is coded 0 for males and 1 for females, and Lei is graded 1 to 4. Of the variables we measured, the single variable associated with the least variance was the VC: Wmax = 335 (VC) − 439 kpm/min (SEE, 212; r, 0.86), where VC is in liters.

Maximal oxygen intake ($\dot{V}O_2$max) was closely correlated to Wmax, and similar predictive equations were obtained for both variables (table 2) in which height (r, 0.78 for the total population), age (r, −0.43) (figure 2), and weight (r, 0.66) contributed significantly to the prediction equations in males and females; however, once height and age had been

TABLE 2

PREDICTIVE EQUATIONS FOR VARIABLES AT MAXIMAL POWER OUTPUT*

Variable	Sex	Height (cm)	Age (yr)	Weight (kg)	Constant	SEE	r
Power output, kpm/min	M	25.3	- 9.06		- 2759	245	0.721
	F	9.5	- 9.21	6.1	- 756	177	0.668
	- 288	20.4	- 8.7		- 1909	218	0.658
	- 249	16.2	- 9.5	5.6	- 1569	213	0.663
Oxygen intake, l/min	M	0.034	- 0.026	0.022	- 3.76	0.483	0.799
	F	0.025	- 0.016	0.010	- 2.28	0.388	0.655
	- 0.624	0.046	- 0.021		- 4.31	0.458	0.869
	- 0.492	0.032	- 0.024	0.019	- 3.17	0.441	0.881
Heart rate, beats/min	M		- 0.80		206	11.6	0.723
	F		- 0.63		198	8.9	0.730
			- 0.72		202	10.3	0.721
Oxygen pulse, ml/beat	M	0.342			- 44.0	3.3	0.593
	F	0.190			- 21.4	2.3	0.414
	- 3.3	0.284			- 26.9	2.8	0.865

* For equations applicable to both sexes male is coded 0, female is coded 1. Blank spaces indicate an insignificant effect for the variable in question

entered into the analysis the addition of weight contributed only a small reduction in variance (change in r^2, 0.021). When $\dot{V}o_2max$ was expressed per kilogram, the effect of age was described by the following equations: males: $\dot{V}o_2max/kg = 55 - 0.44$ yr ml/min/kg (SEE, 6.5; r, 0.71); females: $\dot{V}o_2max/kg = 43 - 0.36$ yr ml/min/kg (SEE, 6.6: r, 0.64). The $\dot{V}o_2max$ was correlated to lean thigh volume in males and females (figure 3), as expressed in the following equation derived for the total population: $\dot{V}o_2max = 0.306$ (tv) + 0.082 L/min (SEE, 0.558; r, 0.79), where lean thigh (muscle plus bone) volume (tv) is the sum of both thighs in liters. As with Wmax, VC was closely correlated to $\dot{V}o_2max$: $\dot{V}o_2max = 0.74$ (VC) - 1.04 L/min (SEE, 0.47; r, 0.86) (figure 4).

Taken by itself, the grade of leisure activity had a highly significant ($p < 0.01$) and linear effect on $\dot{V}o_2max$, even when the effects of age and height had been accounted for; when data from both sexes were combined, $\dot{V}o_2max$ increased from 1.97 ± 0.80 L/min in grade 1 to 2.15 ±0.76 in grade 2, 2.84 ± 0.85 in grade 3, and 3.10 ± 1.22 in grade 4 (ANOVA, $p < 0.001$). The following equation expresses the effect of leisure activity on the prediction of $\dot{V}o_2max$ from age, sex, height, and weight (table 2): $\dot{V}o_2max = 0.025$ (Ht) − 0.023 (Age) − 0.542 (Sex) + 0.019 (Wt) + 0.15 (Lei) − 2.32 (SEE, 0.415; r, 0.892).

Statistically, the effect of leisure activity was similar to the effect of age alone (r, 0.47 in both cases). A comparison between the effects of age and leisure activity may also be stated in equivalent terms—the difference in $\dot{V}o_2max$ between subjects exercising less than 1 h/wk and those exercising more than 6 h/wk is the same as between a 60-yr-old male and a 25 yr-old male.

Occupational activity did not exert a significant effect, but few of our subjects were in highly active occupations.

The HRmax showed a linear decline with age in both sexes (figure 5 and table 2), with no other factors being identified. Maximal oxygen pulse ($\dot{V}o_2max$/HR max) was related to heigh: (r, 0.79) in the total population (figure 6) and in males and females separately (table 2). For a given height, females had a lower maximal oxygen pulse; this effect may be allowed for in the prediction equation given in table 2. Age had by itself a minor effect on maximal oxygen pulse (r, −0.32; p = 0.013) and did not reduce the variance of the linear regression with height.

Systolic blood pressure at maximal power output increased linearly with increasing age from a mean of 165 mmHg at ages 15 to 25 yr to 184 mmHg at ages 55 to 65 yr.

Ventilation at maximal exercise was related to the subject's VC and FEV_1: $\dot{V}Emax = 26.3$ (VC) − 34 L/min (SEE, 23.1; r, 0.78); $\dot{V}Emax = 30.6$ (FEV_1) − 29 L/min (SEE, 23.9; r, 0.76). The maximal tidal volume showed a relationship to VC, with no effect of age or sex being found: Vrmax = 0.74 (VC)− 1.11 L (SEE, 0.54; r, 0.86).

Evolution of Variables through Progressive Exercise

The results obtained at all power outputs were examined to obtain predictive relationships. Oxygen intake increased linearly with power output: $\dot{V}o_2 = 0.19$ (W) + 0.288 L/min (SEE, 0.162; r, 0.98), where W is expressed as a multiple of 100 kpm/min. This relationship was examined in individual subjects to obtain a slope ($S\dot{V}o_2/W$) and intercept ($\dot{V}o_2$ at zero W) for each subject. Small but significant ($p < 0.01$) effects of age and weight were found in the slope, suggesting that increasing age was associated with a reduction in the increase in $\dot{V}o_2$

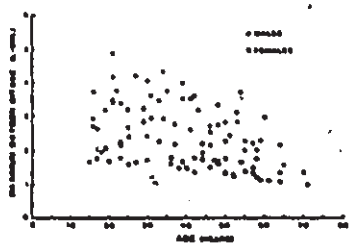

Fig. 2. Oxygen intake at maximal exercise related to age in males and in females.

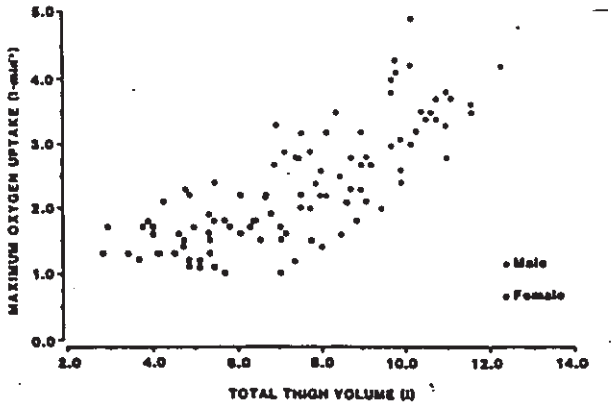

Fig. 3. Maximal oxygen uptake related to lean thigh volume (muscle and bone, sum of both thighs) in males and females.

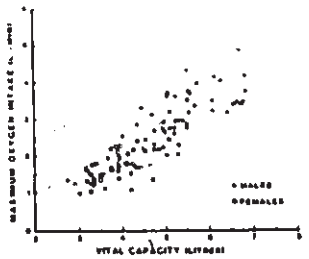

Fig. 4. Oxygen intake at maximal exercise related to vital capacity in males and females.

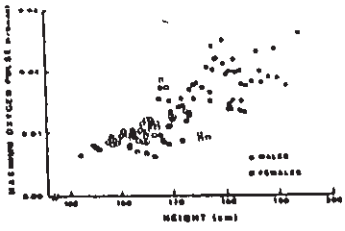

Fig. 6. Oxygen pulse at maximal exercise ($\dot{V}O_2$max/HRmax) related to height in males and in females.

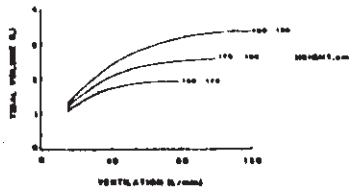

Fig. 7. Tidal volume in exercise related to ventilation and height. Curves were constructed from data obtained at each power output in subjects categorized into 3 groups according to height. Mean coefficient of variance = 12% (SD/x times 100).

with increasing work and that weight had a positive effect; both these effects were quantitatively small: $S\dot{V}O_2/W$ = 0.16 − 0.00046 (Age) + 0.00050 (Wt) (SEE, 0.00018; r, 0.44), where $\dot{V}O_2$ is expressed in L/min and W in 100 kpm/min.

The intercept was also influenced by size, almost equally by height and weight: $\dot{V}O_2$ at zero W = 0.0055 (Wt) − 0.027 L/min (SEE, 0.027; r, 0.47); $\dot{V}O_2$ at zero W = 0.0059 (Ht) − 0.670 L/min (SEE, 0.018; r, 0.47).

The linear increase in heart rate with $\dot{V}O_2$ was used to construct a regression equation for each subject to obtain a slope (SHR/$\dot{V}O_2$ beats/L) that was significantly related to size variables but was more variable for females than for males. Males: SHR/$\dot{V}O_2$ = 144 − 0.59 (Ht) beats/L (SEE, 9.3; r, 0.41); females: SHR/$\dot{V}O_2$ = 170 − 0.65 (Ht) beats/L (SEE, 14.6; r, 0.24); males and females: SHR/$\dot{V}O_2$ = 116 − 0.61 (Ht) + 15.5 (Sex) beats/L (SEE, 12.2; r, 0.72).

Age had no effect on these relationships, but there was a significant negative correlation to activity grade, the slope being steeper at lower grades of leisure activity, consistent with the known effect of training on heart rate (p = 0.03).

Systolic blood pressure (BPs) increased linearly with increases in power output: BPs = 123 + 0.045 (W) mmHg (SEE, 21; r, 0.69), where W is expressed in kpm/min. The slope of this relationship was not influenced by height but increased with increasing age; the slope increased from 4.0 mmHg for each 100 kpm/min increase in power at age 20 by 1.0 mmHg per decade, as expressed in the following equation: SBPs/W = 1.8 + 0.105 (Age) mmHg/100 kpm/min.

The ventilatory "anaerobic threshold" (Vat) was identified for each subject as the $\dot{V}O_2$ at which $\dot{V}E$ increased disproportionately to $\dot{V}O_2$, with an increase in the O_2 concentration in mixed expired gas and no change in CO_2 concentration (7). Mean Vat was 1.64 L/min ± 0.416 in males and 1.23 ± 0.299 in females; Vat was closely related to $\dot{V}O_2$max, and once this variable was included in a regression equation, no other variable (age, height, sex, weight) showed a significant effect: Vat = 0.35 ($\dot{V}O_2$max) + 0.60 L/min (SEE, 0.257; r, 0.79). This relationship tends to suggest that the higher the $\dot{V}O_2$max, the lower the ratio of Vat/$\dot{V}O_2$max. The Vat occurred at 56% (SD, 12) $\dot{V}O_2$max in males and at 74% (SD, 14) $\dot{V}O_2$max in females, consistent with the relationship of Vat to $\dot{V}O_2$max in the total population. A similar effect was identified for age; Vat/$\dot{V}O_2$max increased from 60 ± 16% at age 20 to 75 ± 17% at age 60 ($p < 0.05$). There was little change in Vat/$\dot{V}O_2$max with leisure activity; Vat/$\dot{V}O_2$max declined from 69 ± 16% in Grade 1, to 59 ± 19% in Grade 4 activity ($p < 0.05$).

Vat showed significant ($p < 0.001$) relationships to size variables, increasing with increases in height (r, 0.60) and weight (r, 0.54) and decreasing with age (r − 0.40), as expressed in the following equations derived for the total population: Vat = 0.024 (Ht) − 0.0074 (Age) − 2.43 L/min (SEE, 0.316; r, 0.651); Vat = 0.011 (Ht) − 0.0094 (Age) + 0.012 (Wt) − 0.92 L/min (SEE, 0.306; r, 0.68).

Ventilation below the Vat increased linearly with $\dot{V}O_2$: $\dot{V}E$ = 21.8 ($\dot{V}O_2$) + 5.11 L/min (SEE, 5.4; r, 0.87). A closer relationship was found to carbon dioxide output ($\dot{V}CO_2$): $\dot{V}E$ = 23.9 ($\dot{V}CO_2$) + 5.4 L/min (SEE, 3.6; r, 0.94); $\dot{V}O_2$ and $\dot{V}CO_2$ are expressed in L/min in these equations.

There was a nonlinear increase in VT with increasing $\dot{V}E$; at any $\dot{V}E$, VT increased with increasing height (figure 7) or VC. The pattern of breathing was also analyzed in terms of the time spent in inspiration (Ti) in relation to the total respiratory time (Ttot, 60/f) in a representative sample of 40 subjects. Both Ti and Ttot progressively shortened with increasing exercise; Ti/Ttot remained relatively unchanged, being 0.36 ± SEM 0.052, and 0.43 ± 0.035, at standard levels of $\dot{V}E$ of 20 and 60 L/min, respectively.

Variables Measured at Two Standard Power Outputs

A third approach taken to the interpretation of submaximal exercise test results was the analysis of measurements made at two submaximal power outputs, 300 and 600 kpm/min.

At 300 kpm/min (tables 3A and 3B), there were no differences between the sexes and no effects of age in $\dot{V}O_2$, $\dot{V}CO_2$, R, or $\dot{V}E$; VT was higher and HR was lower in males than in females ($p < 0.001$), but there was no effect of age on these two variables. Systolic blood pressure was similar in males and females but increased significantly ($p < 0.01$) with age. When variables at this power output were analyzed for the effect of height (table 4A), no differences were apparent between the sexes in any variable except HR; at each height, HR was about 20 beats/min higher in females than in males. Significant increases with height were found for $\dot{V}O_2$ ($p < 0.001$), $\dot{V}CO_2$ ($p < 0.001$), $\dot{V}E$ ($p < 0.01$), and VT ($p < 0.01$). No effect of height was found on HR, unless expressed in terms of $\dot{V}O_2$; oxygen pulse ($\dot{V}O_2$/HR) increased with increasing height ($p < 0.001$). There was no effect of height on BPs. Rating of

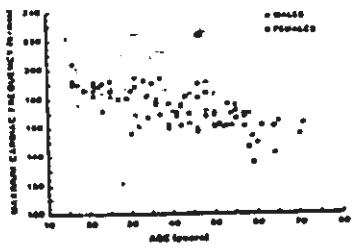

Fig. 5. Cardiac frequency at maximal exercise related to age in males and in females.

TABLE 3A

MEASUREMENTS AT 300 KPM/MIN IN AGE CATEGORIES (MALES)

Age (yr)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min)	BPs (mmHg)
15–24	1.00	0.82	0.81	22.9	1.60	104	130
SD	0.190	0.166	0.065	4.08	0.859	11.7	13.3
25–34	0.95	0.79	0.83	24.5	1.91	95	127
SD	0.153	0.199	0.128	10.5	0.719	10.1	*
35–44	0.91	0.73	0.81	24.3	1.78	92	125
SD	0.109	0.100	0.077	5.62	0.525	15.5	*
45–54	0.87	0.71	0.82	22.8	1.34	93	125
SD	0.126	0.121	0.074	5.85	0.264	9.7	*
55 +	0.68	0.73	0.83	23.6	1.44	95	167
SD	0.105	0.066	0.073	2.78	0.314	10.9	*
Total	0.92	0.76	0.82	23.6	1.63	98	133
SD	0.146	0.143	0.083	6.37	0.604	12.1	*

Definition of abbreviations: $\dot{V}O_2$ = oxygen intake; $\dot{V}CO_2$ = carbon dioxide output; R = respiratory exchange ratio; $\dot{V}E$ = minute ventilation; VT = tidal volume; HR = heart rate; BPs = systolic blood pressure

TABLE 3B

MEASUREMENTS AT 300 KPM/MIN IN AGE CATEGORIES (FEMALES)

Age (yr)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min)	BPs (mmHg)
15–24	0.82	0.67	0.82	20.7	1.10	123	138
SD	0.061	0.062	0.063	2.61	0.104	16.2	*
25–34	0.87	0.72	0.84	21.7	1.23	124	142
SD	0.162	0.114	0.157	2.74	0.305	20.6	*
35–44	0.82	0.71	0.87	23.0	1.26	111	146
SD	0.084	0.053	0.067	2.20	0.231	13.4	*
45–54	0.85	0.70	0.82	22.0	1.31	117	151
SD	0.111	0.093	0.065	4.42	0.213	15.6	*
55–65	0.79	0.69	0.87	22.5	1.08	121	155
SD	0.138	0.132	0.056	5.02	0.269	13.6	*
Total	0.83	0.70	0.85	21.9	1.19	119	133
SD	0.116	0.084	0.069	3.7	0.243	15.9	15.0

For definition of abbreviations, see table 3A.
* Too few observations to calculate SD.

perceived exertion declined with increasing height.

At 600 kpm/min (tables 5A and 5B) in males, there was a trend for $\dot{V}O_2$ to decrease with age ($p = 0.02$), but $\dot{V}CO_2$ and $\dot{V}E$ were similar at all ages; R increased with increasing age ($p < 0.005$). In contrast, in females at 600 kpm/min, significant ($p < 0.001$) increasing effects for age were found in $\dot{V}CO_2$, R, $\dot{V}E$ and VT, with $\dot{V}O_2$ being similar in all age groups. For both males and females, HR was unaffected by age, but BPs increased with age ($p < 0.02$). When variables were analyzed to identify the effect of height (table 4B), $\dot{V}O_2$ increased with increasing height, but $\dot{V}CO_2$, R, and $\dot{V}E$ decreased ($p < 0.001$); BPs was unaffected by height. For a given height the only variable to show a significant difference between the sexes was HR, which was 20 to 40 beats/min higher in females. The rating of perceived effort decreased with increasing size.

Discussion

The present study represents an attempt to obtain predictive data for the analysis of the results of a standarized progressive exercise test on a cycle ergometer (5) used to assess patients with impaired exercise tolerance. As such it may be criticized on two main grounds: first, that the population was not large enough to provide an adequate sample to establish "normal standards," and second, that no new information was obtained regarding the physiologic responses to exercise in health. Both criticisms are to some extent valid, as many studies have presented data obtained in larger numbers of subjects, and the responses found in this study are in agreement with concepts that have been widely accepted for decades. The contribution made by the study lies in the weighting of different factors that influence performance to provide improved precision in standards used to interpret clinical exercise tests.

Although the number of subjects entered into the present study represented only a modest population sample compared with the many studies of maximal exercise performance, care was taken to obtain a distribution of some of the main variables that influence exercise capacity that was representative of the population referred for exercise testing. We wished to avoid a weighting of the results by uneven grouping of age, size, or fitness by recruiting a population containing equal numbers of males and females, and in age groups, a range of heights and a level of habitual activity representative of the general population (13). A number of findings suggested that the sample represented a similar population to other studies of exercise and pulmonary function. First, the relation between height and weight was the same as in the general population (13). Second, the values for $\dot{V}O_2$max, whether expressed in absolute terms (figure 2) or per kilogram body weight, were almost identical to those in the literature, as discussed in more detail below. Third, regression equations calculated from our data to relate vital capacity and FEV_1 to sex, height, weight, and age yielded similar prediction equations to those in current use (14). The number of subjects represented a compromise between a true population study and the number of subjects in whom we could make accurate measurements of all the variables that we thought important.

Previous population studies of $\dot{V}O_2$max in Europe (1–3) and North America (1, 15,16) have shown a remarkable uniformity in the findings despite different testing methods, protocols, and criteria for the measurement. In North America, most have employed an incremental treadmill protocol (15), and some have used discontinuous "supramaximal" exercise to establish a plateau value for $\dot{V}O_2$max, a technique that is not applicable to clinical situations. Because $\dot{V}O_2$max measured in treadmill exercise is about 10% higher than in cycling (2) in young subjects, one approach is to use as predicted $\dot{V}O_2$max for cycling tests 90% of the value established for treadmill exercise, when interpreting the results of cycle ergometry (6). Dehn and Bruce (15) reviewed 17 cross-sectional studies in males, comprising 700 observations in treadmill exercise to derive the following equation: $\dot{V}O_2$max = 57 − 0.39 yr ml/min/kg, which is closely comparable

TABLE 4A

MEASUREMENTS AT 300 KPM/MIN IN HEIGHT CATEGORIES

Height (cm)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min) M	HR (beats/min) F	BPs (mmHg)	RPE
< 160	0.83	0.74	0.90	22.5	1.14		128	143	3.3
SD	0.152	0.139	0.096	3.89	0.229		11.4	31.8	*
160–165	0.80	0.68	0.85	21.4	1.16		118	143	2.0
SD	0.096	0.090	0.065	4.16	0.232		15.3	28.1	*
165–170	0.82	0.66	0.81	20.9	1.23	97	115	131	1.8
SD	0.112	0.066	0.075	3.47	0.256	15.4	16.3	25.2	*
170–175	0.88	0.72	0.82	22.9	1.48	96	114	130	2.0
SD	0.135	0.121	0.068	4.79	0.330	12.8	21.1	*	*
175–180	0.91	0.71	0.79	22.4	1.60	94		125	2.0
SD	0.066	0.068	0.068	4.46	0.634	17.0		*	*
180–185	0.95	0.80	0.84	25.3	1.51	98		135	1.7
SD	0.081	0.101	0.084	3.71	0.311	11.0		*	*
> 185	1.09	0.93	0.86	28.3	2.27	94		120	1.0
SD	0.178	0.167	0.129	10.1	0.920	11.0		*	*
Total	0.87	0.73	0.83	22.7	1.40	96	119	132	*
SD	0.139	0.124	0.087	5.08	0.502	12.4	15.7	23.2	*

Definition of abbreviations: RPE = rating of perceived exertion. For other definitions, see table 3A.
* Too few observations to calculate SD.

TABLE 4B

MEASUREMENTS AT 600 KPM/MIN IN HEIGHT CATEGORIES*

Height (cm)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min) M	HR (beats/min) F	BPs (mmHg)	RPE
< 160	1.30	1.54	1.19	43.2	1.65		163	159	6.0
SD	0.117	0.199	0.144	6.48	0.196		9.6	13.9	†
160–165	1.30	1.44	1.11	41.7	1.72		153	151	4.7
SD	0.119	0.189	0.136	7.43	0.258		10.8	16.1	†
165–170	1.33	1.29	0.97	36.4	1.78	124	147	144	4.1
SD	0.128	0.178	0.123	6.56	0.391	18.4	12.3	12.3	†
170–175	1.42	1.38	0.98	38.9	1.89	115	144	158	3.8
SD	0.153	0.156	0.148	7.63	0.369	10.8	21.5	31.8	†
175–180	1.51	1.31	0.87	36.8	2.24	114		150	3.0
SD	0.145	0.078	0.077	5.43	0.679	21.7		17.0	†
180–185	1.44	1.32	0.92	37.0	2.01	111		153	3.1
SD	0.086	0.099	0.063	2.41	0.353	11.3		13	
> 185	1.55	1.36	0.88	36.3	2.86	109		149	2.4
SD	0.105*	0.113	0.042	4.7	1.109	17.3		18.0	†
Total	1.39	1.36	0.99	38.4	1.96	116	151	151	†
SD	0.148	0.167	0.147	6.49	0.582	18.0	13.0	18.2	†

For definition of abbreviations, see table 3A.
* There were no significant differences between males and females except for heart rate.
† Too few observations to calculate SD.

to that reported above. Similarly, the same relationship derived by Drinkwater and colleagues (16) in a review of 29 studies of females was close to that of the present study: $\dot{V}O_2max = 44 - 0.31$ yr ml/min/kg. The equations reported in these 2 studies predict $\dot{V}O_2max$ to be 41.4 ml/kg/min for a 40-yr-old male and 31.6 ml/kg/min for a 40-yr-old female, compared with 37.4 and 28.6 ml/kg/min, respectively, in the present study, both values being some 10% lower, consistent with the established differences between treadmill and cycling exercise. The values of $\dot{V}O_2max$/kg in the present study are closely comparable to those of the large studies of Shephard (1) and Åstrand (3) in which cycle ergometry was employed.

Buskirk and Taylor (4) made the important observation that $\dot{V}O_2max$ was more closely related to indexes of fat-free weight or active tissue volume than to total body weight, and pointed out that although the capacity to perform running work will be related to $\dot{V}O_2max$ per kilograms body weight, when assessing the capacity of cardiorespiratory mechanisms $\dot{V}O_2max$ should be considered in relation to lean body mass, or the obese will be wrongly assessed as impaired. A recent study by Hansen and associates (6) of 77 shipyard workers, one third of whom were obese (weight greater than 120% of that expected for height), supports an approach that uses age, sex, and height to predict $\dot{V}O_2max$ (5). Hansen and associates (6) were able to show that when $\dot{V}O_2max$ was predicted by a height-derived "normal" weight using the equation of Bruce and colleagues (17), the values were close to those measured in a progressive cycle ergometer test similar to the one used in the present study. The correlation coefficients and errors of estimate of the equations derived in our study are closely comparable to those of Bruce and colleagues (17) for males, and Drinkwater and coworkers (16) for females; the variance in results also agrees with the large surveys reported by Shephard (1). In deciding which equations are most appropriate in interpreting test results, it should be noted (table 2) that although correlation coefficients are higher for the equations derived from the total population, the estimation errors are in some instances smaller when males and females are considered separately.

The improvement in $\dot{V}O_2max$ that follows a training program of increased physical activity is well established. Thus, a major factor that may account for the variation in $\dot{V}O_2max$ in subjects of given age, sex, and height is their "fitness." It was of interest that a crude index of habitual leisure time activity significantly reduced this variation, even though we excluded subjects in competitive athletic training. For some applications, it may be desirable to take this factor into account in a prediction of $\dot{V}O_2max$; an equation that included the leisure activity grade was able to predict the $\dot{V}O_2max$ of our subjects with a residual variance of 20% (figure 8).

Two other approaches were examined in the present study to estimate $\dot{V}O_2max$ from estimates of lean thigh volume (figure 3) and measurements of VC (figure 4). Buskirk and Taylor (4) found that fat-free body weight (underwater weighing and thiocyanate space) correlated closely (r, 0.85) with $\dot{V}O_2max$ in young males. Also Davies and Sargeant (18) and Davies and coworkers (19) reported that lean leg volume measurements correlated closely with $\dot{V}O_2max$ in subjects of varying racial characteristics and in the obese and the nonobese. Although there are inaccuracies inherent in the measurement of lean thigh volume (18), in the present

TABLE 5A

MEASUREMENTS AT 600 KPM/MIN IN AGE CATEGORIES (MALES)

Age (yr)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min)	BPs (mmHg)
15–24	1.53	1.39	0.92	35.9	2.31	122	152
SD	0.116	0.088	0.073	2.62	1.06	14.6	11.8
25–34	1.48	1.28	0.86	34.0	2.31	112	143
SD	0.093	0.123	0.083	6.00	0.656	12.6	25.9
35–44	1.50	1.32	0.88	37.6	2.35	111	148
SD	0.144	0.075	0.074	6.21	0.656	20.1	10.3
45–54	1.42	1.28	0.90	36.5	1.92	112	157
SD	0.092	0.102	0.065	2.73	0.360	11.6	12.5
54–65	1.35	1.35	1.00	36.7	2.07	123	178
SD	0.140	0.149	0.100	4.32	0.262	13.0	24.9
Total	1.46	1.35	0.91	36.6	2.19	116	164
SD	0.128	0.115	0.089	4.33	0.58	15.0	19.7

For definition of abbreviations, see table 3A.

TABLE 5B

MEASUREMENTS AT 600 KPM/MIN IN AGE CATEGORIES (FEMALES)

Age (yr)	$\dot{V}O_2$ (L/min)	$\dot{V}CO_2$ (L/min)	R	$\dot{V}E$ (L/min)	VT (L)	HR (beats/min)	BPs (mmHg)
15–24	1.30	1.30	0.99	36.1	1.40	156	140
SD	0.129	0.241	0.122	8.65	0.193	15.0	12.4
25–34	1.38	1.27	0.93	36.3	1.70	150	144
SD	0.190	0.149	0.168	7.87	0.280	17.7	16.2
35–44	1.31	1.47	1.10	41.2	1.81	154	152
SD	0.106	0.123	0.123	3.78	0.312	13.8	10.3
45–54	1.35	1.48	1.10	42.3	1.85	151	157
SD	0.120	0.160	0.129	5.38	0.260	12.1	11.3
55–65	1.27	1.53	1.21	46.8	1.78	155	162
SD	0.136	0.199	0.118	8.00	0.271	12.2	20.5
Total	1.32	1.40	1.07	40.4	1.70	153	149
SD	0.137	0.203	0.132	6.95	0.268	14.2	13.8

For definition of abbreviations, see table 3A.

study a close (r, 0.78) relationship was found between lean thigh volume and $\dot{V}O_2$max. This measurement may be of help in predicting $\dot{V}O_2$max, but as there were few obese subjects in the present study, we were unable to show that it was any better than age and height (table 2). The interesting finding of Kannel and coworkers (20), in the large prospective Framingham study, that VC was predictive of outcome from coronary artery disease prompted an examination of VC as a predictor of $\dot{V}O_2$max. As a single variable, VC was a powerful predictor of $\dot{V}O_2$max (r, 0.86), and the relationship was relatively unaffected by sex and age. Thus, as the vital capacity is closely related to other size variables, deteriorates predictably with age, and is probably related to the size of other organs that may influence exercise performance (such as the heart), it may represent a reliable predictor of maximal exercise capacity in subjects free of respiratory disease. However, the variance is no less than the equations employing sex, age, and height (table 2), and as in many subjects referred for exercise testing, it may not be possible to exclude significant respiratory disease; the predictive equations in table 2 are more applicable generally. The results support the conclusion reached by Hansen and colleagues (6) recently that age and height are more reliable as predictors of $\dot{V}O_2$max than are age and weight.

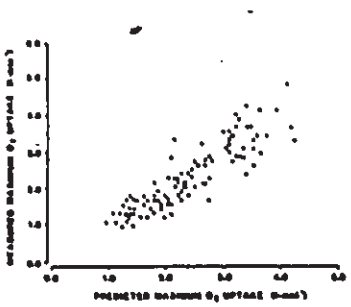

Fig. 8. Concordance between $\dot{V}O_2$max predicted from height, age, sex, weight, and leisure activity grade, and the measured $\dot{V}O_2$max.

Of the other variables measured at maximal exercise, heart rate was shown to vary as a function of age in males and females, the relationship (figure 5) being indistinguishable from that presented by many larger population studies in the past 50 yr (2). The relationship between heart rate and O_2 intake yields information regarding the cardiac stroke volume that depends on the well-established linear and precise relationship between cardiac output and $\dot{V}O_2$ (21). Thus, the "oxygen pulse"[2] at maximal exercise ($\dot{V}O_2$max/HRmax) reflects maximal stroke volume. The values obtained in the study suggest that a normal value may be predicted from height (figure 6) together with sex, the correlation coefficients (table 2) being high enough for predictive purposes. The values presented here are in agreement with those of Hansen and associates (6) in obese and nonobese males. The ventilation in maximal exercise ($\dot{V}E$max) was closely related to maximal $\dot{V}CO_2$; the relationships of $\dot{V}E$max to the FEV_1 and VC indicate that in normal subjects the ventilation in maximal exercise reaches about 70% of the maximal voluntary ventilation, either estimated from FEV_1 times 35 or directly measured (6, 22).

Although normal values for many variables at maximal exercise are reasonably well established, data at submaximal exercise levels are limited to studies of relatively small numbers of subjects and are not easy to use in interpreting exercise tests carried out in subjects of different ages and sizes. One of the objectives of the present study was to obtain data that might improve such predictive standards for heart rate, ventilation and tidal volume, and blood pressure in submaximal exercise in a group of subjects defined in terms of maximal exercise capacity. Several approaches have been used in the past, and some of these were tested in the present study; all use the general principle of relationships between dependent and independent variables, either through the whole exercise test by constructing regression equations or at standard submaximal power outputs or oxygen intakes. An approach that uses the measurements obtained at all the incremental power outputs is attractive, in that expressions may be derived to describe the evolution of responses to increasing exercise. Also, by using all the measurements, variation may be minimized by reducing the reliance on a single value. In the present study, as in many previous reports, a close relationship was

found between increasing power (W) and $\dot{V}O_2$ that is conventionally used as an index of the "mechanical efficiency" of the task being carried out. The results of the present study, in agreement with many studies in the past (2 and 3 among others), demonstrated that in cycling exercise there is little variation in efficiency. The small effect that size and age had on the slope of the relationship is of little importance within the ranges of size and age usually encountered in an exercise laboratory. However, body size, by influencing the intercept of the relationship in a subject through its effect on the $\dot{V}O_2$ at rest or during unloaded cycling, will influence the $\dot{V}O_2$ obtained at a given power output. Wasserman and Whipp (23) pointed out that the oxygen cost of unloaded cycling was linearly related to body weight; the coefficient calculated by Hansen and associates (6) to express the increase in $\dot{V}O_2$ in unloaded cycling as a function of weight (0.00589 Wt) is very similar to the effect of weight on the intercept of $\dot{V}O_2/W$ ($\dot{V}O_2$ at zero W) in individual subjects of the present study (0.0055 Wt).

The heart rate response to exercise may be described by a linear regression equation that relates the increase in heart rate to the increase in $\dot{V}O_2$ through the test ($SHR/\dot{V}O_2$); this slope was proposed as an index of physiologic strain by Spiro and colleagues (24) in an investigation of the normal responses to an identical exercise protocol to the one used in the present study. Their findings were similar to those of the present study: $SHR/\dot{V}O_2$ was 42 beats/L in young and old males, 63 beats/L in women younger than 40, and 71 beats/L in women older than 40; the differences between males and females were accounted for by differences in lean body mass, in agreement with the findings of Cotes and coworkers (25). In the present study, height was the best predictor of this slope in males and females, and the prediction was not significantly influenced by age. Using the above mentioned prediction equations, the slope was 44 beats/L in a male 170 cm in height and 66 beats/L in a female 160 cm in height. The precision of the calculated slope depends on an adequate number of observations and an absence of a spurious tachycardia at the lower work loads; these two factors may account for the variability found in the present study in regression equations relating slope to height in female subjects. Thus, the O_2 pulse at maximal exercise is probably preferable to the $SHR/\dot{V}O_2$ as an index of stroke volume in routine exercise studies. If a prediction is required for the slope of HR to $\dot{V}O_2$, this also may be indirectly obtained from predicted values for HRmax and $\dot{V}O_2$max (5).

Systolic blood pressure increased linearly with power output in all subjects, but the increase was greater in older subjects. This finding is in agreement with the findings of Tornvall (26) and Granath and coworkers (27) and suggests that the interpretation of blood pressure changes with exercise should take the subject's age into account; an increase that may be within the normal range for an elderly subject may be outside the normal range for a younger subject. Although the increase in blood pressure with exercise for various stages of essential hypertension were well described by Sannerstedt (28), the clinical implications of an abnormal rise in blood pressure in exercise have yet to be established.

The increase in ventilation with exercise showed a close relationship to the increase in CO_2 output, as established by many studies in the past (23), and indicated that the component responses in terms of alveolar ventilation and the ratio of dead space to tidal volume are quite uniform in normal subjects and relatively unaffected by age and size. Although larger subjects show larger tidal volumes at a given power output, thus tending to reduce the V_D/V_T ratio, they also have a larger airway dead space, which presumably offsets this effect; pulmonary gas exchange mechanisms in exercise were reviewed in a recent symposium (6, 29). As demonstrated in previous studies (30), the increase in tidal volume with exercise is not linear, tending to increase mainly at low and moderate power outputs and relatively less as exercise becomes heavy. Some researchers have analyzed the V_T response to increasing $\dot{V}_E$ into 2 intersecting straight lines—a lower linearly increasing V_T up to a maximal value, and an upper portion in which increases in ventilation are brought about by increases in the frequency of breathing, with V_T remaining constant (31). The finding of a curvilinear response of V_T to $\dot{V}_E$, the position of the curve in an individual being influenced by height (figure 7) or lung size as reflected in VC, is consistent with the curvilinear relationship between volume changes in the total respiratory system and respiratory muscle forces (32), and of optimization of respiratory muscle effort (33).

A final approach to interpretation of exercise data that has been used in the past is based on measurements made at standard power outputs or $\dot{V}O_2$. It is clear from our data that in addition to age (tables 3 and 5) the effect of height (table 4) must be taken into account also in such an analysis. As height in our population was fairly evenly distributed, it may account for differences between the present results and previous studies. In the study of Spiro and colleagues (24) data were analyzed into 4 groups according to sex and age (20 to 40 and older than 40 yr of age). Otherwise, similar conclusions may be drawn from the data, as outlined previously for the continuous variable approach. Sensitivity of the analysis at a standard power output is likely to be similar, but an approach based on measurements at many levels of exercise is likely to be more precise because of the reduction in random variation that may occur at a single power output.

Measurement of the "anaerobic threshold" has been popularized by Wasserman and colleagues (7) as an important index of exercise performance and fitness. In the present study, we used their criteria to identify the $\dot{V}O_2$ at which $\dot{V}_E$ increased disproportionately in relation to $\dot{V}O_2$ because of a relative increase in $\dot{V}CO_2$—the ventilatory anaerobic threshold (Vat). Although initially the normal variation of Vat expressed as a percentage of $\dot{V}O_2$max was thought to be quite small (34), subsequent applications of the measurement to population studies have shown a wider variation; the findings of Hansen and associates (6) are almost identical to those of the present study; these workers reported a normal range of Vat in their male population of 40 to 78% (mean, 56%) of $\dot{V}O_2$max and proposed a normal lower limit of 40% of predicted $\dot{V}O_2$max. The variation in measurements of Vat in the present study was higher than that of $\dot{V}O_2$max when relationships to age and height were examined, with a correlation coefficient of 0.65, compared with 0.87, as was also suggested by the results of some but not all previous studies. In an early study, Wasserman and colleagues (7) reported anaerobic threshold measurements in 85 healthy subjects that varied between 45 and 180 W "depending on age and physical fitness," but comparisons with $\dot{V}O_2$ were not made; figure 7 of their report indicated higher average values for males than for females. Although mean Vat was higher in the 31 male subjects reported by Weltman and Katch (35) than the male subjects in our study, they were young subjects with relatively high $\dot{V}O_2$max (51 ml/min/kg); our

data are similar if allowance is made for these differences. Yoshida and coworkers (36) in young males found a variance of 12% in $\dot{V}O_2max$ compared with 20% for Vat, but Sue and Hansen (37) report a similar variance of 18% for both measurements in the shipyard workers studied by Hansen and associates (6). In a large European study of 66 males and 50 females, Reinhard and coworkers (38) reported values for $\dot{V}O_2max$ as well as for Vat. Although mean $\dot{V}O_2max$ and Vat were both lower than in the present study, similar qualitative effects of age and sex are apparent in their data, with increases in $Vat/\dot{V}O_2max$ as $\dot{V}O_2max$ declines. This is illustrated by the differences in the slope of the decline of $\dot{V}O_2max$ and Vat with age; in males, $\dot{V}O_2max$ declined by 0.021 L/min for each year of age, compared with only 0.0068 L/min for Vat. The values in our study were almost identical (0.021 and 0.0070, respectively); thus, as $\dot{V}O_2max$ declines with age faster than Vat, the ratio of Vat to $\dot{V}O_2max$ increases. Although Hansen and associates (6) concluded that the normal lower limit for Vat is 40% of the predicted $\dot{V}O_2max$, the results of the present study suggest that a predicted normal value should be based on the actual predicted $\dot{V}O_2max$ rather than on a fixed proportion of $\dot{V}O_2max$ because of the positive intercept that was found in the relationship of Vat to $\dot{V}O_2max$. This finding is the mathematical explanation for $Vat/\dot{V}O_2max$ being higher in females than in males, for the increase found with age, and for the similar $Vat/\dot{V}O_2max$ across activity grades. Although Vat increased in absolute terms from 1.34 in activity Grade 1 to 1.44 in Grade 2, 1.68 in Grade 3, and 1.83 L/min in Grade 4, consistent with increasing fitness, when expressed as $Vat/\dot{V}O_2max$ the values were 68, 67, 59, and 59%, respectively. Although the appearance of a Vat usually indicates lactate production (8) and is thus a useful observation in clinical exercise tests, a number of mechanisms are likely to contribute to it (39), making the physiologic implications of the variability and apparent dependence on $\dot{V}O_2max$ of uncertain significance.

We have presented the results of this study in several ways, partly to allow investigators to compare studies in other laboratories with those of the present study and partly to allow a flexible approach to the interpretation of exercise test results; the choice of independent variables was also kept flexible for similar reasons. Strictly, our results may be applied only to studies carried out using the described protocol; however, the strong linkage between circulatory variables and $\dot{V}O_2$ and ventilatory variables and $\dot{V}CO_2$ probably means that the relationships will hold for other protocols if the variables are first related to these "metabolic loads" (23).

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Maximal short term exercise capacity in healthy subjects aged 15-70 years

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Summary

1. Fifty males and 50 females, 15–71 years of age, exercised maximally for 30 s on an isokinetic ergometer at a pedalling frequency of 60 rev./min. Results were compared with maximal oxygen uptake ($\dot{V}O_2$max.) obtained in a progressive incremental exercise test.

2. Total work in 30 s was higher in males than females, declined linearly by about 6% per decade of age ($r = -0.65$), and was related closely to height ($r = 0.75$) and to lean thigh volume estimated anthropometrically ($r = 0.84$). A close association with vital capacity ($r = 0.86$) was also found that accounted statistically for the combined effects of age and height.

3. The percentage decline in power during 30 s (fatigue index) was lower in subjects reporting greater leisure activity.

4. A close relationship was found between total work in 30 s and $\dot{V}O_2$max. ($r = 0.86$), with vital capacity and leisure activity exerting additional influences on $\dot{V}O_2$max. ($P < 0.001$; multiple $r = 0.93$).

5. The well-established reduction with age in $\dot{V}O_2$max. is associated with an apparent parallel reduction in the power output capacity of large muscle groups recruited in heavy dynamic leg exercise.

Key words: ageing, body size, exercise, fatigue, leisure activity, oxygen uptake, thigh volume.

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Introduction

The major factors that influence maximum aerobic power ($\dot{V}O_2$max.) in an untrained population have been recognized for many years as being those of gender, age and body size [1–4], with other factors playing a less important role. Recently attention has been drawn to other indices of maximal exercise performance, such as the maximum power developed in short term, theoretically 'anaerobic' exercise [5], but the values to be expected in normal subjects of different sexes, age and size have not been as well defined as for $\dot{V}O_2$max. The studies described in the present paper provide such values, and in particular focus on the influence of age on indices of maximal exercise performance.

There are a number of physiological changes that accompany the steady fall in aerobic exercise capacity that occurs with age [6]. Central mechanisms influencing oxygen transport, such as the maximum cardiac output and heart rate [2, 6–8], and pulmonary gas exchange function [9], decline steadily after the third decade. Also there are decreases in total muscle mass and the number of functioning motor units [10–12]; this is associated with histochemical and histological evidence of denervation in ageing muscle and a reduction in muscle strength that accelerates after the age of 65 [10–13]. Although the age-related changes in muscle structure and function and in central oxygen delivery mechanisms appear to occur in parallel and are of similar magnitude [14], it remains to be determined if the same factors underlie these changes, or whether they occur independently.

Cross-sectional and longitudinal studies of healthy subjects have suggested that the rate of decline in aerobic exercise capacity with increasing

years is influenced by the level of habitual physical activity, with more active subjects exhibiting a slower rate of decline than sedentary subjects [6, 15, 16]. Whether the effects of habitual physical activity are mediated through the maintenance of cardiac function or by lessening the decline in muscle function is not established. In part, the difficulty in answering these questions is related to uncertainty as to the expected values for maximal muscle power in healthy subjects of various ages. The present study employed an isokinetic cycle ergometer [17] to measure power output and related variables during a brief period of maximal exercise in healthy subjects aged 15–71 years. As the test lasted 30 s only, central oxygen delivery mechanisms theoretically should play relatively little part in meeting the energy requirements of the exercise. In addition to providing data related to normal values of maximum exercise capacity the study compared results obtained with this procedure, with measurements of maximal oxygen intake in a standard progressive incremental exercise test, to examine possible associations between aerobic performance and maximal glycolytic ('anaerobic') capacity.

Methods

The subjects were 100 healthy volunteers, 50 males and 50 females, who were recruited to provide a wide distribution of ages (from 15 to 71 years, Table 1), and of height (males 165–194 cm; females 152–176 cm). Recruitment was by advertisement in local publications and a local radio station; responders were asked their age and height and recruited into the study if a vacancy existed in their respective category; other exclusion criteria included competitive athletes (past or present), and history of serious cardiac or pulmonary disease. At the time of the study spirometric evidence of significant airflow obstruction (>2 SD below predicted values), abnormal resting electrocardiograph, medications known to influence exercise performance, and systemic hypertension (resting blood pressure of 150/90 mmHg or above) constituted additional exclusion criteria. Subjects gave signed informed consent after description of the procedures and possible risks and the study was approved by the institution's ethics committee. No remuneration was offered to subjects.

A smoking history and information on occupational physical activity and activity during leisure hours was obtained by questionnaire. There were 50 life-long non-smokers; 31 subjects had stopped smoking for at least a year before; 13 smoked less than 15 cigarettes per day, and six smoked more than 15 cigarettes per day. Occupational activity was categorized in three grades: sedentary (54 subjects), occupations requiring some standing and walking (30) and those requiring constant activity (10); in six subjects occupational activity was not categorized. Leisure activity was classed into four grades according to the hours per week spent in recreational activity (1, comprising 26 subjects, exercised for less than 1 h; 2, 42 subjects, for 1–3 h; 3, 23 subjects, for 3–6 h; and 4, nine subjects, exercised for more than 6 h); 'activity' was defined as any exertion associated with at least a doubling of the resting metabolic rate [18]. Smoking, occupational and leisure activities were similar in males and females.

Measurements of vital capacity (VC) and the forced expired volume in 1 s (FEV_1) were obtained with a calibrated dry spirometer (Vitalograph); the best of three attempts was recorded for both measurements.

TABLE 1. *Anthropometric variables for male and female subjects*

Mean values ± SD are shown.

	Males						Females					
Age (years)	n	Height (cm)	Weight (kg)	FEV_1 (l)	VC (l)	Lean thigh volume (l)	n	Height (cm)	Weight (kg)	FEV_1 (l)	VC (l)	Lean thigh volume (l)
15–24	11	183	78	5.0	5.9	10.3	10	166	60	3.6	4.1	6.9
SD		6.6	10.7	0.48	0.73	0.65		4.6	5.9	0.16	0.31	1.29
25–34	10	178	79	4.7	5.7	9.3	10	167	59	3.8	4.4	6.2
SD		6.0	10.6	0.64	0.75	1.74		4.3	4.9	0.35	0.49	1.31
35–44	9	179	79	4.6	5.7	9.1	10	162	60	3.1	3.6	5.1
SD		7.4	8.6	0.50	0.66	1.57		5.0	7.2	0.44	0.53	0.88
45–54	10	176	78	4.0	4.7	9.3	10	164	65	2.9	3.8	4.5
SD		7.2	10.9	0.69	0.59	1.46		5.3	8.6	0.51	0.56	1.45
55–71	10	177	78	3.6	4.7	8.6	10	164	62	2.8	3.4	5.4
SD		6.6	9.3	0.61	0.70	0.83		5.0	3.8	0.47	0.51	0.87

Subjects performed two exercise tests on different days: an incremental multistage progressive exercise test and a 30 s maximal cycling test on an isokinetic ergometer. The incremental test was always carried out first, partly to screen the population for untoward effects of exercise: two older subjects were excluded on the basis of electrocardiographic changes consistent with coronary artery disease.

The progressive multistage exercise test [18] was performed on a calibrated stabilized load cycle ergometer (Elma EM 370); the procedure is described in a separate report [19] in which the submaximal and maximal measurements are presented. In brief, the initial power output was 100 kpm/min (16.3 W) and the power output was increased by 100 kpm/min at the end of each minute until the subject could no longer maintain a required pedal frequency. The subject breathed through a low resistance, low dead space valve, inspired ventilation was measured by dry gas meter, and expired gas was analysed for oxygen and carbon dioxide by a Perkin-Elmer MGA 1100 respiratory mass spectrometer. Measurements were averaged every 15 s and the mean of the highest three measurements of oxygen uptake was taken as the subject's capacity ($\dot{V}O_2$ max.). Although a plateau of $\dot{V}O_2$, with $\dot{V}O_2$ being maintained within ± 5% for 1 min at maximum effort, was usually obtained, this was not a requirement for the measurement.

After the incremental exercise test the isokinetic cycle ergometer test was explained to the subject and a practice test was performed.

On the second occasion lean thigh volume was estimated from anthropometric measurements [20], and the subject carried out a 30 s cycling test on an isokinetic cycle ergometer. The ergometer, and procedure, are described in detail elsewhere [17]. The velocity of pedalling was controlled to a predetermined rate by a 3 h.p. motor, and an electrical speed controller. The pedalling rate in the present study was controlled at 60 rev./min, chosen because it is the velocity at which most exercise tests are carried out [18]. The subject pedalled with maximal effort and the torque exerted on the pedals was recorded via strain gauges bonded to the pedal cranks and connected to a multichannel recorder and computer through a slip ring and a Wheatstone bridge circuit.

The ergometer was calibrated before each test. The optimal saddle height was selected for the subject and feet were secured to the pedals by toe clips and straps. A webbing harness around the hips prevented lateral and forward movement and ensured that the major contribution to force output was from the quadriceps muscles. The subject was instructed to exert maximal effort; constant encouragement was given to the subject during the test, with instructions not to grip the handlebars tightly and to breathe regularly. Heart rate and the electrocardiograph were monitored throughout the test and during recovery. There were no untoward sequelae.

The output from the strain gauges was sampled at 10 ms intervals by a laboratory computer (PDP 11-03, Digital Equipment Corp.) that performed integration with respect to time. Measurements were obtained in each subject of peak instantaneous power, average power and work for each pedal revolution, and total cumulative work during the test. All subjects showed a gradual and smooth decline in power during the 30 s test; the decline in average power by the end of the test was expressed as a percentage of the maximum power achieved during the first few pedal strokes (fatigue index, FI) [17]. Our experience with this test in healthy subjects [21] and patients with coronary artery disease (unpublished work) has indicated that the measurements are repeatable in a given subject with a coefficient of variation less than 10%, and that there is no significant effect of habituation.

Results

Performance in the 30 s test could be characterized equally well by all three variables, maximum peak instantaneous power ($W_{inst.}$), maximum average power ($W_{av.}$) and the total work achieved in the 30 s of the test ($W_{tot.}$), in that the correlation coefficients between them were in excess of 0.98.

Effects of age

Male and female subjects $W_{inst.}$ and $W_{av.}$ showed a decline with age that amounted to about 6% for each decade. For maximum peak power, mean values in the 15–24 years decade were 1037 W in males and 661 W in females, declining to 760 W and 466 W respectively in the subjects over 55 years. Maximum average power was 656 W and 404 W respectively, falling to 455 W and 282 W for the same age groups. Similarly, the total cumulative work was 17.8 kJ and 10.6 kJ with a decline to 12.0 kJ and 7.4 kJ (Fig. 1). Thus the percentage decline with age for all three variables was almost identical, and similar for males and females. Total work was statistically the variable most closely related to age ($r = -0.65$). Values for all three variables in females were about two-thirds of the values obtained in males (Fig. 1). The following regression equations express the

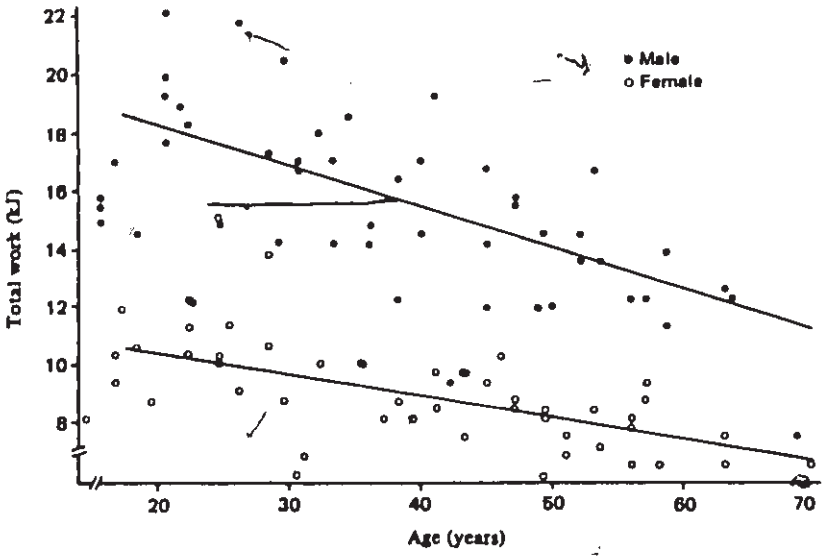

FIG. 1. Total work in 30 s in males (●) and females (○), related to age.

influence of age on these three variables in the two sexes:

Males $W_{inst.} = 1222 - 7.3\,(age)$ W
(SEE 152, $r = -0.59$)
$W_{av.} = 770 - 4.7\,(age)$ W
(SEE 102, $r = -0.58$)
$W_{tot.} = 20.8 - 0.137\,(age)$ kJ
(SEE 2.30, $r = -0.66$)

Females $W_{inst.} = 780 - 5.0\,(age)$ W
(SEE 91, $r = -0.64$)
$W_{av.} = 486 - 3.1\,(age)$ W
(SEE 62, $r = -0.61$)
$W_{tot.} = 12.7 - 0.085\,(age)$ kJ
(SEE 1.41, $r = -0.66$)

where age is in years.

Effects of body size

In both females and males, the power variables $W_{inst.}$ and $W_{av.}$ and also $W_{tot.}$ showed strong correlations to parameters of body size; height ($r = 0.75$ for all variables) was more closely related to all three variables than weight ($r = 0.66$). Analysis of covariance showed that significant ($P < 0.001$) effects of age and sex remained even when height or weight was taken into account. The regression equations linking these variables for the total population were:

$W_{inst.} = 8.2(ht.) - 5.2(age) - 251(sex) - 310$ W
(SEE 119, $r = 0.87$)
$= 6.7(wt.) - 6.5(age) - 249(sex) + 672$ W
(SEE 116, $r = 0.88$)

$W_{av.} = 5.0(ht.) - 3.4(age) - 160(sex) - 175$ W
(SEE 81, $r = 0.87$)
$= 4.3(wt.) - 4.2(age) - 155(sex) + 412$ W
(SEE 78, $r = 0.87$)

$W_{tot.} = 0.125(ht.) - 0.097(age) - 4.5(sex) - 3.14$ kJ (SEE 1.90, $r = 0.89$)
$= 0.108(wt.) - 0.117(age) - 4.4(sex) + 11.5$ kJ (SEE 1.80, $r = 0.90$)

where height (ht.) is in cm, weight (wt.) in kg, age in years and sex is coded 0 for males and 1 for females.

Lean thigh volume (sum of both legs) was closely related to maximum peak power ($r = 0.81$), maximum average power ($r = 0.80$) and total work ($r = 0.84$) (Fig. 2); the regression equation for this last relationship was:

$W_{tot.} = 1.45(tv) + 1.36$ kJ (SEE 2.22, $r = 0.84$)

where lean thigh volume for both legs (tv) is expressed in litres. Similar significant relationships were found for males and females separately.

Analysis of covariance showed that when the relationship between the power variables and thigh volume had been taken into account, an age effect did not remain.

Effects of habitual activity

The occupational level and the time spent in active leisure pursuits did not have a significant

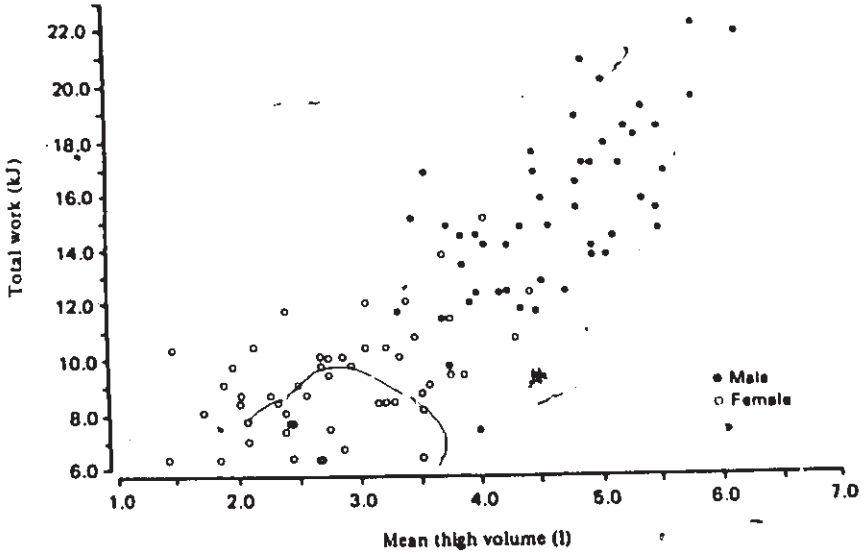

FIG. 2. Total work done in 30 s in males (●) and females (○), related to lean thigh volume, average of the two legs, estimated from anthropometric measurements.

effect on $W_{inst.}$, $W_{av.}$ or $W_{tot.}$, when height, weight and age had been entered into an analysis of covariance.

Fatigue index

In contrast to power variables and the total work, the fatigue index was not related to age, height or weight. The average fatigue index for $W_{inst.}$ and $W_{av.}$ over the 30 s of the test was 26 ± 10.0% in males and 30 ± 9.7% for females (not significant). A weak negative correlation ($r = -0.30$) was found between the fatigue index and leisure activity grade, suggesting a possible association between these two variables ($P < 0.001$). There was no significant relationship between the fatigue index and maximum power variables.

Comparison of 30 s performance with $\dot{V}_{O_2}$ max.

Significant relationships ($P < 0.001$) were obtained between the two power variables and total work and $\dot{V}_{O_2}$max. measured in the incremental exercise test ($r = 0.89$ for all three variables) (Fig. 3). There were significant relationships between $\dot{V}_{O_2}$max. and height ($r = 0.78$), weight ($r = 0.65$) and lean thigh volume ($r = 0.79$). Other variables that were significantly related ($P < 0.001$) to both power variables, to total work ($r = 0.84$) and to $\dot{V}_{O_2}$max. ($r = 0.86$) were the VC and FEV_1; these variables statistically took into account the combined effects of height, age and sex.

Multiple stepwise regression analysis was used to examine associations between measurements of body size, maximum power variables and other variables and $\dot{V}_{O_2}$max. The total work was the variable most closely related to $\dot{V}_{O_2}$max. ($F = 63.9$, $P < 0.0001$), followed by VC (partial $F = 26.2$, $P < 0.001$) and leisure activity (partial $F = 19.3$, $P < 0.001$). The multiple regression equation for this relationship was:

$$\dot{V}_{O_2}\text{max.} = 0.123\,(W_{tot.}) + 0.30\,(\text{VC}) + 0.17\,(\text{leisure}) - 0.898\ \text{l/min}$$
$$(\text{SEE}\ 0.415, r = 0.93)$$

Thus only 13% of the variance ($r^2 = 0.87$) in measurements of $\dot{V}_{O_2}$max. was left unexplained once the total work, VC and leisure activity had been entered into the stepwise regression procedure. A similar expression was derived where age, sex, height and weight replaced the constant for VC:

$$\dot{V}_{O_2}\text{max.} = 0.131\,(W_{tot.}) + 0.016\,(\text{ht.}) - 0.008\,(\text{age}) - 0.059\,(\text{sex}) + 0.007\,(\text{wt.}) + 0.145\,(\text{leisure}) - 2.44\ \text{l/min}$$
$$(\text{SEE}\ 0.458, r = 0.92).$$

Discussion

The present study employed a recently developed isokinetic cycle ergometer that enables power and

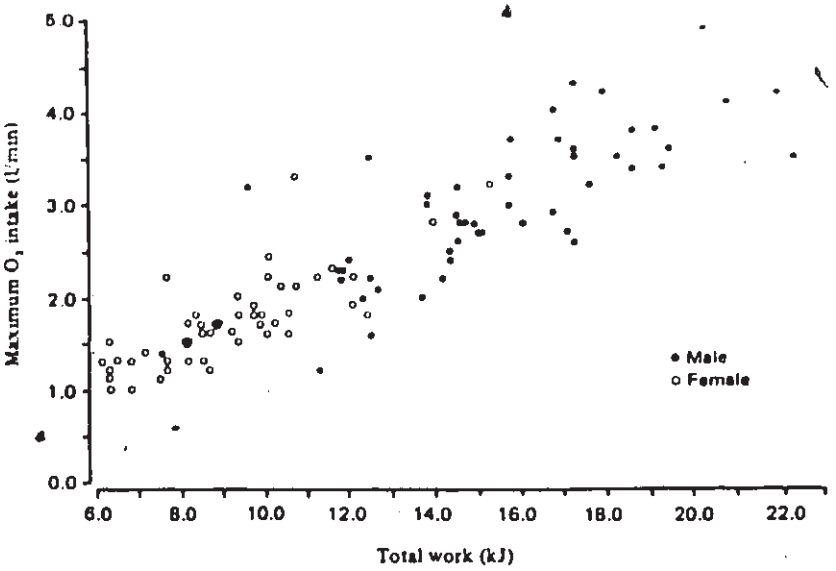

FIG. 3. Total work in 30 s related to maximal oxygen intake measured in an incremental progressive exercise test.

fatigue to be measured precisely during dynamic cycling exercise [17]. The results provided information regarding the factors that influence muscle power in healthy subjects, the effects of ageing, and associations between maximal short-term muscle power and maximal exercise aerobic capacity ($\dot{V}O_2$max.).

The measurements were made in males and females, selected to provide a wide distribution of age and height. Although the study may be criticized on the grounds that the sample size was too small to be representative of the population, we sought to minimize this effect by selecting for as even a distribution as possible for age and height. The measurements made in the study to characterize the population were comparable with established normal values for pulmonary function data [22] and maximal exercise test results [6, 7, 19, 23].

Although the majority of studies that have investigated maximal exercise capacity have been concerned with aerobic power as reflected in $\dot{V}O_2$max., recently there has been an increased interest in maximal short-term or anaerobic power, in which the duration of exercise is 30 s or less. In meeting the demands of maximal exercise for a short time, there is less reliance on muscle oxygen delivery than in exercise of lesser intensity or longer duration, and large increases in muscle [24] and plasma [21] lactate concentrations are found. Several studies have [illegible] the Wingate test [25], in which the subject pedals as fast as possible on a mechanically braked ergometer; after maximal pedal velocity is reached a standardized resistance is imposed; during 30 s the pedal velocity is monitored and declines progressively; power is calculated from the resistance and the number of revolutions of the flywheel during each 5 s of the test. Although the methods used in the present study are more complex, they have the advantages of measuring torque directly and at a constant velocity; thus no assumptions have to be made regarding changes in frictional and inertial forces during the procedure. The relationship between peak power and crank velocity is a parabola, with a peak of optimal velocity between 120 and 160 rev./min [21]; although our choice of 60 rev./min may not have represented the cycling frequency required for our subjects' maximum power, it is the frequency associated with the least fatigue and subjective stess [21], and also that employed in most cycle ergometer tests [18]. Furthermore, it has been our experience that elderly or infirm subjects find difficulty in pedalling at the very high frequencies that are required in the Wingate test. The Wingate test was not used in a study comparable with the present one (O. Bar-Or, personal communication), but comparison of our results in the younger subjects with those reported for the Wingate test [24, 25] suggests that the average power measurements are similar.

As in previous studies that used an isokinetic ergometer in healthy young subjects [21], we found close relationships between the maximum peak and average power, and the total cumulative work indicating that any one of the three could be used as an index of maximal performance during this type of exercise, but for most relationships the total cumulative work during the test gave the least variation. Total work was higher in males (mean 15.6 kJ) than females (9.1 kJ) and showed a gradual decline with age that on average amounted to 6% per decade of age in both sexes (Fig. 1).

At any given age there were associations between the power variables and height and, less significantly, weight. For subjects of given size, age was significantly related to power variables, but when thigh volume measurements were used in an analysis of covariance, age was not found to exert an independent effect. The estimate of lean thigh volume from anthropometry [20] is potentially subject to error, but it is of interest that it was the measurement to which power was most closely related. This finding is consistent with previously established relationships between maximum power during cycling and the size of muscles [5, 21]. Although the small age-related decline in lean thigh volume ($r = -0.25$, $P < 0.01$) may thus contribute to the reductions in power, this decline in our subjects was not uniform with age. When data in males and females were combined, lean thigh volume (sum of both thighs) was found to fall by 11% between age 20 and 30 years (from 8.66 litres to 7.67 litres); a further 8% between 30 and 40, to 6.94 litres, but no further significant reductions occurred, mean values being 6.78 litres at age 50 and 6.9 litres at 60 years. As discussed below, changes in muscle function with age are only partly related to size, several other factors making significant contributions. Aniansson *et al.* [13] showed that reductions with age in muscle strength and the ability to perform a step test correlated with reductions in body cell mass and muscle fibre area. Although these workers did not find histological evidence of muscle degeneration, Grimby *et al.* [26] found changes consistent with denervation and reinnervation in 80 year-old subjects; because falls in lean body mass were more marked than in fibre area, they suggested that a loss of fibres also occurred in old age.

Whereas the maximal power variables measured during the isokinetic cycle ergometer test appear to reflect mainly muscle size, the relative extent to which power declines during the 30 s (fatigue index) was found to be unaffected by age and size; indeed the relationship between average thigh volume and fatigue was positive, suggesting that the larger the thigh muscle volume the greater the relative fatigue during the test. The explanation appears to lie in the fact that subjects with larger thighs achieved the higher maximum peak power; although the decline from these maximum values was greater, the total work accomplished in the 30 s (a composite function of both maximum power and the fatigue index) is still greater in subjects with greater muscle mass.

The finding in the present study of a decline with age in the peak power and total work in 30 s, without a significant change in the fatigue characteristics, may indicate a relative preservation of oxidative fatigue-resistant muscle fibres in older subjects. This hypothesis is consistent with the finding in several studies of a reduction in the size and number of fast twitch type 2 fibres with age [2, 4, 10, 27]. Such age-related changes may be due to the motoneuron dysfunction shown by Campbell *et al.* [12] to occur with age.

Although the measurement of $\dot{V}O_2$ max. conventionally is used to assess aerobic power, whereas the total work in 30 s mainly assesses the muscles' maximal glycolytic, or 'anaerobic', capacity, a close relationship was obtained between the two measurements. Also the two indices appeared to decline with age at similar rates. The high correlation between total work and $\dot{V}O_2$ max. suggests that there are common factors contributing to both measures; taken with the relationship between thigh volume and $\dot{V}O_2$ max. this finding suggests that in the average population the size of muscles and their capacity to generate power are important factors contributing to the maximal aerobic power in exercise [28]. However, if we had included well-trained endurance athletes in our population their $\dot{V}O_2$ max. would be high in relation to the maximum isokinetic power (Fig. 3) [17]. Gollnick *et al.* [29] showed specific training effects in terms of muscle enzyme activities in athletes trained for different events that provide a biochemical basis for the fatigue resistance and high $\dot{V}O_2$ max. of endurance athletes.

A number of studies have shown that, for any given age and size, active individuals tend to have a higher $\dot{V}O_2$ max. than inactive subjects [30]. Furthermore, longitudinal studies have established that the decline in $\dot{V}O_2$ max. with age is less in the active than inactive [15, 31, 32]. In the present study a simple categorization of subjects according to weekly leisure activity hours had a positive influence on $\dot{V}O_2$ max. and was associated with a reduction in fatigue during the 30 s maximum cycling test. Thus these findings raise the possibility that the maintenance of muscle quantity and quality are both important in the maintenance

of $\dot{V}O_2$max. with increasing age. Although the well-established linear decline in maximum cardiac frequency with age [7] has been used as an indication that the fall in $\dot{V}O_2$max. is determined by 'central' factors related to intrinsic myocardial properties [33], it may be argued that the function of muscles may be at least as important.

Our findings have implications for the maintenance of muscle size and function and of their aerobic power with age. It seems likely that subjects in active occupations will maintain muscle size and the muscle's aerobic capacity will be maintained by regular leisure activity. In addition, the possibility is raised that exercise carried out at any age may help to improve muscles' aerobic capacity and thus help to maintain $\dot{V}O_2$max. Aniansson & Gustafsson [34] trained 12 70 year-old subjects and showed increases in the area of type 2 fibres associated with improvements in muscle function. In spite of evidence of neurally mediated degeneration of muscle with age [12], muscle remains susceptible to improvement by training techniques.

Acknowledgments

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Appendix 3. Descriptive Characteristics of Subjects (means and SD)

Age (yrs.)	Weight (kg)	Height (cm)	Vital capacity (l)	Lean thigh volume (l)	Resting blood pressure (mmHg) Systolic	Diastolic	Leisure* activity	Smoking category
27.4	71.0	176.3	5.335	3.68	118.0	78.8	1.4	1.3
2.9	8.5	5.8	0.585	0.35	14.4	4.5	0.7	
65.0	73.3	170.6	4.032	3.44	136.0	85.8	1.5	2.1
3.3	8.3	4.7	0.464	0.33	15.5	8.7	0.5	0.8

* Leisure activity was classified as follows:

1 = inactive
2 = 1-3 hrs/wk
3 = > 3-6 hrs/wk
4 = > 6 hrs/wk

Smoking category was classified as follows:

1 = never
2 = previous smoker
3 = < 15 cigs./day (pipe)
4 = > 15 cigs./day

Appendix 4. Employment Profile of Subjects

	GROUP	
	20-30 yrs.	60-70 yrs.
Sales	3	3
Professional	4	1
Managerial	0	2
Retired	0	6
Students	2	0
Unemployed	1	0
TOTAL	N = 10	N = 12

The sales category includes real estate, insurance and Xerox company.

The professional category includes engineers, physician, computer programmer and systems analyst.

The managerial category includes retail store and parking lot managers.

The two students were in graduate studies (pathology and engineering).

Appendix 5. Clinical and Sociodemographic Data Questionnaire

DATE OF EXAMINATION _____ P_B _____ HUMIDITY _____ TEMPERATURE _____

NAME _____ CODE _____ DATE OF BIRTH _____ AGE _____

OCCUPATION _____ HEIGHT _____ WEIGHT _____

Clinical Data

BLOOD PRESSURE _____

Do you experience chest pain with activities such as walking, running or lifting? Yes ____ No ____

Do you have:	Yes	No
hypertension		
diabetes		
lung disease		
heart disease		
asthma		
bronchitis		
other (specify)		

If yes, give details _____

_____

_____

Are you presently taking any meditations? Yes ____ No ____

If yes, give details _____

_____

Appendix 6. Physical Activity and Smoking History Questionnaire

Physical Activity

Do you undertake physical activity such as walking, jogging, swimming, skiing, etc.?

If yes, indicate:

Nature of activity	Duration/session	Frequency/week
_____	_____	_____
_____	_____	_____
_____	_____	_____

Is your occupation physically demanding? Yes ____ No ____

Indicate whether it involves: mostly sitting _____

mostly walking/standing _____

manual work/climbing stairs _____

PHYSICAL ACTIVITY CODE: Leisure: _____

Occupation: _____

Smoking History

Have you smoked in the last 4 weeks? Yes ____ No ____

If yes, indicate number of cigarettes smoked/day ____

If no, have you ever smoked? Yes ____ No ____

When did you stop smoking? _____

SMOKING CODE: _____

Appendix 7. Health Status Form*

PATIENT'S NAME _____ PHYSICIAN'S NAME _____

ADDRESS _____ ADDRESS _____

_____ _____

_____ _____

TEL. NO. _____

EXAMINATION DATE _____

Does Mr. _____ have any clinical evidence of the following clinical disorders which might limit exercise or be associated with risk:

	Yes	No
Heart disease	____	____
Lung disease	____	____
Hypertension	____	____
Diabetes	____	____
Asthma	____	____
Bronchitis	____	____
Arthritis	____	____
Other condition that would exclude participation in an exercise program	____	____

If yes, please give details _____

_____

_____

Has the patient been on any medication over the last two months?

Yes ____ No ____

If yes, please give details _____

_____

Patient's resting blood pressure _____

Has the patient had a recent electrocardiogram? Yes ____ No ____

If yes, please indicate date _____ and result _____

_____

If you have any other comments you wish to offer regarding Mr. _____ participation in this study, please indicate below.

Date _____ Physician's signature _____

_____

* Completed by physician of subjects in older group only.

Appendix 8. Physician Information Outline*

Dear Dr. _____

RE: "Effect of Training on Muscle Function and Aerobic Capacity in Young and Older Subjects"

Your patient, Mr. _____ has consented to participate in the above study which involves participation in a 12-week training program, three times a week, for one hour. The intensity of the program will be based on the individual subject's maximal oxygen uptake which will be determined via a graded exercise test. Blood pressure and heart rate will be monitored throughout the test. In addition, muscle power in the legs will be determined via a maximal effort, short term, cycling test which lasts for 30 seconds using a constant velocity ergometer.

The purpose of our study is to investigate the effects of training on physical work capacity and muscle function and to examine how these may differ in young and older subjects. Our sample consists of two groups of healthy male volunteers, 20-30 and 60-70 years of age who do not undertake regular physical activity.

We are seeking your assistance in determining Mr. _____ general health status and we would be most grateful if you complete the attached form and return it to us as soon as possible. A stamped self-addressed envelope is enclosed.

If you have any questions, please feel free to contact us. Thank you for your cooperation.

Yours sincerely,

---

* Mailed to physician of subjects in older group only.

Appendix 9. Consent Form

Our purpose is to investigate the effect of training on your ability to exercise for long periods of time (heart and lung function) and short periods of time (muscle function) and to examine how this may differ in young and older subjects. If you consent to participate in this study, you will be required to attend the one-hour exercise sessions, three times a week for a total of 12 weeks. **IT IS ESSENTIAL THAT YOU ATTEND ALL SESSIONS.**

A series of tests will be performed before and after the 12-week training program. These are:

1. A graded exercise test measuring heart and lung function.
2. A test measuring muscle strength in the legs.
3. A test measuring lung capacity.

1. **Graded exercise test:** This will be done on a cycle ergometer. The work will begin at a very easy level and will gradually become more difficult. We would like you to continue to exercise until you are limited by fatigue or extreme discomfort. We will be closely monitoring your test; if we see any reason to stop the test prematurely, we will do so at once. While exercising, you will breathe through a mouthpiece to measure how much oxygen you use. We will ask you to use a noseclip to make sure that all the air is inhaled and exhaled through the mouth. Electrodes will be attached to your chest to monitor your heart rhythm. A cuff around your arm will be inflated every 2 minutes to measure your blood pressure. This test will take approximately one hour.

2. **Test measuring muscle strength:** This test involves pedalling on a cycle ergometer whose pedals are driven by a motor. You will be asked to pedal as hard as you can for a few seconds at different speeds of the pedals. Three small electrodes will be attached to your chest to monitor your heart rate and rhythm. After this test, a small quantity of blood will be sampled from a superficial vein in your arm. We will also measure the circumference of your thigh, using a tape measure, to see how the size of your leg muscles affects muscle function. The time required for these procedures will be approximately 30 minutes.

3. **Test measuring lung capacity:** This test involves taking a deep breath in and then breathing out through a mouthpiece. It takes approximately 15 minutes.

It is possible that certain adverse changes may occur during the tests. These may include abnormal blood pressure, fainting, disorders of heart beat, and very rarely, instances of heart attack. However, the likelihood of such risks in the controlled environment of the laboratory is small. Appropriate equipment and trained personnel are available in the laboratory to deal promptly with any unusual situations.

After the completion of the study, we will send you a written report of the results of your tests. If you have further questions, we will be pleased to discuss them with you.

Participation in this study is voluntary. You may withdraw your consent at any time, even after signing this form. All information gathered will be confidential.

If you agree to participate, please accept our thanks and indicate your consent by signing below.

_____ _____

SUBJECT SIGNATURE WITNESS SIGNATURE

_____ _____

SUBJECT NAME (please print) WITNESS NAME (please print)

_____ _____

DATE

_____

_____

SUBJECT ADDRESS & POSTAL CODE
(please print)

Home: _____ Work: _____
SUBJECT TELEPHONE NUMBER

- -

I have explained the nature of the study to the subject and I believe that he has understood it.

_____ _____ _____

NAME **SIGNATURE** **DATE**

Appendix 10. Criteria for Stopping the Multistage Exercise Test*

General Signs and Symptoms	Severe chest pain suggestive of angina Severe dyspnoea Dizziness or fainting Marked apprehension, mental confusion or lack of coordination Sudden onset of pallor and sweating Onset of cyanosis
Electrocardiographic Signs	Frequent ventricular premature beats, particularly when showing the R on T wave, frequent runs of three or more, and paroxysmal ventricular tachycardia Atrial fibrillation when absent at rest Second or third degree heart block Ischaemic changes: marked ST depression, T wave inversion, or the appearance of a Q wave Appearance of bundle branch block pattern
Blood Pressure Signs	Any fall in systolic pressure below the resting value A fall of more than 22 mmHg in systolic pressure occurring after the normal exercise rise Systolic blood pressure in excess of 300 mmHg or diastolic pressure in excess of 140 mmHg

* Jones and Campbell (1982).

Appendix 11. Equations Used in the Calculation of Ventilation (V_E), Oxygen Intake (VO_2) and Carbon Dioxide Output (VCO_2)

$$V_E \text{ (BTPS) (l/min)} = \left[\frac{\text{volume} \times 60_S}{t}\right] \times \frac{[(P_B - 29) \times (P_B - 47)]}{\frac{310}{273 + T}}$$

$$VO_2 \text{ (STPD) (l/min)} = \left[\frac{FIO_2 \times [(1 - FEO_2) - FECO_2]}{(1 - FIO_2) - FEO_2}\right] \times V_E \text{ (STPD)}$$

$$VCO_2 \text{ (STPD) (l/min)} = (FECO_2 - FICO_2) \times V_E \text{ (STPD)}$$

Appendix 12. Equations Used in the Calculation of Cardiac Output and Mean Arterial Pressure

$$PvCO_2^* \text{ (mmHg)} = P_{EQ}CO_2 - (0.24\ P_{EQ}CO_2 - 11.0)$$

$$PaCO_2^* \text{ (mmHg)} = 5.5 + 0.9\ P_{ET}CO_2 - 0.0021\ VT$$

$$CCO_2^* \text{ (ml/100 ml)} = 10\ (0.396\ \text{Log}\ PCO_2 + 2.4)$$

$$Pa \text{ (mmHg)} = Pd + 0.33\ (Ps - Pd)$$

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* Jones and Campbell, 1982, pp. 237-239.
Berne and Levy, 1977, p. 103.

Appendix 13. Twenty-30 years: Paired Values* of Repeat Measurements for Each Subject during 30 Seconds of Maximal Cycling on an Isokinetic Ergometer

Subject	Pedalling Frequency (rpm)	Max. Average Power (w)		Total Work (kJ)		Fatigue Index (%)	
		Day 1	Day 2	Day 1	Day 2	Day 1	Day 2
1	60	652.2	636.0	16.1	16.6	34.2	33.5
2	60	499.9	567.3	14.0	15.1	25.0	32.2
3	60	821.3	753.3	20.6	18.4	27.1	29.8
4	110	930.3	931.2	15.7	16.3	63.6	72.4
5	110	1059.0	952.7	17.0	18.7	70.1	60.1
6	60	638.9	609.7	16.2	16.5	29.3	17.6
7	60	633.0	665.7	16.6	17.4	21.6	23.0
8	110	749.9	695.7	13.7	14.7	60.0	55.7
9	110	868.2	818.7	15.4	15.1	62.2	60.3
10	110	834.4	830.1	13.9	13.3	69.2	72.5
	Mean	768.7	746.0	15.9	16.2	46.2	45.7
	S.D.	166.3	133.8	2.0	1.7	20.3	20.7

* not significant

Appendix 14. Sixty-70 years: Paired Values* of Repeat Measurements for Each Subject during 30 Seconds of Maximal Cycling on an Isokinetic Ergometer

Subject	Pedalling Frequency (rpm)	Max. Average Power (w)		Total Work (kJ)		Fatigue Index (%)	
		Day 1	Day 2	Day 1	Day 2	Day 1	Day 2
1	110	746.6	683.1	12.1	10.9	66.4	68.3
2	60	453.0	453.1	10.0	11.1	35.4	41.9
3	110	581.4	637.0	9.7	10.9	60.1	59.1
4	60	397.5	396.4	10.4	10.3	22.4	19.6
5	110	600.8	665.0	11.3	12.6	62.1	55.5
6	60	460.9	437.9	12.7	12.1	24.6	22.9
7	60	404.0	381.1	11.1	10.5	16.7	15.6
8	110	648.2	858.5	12.1	12.7	61.1	67.5
9	60	530.5	515.2	11.8	11.5	49.9	52.7
10	60	497.8	522.6	11.0	11.6	41.2	43.7
11	60	551.8	546.9	14.6	14.6	29.2	33.1
12	110	560.3	630.9	9.8	11.1	63.4	65.2
	Mean	536.1	560.6	11.4	11.7	44.4	45.5
	S.D.	102.1	139.8	1.4	1.2	18.3	19.0

* not significant

Appendix 15. Mean Training Heart Rate and Power Output in 60-70 and 20-30 year old Males During 12 Weeks of Endurance Training

		Old		Young	
		Heart Rate (beats/10s)	Power Output (kpm/min)	Heart Rate (beats/10s)	Power Output (kpm/min)
Week	1	19-21	433	22-24	645
	2	20-22	508	25-27	765
	3	22-24	594	27-29	890
	4	22-24	598	27-29	890
	5	22-24	685	27-29	950
	6	22-24	729	27-29	1010
	7	22-24	742	27-29	1050
	8	22-24	788	27-29	1055
	9	22-24	800	27-29	1085
	10	22-24	817	27-29	1120
	11	22-24	888	27-29	1150
	12	22-24	900	27-29	1200

Appendix 16. Individual values before and after training in oxygen intake, carbon dioxide output, ventilation, tidal volume, heart rate, cardiac output and systolic blood pressure during the progressive incremental cycle ergometer test. The increments in power output in the 20-30 year-old group (subject number 1-10) were 200 kpm/min at the end of 4 minutes. The increments in power output in the 60-70 year-old group (subject number 11-22) were 150 kpm/min at the end of 4 minutes.

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
1	200								
		PRE	.704	.603	16.0	.991	91	*******	138
		POST	.805	.636	18.9	1.406	102	10.3	135
	400								
		PRE	.980	1.069	26.3	1.424	120	14.1	140
		POST	1.116	1.022	26.3	1.755	102	11.5	150
	600								
		PRE	1.305	1.385	33.9	1.825	137	14.7	155
		POST	1.496	1.426	33.3	1.914	116	13.5	155
	800								
		PRE	1.708	1.845	42.8	2.144	151	16.0	170
		POST	1.828	1.822	42.7	2.128	132	16.1	165
	1000								
		PRE	2.410	2.917	80.6	2.905	189	16.3	190
		POST	2.272	2.364	53.1	2.647	151	*******	178
	1200								
		POST	2.754	2.972	68.6	2.926	163	18.2	180
	1400								
		POST	3.440	3.852	95.2	3.055	194	20.0	185
	1600								
		POST	3.965	4.652	127.1	3.154	196	22.6	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
2	200	PRE	.759	.726	20.4	1.697	121	8.2	130
		POST	.679	.575	19.0	1.731	102	*******	112
	400	PRE	1.042	1.111	27.9	2.245	138	12.3	145
		POST	1.027	.856	21.7	2.554	112	10.9	120
	600	PRE	1.411	1.536	38.4	2.438	176	14.2	190
		POST	1.381	1.330	32.1	2.361	135	13.9	150
	800	PRE	1.815	2.009	56.6	2.742	198	14.7	195
		POST	1.797	1.891	47.4	2.564	170	15.3	170
	1000	POST	2.168	2.302	49.2	2.604	178	15.8	194
	1200	POST	2.548	2.828	87.2	2.585	185	*******	212
	1400	POST	2.810	3.244	112.0	2.718	197	19.0	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
3	200	PRE	.784	.682	18.0	1.042	94	10.2	130
		POST	.781	.622	17.9	.937	92	12.0	135
	400	PRE	1.129	1.035	25.9	1.376	108	13.6	140
		POST	1.031	.871	22.4	1.136	101	15.3	144
	600	PRE	1.410	1.345	29.8	1.430	121	15.3	155
		POST	1.410	1.267	31.2	1.537	112	16.4	160
	800	PRE	1.739	1.835	38.8	1.978	137	17.0	170
		POST	1.753	1.622	36.8	1.684	131	17.8	174
	1000	PRE	2.162	2.291	47.4	2.262	168	19.0	********
		POST	2.016	2.068	44.8	2.027	152	19.1	188
	1200	PRE	2.608	2.912	64.1	2.545	178	20.0	195
		POST	2.460	2.464	54.6	2.381	161	20.1	210
	1400	PRE	3.156	3.662	86.7	2.869	196	21.9	200
		POST	2.910	3.058	69.5	2.614	179	20.7	210
	1600	POST	3.308	3.649	87.4	2.814	190	21.0	214
	1800	POST	3.826	4.397	111.8	3.042	197	*******	********
	2000	POST	4:056	4.700	126.9	2.890	217	24.7	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

	TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
200	PRE	.900	.733	21.1	.962	105	10.5	120
	POST	.807	.685	19.7	1.046	94	10.7	135
400	PRE	1.213	1.082	28.6	1.200	122	12.6	140
	POST	1.188	1.074	30.8	1.300	96	12.7	140
600	PRE	1.656	1.575	42.8	1.522	139	15.1	155
	POST	1.512	1.405	39.0	1.588	116	13.4	140
800	PRE	2.052	2.089	54.9	1.890	152	16.9	165
	POST	1.868	1.812	47.7	1.942	130	14.6	154
1000	PRE	2.506	2.687	75.0	2.221	191	17.0	180
	POST	2.246	2.282	60.2	2.191	151	15.2	165
1200	PRE	2.660	3.109	86.2	2.450	191	18.6	********
	POST	2.670	2.786	76.1	2.328	161	*******	180
1400	POST	3.215	3.522	106.4	2.507	178	*******	********
1600	POST	3.453	3.837	128.9	2.567	189	19.2	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
5	200	PRE	.821	.728	20.6	1.430	*******	9.5	150
		POST	.845	.664	18.0	2.336	116	9.2	130
	400	PRE	1.112	1.108	31.0	1.939	139	11.9	175
		POST	1.039	.900	24.3	1.819	128	11.5	135
	600	PRE	1.495	1.543	42.8	2.194	153	13.9	170
		POST	1.359	1.261	32.4	1.905	142	13.5	152
	800	PRE	1.858	2.045	57.9	2.754	170	15.6	190
		POST	1.670	1.682	42.7	2.174	157	14.8	170
	1000	PRE	2.265	2.517	74.0	2.906	191	17.2	190
		POST	2.070	2.181	56.1	2.503	187	15.8	180
	1200	POST	2.442	2.728	73.4	2.840	192	17.5	180

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
6	200	PRE	.735	.799	30.9	2.121	*******	*******	130
		POST	*******	*******	*******	*******	*******	*******	********
	400	PRE	1.072	.944	29.5	2.070	114	9.7	********
		POST	1.038	.942	25.3	1.701	114	11.4	132
	600	PRE	1.444	1.387	37.3	2.657	134	12.5	155
		POST	1.342	1.243	28.2	1.900	133	13.8	144
	800	PRE	1.880	2.015	50.6	2.770	159	15.1	165
		POST	1.690	1.757	36.8	2.584	152	14.9	160
	1000	PRE	2.477	2.800	94.4	2.030	193	18.7	175
		POST	2.187	2.425	51.0	2.339	173	17.2	168
	1200	POST	2.591	3.000	72.0	2.424	185	*******	180
	1400	POST	2.989	3.587	108.6	2.376	200	21.2	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

	TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
7 200	PRE	.718	.655	18.7	1.302	86	9.6	135
	POST	.783	.610	18.5	1.114	75	9.7	130
400	PRE	1.100	1.005	26.1	1.755	106	12.1	135
	POST	1.007	.874	23.2	1.368	99	12.2	150
600	PRE	1.425	1.433	35.6	2.153	129	14.4	170
	POST	1.305	1.220	29.2	1.766	109	14.8	170
800	PRE	1.750	1.804	42.6	2.611	154	16.3	180
	POST	1.664	1.582	38.3	1.998	126	16.1	180
1000	PRE	2.147	2.316	56.0	2.780	162	18.4	190
	POST	2.050	2.015	47.7	2.506	151	17.5	190
1200	PRE	2.624	2.975	75.1	3.300	193	19.0	195
	POST	2.322	2.352	56.8	2.648	160	19.1	200
1400	PRE	3.382	3.964	119.2	3.514	198	21.4	197
	POST	2.731	2.871	69.5	2.972	180	20.3	200
1600	POST	3.045	3.347	88.9	2.911	189	20.1	210
1800	POST	3.471	3.986	118.4	3.126	204	*******	********
2000	POST	3.874	4.368	144.2	2.929	204	25.3	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

TEST		OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/ MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
200	PRE	.708	.705	27.4	1.419	100	8.2	130
	POST	.734	.622	17.9	1.037	100	10.3	********
400	PRE	1.079	1.017	28.1	1.360	116	11.3	150
	POST	1.122	1.088	29.1	1.323	110	12.2	********
600	PRE	1.453	1.497	41.1	1.765	144	13.3	175
	POST	1.379	1.391	37.6	1.566	123	13.2	********
800	PRE	1.896	2.062	61.7	2.105	167	14.1	185
	POST	1.787	1.960	54.4	1.893	147	14.6	********
1000	PRE	2.445	2.750	94.6	2.284	188	16.0	190
	POST	2.181	2.384	69.8	2.309	174	15.1	********
1200	POST	2.617	2.901	100.0	2.288	187	*******	********
1400	POST	2.754	3.131	115.4	2.364	192	17.5	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/ MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
9	200	PRE	.678	.576	17.6	.847	115	9.9	120
		POST	.651	.556	17.3	.734	103	9.0	130
	400	PRE	1.065	.949	27.2	.949	125	11.1	130
		POST	..958	.908	24.9	.920	116	11.9	150
	600	PRE	1.436	1.423	35.8	1.269	157	15.5	160
		POST	1.304	1.268	30.6	1.249	128	14.4	170
	800	PRE	1.882	2.040	53.7	1.616	166	17.3	180
		POST	1.728	1.787	41.4	1.605	145	15.7	190
	1000	PRE	2.295	2.596	67.0	1.952	176	18.9	185
		POST	2.146	2.347	53.3	2.003	172	17.3	192
	1200	POST	2.615	3.040	75.8	2.062	184	16.8	195
	1400	POST	2.859	3.675	97.3	2.240	204	19.3	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

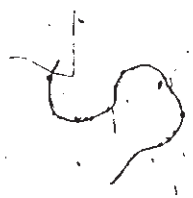
		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
10	200								
		PRE	.672	.591	19.7	.796	95	7.7	120
		POST	.781	.672	22.7	.942	103	10.3	128
	400								
		PRE	1.038	1.061	30.8	1.114	117	10.9	135
		POST	1.118	1.012	30.5	1.304	125	12.1	132
	600								
		PRE	1.402	1.488	40.7	1.472	138	12.0	150
		POST	1.445	1.403	40.5	1.617	139	13.2	150
	800								
		PRE	1.876	2.096	58.8	1.802	169	13.4	170
		POST	1.801	1.780	49.2	2.041	153	14.6	155
	1000								
		PRE	2.458	3.070	104.5	2.306	202	16.5	190
		POST	2.225	2.305	61.6	2.561	163	15.3	170
	1200								
		POST	2.750	2.979	81.8	2.896	188	16.5	174
	1400								
		POST	3.314	3.738	109.6	3.214	198	*******	********
	1600								
		POST	3.425	3.915	129.8	3.008	215	19.0	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
11	150								
		PRE	.553	.532	18.2	1.172	88	7.1	150
		POST	.678	.550	17.5	1.130	84	8.7	125
	300								
		PRE	.803	.789	26.8	1.462	99	8.7	155
		POST	.950	.788	24.2	1.354	95	11.0	140
	450								
		PRE	1.034	1.070	35.5	1.579	128	10.0	165
		POST	1.174	1.046	32.8	1.458	101	11.7	150
	600								
		PRE	1.285	1.400	49.9	1.811	146	10.7	175
		POST	1.468	1.345	41.2	1.629	116	12.1	160
	750								
		PRE	1.594	1.788	68.8	1.919	180	11.1	190
		POST	1.792	1.712	51.9	1.805	132	13.4	165
	900								
		POST	2.094	2.108	67.1	1.892	147	13.7	180
	1050								
		POST	2.272	2.464	83.3	1.785	168	14.7	********



PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

(L/MIN)	TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (ML) MIN)	TIDAL VOLUME (L/MIN)	HEART RATE (BEATS/ (MM HG)	CARDIAC OUTPUT	SYSTOLIC BLOOD PRESSURE
12 150								
	PRE	.590	.625	17.2	1.091	95	9.3	155
	POST	.685	.598	18.9	1.062	93	9.5	144
300								
	PRE	.785	.842	22.7	1.420	101	11.0	170
	POST	.900	.853	26.1	1.482	107	*******	162
450								
	PRE	1.068	1.246	34.7	1.762	122	12.8	175
	POST	1.122	1.106	32.6	1.732	115	12.2	176
600								
	PRE	1.376	1.671	53.6	1.882	145	12.8	205
	POST	1.391	1.428	41.3	2.149	120	13.1	190
750								
	POST	1.662	1.768	53.3	2.246	130	13.7	202
900								
	POST	1.916	2.117	67.5	2.450	142	*******	208
1050								
	POST	2.193	2.494	88.2	2.468	157	16.4	215

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
13	150	PRE	.545	.452	15.3	.888	82	7.1	138
		POST	.583	.486	16.5	.931	75	8.9	128
	300	PRE	.759	.716	21.9	1.168	92	9.1	150
		POST	.834	.746	22.9	1.170	64	10.7	140
	450	PRE	1.021	1.071	31.0	1.507	108	11.3	150
		POST	1.042	.955	26.8	1.437	93	11.6	140
	600	PRE	1.316	1.412	41.7	1.929	131	12.6	165
		POST	1.302	1.282	36.0	1.654	108	13.2	160
	750	PRE	1.599	1.799	58.4	2.304	151	12.6	185
		POST	1.590	1.600	44.4	1.810	130	14.2	178
	900	PRE	2.001	2.435	99.2	2.758	178	15.6	195
		POST	1.852	1.955	56.2	2.154	142	14.8	194
	1050	POST	2.150	2.354	69.9	2.330	151	15.8	210
	1200	POST	2.420	2.744	81.5	2.420	167	*******	215
	1350	POST	2.510	2.728	99.7	2.524	184	17.8	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
14	150								
		PRE	.591	.504	17.8	1.086	104	7.1	170
		POST	.610	.505	17.6	1.116	88	8.0	148
	300								
		PRE	.811	.791	26.0	1.470	126	9.2	192
		POST	.821	.752	24.0	1.481	109	10.9	155
	450								
		PRE	1.018	1.110	37.2	1.952	151	10.9	194
		POST	1.044	1.026	33.0	1.695	123	*******	180
	600								
		PRE	1.318	1.508	55.8	2.233	168	11.7	195
		POST	1.318	1.338	41.9	2.023	136	12.4	180
	750								
		POST	1.582	1.742	55.6	2.402	152	13.1	194
	900								
		POST	1.842	2.109	67.3	2.524	162	13.1	210
	1050								
		POST	2.107	2.588	86.4	2.600	186	14.8	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
15	150								
		PRE	.611	.557	15.1	1.161	82	9.0	125
		POST	.651	.596	16.1	1.371	90	10.3	135
	300								
		PRE	.820	.770	19.2	1.344	94	10.6	135
		POST	.897	.863	22.2	1.402	102	11.8	145
	450								
		PRE	1.052	1.077	26.4	1.717	108	12.2	152
		POST	1.066	1.049	26.9	1.531	116	13.0	155
	600								
		PRE	1.372	1.475	35.2	2.242	121	14.2	160
		POST	1.324	1.359	35.0	1.781	128	14.1	190
	750								
		PRE	1.717	1.915	49.1	2.548	144	15.2	185
		POST	1.598	1.680	43.7	2.431	139	14.8	195
	900								
		PRE	2.014	2.392	66.8	2.723	173	17.4	********
		POST	1.896	2.160	55.7	2.160	154	15.8	204
	1050								
		POST	2.148	2.545	79.4	2.818	175	*******	********
	1200								
		POST	2.218	2.598	86.0	2.620	188	17.5	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
16	150	PRE	.680	.494	20.2	1.193	104	6.3	185
		POST	.716	.560	18.0	1.321	99	8.4	155
	300	PRE	.860	.715	26.7	1.645	116	7.8	190
		POST	.989	.836	25.2	1.555	106	10.6	160
	450	PRE	1.111	.971	37.0	1.897	138	9.3	198
		POST	1.170	1.001	29.5	1.545	118	11.8	190
	600	PRE	1.368	1.258	49.9	2.030	147	10.0	205
		POST	1.453	1.294	37.4	1.719	126	12.9	190
	750	PRE	1.650	1.596	67.1	2.156	159	11.2	220
		POST	1.747	1.648	47.7	1.763	133	13.8	198
	900	POST	2.022	2.038	58.6	2.048	149	15.1	215
	1050	POST	2.282	2.334	65.6	2.115	155	*******	215
	1200	POST	2.517	2.703	84.7	2.362	173	*******	225
	1350	POST	2.575	2.736	86.3	2.404	177	18.0	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTILATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
17	150								
		PRE	.589	.496	14.9	1.434	77	8.4	145
		POST	.719	.645	20.6	1.501	77	9.6	135
	300								
		PRE	.776	.692	20.4	1.579	89	9.2	160
		POST	.878	.809	25.1	1.760	88	10.8	150
	450								
		PRE	1.099	1.086	32.0	2.282	112	11.8	175
		POST	1.157	1.077	31.1	1.975	100	12.6	170
	600								
		PRE	1.375	1.424	43.4	2.588	126	12.7	195
		POST	1.475	1.433	42.1	2.553	117	14.0	175
	750								
		PRE	1.680	1.816	58.6	2.858	151	13.8	210
		POST	1.829	1.739	49.2	2.557	138	15.2	190
	900								
		POST	2.086	2.172	63.6	2.714	152	16.4	205
	1050								
		POST	2.343	2.528	85.1	2.709	165	*******	********
	1200								
		POST	2.401	2.626	96.4	2.732	168	18.1	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
18	150								
		PRE	.660	.561	18.4	1.077	95	7.0	150
		POST	.654	.570	18.0	1.002	83	8.4	140
	300								
		PRE	.816	.814	26.5	1.404	108	8.7	165
		POST	.790	.784	22.7	1.136	94	9.5	155
	450								
		PRE	1.073	1.122	37.8	1.575	127	10.0	195
		POST	1.136	1.098	32.3	1.371	107	*******	170
	600								
		PRE	1.378	1.551	58.0	1.974	145	11.3	205
		POST	1.382	1.366	40.2	1.731	119	12.8	195
	750								
		POST	1.667	1.733	54.2	1.816	127	13.4	210
	900								
		POST	1.909	2.168	77.9	2.115	144	*******	225
	1050								
		POST	1.965	2.272	88.5	2.136	147	15.3	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
19	150								
		PRE	.616	.569	18.7	.940	81	6.8	130
		POST	.576	.496	16.2	.934	69	7.4	100
	300								
		PRE	.828	.779	24.2	1.225	90	8.5	130
		POST	.789	.687	21.8	1.232	82	8.7	112
	450								
		PRE	1.122	1.172	35.6	1.596	111	10.6	160
		POST	1.034	.953	28.0	1.501	105	9.9	132
	600								
		PRE	1.388	1.551	49.8	2.106	142	11.8	200
		POST	1.314	1.382	40.8	1.713	121	11.9	142
	750								
		POST	1.598	1.776	54.8	2.229	138	*******	202
	900								
		POST	1.954	2.320	87.3	2.638	173	15.6	209

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
20	150								
		PRE	.661	.572	17.1	.951	115	8.6	180
		POST	.588	.548	17.6	1.048	107	8.9	170
	300								
		PRE	.891	.866	23.8	1.365	135	10.4	190
		POST	.904	.808	23.5	1.404	116	12.0	175
	450								
		PRE	1.132	1.153	30.3	1.673	142	12.2	198
		POST	1.137	1.044	29.9	1.635	123	13.3	182
	600								
		PRE	1.424	1.538	43.3	1.961	156	12.9	205
		POST	1.412	1.321	36.5	1.815	132	14.7	194
	750								
		PRE	1.792	2.088	78.9	1.724	165	12.6	215
		POST	1.697	1.653	45.8	2.066	147	15.5	205
	900								
		POST	1.948	2.006	62.1	2.050	153	14.6	205
	1050								
		POST	2.213	2.406	83.4	2.262	167	*******	220
	1200								
		POST	2.423	2.790	117.0	2.095	175	18.1	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

		TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
21	150	PRE	.623	.465	14.7	1.375	104	7.5	180
		POST	.688	.604	15.0	1.596	93	9.5	170
	300	PRE	.826	.699	21.2	1.529	117	8.4	185
		POST	.833	.784	18.7	2.198	99	10.3	190
	450	PRE	1.050	1.000	29.2	1.996	128	10.8	205
		POST	1.148	1.058	23.2	2.348	109	12.7	198
	600	PRE	1.326	1.303	40.4	2.122	152	11.8	220
		POST	*******	*******	26.1	*******	123	*******	********
	750	PRE	1.685	1.747	56.2	2.302	168	13.2	225
		POST	1.663	1.739	38.8	2.526	138	14.4	210
	900	POST	1.946	2.106	44.4	2.622	158	15.8	240
	1050	POST	2.163	2.452	52.8	2.719	181	*******	240
	1200	POST	2.223	2.524	57.1	2.606	190	17.3	********

PROGRESSIVE INCREMENTAL EXERCISE TEST DATA

FOR

EACH SUBJECT

	TEST	OXYGEN UPTAKE (L/MIN)	CO2 UPTAKE (L/MIN)	VENTI-LATION (L/MIN)	TIDAL VOLUME (ML)	HEART RATE (BEATS/ MIN)	CARDIAC OUTPUT (L/MIN)	SYSTOLIC BLOOD PRESSURE (MM HG)
22 150	PRE	.442	.375	15.6	1.042	87	5.9	170
	POST	.601	.513	17.1	.827	77	6.9	160
300	PRE	.665	.604	25.4	1.190	95	6.7	175
	POST	.808	.766	22.9	1.166	86	*******	160
450	PRE	.838	.814	34.2	1.410	122	8.1	175
	POST	1.067	1.042	29.7	1.411	104	10.8	170
600	PRE	1.088	1.118	49.2	1.614	153	8.7	195
	POST	1.360	1.408	40.0	1.897	126	11.9	180
750	PRE	1.309	1.474	72.3	2.152	163	9.9	215
	POST	1.598	1.713	51.4	2.068	144	12.9	190
900	POST	1.702	1.978	68.0	1.990	159	13.9	********

Appendix 17. Individual values before and after training in maximal peak power, maximal average power, total work, fatigue index and lactate during the 30 second maximal power output test. Values are listed at both 60 and 110 rpm.

MAXIMAL ISOKINETIC CYCLING DATA

FOR

EACH SUBJECT

60 RPM

SUBJECT NUMBER	PRE-TEST					POST-TEST				
	MAXIMUM PEAK POWER (WATTS)	MAXIMUM AVERAGE POWER (WATTS)	CUMU-LATIVE WORK (KJ)	FATIGUE INDEX (%)	LACTATE (MMOLES PER L)	MAXIMUM PEAK POWER (WATTS)	MAXIMUM AVERAGE POWER (WATTS)	CUMU-LATIVE WORK (KJ)	FATIGUE INDEX (%)	LACTATE (MMOLES PER L)
					20-30 YRS					
1	1055.2	652.2	16.148	34.2	13.74	1098.4	670.8	17.812	25.1	13.29
2	728.4	499.9	14.048	25.0	11.77	765.2	464.4	13.034	13.9	8.50
3	1295.6	821.3	20.560	27.1	11.39	1231.6	762.0	18.813	28.9	13.21
4	1077.6	664.3	17.435	26.8	9.27	1060.3	718.6	17.332	38.8	12.22
5	1130.7	701.7	17.785	25.2	12.68	1061.7	680.6	17.754	26.0	10.44
6	1074.8	638.9	16.157	29.3	13.29	1160.5	671.2	18.008	23.8	13.45
7	978.8	633.0	16.631	21.6	10.18	970.8	629.2	17.240	21.9	10.32
8	955.2	606.0	15.810	30.4	14.24	979.3	607.7	15.364	32.8	12.18
9	901.5	641.4	15.788	35.1	17.23	815.3	570.0	14.942	31.1	*******
10	1056.5	644.5	15.670	37.2	13.82	898.0	629.0	15.646	32.0	13.93
					60-70 YRS					
11	761.9	495.5	12.178	32.5	8.78	796.2	502.0	11.934	36.5	7.99
12	722.6	453.0	10.042	35.4	10.87	709.2	467.0	12.545	29.5	9.97
13	736.3	515.2	10.929	46.1	10.00	684.5	452.5	10.897	35.7	*******
14	614.1	397.5	10.364	22.4	11.48	783.8	456.1	11.487	19.4	9.73
15	780.3	460.9	12.680	24.6	8.17	808.5	534.4	13.810	25.6	6.41
16	702.6	487.6	12.241	35.6	10.08	700.3	495.3	12.133	30.7	7.83
17	551.1	381.1	10.466	15.6	3.54	614.4	381.2	10.755	11.1	5.02
18	715.1	431.2	10.350	36.8	10.95	756.2	472.4	11.150	32.7	7.52
19	949.0	530.5	11.819	49.9	*******	683.8	462.1	9.971	40.9	*******
20	810.2	497.8	11.050	41.2	10.32	657.9	412.7	10.429	26.1	7.12
21	858.5	551.8	14.603	29.2	9.25	1003.9	625.1	15.277	37.1	7.67
22	714.0	423.3	9.641	42.0	6.03	756.0	477.1	10.442	43.3	6.12

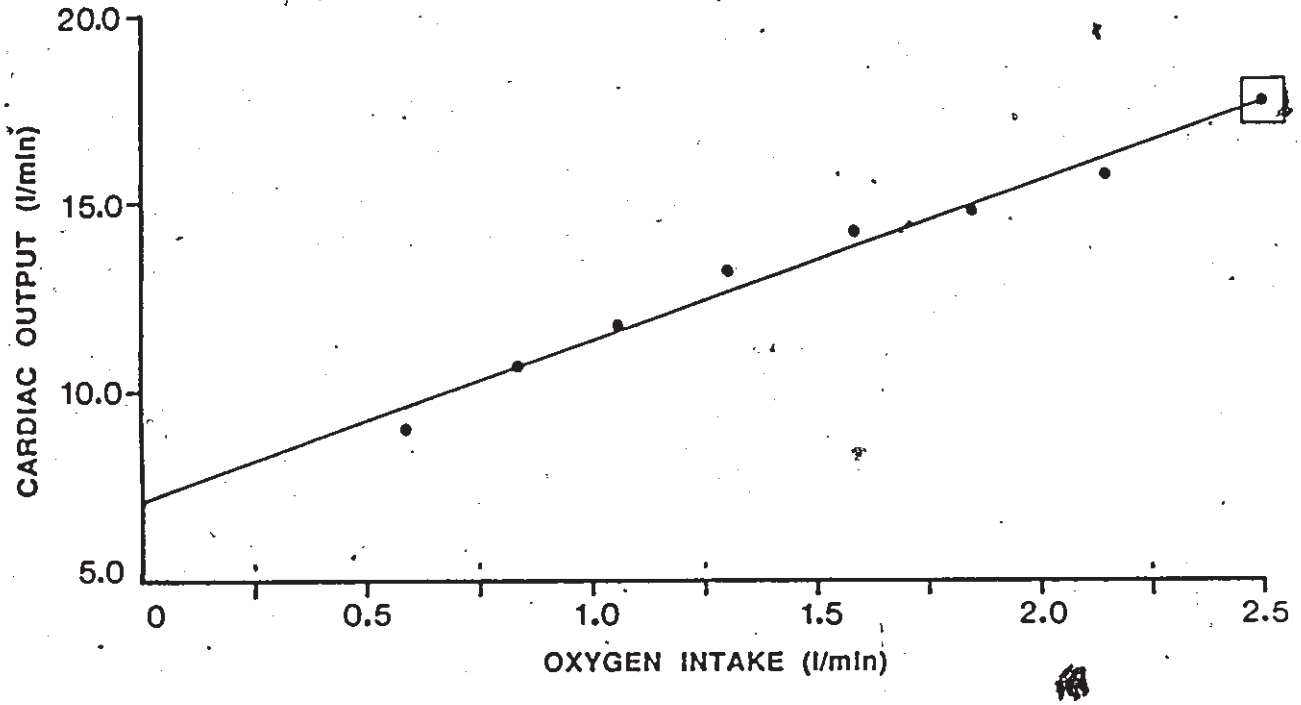
MAXIMAL ISOKINETIC CYCLING DATA

FOR

EACH SUBJECT

110 RPM

SUBJECT NUMBER	PRE-TEST MAXIMUM PEAK POWER (WATTS)	PRE-TEST MAXIMUM AVERAGE POWER (WATTS)	PRE-TEST CUMULATIVE WORK (KJ)	PRE-TEST FATIGUE INDEX (%)	PRE-TEST LACTATE (MMOLES PER L)	POST-TEST MAXIMUM PEAK POWER (WATTS)	POST-TEST MAXIMUM AVERAGE POWER (WATTS)	POST-TEST CUMULATIVE WORK (KJ)	POST-TEST FATIGUE INDEX (%)	POST-TEST LACTATE (MMOLES PER L)
					20-30 YRS					
1	1489.6	853.6	14.419	67.6	13.64	1610.9	939.5	18.342	60.7	15.51
2	1082.9	777.2	14.799	85.3	10.83	990.5	580.2	13.090	52.6	8.89
3	1735.9	1049.5	21.496	54.9	15.03	1896.1	1095.4	21.682	57.7	11.87
4	1496.0	930.3	15.704	63.6	15.68	1285.2	823.0	15.650	62.3	8.74
5	1695.5	1059.0	17.065	70.1	17.08	1638.2	1114.8	20.361	64.7	13.05
6	1380.2	763.5	14.107	57.8	11.85	1506.2	902.3	17.952	58.1	12.06
7	1359.0	802.5	16.156	57.2	15.15	1360.3	801.6	16.533	57.2	13.32
8	1254.1	749.9	13.663	60.0	12.15	1130.6	721.4	14.305	47.7	*******
9	1179.6	868.2	15.379	62.2	18.72	1100.6	811.9	15.774	60.2	13.93
10	1264.1	834.4	13.878	69.2	10.83	1153.5	752.1	14.835	59.5	11.79
					60-70 YRS					
11	1078.7	746.6	12.126	66.4	12.62	1091.6	690.8	12.288	59.0	6.99
12	1064.4	657.6	9.865	67.4	11.71	1054.3	675.1	11.839	63.9	*******
13	810.9	581.4	9.727	60.1	13.33	717.9	551.4	10.593	51.7	10.08
14	785.2	470.9	10.992	46.9	9.21	823.4	516.8	11.861	48.6	9.13
15	1175.1	718.9	12.188	62.7	9.97	1076.1	678.1	13.550	67.5	9.49
16	953.2	600.8	11.275	62.1	13.09	986.9	746.7	15.709	49.4	7.49
17	807.7	491.0	10.426	43.8	8.57	853.1	527.3	13.068	33.3	5.02
18	1063.6	858.5	12.687	67.5	7.86	1039.9	678.4	12.008	59.7	8.42
19	948.2	625.1	10.978	61.9	*******	950.7	660.5	12.449	58.0	9.21
20	860.4	502.4	9.812	47.9	11.90	814.6	515.1	11.748	40.8	*******
21	1171.2	689.4	14.152	57.7	8.49	1245.8	793.7	15.604	58.2	7.12
22	886.8	560.3	9.799	63.4	8.22	957.3	609.0	10.129	60.3	5.10



Appendix 18. Example of extrapalation of cardiac output at peak exercise by using linear regression. Extrapolated value at peak exercise indicated by large square.

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