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NORTH LONDON POLY.

MAXIMAL SHORT-TERM POWER OUTPUT

FROM HUMAN MUSCLE

by

PATRICIA DOLAN

A thesis submitted in partial fulfilment of the requirements for
the Degree of Doctor of Philosophy of the
Council for National Academic Awards

The Polytechnic of North London

May 1985

MAXIMAL SHORT-TERM POWER OUTPUT FROM HUMAN MUSCLE
by
PATRICIA DOLAN

Maximal short-term power output was determined in adults and children and its variability under different physiological conditions was examined.

Measurements were made using an isokinetic cycling technique which enabled power output to be determined at different pedalling speeds during brief intense exercise.

Maximal power was attained at a velocity of 118 ± 17 rev. min^{-1} and in adults was positively correlated to the percent type II fibre area. When standardised for muscle size maximal power was significantly greater in adults than children which may reflect a greater contribution from type II fibres to the muscle cross-sectional area in adults.

Measurement of maximal power following prior submaximal exercise revealed an increasing decrement as a result of increasing either the prior exercise duration up to 6 minutes or the intensity beyond 60% VO_2max while lower work levels produced an improvement. Recovery of maximal power following heavy submaximal exercise was very rapid and appeared to reflect the pattern of phosphorylcreatine resynthesis. This recovery occurred within one minute although extending the recovery period produced further improvements in performance suggesting a warming-up effect of the previous exercise.

The effects of both active and passive warm-up were examined in later experiments where results indicated an improvement in maximal power of ~2.5% for each $^{\circ}\text{C}$ rise in muscle temperature. This increase was similar independent of the method of warm-up indicating that the beneficial effect of active warm-up was almost entirely attributable to the elevated muscle temperature.

In the final part of this thesis where maximal power was measured following prolonged concentric and eccentric exercise the latter produced a considerable decrement which persisted for several days. This loss of power could not be explained by a depletion of metabolic substrate and was attributed to muscle damage as a result of the high forces generated during eccentric contractions.

ACKNOWLEDGEMENTS

I should like to take this opportunity to express my gratitude to Professor R. Goldsmith, my external supervisor, and Dr. David Jones for their advice and encouragement during the preparation of this thesis. I should also like to thank Carolyn Greig and Zehra Jax for their assistance and continued enthusiasm.

My thanks are also given to the Polytechnic of North London who provided the facilities and my salary, and to the staff of the Department of Human Metabolism, University College Hospital Medical School for their help with the biochemical analyses.

To all the subjects who took part in the experiments I am most grateful; without them this work would not have been possible.

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APPENDIX VII

Reprint of: Dolan, P., Greig, C. and Sargeant, A.J. (1985) Muscular weakness following prolonged eccentric and concentric exercise in man. J. Physiol. In Press

CHAPTER 1

Introduction

Introduction

The current preoccupation with physical fitness in this country has led to a marked increase in the number of people who now participate in some form of regular exercise. In the majority of cases such exercise is aimed at improving the individual's aerobic fitness rather than their short-term performance capacity. Nevertheless many everyday activities may depend more upon the ability to perform brief bursts of strenuous exercise than on the capacity to sustain prolonged periods of aerobic work. Despite this fact man's capacity to generate power during maximal exercise of short duration has until recently been little investigated.

Such neglect may be related at least in part to methodological problems. Previous investigations have shown that the maximum force which a muscle may exert decreases with increasing speed of contraction (Fenn and Marsh, 1935; Wilkie, 1950). As a mathematical consequence of this the power which can be generated by the muscle initially increases with the speed of shortening to reach a maximum value at some intermediate or 'optimum' velocity. If the speed of contraction is increased beyond this point then power output is reduced. Thus maximum power is only achieved when the muscle operates at its optimum velocity for power output as defined by its force-velocity relationship. To measure maximal power under circumstances where the speed of contraction is not controlled it is therefore essential to match the load to the capacity of the active muscles to ensure that they operate at optimum velocity.

In earlier studies maximal short-term power output from human

muscle has been measured during both stair-climbing (Margaria, Aghemo and Rovelli, 1966) and cycling (Asmussen and Boje, 1945 ; Bar-Or, Dotan and Inbar, 1977). Other workers have used force platforms to measure the force exerted and hence the power generated during standing jumps (Davies and Young, 1983). However in such freely accelerating efforts the contracting muscles may be operating at their optimum velocity for only a very brief period of time and maximal power may not be achieved.

In order to overcome this problem Sargeant (1980) devised a method for measuring short-term power output of human muscle whereby the velocity during a brief maximal cycling exercise could be held constant despite the efforts of the subject to increase it. Force exerted and power generated throughout the test could therefore be measured under isokinetic conditions enabling maximal power at a given velocity to be determined. The absence of an acceleration phase at the start of the test meant that subjects were able to attain maximal power very rapidly before lack of substrate would be likely to limit performance. By repeating the testing procedure over a range of pedalling speeds the force-velocity and power- velocity relationships during the cycling exercise could be determined and the optimum velocity for power output identified .

In the present thesis this technique has been employed to examine the force-velocity relationship of human muscle in vivo in order to determine maximal short-term power output. The influence of physical factors such as age, sex, muscle size and fibre type composition upon this relationship is examined. In addition the effects of physiological changes within the muscle upon subsequent

short-term power output are investigated.

The next part of this introductory chapter reviews the work which has given rise to our current understanding of muscle metabolism and upon which the studies carried out in the present thesis were founded.

Muscle Metabolism During Short-term Exercise

During strenuous exercise lasting less than a minute man relies almost entirely upon anaerobic processes to provide the energy for muscular contraction. It was suggested by Margaria (1968) that the power developed during the first few seconds of such activity would depend solely upon the splitting of high-energy phosphate (adenosine triphosphate (ATP) and phosphorylcreatine (PC)) within the active muscle fibres. For a long time however the importance of ATP and PC in the contractile process went unrecognised and it was generally believed that the initial anaerobic event at the onset of exercise was the breakdown of glycogen to lactic acid which was therefore assumed to power muscular contraction.

This, belief which was widespread in the early 1920's, came about mainly as a result of a number of investigations independently carried out by Meyerhof (1920 a;b;c; 1921 a,b; 1922) and by Hill and his associates (Hill and Lupton, 1923; Hill, Long and Lupton, 1924 a,b; Furusawa, Hill, Long and Lupton, 1924). In the course of their experiments these workers demonstrated that the breakdown of glycogen during muscular contraction was accompanied by an equivalent production of lactate and that the latter disappeared at the end of exercise following a similar time course to the decline in oxygen uptake.

The theory which eventually grew from these findings appeared to explain virtually all the metabolic events which accompanied muscle contraction. However despite the initial widespread acceptance that this work received several observations which were not compatible with its proposals were made over the next few years .

In the early 1930's Margaria, Edwards and Dill (1933) found that the removal of lactate following exercise did not in fact coincide with the decline in oxygen consumption and that there was after work of all intensities an oxygen debt that was unrelated to the production of lactic acid. It was also shown by Lundsgaard (1930 a;b) that muscles poisoned with iodoacetate, an inhibitor of glycolysis, were able to contract without the formation of lactate but with an accompanying reduction in PC. These findings demonstrated that the development of tension was not a direct consequence of lactate production and that the splitting of PC represented a more immediate source of energy for muscular contraction than the anaerobic breakdown of glycogen.

Not long after Lundsgaard's important experiments, Lohmann in 1931 discovered another energy-rich compound in muscle which he identified as ATP. He was able to demonstrate that one of its breakdown products, namely adenosine diphosphate (ADP), was a co-factor required for the splitting of PC (Lohmann,1934). The implications of this finding were evident; in order for PC to deliver energy to the contracting muscle ATP had first to be split into ADP and inorganic phosphate indicating that ATP rather than PC constituted the initial source of energy for muscular contraction.

Over the next few years immense progress was made in the understanding of muscle metabolism and in the field of biochemistry as a whole. The glycolytic pathway and the reactions involved in oxidative phosphorylation were elucidated and again ATP was found to be the key substrate responsible for the transfer of energy. It was also confirmed in the course of these investigations that PC had no independent pathway for delivering energy to the contraction process and that it could act only via the transfer of phosphate to ADP.

With the re-introduction of the needle biopsy technique by Bergstrom in 1962 and the development of more refined biochemical procedures it became possible to study in man many features of skeletal muscle which had previously only been demonstrated in minced muscle or animal muscle preparations. These new advances allowed certain metabolites such as lactate, ATP and PC to be determined in human muscle with relatively little or no discomfort to the subject and consequently opened new avenues of investigation to the muscle physiologist.

In 1967 Hultman, Bergstrom and McLennan-Anderson employed these techniques to follow levels of ATP and PC in human muscle during various stages of exercise and recovery. In the course of their investigation these workers demonstrated a rapid initial reduction of both PC and ATP in the working muscles during the first two minutes of exercise and in the case of PC found the extent of this depletion to be related to the previous work load. At all exercise intensities they observed a more marked decline in PC than in ATP and found that even at the highest work loads when PC levels had fallen to practically zero, ATP concentrations were only reduced to about 60% of their

initial value, indicating that a large store of ATP remained intact.

During recovery the subsequent resynthesis of ATP and PC proved to be very rapid being virtually complete in the majority of cases within a few minutes of terminating work. Using this data a half-time of 30-40 seconds could be calculated for the resynthesis of high-energy phosphate in man. This corresponded remarkably well with the value of 25-30 seconds determined initially by Margaria et al (1933) and later by a number of other workers (De Moor, 1954; Henry and De Moor, 1956; Margaria, Mangili, Cuttica and Cerretelli, 1965; Di Prampero, Davies, Cerretelli and Margaria, 1970; Knuttgen, 1970) as the half-time for the repayment of the alactic oxygen debt.

Around this time many other workers reported marked reductions in muscle PC accompanied by lesser changes in ATP as a result of physical exercise in man. Karlsson and Saltin (1970) showed that not only was depletion maximal for a given work load after only two minutes of work but that there was also no further depletion as a result of increasing the intensity of exercise to work loads above 100% of the individual's maximal oxygen uptake ($\dot{V}O_2\text{max}$).

In a later investigation Karlsson, Diamant and Saltin (1971) followed muscle concentrations of PC and ATP at exercise intensities ranging from 60-100% of the $\dot{V}O_2\text{max}$ of their subjects and found the extent of high-energy phosphate depletion to be almost linearly related to the relative work load in keeping with the original findings of Hultman et al (1967). However when Knuttgen and Saltin (1972) examined high-energy phosphate concentrations over an even

wider range of submaximal work loads they found that depletion was much more marked at the higher intensities than at those below 60% $\dot{V}O_2$ max where reductions in both PC and ATP were relatively slight.

In general then it appeared that for a given work load depletion of the high-energy phosphate store was maximal after only 2 minutes of exercise and that the extent of this depletion was linearly related to the previous work load where this was between 60-100% $\dot{V}O_2$ max.

On the basis of these findings Margaria et al (1971) attempted to demonstrate a relationship between high-energy phosphate depletion and maximal power output in man. These workers examined the effect of performing different levels and durations of submaximal exercise prior to the performance of a short stair-climbing test devised to measure maximal anaerobic power.

The maximal power attained in the test was shown to be inversely related to the intensity of the preceding exercise although it was not affected by increasing the duration of this exercise beyond three minutes. The loss in power output following submaximal exercise therefore appeared to reflect the pattern of high-energy phosphate depletion found in earlier investigations.

When Margaria and his associates calculated the energy derived during the test from the splitting of high-energy phosphate, over and above that involved in the previous steady-state exercise, they found that this decreased in a linear fashion with the intensity of prior exercise. The energy provided via glycolysis however remained the same and was therefore unaffected by the previous work load. These findings led Margaria and his associates to conclude that the loss in power

output following steady-state exercise was due to a depletion of high-energy phosphate within the active muscle fibres.

The existence of such a relationship between the prevailing high-energy phosphate concentration and maximal short-term power output suggests that measurement of the latter might provide a functional index of the available energy capacity. According to Margaria's findings any change in the amount of unsplit high-energy phosphate ought to be reflected in the power output measurement. Hence the recovery in maximal power output following exercise should be related to the resynthesis of high-energy phosphate in the fatigued muscle.

In a number of investigations direct measurements of high-energy phosphate resynthesis following exercise have been made in human muscle although in such studies there seems to be no general agreement regarding the rate at which this process occurs. Several studies (Karlsson, Bonde-Petersen, Henriksson and Knuttgen, 1975; Karlsson, Funderburk, Essen and Lind, 1975) reported rates similar to those originally observed by Hultman et al (1967) while others (Karlsson and Saltin, 1971; Saltin and Essen, 1971) obtained results which indicated a much slower rate of resynthesis.

It was suggested by Harris, Edwards, Hultman and Nordesjo (1976) that the conditions of the preceding exercise might have some influence upon the subsequent rate of high-energy phosphate resynthesis. In order to investigate this they followed the rate of PC resynthesis in the human quadriceps muscle during recovery from both isometric and dynamic exercise.

Like earlier workers Harris and his associates found that the

reduction in ATP following both types of exercise was only slight and in their experiments accounted at most for only 5% of the total decrease in high-energy phosphate. PC depletion in contrast was very marked with post-exercise levels falling as low as 15% of their initial value.

During recovery they found that resynthesis of PC occurred very rapidly in the initial stages although it was not complete until approximately 20 minutes after terminating exercise. This process appeared to proceed exponentially with respect to time, a finding which was consistent with earlier observations made on both human (Hultman et al, 1967) and animal (Sacks and Sacks, 1935; Di Prampero and Margaria, 1969; Piiper and Spiller, 1970) muscle. However despite this observation Harris and his colleagues were unable to derive a single exponential equation to fit their data which appeared instead to describe a biphasic process. They therefore assumed that PC resynthesis occurred in two stages consisting of a fast and a slow recovery component.

Half-times for the fast component were estimated at 21 and 22.5 seconds following dynamic and isometric exercise respectively, while for the slow component a $t_{1/2}$ of more than 170 seconds was calculated for both types of exercise.

The authors pointed out that if they were correct in their analysis of the data then treatment of PC resynthesis as a single exponential process would result in the half-time being incorrectly estimated to lie somewhere between the values which they had calculated for the two components of the biphasic model. The actual

half-times obtained would therefore depend upon the recovery period over which resynthesis was followed. The authors suggested that this explanation might account for the widely differing half-times previously obtained to describe the resynthesis of PC in human muscle.

Harris et al (1976) also noted in the course of their investigation that the pattern of PC resynthesis which they had observed in the quadriceps muscle was very similar to that of strength recovery following exhaustive exercise in man (Clarke, 1962; Clarke and Stull, 1969; Stull and Clarke, 1971; Edwards, Nordesjo, Koh, Harris and Hultman, 1971). Both exhibited fast and slow recovery components which followed approximately the same time course and both appeared to proceed more slowly following isometric as opposed to dynamic exercise. Furthermore occlusion of the circulation to the fatigued muscle during recovery which had previously been shown to inhibit strength recovery (Edwards et al, 1971) was also found by Harris et al (1976) to completely abolish the resynthesis of PC. These findings suggest that recovery in isometric force may be related to the resynthesis of PC or that both processes are governed by some other factor such as the recovery in intramuscular pH.

Factors Influencing Maximal Short-term Power Output

From the above discussion it would appear that the capacity to exert force and generate power during maximal exercise of a few seconds duration is related to the level of unsplit high-energy phosphate in the active muscles. Nevertheless there is little functional data in the literature to support this assumption which is based mainly on the work of Margaria et al (1971) who found that the

decrement in power output following different levels of prior exercise reflected the pattern of high-energy phosphate depletion (cf. Hultman et al, 1967; Karlsson et al, 1971).

Another effect of prior exercise which Margaria et al did not discuss was its influence upon muscle temperature. A number of workers have reported considerable improvements in performance during maximal exercise of brief duration as a result of previous warming-up (Asmussen and Boje, 1945; Bergh and Ekblom, 1979; Sargeant, 1981). Thus the effect of prior exercise of a few minutes duration upon subsequent short-term power output may be twofold with the increase in muscle temperature tending to counteract the effect of high-energy phosphate depletion. Which of the two effects predominates may be dependent upon the prior exercise intensity.

Following prior exercise of longer duration the situation is somewhat different. In a recent investigation Edwards, Hill, Jones and Merton (1977) examined the effect of prolonged exercise upon maximal isometric force of the quadriceps muscle. They found that such exercise, especially where it has a large eccentric component, produces a long-lasting decrement in force which persists for several hours or even days and cannot therefore be attributed to a depletion of high-energy phosphate.

Edwards, Mills and Newham (1981) showed that this type of fatigue occurred primarily at low frequencies of stimulation and that it was still marked even when muscle metabolites were restored to pre-exercise levels. It was suggested by Jones (1981) that this relative loss of force at low frequencies of stimulation might be due to either

a reduced release of calcium from the sarcoplasmic reticulum for each excitatory action potential or a change in the affinity of troponin to bind calcium.

The former might occur if calcium homeostasis in the muscle fibres was upset and it was suggested by Wrogeman and Pena (1976) that such a situation could arise as a result of damage to the membrane of the sarcoplasmic reticulum.

Evidence of structural disturbances in the myofibrillar band pattern following prolonged exercise has since been found in biopsy samples taken from both animal (Armstrong, Ogilvie and Schwane, 1983) and human (Newham, McPhail, Mills and Edwards, 1982; 1983) muscle and it is now believed that such mechanical damage to the sarcomere structure might be responsible for the delayed recovery in isometric force.

Whether the muscles ability to generate force during powerful dynamic contractions is effected in the same way as isometric force by prolonged exercise is not known. The effect of prolonged exercise upon maximal performance capacity has been little investigated. It may be that the effect of low frequency fatigue depends to a large extent upon the physiological stimulation frequencies employed to produce the contraction in vivo.

Finally, this discussion would not be complete without considering the effects of muscle size and fibre type composition upon the capacity for power output. Studies on animal muscle suggest that type II fibres are associated with greater force development than type I fibres (Barany and Close, 1971; Burke and Tsairis, 1973). However

data regarding the effects of muscle fibre type distribution on maximal force development in human muscle are somewhat conflicting. Nygaard et al (1981) found no relationship between maximal isometric force and muscle fibre composition although they found that the type II fibres produced relatively more force than the type I fibres during dynamic contractions. However in a more recent study Schantz et al (1983) measured maximal voluntary strength in man at a number of angular velocities and found that maximal force when standardised for the cross-sectional area did not show any significant correlation to muscle fibre type composition at the speeds studied. It may be that differences in maximal force between the two main fibre types depend to a large extent upon the contraction speeds studied. Further studies are therefore needed in order to explain the above discrepancies.

Aims of the Investigation.

The investigations described in this thesis sought to assess maximal power output from human muscle under various physiological conditions. In the first part of the thesis the force-velocity and power-velocity characteristics of human muscle during isokinetic cycling were determined to enable the optimum velocity for power output to be identified. The influence of physical characteristics such as age, sex, muscle size and fibre type composition upon these relationships was determined.

In subsequent parts of the thesis the effect of varying the metabolic condition within the muscle upon subsequent short-term power output was investigated. This was effected by prior exercise, both short-term and long-term, and by active and passive warm-up.

CHAPTER 2

Force-Velocity Relationship and
Maximal Short-Term Power Output
of Human Muscle.

INTRODUCTION

The relationship between maximal force development and contraction velocity was first investigated by Hill in 1922 who found that the work done or force exerted by human muscle was inversely and linearly related to its speed of shortening. In contrast to these early findings however Gasser and Hill (1924) and Fenn and Marsh (1935) in their studies on isolated muscle repeatedly observed an exponential relationship between force and contraction velocity. Fenn and Marsh suggested that the linear relationship observed in human muscle by Hill was due to the force exerted in vivo being modified by nervous inputs. It should be pointed out however that the forces measured in vitro are the result of a single muscle contracting at a certain velocity. In vivo the situation is more complex since even the most simple of movements may involve more than one muscle and often movement across more than one joint.

In 1950 Wilkie (4) conducted an experiment on the human elbow flexors which involved movement across only a single joint and he found an almost identical force-velocity relationship in his in vivo experiments to that previously determined for isolated muscle (Fenn and Marsh, 1935; Hill, 1938). As a consequence of this relationship power output increased initially with the speed of contraction to reach a maximum value at some intermediate or 'optimum' velocity between zero and the muscles maximum speed of shortening. In a later investigation Binkhorst, Hoofd and Vissers (1977) obtained similar results for the handgrip muscles (Fig.1). These results underlined the importance when assessing muscle's maximal capacity for power

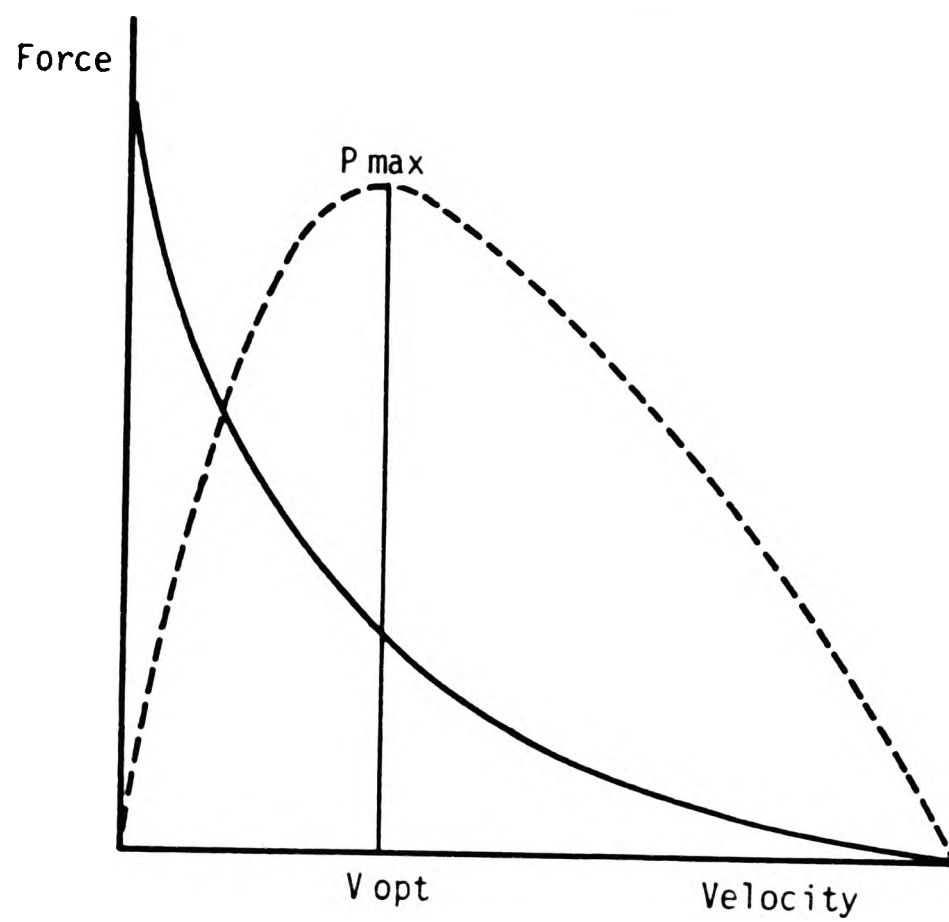


Fig.1. The force developed and power generated by the handgrip muscles at different contraction velocities (from Binkhorst, Hoofd and Vissers, 1977).

output of controlling either the load or the speed of contraction to ensure that the muscle operates at its optimum velocity for power output as defined by its force-velocity relationship.

In previous studies maximal power output from human muscle has been measured during various forms of exercise including stair-climbing (Margaria et al, 1966), cycling (Asmussen and Boje, 1945; Bar-Or et al, 1977) and standing jumps performed on a force platform (Davies and Young, 1983). However in such freely accelerating sprint efforts the muscle works at its optimum velocity for only a very short period of time making a measurement of maximal power output very difficult.

More recently the force-velocity and power-velocity relationships of human muscle have been investigated using isokinetic devices such as the Cybex dynamometer (Thorstensson, Grimby and Karlsson, 1976; Coyle, Costill and Lesmes, 1979). However such apparatus is limited in its application since maximal torque can only be reliably measured at low velocities below 25% of the maximum voluntary contraction velocity.

The development of the isokinetic ergometer referred to in the previous chapter (Sargeant, Hoinville and Young, 1981) made it possible to measure maximum force and power output under constant velocity conditions over a wide range of pedalling speeds. It was the aim of the present study, using this technique, to establish the force-velocity and power-velocity relationships of human muscle during cycling in order to determine maximal short-term power output. The extent to which these relationships were influenced by age, sex and

muscle size was investigated. In addition the fibre type composition of the quadriceps muscle was determined by needle biopsy in a subgroup of the adult subjects. This enabled the effect of muscle fibre type upon the capacity for maximal short-term power output to be determined.

SUBJECTS

40 adults (16 females and 24 males) together with 25 male children (mean age:13.7 years) were studied. Informed consent was given by all subjects who participated in the investigation and in the case of the children this was also obtained from the school authorities and the children's parents.

Physical characteristics of the subjects including measurements of lean body mass (LBM) upper leg muscle (plus bone) volume (ULV) and maximal oxygen uptake ($\dot{V}O_2\text{max}$) are shown in Tables Ia-Ic.

In a subgroup of the adult subjects (n=22) muscle fibre type was also determined by needle biopsy and values shown represent the percent cross-sectional area of Type II fibres expressed in terms of the total cross-sectional fibre area.

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Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_{2\max}$ l min ⁻¹	ULV litres	% CSA type II
MJ	25	185.5	81.4	67.5	-	5.15	50
MT	28	178.6	71.1	64.3	4.72	4.60	4
MW	28	184.5	63.1	55.9	-	4.45	32
TR	31	178.2	68.4	60.3	-	4.66	39
AS	33	179.0	71.6	62.0	3.20	5.49	52
JJ	22	170.4	59.6	53.8	3.40	4.34	49
AY	30	176.0	81.0	69.9	4.41	5.61	45
EZ	28	172.4	75.0	62.0	3.90	4.81	56
TW	25	178.4	58.7	53.4	-	4.06	25
ME	22	179.9	71.3	64.2	-	4.64	10
BS	25	176.5	75.0	67.8	4.20	5.12	65
LM	26	168.4	78.4	64.3	3.61	4.97	57
JO	25	183.8	74.2	66.6	4.20	4.76	61
VW	23	184.0	77.5	73.0	3.61	5.40	
AT	42	170.8	73.8	66.0	3.90	4.91	
KD	22	169.3	66.3	58.2	3.76	3.39	
SM	21	178.6	84.5	72.1	4.81	5.66	
PJ	26	162.8	66.7	50.7	3.20	3.20	
MN	27	170.3	59.2	53.9	3.40	3.91	
JS	21	169.2	64.0	54.6	3.39	4.34	
JR	26	181.1	70.2	58.9	-	4.02	
RC	23	182.4	68.5	56.2	-	4.63	
RA	31	169.8	65.2	58.0	3.57	4.62	
DM	30	175.7	74.5	60.4	3.30	4.22	

Table 1a Physical characteristics of male adults

Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_{2max}$ l min ⁻¹	ULV litres	% CSA type II
LP	22	155.0	46.1	30.9	1.87	2.22	55
LT	26	166.1	58.8	47.0	-	3.02	16
VS	31	167.4	60.2	48.3	-	3.48	32
WD	29	167.5	60.6	46.9	-	3.14	9
CG	21	166.9	50.7	39.8	2.41	2.58	55
NI	27	159.3	55.9	39.4	2.61	2.86	45
DN	32	177.4	69.1	50.1		4.07	44
OR	22	160.7	67.4	47.4	2.38	2.89	58
JA	24	173.7	68.7	50.2	3.04	3.94	49
PD	27	164.6	56.6	43.2	2.63	2.55	-
FC	25	165.5	60.7	44.3	2.17	3.85	-
KD	22	150.8	49.7	36.5	-	2.19	-
NR	24	153.9	52.5	38.3	-	2.94	-
RR	27	164.9	61.1		-	2.84	-
JS	45	166.2	55.0	44.2	3.23	3.17	-
KB	24	162.7	49.0	39.9	3.09	3.31	-

Table 1b Physical characteristics of female adults

Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_2$ max l min ⁻¹	ULV litres
LC	14	172.0	58.6	49.3	2.93	4.43
GC	14	149.0	49.1	39.6	-	3.03
VD	13	155.5	47.1	37.9	2.12	3.35
MG	13	145.5	45.6	34.9	-	3.62
DM	14	164.5	48.2	42.4	-	3.12
MT	13	165.5	46.8	41.4	2.21	3.02
MB	14	144.0	40.3	33.6	1.99	2.63
AC	13	164.0	53.6	43.7	3.06	3.45
JE	14	156.2	45.5	39.3	2.38	2.82
RF	14	164.0	48.4	39.1	2.16	3.12
PA	14	163.0	59.2	50.3	2.62	4.51
FA	14	173.5	48.1	41.8	2.59	3.61
PF	13	168.8	65.9	52.9	-	4.21
KK	14	164.0	55.8	45.6	-	4.64
MO	14	174.8	55.6	47.6	2.64	4.76
TM	14	161.7	54.8	43.7	-	4.46
CV	14	151.8	39.1	33.9	-	2.88
TH	14	162.5	48.8	40.8	-	4.12
PK	13	137.5	31.1	26.8	1.94	2.11
FK	14	180.0	59.7	52.7	-	4.49
SM	14	161.0	42.1	36.1	2.8	3.12
JO	14	175.0	52.9	44.8	2.96	4.06
PP	13	162.1	49.7	43.5	2.72	3.29
RS	13	173.4	58.8	47.7	2.99	3.57
DC	14	162.5	66.3	47.9	3.02	3.49

Table 1c Physical characteristics of male children

METHODS

Force exerted and power generated were measured during the performance of a short (20 sec) maximal test which was carried out on a specially modified cycle ergometer (Sargeant, Hoinville and Young, 1981). This ergometer was essentially isokinetic due to the addition of a 3-horse power electric motor which was employed to drive the cranks at constant velocity . The inclusion of a variable-speed gear box in this arrangement enabled the velocity to be adjusted to give pedalling speeds in the range 23-180 revolutions per minute (Fig. 2). Subjects were required to perform the maximal test at 4 or more velocities. This enabled the force-velocity and power-velocity characteristics of the muscles employed during exercise to be assessed.

Force and Power Measurements.

In order to determine the power generated during the cycling test force exerted on the pedals was continuously monitored by silicon strain gauges bonded approximately midway along the trailing and leading edges of each crank. Output from these gauges was relayed to brass slip-rings which were mounted on discs attached inside each crank so that disc and crank rotated simultaneously (Fig.3). Phosphorbronze metal pick-ups in contact with the slip-rings relayed the output from each crank via separate Wheatstone bridge circuits to an ultraviolet oscillograph recorder (S.E. Laboratories Ltd. - Type 3006). In this way force exerted was recorded simultaneously though independently for each leg on the same trace as shown in Fig.4.

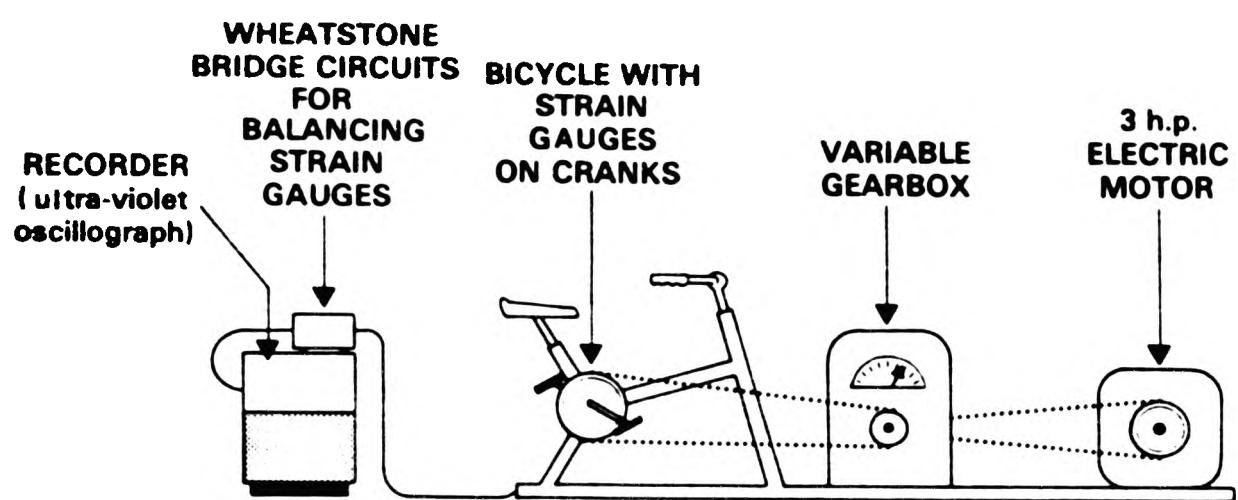


Fig.2. Schematic diagram of the isokinetic ergometer and recording apparatus.

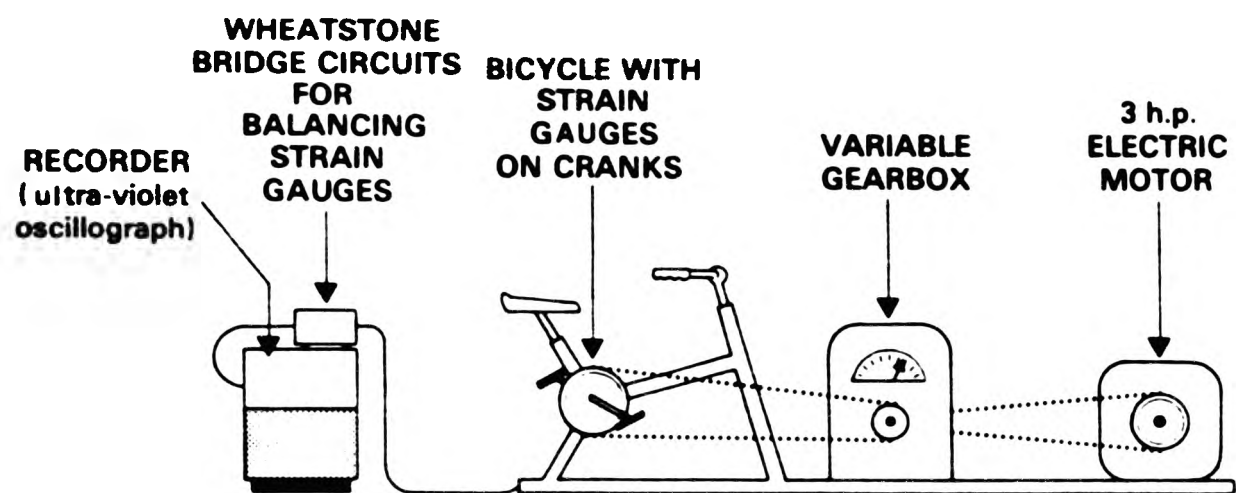


Fig.2. Schematic diagram of the isokinetic ergometer and recording apparatus.

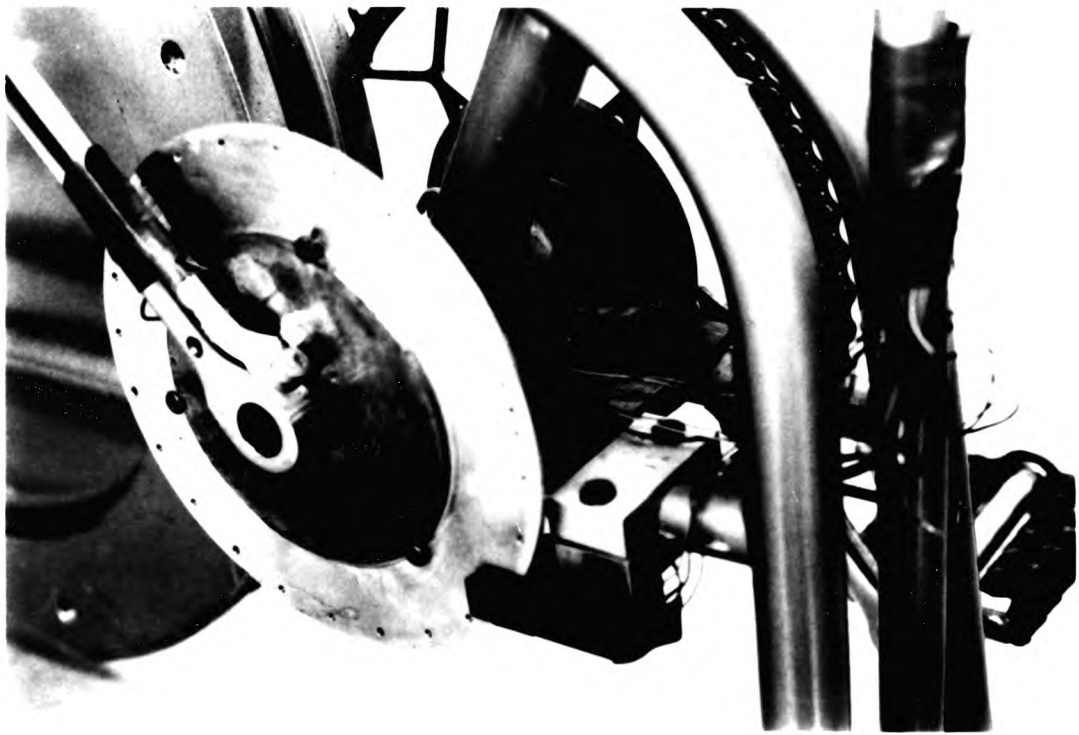


Fig.3. The force-measuring apparatus attached between the cranks of the cycle ergometer.

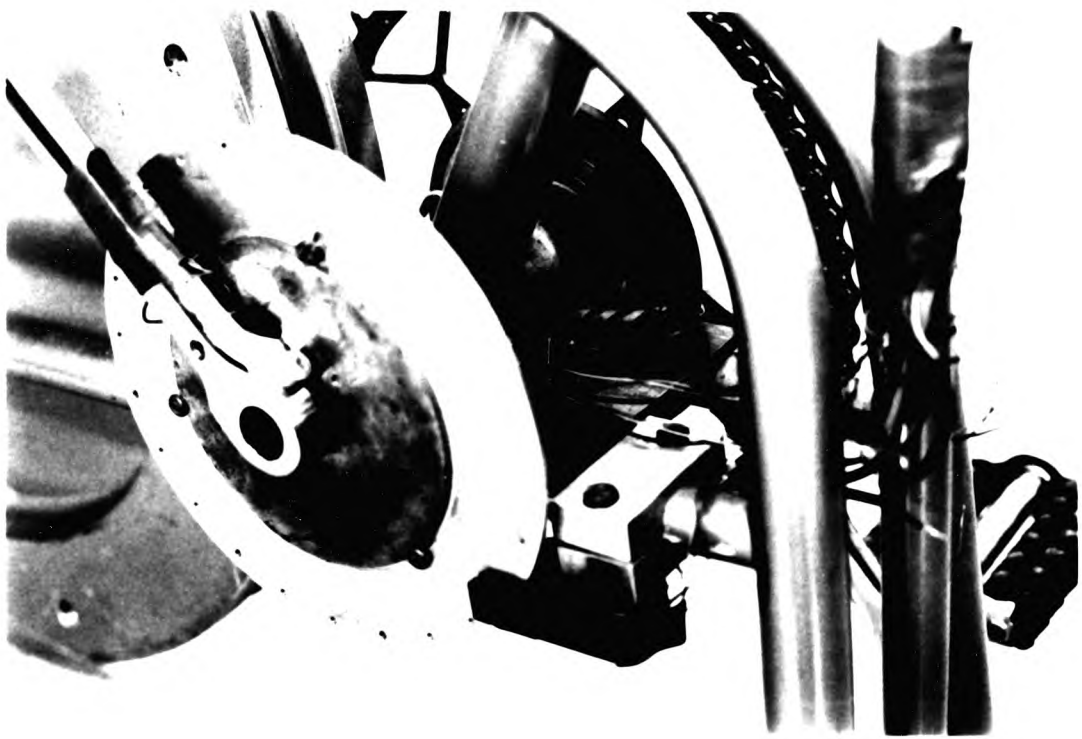


Fig.3. The force-measuring apparatus attached between the cranks of the cycle ergometer.

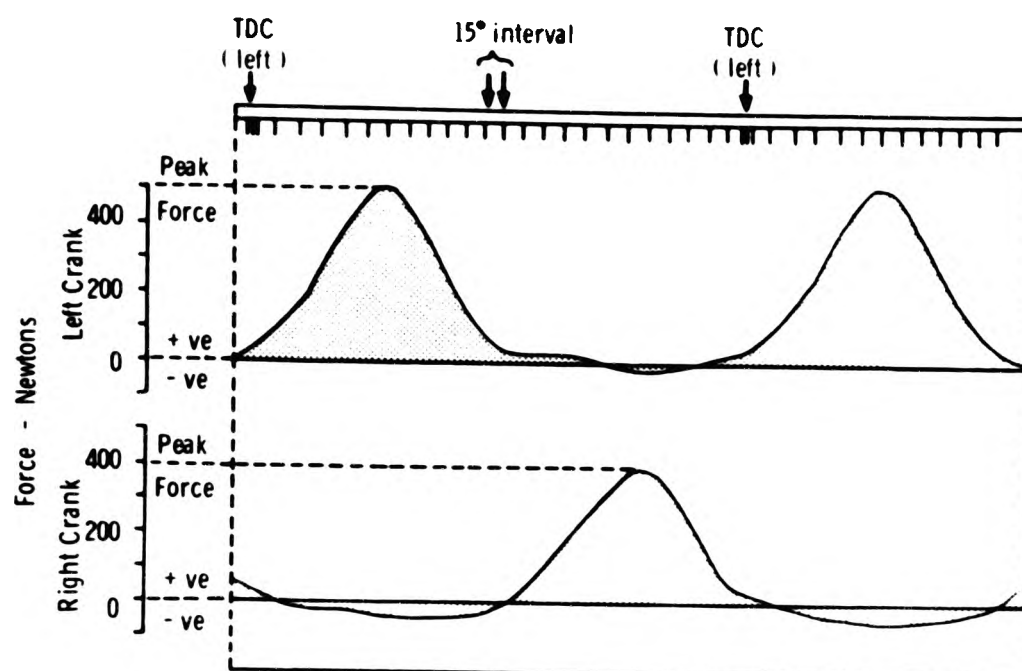


Fig.4. A force recording made during 20 seconds of maximal cycling exercise at a crank velocity of $110 \text{ rev. min}^{-1}$. Force exerted is measured separately for each leg. Markers at the top of the trace indicate each 15° rotation of the crank, top dead centre (TDC) of the left-hand crank being shown by a triple marker as a point of reference.

Through another channel on the ultraviolet recorder crank position was indicated on the force recording by a photoelectric transistor and lamp which were aligned and mounted either side of the left slip-ring disc. The rim of this disc was interrupted at 15° intervals by a series of holes so that when the ergometer was in operation corresponding markers were produced on the force record, top dead centre (TDC) of the left-hand crank being indicated by a triple marker as a point of reference (Fig.4).

Calibration of the ergometer was carried out while stationary by measuring the deflection when a known force was exerted on the cranks. Each crank was calibrated separately at a position 90° past TDC to allow measurement of effective force tangential to the arc of rotation.

Once the system was calibrated subjects were next seated on the ergometer and the saddle height was adjusted to a position comfortable for normal cycling. This saddle height was recorded and was then used for all subsequent tests to ensure that maximum force was exerted at a constant knee angle throughout the investigation. Toe straps were used to secure the balls of the feet over the pedal spindles and before exercising at the slower velocities where the force exerted would be likely to exceed their body weight subjects were secured to the seat of the ergometer by a strap fastened around their hips and anchored at a suitable point to the ergometer frame. This arrangement prevented subjects from being raised out of the saddle, enabling them, by pushing back against the strap, to exert maximum force whilst remaining seated.

With the gear box set to produce the slowest pedalling speed the motor was switched on and the velocity increased until the required pedalling frequency was attained. During this time subjects were asked to refrain from exerting any force on the pedals while they were allowed a little time (5-10 sec) to become accustomed to the speed. They were next instructed to make a maximal effort for 20 sec in an attempt to speed up the motor although the motor-gearing system operated in such a way that the velocity remained virtually unchanged, the variation in speed over the 20 sec period being less than 4% at all velocities.

Throughout the 20 sec period two parameters, peak force (PF) and peak power (PP) were determined for each leg independently. Peak force was the greatest force exerted during each revolution which was attained at $\sim 90^\circ$ past TDC (Fig.4) while peak power was the power generated at the point in each revolution where peak force occurred.

In previous investigations no significant difference has been found in the force exerted by the preferred and other limb in normal subjects (Sargeant, Hoinville and Young, 1981; Sargeant, 1976). In the present investigation all values of peak force and peak power have therefore been expressed as a mean of the right- and left-leg values.

Maximum values of peak force (PFmax) and peak power (PPmax) were calculated by taking a mean of the three highest consecutive values to be attained during the 20 sec period. By repeating this procedure at each of the different pedalling speeds subjects' force-velocity and power-velocity relationships during the cycling exercise

could be determined.

Muscle Size

Upper leg muscle (plus bone) volume (ULV) measured between the knee-joint space and the gluteal fold was determined for both legs in each subject to enable data to be standardised for muscle size. Bone volume as calculated from x-ray measurements (Sargeant, 1976) has been shown to constitute a relatively small but constant proportion ($11 \pm 1\%$) of the total muscle plus bone volume of the legs of normal adult males. It therefore seemed reasonable to assume that its inclusion in the measurement was likely to make only a small systematic difference when estimating the size of the upper leg muscle.

In the present investigation an anthropometric technique originally proposed by Zook (1932) and later developed and validated by Jones and Pearson (1969) was used to assess upper leg muscle (plus bone) volume.

This involved division of the upper leg between the knee-joint space and the gluteal fold into a number of segments each of which was treated as a truncated cone (Fig.5). By measuring the circumferences of the two parallel surfaces and the perpendicular distance between them the volume of each segment could be calculated and the total upper leg volume determined. Using Harpenden calipers skinfold measurements were then taken at the midline of the anterior and posterior thigh midway between the gluteal fold and the knee-joint space in order to determine the subcutaneous fat thickness.

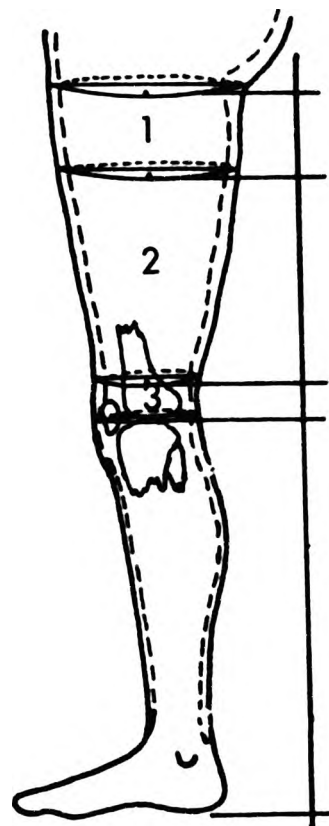


Fig.5 Diagram of the leg showing the sites at which anthropometric measurements were made for determination of upper leg volume. (After Jones and Pearson, 1969).

Using the appropriate regression equations derived by comparison of skinfold measurements with direct X-ray determinations of subcutaneous fat thickness (Jones and Pearson, 1969) these caliper readings were converted to give a measurement of the subcutaneous fat layer. The mean fat thickness derived from the anterior and posterior values was then deducted from the initial radius at each of the previously measured sites and using these new radii the muscle (plus bone) volume of each segment and hence of the complete upper leg was determined.

Estimations of total leg volume measured in this way have been shown to be highly correlated with volumes determined using both water displacement (Jones and Pearson, 1969; Davies, Barnes and Godfrey, 1972; Katch, Amuchie and Michael, 1973) and X-ray techniques (Sargeant, 1976) while more recently anthropometric measurements of upper leg muscle (plus bone) volume were shown to be highly correlated with volumes estimated using X-ray computerised tomography (Edwards, Grindrod, Narici, Rutherford and Sargeant, 1984).

The anthropometric method used in the present investigation would therefore appear to represent an accurate method of determining gross muscle size and it has the added advantage over X-ray techniques that it is non-invasive and can therefore be used to make repeated observations on the same subject.

Determination of Maximal Oxygen Uptake

In a number of subjects maximal oxygen uptake was measured directly during progressive exercise on an electrically-braked (Lode) cycle ergometer. Starting at zero load subjects were required

to pedal at $\sim 60 \text{ rev. min}^{-1}$ for 5 min at each of a number of successively increasing work loads intended to span the range of the subjects work capacity in 4 or 5 steps. Expired air was collected over the final 2 minutes of each work load using standard Douglas bag technique and this was analysed for CO_2 and O_2 using an infra-red carbon dioxide analyser (P.K. Morgan) and a paramagnetic oxygen analyser (Servomex-OA 150) respectively.

The CO_2 analyser was calibrated at the beginning of each test using atmospheric air and a cylinder gas composed of $\sim 4\% \text{ CO}_2$, $17\% \text{ O}_2$ and $79\% \text{ N}_2$, the exact composition having previously been determined by Haldane analysis. The O_2 analyser was calibrated using atmospheric air and oxygen-free nitrogen, the cylinder gas mixture being used to check the calibration in the respiratory range. Expired volume was measured using a dry gas meter (Parkinson Cowan CD4).

Cardiac frequency was measured throughout the exercise using lightweight disposable electrodes (Devices Sales Ltd.) to pick up an ECG signal which was amplified and then recorded on an ultraviolet oscillograph (S.E.Labs.Ltd.).

The criterion used to confirm that maximal levels of oxygen uptake were attained was that oxygen uptake showed no further rise with increasing work load. In some subjects however this was not always easy to apply. Some individuals even though encouraged to keep going for as long as possible could only sustain maximal levels for a relatively short period. In order to overcome this difficulty when it arose duplicate measurements were made on a subsequent day at different supramaximal loads.

Estimation of Lean Body Mass

Lean body mass was estimated as previously described by Durnin and Rahaman (1967) who used the relationship between skinfold thickness and body density to assess body fat. Skinfold measurements were taken on the left-hand side of the body at four sites as follows; biceps and triceps (measured over the mid-point of the muscle belly), subscapular (measured at an angle of $\sim 45^\circ$ just below the tip of the inferior angle of the scapular) and suprailiac (measured in the mid-axillary line just above the iliac crest). By inserting the log of the sum of the four skinfold thicknesses into the appropriate regression equation a prediction of body density was made and body fat was then estimated using this predicted value in the Siri equation (Siri, 1956) given below:

$$\text{Fat (\%)} = [(4.95/\text{density}) - 4.5] \times 100$$

Using this technique Durnin and Rahaman achieved correlation coefficients in the region of -0.80 between total skinfold thickness and body density and this enabled them to make an estimation of body fat from skinfold measurements with a ^{standard} error of $\sim \pm 3.5\%$.

Determination of Muscle Fibre Type

In a subgroup of the adult subjects the relative contribution of type I and type II fibres to the total cross-sectional fibre area was estimated in needle biopsy samples taken from the lateral part of the quadriceps muscle at the junction of the distal and middle thirds of the thigh. Samples were immediately frozen in liquid nitrogen-chilled Freon and stored at -80°C .

In order to classify type I and type II fibres transverse 10 micrometre frozen sections were cut in a cryostat at -20°C and these were stained to show the activity of myosin adenosine triphosphatase (myosin ATPase) after pre-incubation at pH 9.4 (Edwards, Young and Wiles, 1980). Fibres were then classified as type I (lightly stained at pH 9.4) or type II (heavily stained) and the relative frequency of the two fibre types was determined for each biopsy specimen.

The mean cross-sectional area of type I and type II fibres was then calculated from measurements of at least 100 fibres of each type. Having previously determined the relative frequency of each fibre type their relative contribution to the overall cross-sectional fibre area of the biopsy sample was determined.

Statistical Analyses

Where summarised data is presented this is given as means and standard deviations throughout the text.

In order to compare different sets of data student's 't' test was used. Comparison between different groups of subjects was carried out using a grouped t-test while paired t-tests were used to compare results within the same individual.

Linear regression equations were derived using the sum of least squares method as described in standard statistics textbooks.

RESULTS

Force-Velocity Relationship.

Maximum values of peak force during the 20 sec test were generally attained within a few revolutions from the start after which there was a gradual fall in force exerted over the remainder of the 20 sec period as the muscle fatigued (Fig. 6).

Maximum peak force was velocity-dependent with the greatest force being attained at the slowest cycling speed (Fig.7). In the majority of subjects maximum peak force was inversely and linearly related to crank velocity.

At any given velocity intersubject differences in the maximum peak force achieved were considerable. This variation was partly accounted for by expressing the force exerted in terms of the upper leg muscle (plus bone) volume as described earlier. When data was standardised in this way the relationship between maximal peak force (N. litre_{ULV}⁻¹) and crank velocity (rev.min⁻¹) in the three groups of subjects studied could be described by the following regression equations (Fig. 8);

$$\text{Male Adults: } y = 252.65 - 1.0765x \quad (n=114, r=-0.8508, p<0.001)$$

$$\text{Female Adults: } y = 254.37 - 1.0499x \quad (n=64, r=-0.7274, p<0.001)$$

$$\text{Male Children: } y = 219.95 - 0.9522x \quad (n=92, r=-0.4032, p<0.001)$$

where $y = \text{PFmax (N.litre}_{\text{ULV}}^{-1})$ and $x = \text{crank velocity (rev min}^{-1})$

Students t-test was used to compare the slope of this

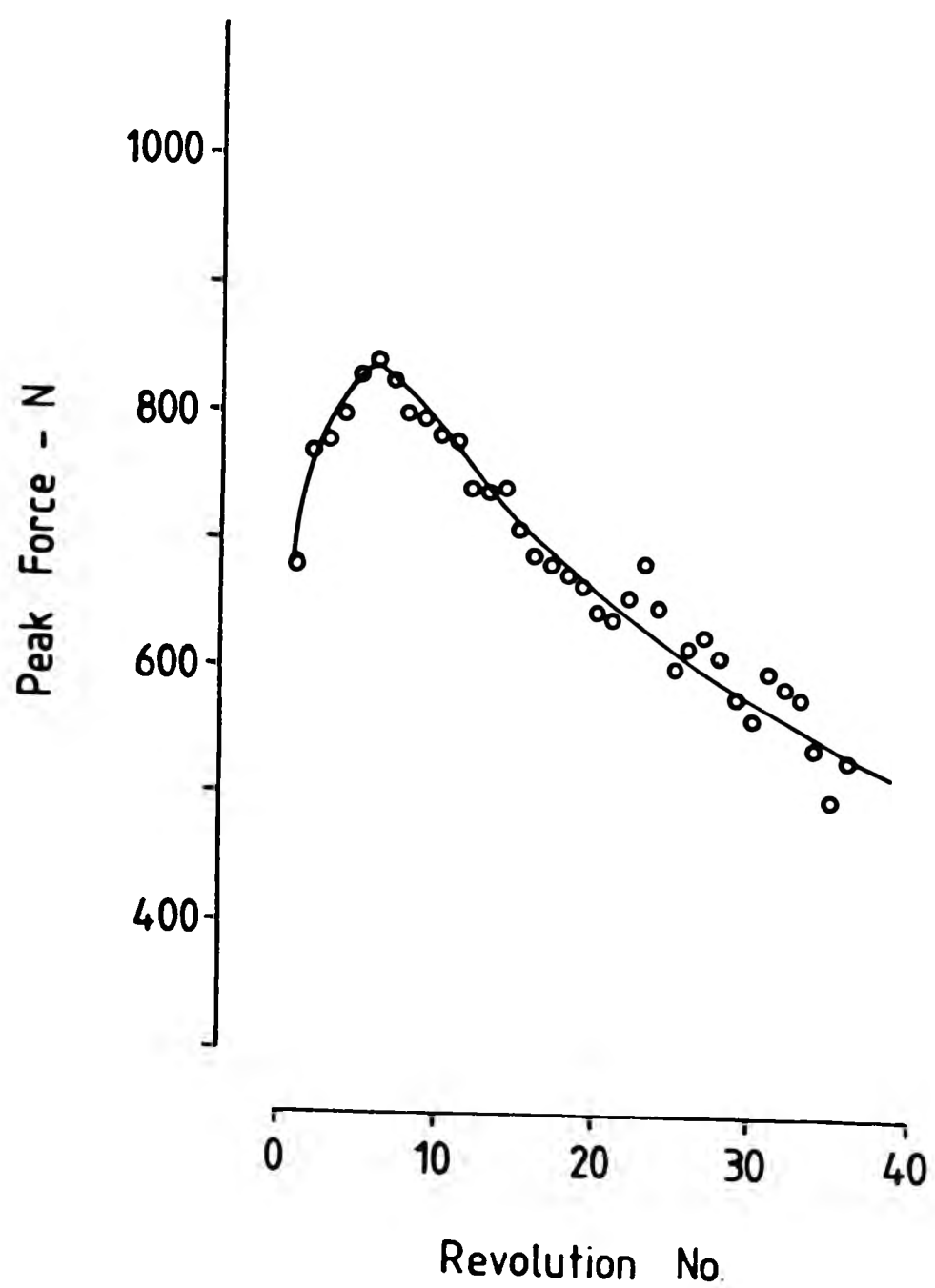


Fig.6 Peak force (mean of right and left legs) for each revolution during 20 seconds maximal exercise at 110 rev. min⁻¹. (Subject A.S.)

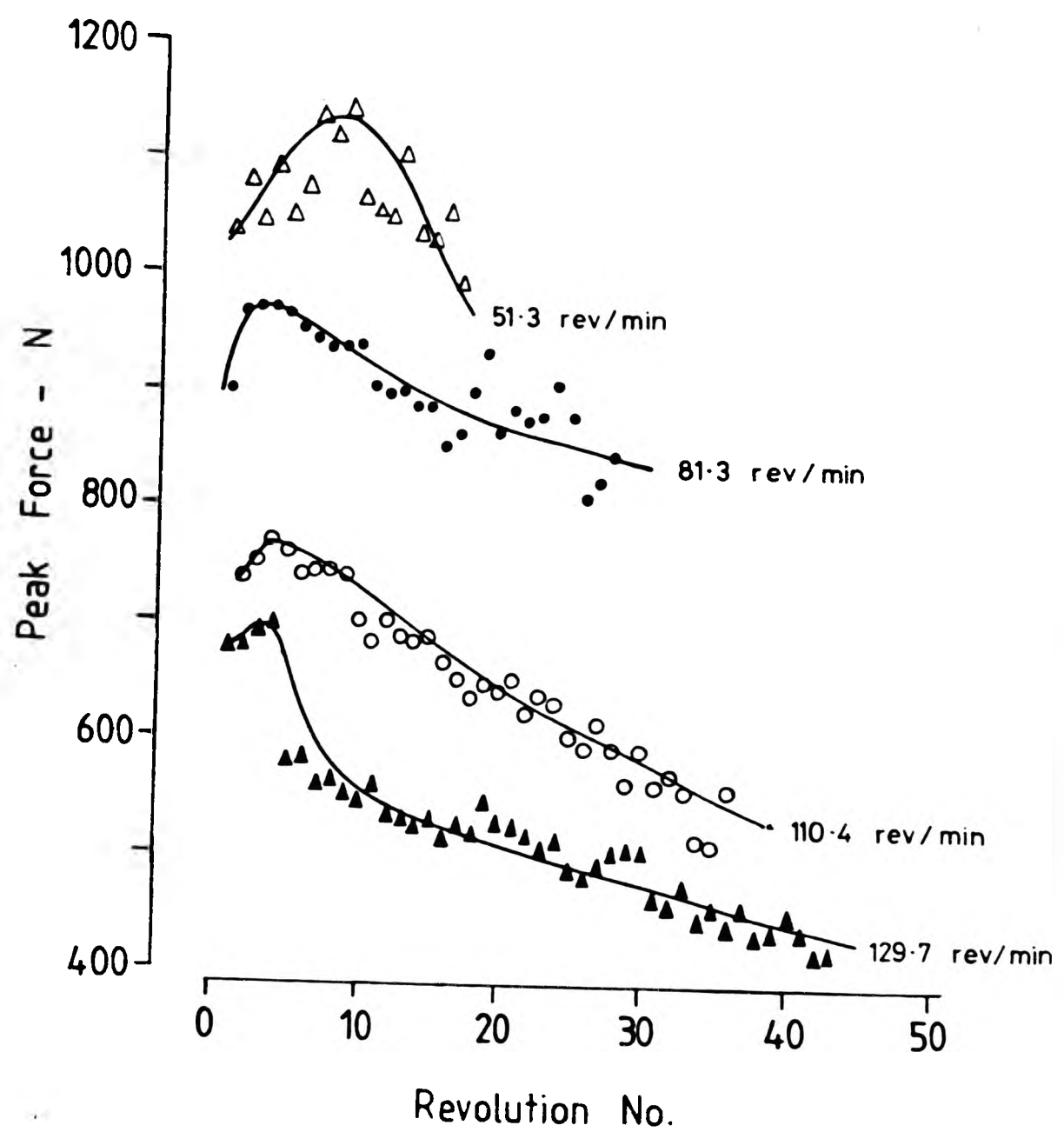


Fig.7 Peak force for each revolution during 20 seconds maximal exercise at 51.3 (Δ), 81.3 (\bullet), 110.4 (\circ) and 129.7(\blacktriangle) rev. min^{-1} . (Subject S.M.).

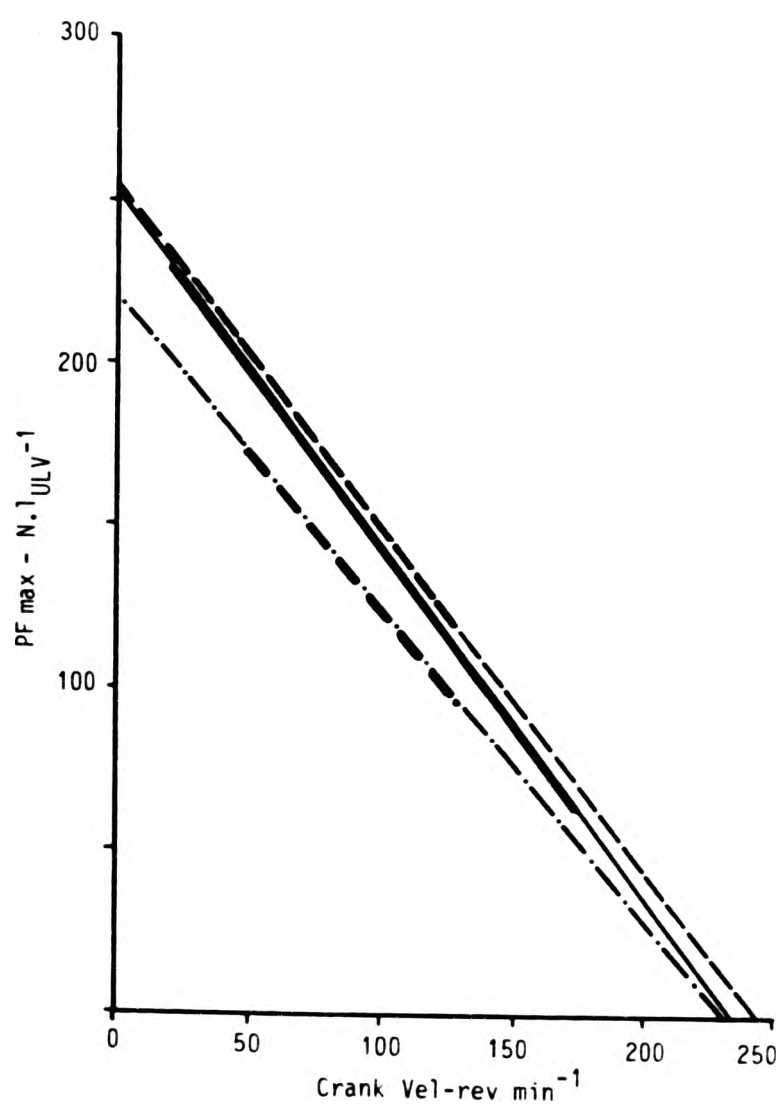


Fig.8 The relationship between maximum peak force (standardised for the upper leg muscle (plus bone) volume ($PF_{max} l. ULV^{-1}$)) and crank velocity ($rev. min^{-1}$) is shown for male adults (solid line), female adults (broken line) and 13 year old boys (dotted line). Actual data points have been omitted for the sake of clarity. Lines indicate linear regression based on individual data from subjects of each group. The heavy part of the line denotes the limits of the experimental data.

relationship between individuals in one group with those of another. No significant difference was found between male and female adults although the adults achieved significantly higher forces per unit of muscle mass when compared to the 13 year old boys (male adults $p < 0.002$; female adults $p < 0.05$).

Determination of Maximal Short-term Power Output

Since the maximal tests were performed at constant velocity peak power throughout the 20 sec period followed a similar pattern to peak force generally reaching maximum values near the start and declining thereafter over the remainder of the exercise period (Fig.9). As a mathematical consequence of the linear relationship observed between maximal peak force and crank velocity maximal peak power was a parabolic function of velocity over the range of speeds studied (Fig. 10).

The parabola of best fit was determined for each of the subjects from the regression equation describing their force-velocity relationship. Maximal short-term power output was calculated from the apex of this relationship where the velocity represented the optimum for power output.

Values of maximal short-term power output determined at optimum velocity showed considerable variation in the present group of subjects with individual values ranging from 444 - 1536W. These differences were partly resolved by expressing the data in terms of the upper leg muscle (plus bone) volume. This reduced the coefficient of variation for maximal peak power from 29.2 to 20.0% in the present group of subjects (Table 2) giving an overall mean value

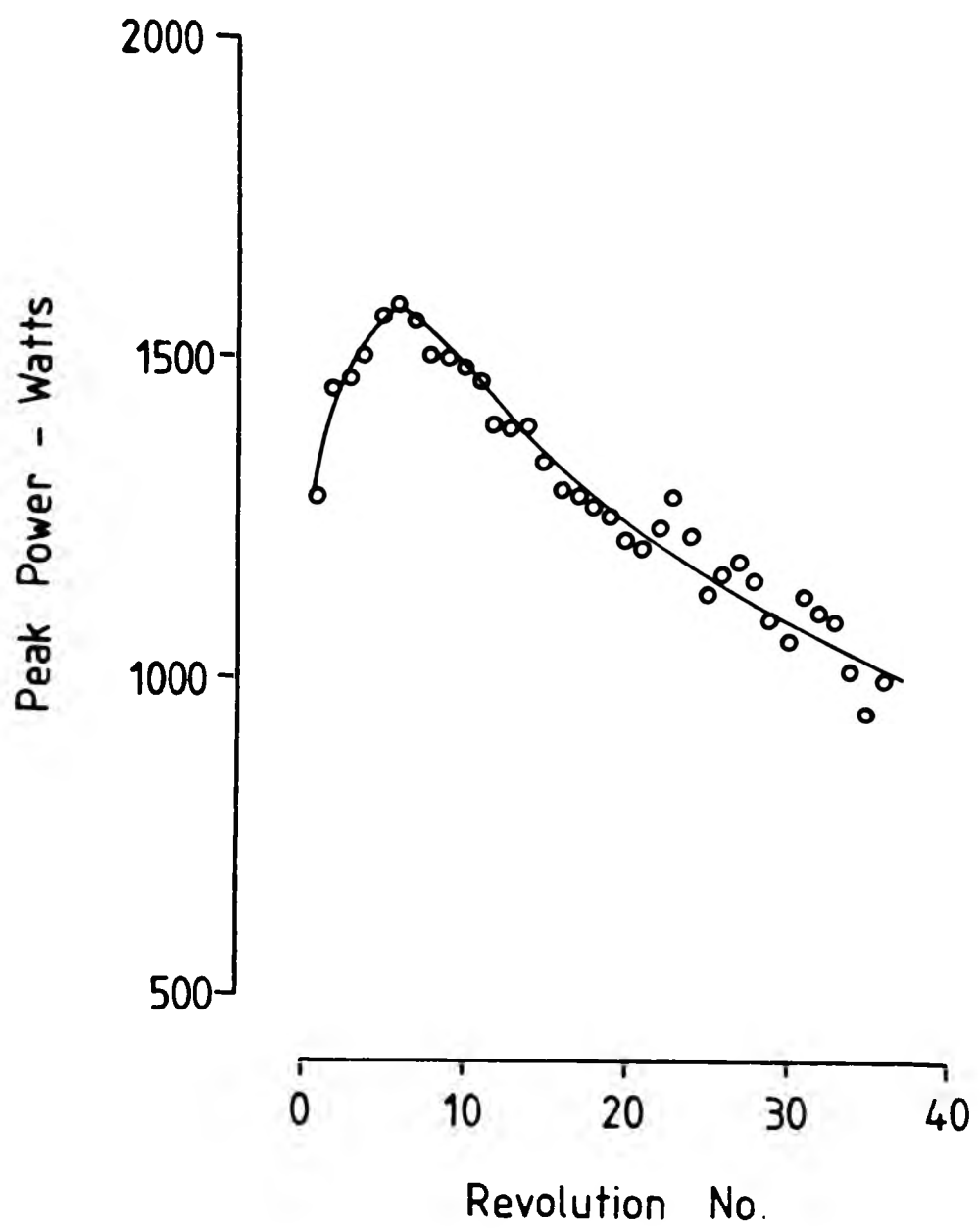


Fig.9 Peak power for each revolution during 20 seconds maximal exercise at 110 rev. min⁻¹. (Subject A.S.).

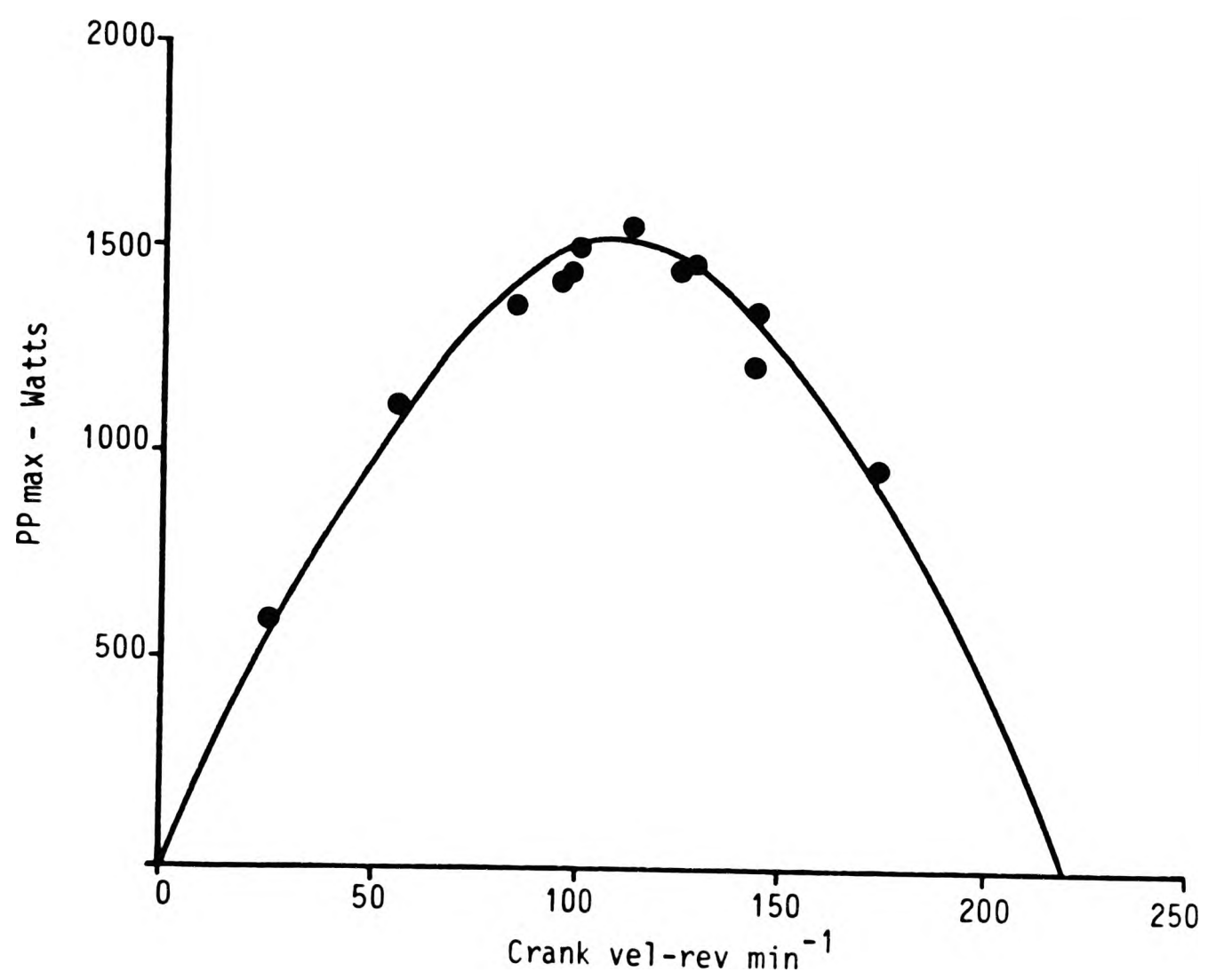


Fig.10 Relationship between maximal peak power (PPmax) and crank velocity in subject A.S.

of $253 \pm 50 \text{ W litre}_{\text{ULV}}^{-1}$ (range 159.4 - 342.2W)

When data on male and female adults and male children were considered independently mean values of maximal short-term power output for the 3 groups of subjects were 1239 ± 227 , 820 ± 156 and 795 ± 153 Watts respectively (Table 2, Fig 11). When standardised for the upper leg muscle (plus bone) volume the equivalent mean values were 269 ± 36 , 274 ± 63 and $224 \pm 37 \text{ W.l.}_{\text{ULV}}^{-1}$. Values obtained for the children were significantly lower than those obtained for both the male ($p < 0.001$) and female ($p < 0.02$) adults although there was no significant difference between the latter 2 groups.

Optimum Velocity

The optimum velocity for maximum power output was defined for each subject by the apex of their power-velocity relationship. Individual values varied from 83-170 rev.min^{-1} . The mean values calculated for each group were 114 ± 15 , 119 ± 20 and $120 \pm 17 \text{ rev.min}^{-1}$ for male adults, female adults and children respectively (Table 2). No significant differences in optimum velocity were observed between the groups thus an overall mean value of $118 \pm 17 \text{ rev.min}^{-1}$ was obtained for all subjects combined.

Effect of Muscle fibre Type.

The effect of fibre type on the force-velocity characteristics of human muscle was examined in a subgroup of the adult subjects ($n=22$) whose muscle fibre type composition was determined by needle biopsy as described earlier.

The optimum velocity for power output in this group of subjects

	V opt (rev min ⁻¹)	PPmax (Watts)	PPmax (W. l ULV ⁻¹)
Male Adults (n = 24)	114.3 ±14.7 (12.9%)	1239 ±227 (18.3%)	269 ±36 (13.5%)
Female Adults (n = 16)	119.5 ±19.9 (16.7%)	820 ±156 (19.1%)	274 ±63 (23.0%)
Male Children (n = 25)	120.0 ±17.4 (14.5%)	795 ±152 (19.2%)	224 ±37 (16.7%)
All Subjects (n = 65)	117.8 ±17.1 (14.5%)	964 ±281 (29.2%)	253 ±50 (19.6%)

Table 2

Optimum velocity (V opt) and maximal peak power (PPmax) calculated at optimum velocity in the three groups of subjects studied. PPmax is given in absolute terms (Watts) and standardised for upper leg muscle plus bone volume (W. litre ULV⁻¹). Values are expressed as the mean ± SD. The coefficient of variation is shown in brackets.

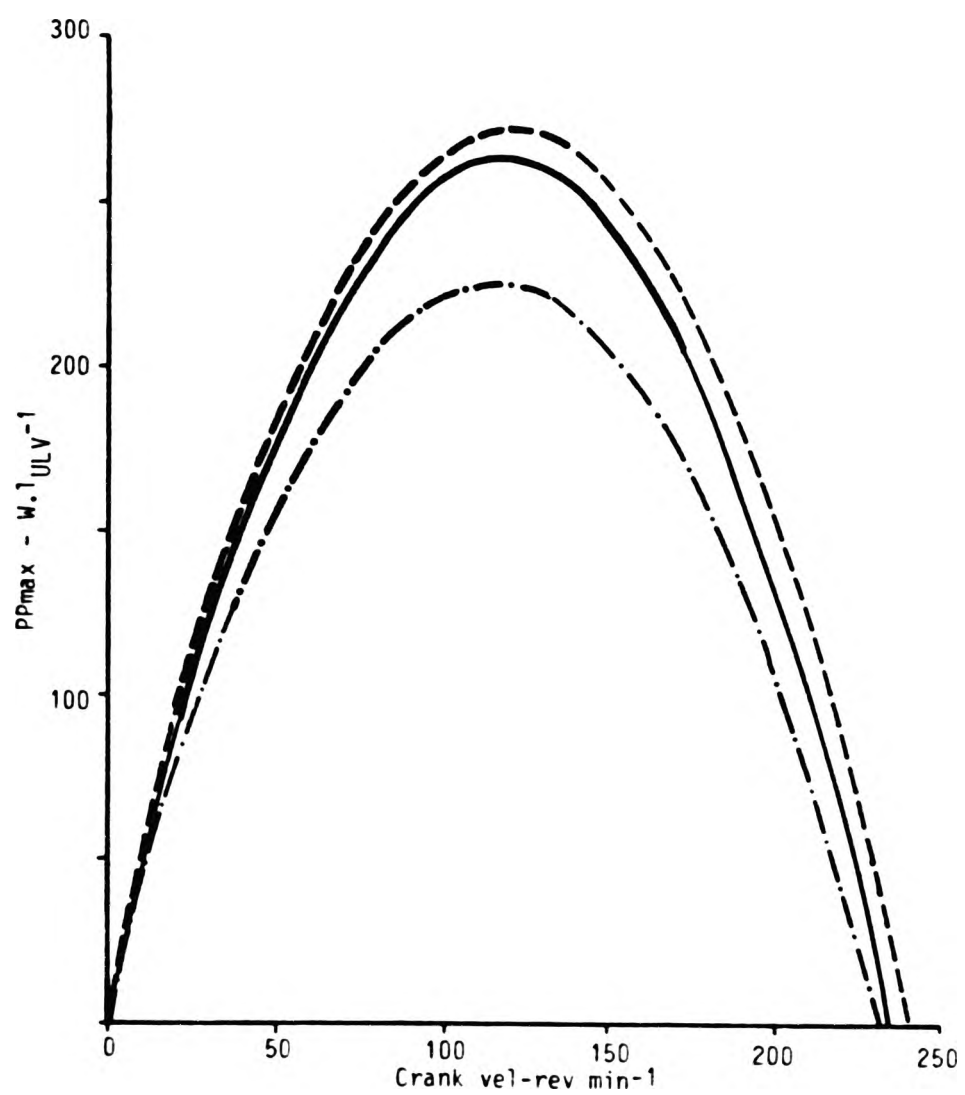


Fig.11 Relationship between maximal peak power (Standardised for the upper leg muscle (plus bone) volume ($W.l. ULV^{-1}$)) and crank velocity in male adults (solid line), female adults (broken line) and 13 year old boys (dotted line). Data points have been omitted. Lines represent the parabola of best fit derived from force-velocity regression equations. The heavy part of the line denotes the experimental limits of the data.

was 112.6 ± 14.0 (Table 3) which was not significantly different from the adult group as a whole (116.4 ± 17.0).

Within this subgroup optimum velocity (V_{opt}) was found to be directly and linearly related to the percent cross-sectional area of type II fibres (% CSA Type II). The relationship between the two was given by the following equation (Fig 12);

$$V_{opt} = 95.941 + 0.404 \times \% \text{ CSA Type II } (n=22; r=0.5233; p<0.05)$$

There was no significant difference between males and females.

Maximal power output predicted at optimum velocity and standardised for the upper leg muscle plus bone volume was also found to increase linearly with the proportion of type II fibres as shown in Fig 13 ($n = 22; r = 0.5053; p < 0.05; PP_{max} = 214.23 + 1.241 \times \% \text{ CSA Type II}$)

Fatiguability was assessed in a number of subjects in whom power output had been measured at a standard velocity (110 rev min^{-1}) close to the optimum. In these subjects a fatigue index was compiled by calculating peak power (mean of three consecutive values) 15 seconds after the attainment of PP_{max} so that the loss in power output could be determined. From these values a mean rate of fatigue (in $W. \text{ sec}^{-1}$) over this period was calculated.

When the loss in power output was expressed as a percent of the maximal peak power attained at the start of the test ($\% \text{ sec}^{-1}$) this fatigue index was found to be correlated to the proportion of type II fibres in the male subjects (Fig.14a) although a similar relationship was not observed in the female subjects.

	V opt (rev.min ⁻¹)	PPmax (Watts)	PPmax (W. l.ULV ⁻¹)
Adults with known fibre type (n = 22)	112.6 ±14.0	1088 ±293	266 ±44
All Adults (n = 40)	116.4 ±17.0	1073 ±292	271 ±48

Table 3

Optimum velocity (V opt) and maximal peak power determined at optimum velocity (PPmax). Mean (±SD) values are shown for all adults and those with known fibre type.

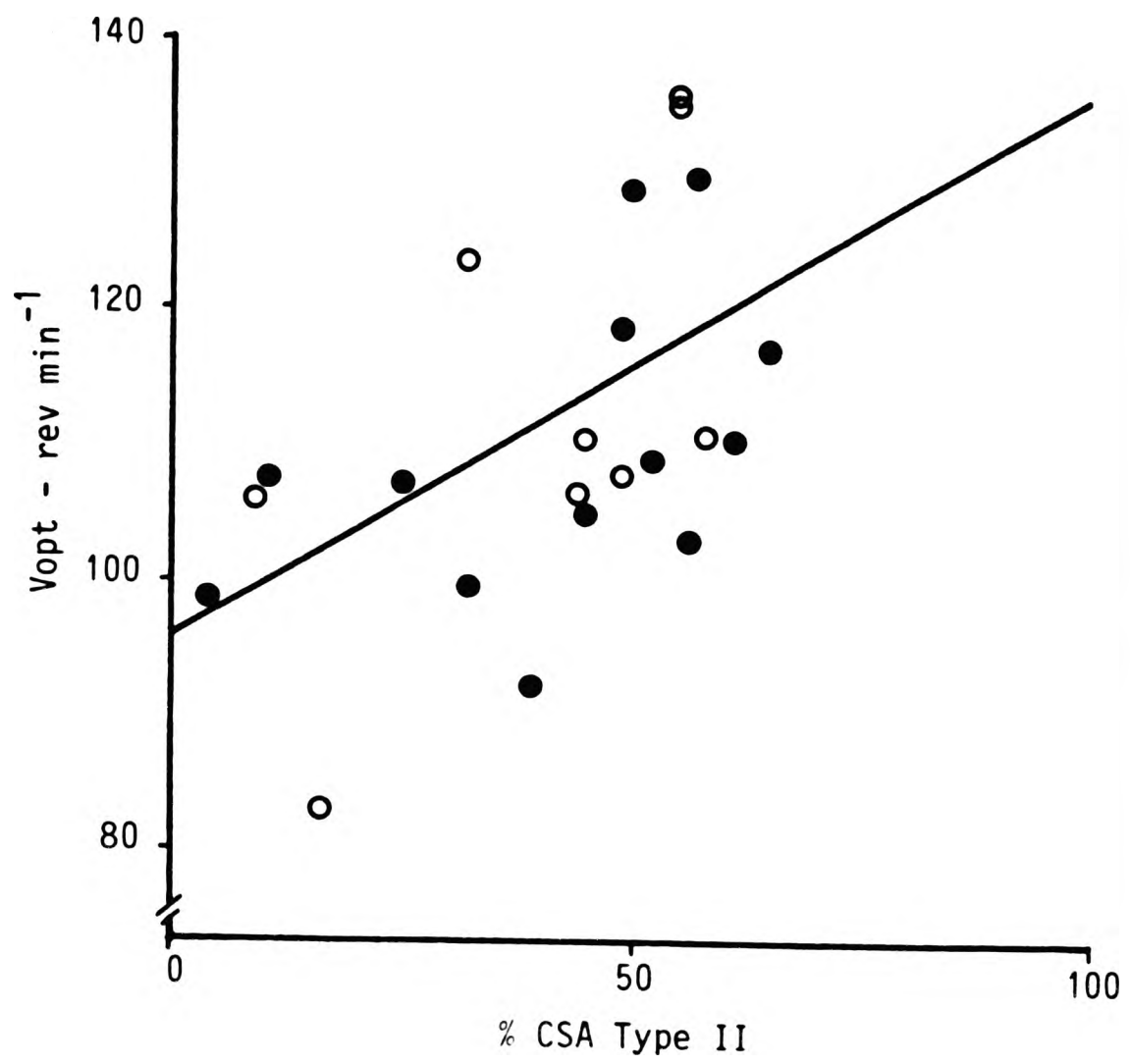


Fig.12 Linear regression line showing relationship between optimum velocity (Vopt) and the percent cross-sectional area of type II fibres (% CSA Type II) in male (closed symbols) and female (open symbols) adults. (n=22; r=0.5233; p<0.05)

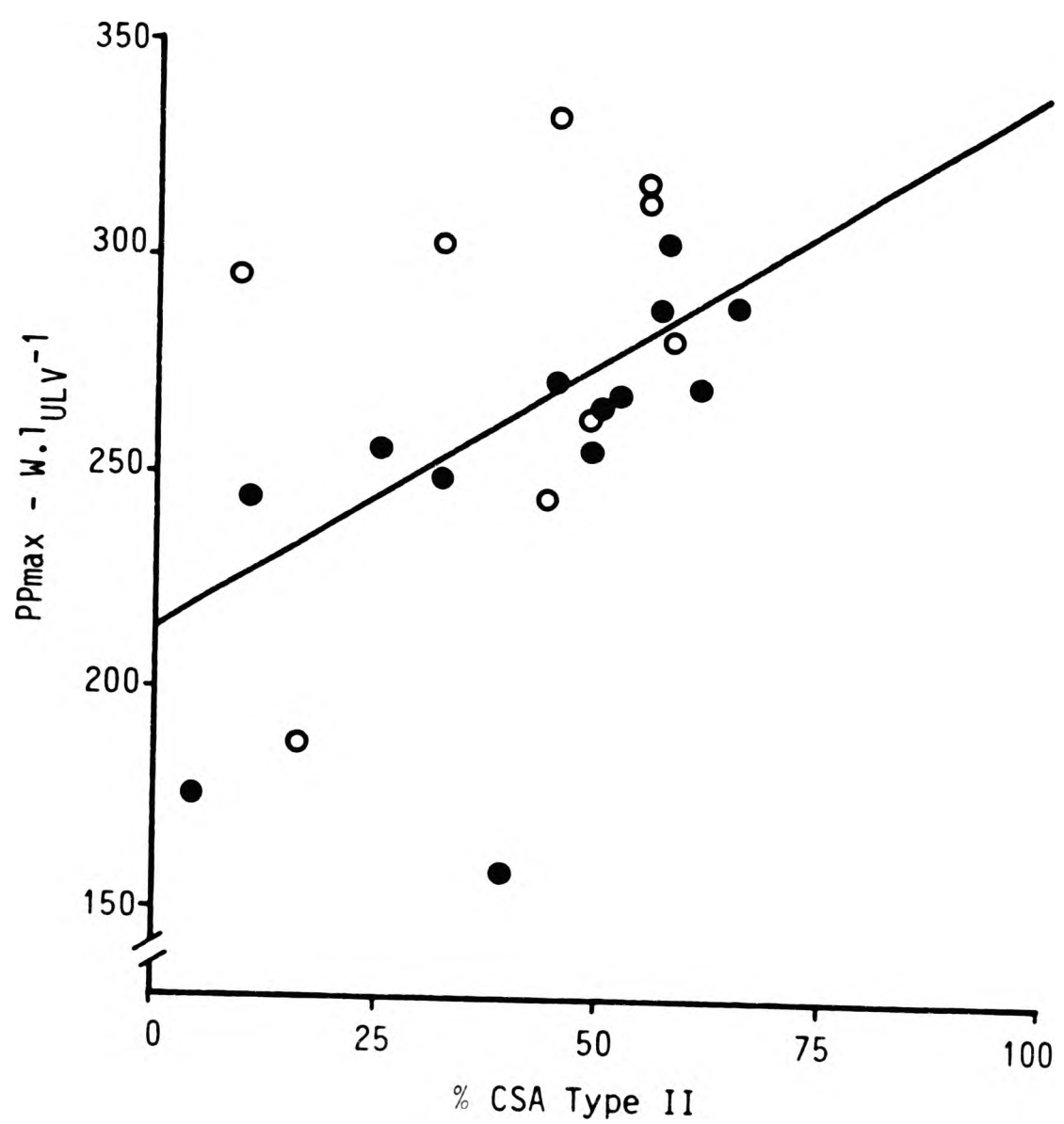


Fig.13 Linear regression line showing relationship between maximal peak power at optimum velocity (standardised for the upper leg muscle (plus bone) volume ($W.l.ULV^{-1}$)) and the percent cross-sectional area of type II fibres (% CSA Type II) in male and female adults ($n=22$; $r=0.5053$; $p<0.05$). Symbols as for Fig.12.

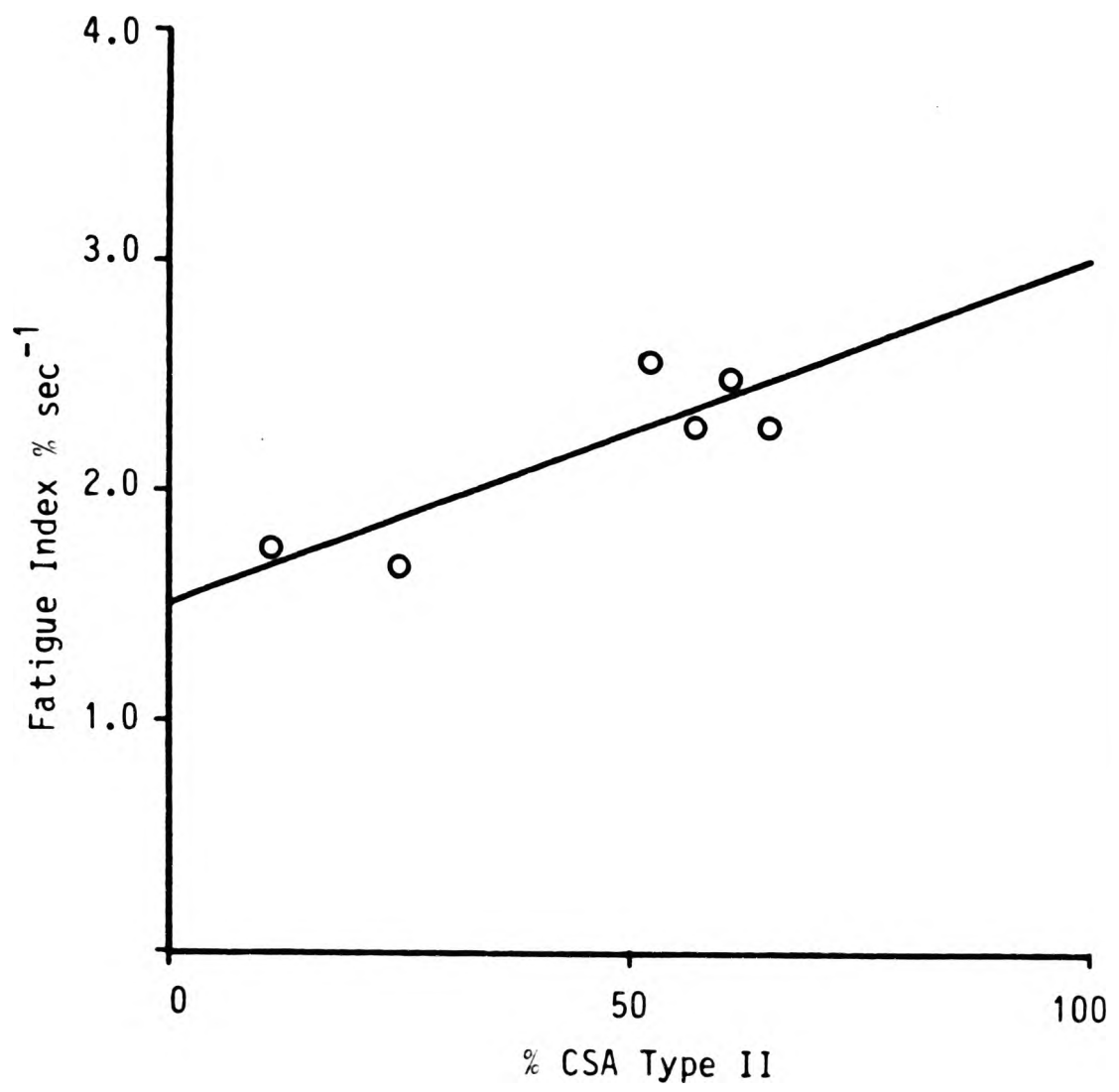


Fig.14 Linear regression line showing relationship between rate of fatigue at $\sim 110 \text{ rev. min}^{-1}$ and the percent cross-sectional area of type II fibres (% CSA Type II) in male adults ($y = 1.501 + 0.015x$; $n=6$; $r=0.871$; $p<0.05$)

Discussion

In this chapter the application of an isokinetic technique for the measurement of maximal force and power output in human muscle is described. This technique has a number of advantages over other methods which are currently used to assess dynamic muscle function in man. First of all the isokinetic ergometer used in the present investigation can be set to give pedalling speeds in the range 23-180 rev.min⁻¹ allowing maximal force and power output to be determined over a wide range of contraction velocities. Since there is no acceleration phase involved in the exercise subjects are also able to achieve maximal levels of power output very rapidly before the metabolic substrates become significantly depleted to the extent where they might limit performance. The test used is easy to perform and although the results obtained are clearly dependent upon the motivation of the subject, the exercise is of such short duration that it is generally well accepted by the subjects.

Measurements made using this technique are highly reproducible as shown in a previous study where the coefficient of variation between 41 paired tests performed over a range of crank speeds was found to be less than 6% (Sargeant et al, 1981). Reproducibility was also tested in the present study by making repeated observations at a single speed, several times over a number of weeks. This was done in 4 subjects and again the coefficient of variation was found to be less than 6% (Table 4).

Subject	Velocity (rev.min ⁻¹)	PPmax (Watts)
M.N.	112.2	1012
	112.4	1059
	112.6	1113
	112.1	1149
Mean ± SD		1083±60
CV		5.57%
A.S.	112.2	1491
	112.4	1411
	112.8	1408
	113.2	1536
	113.0	1485
	111.9	1398
	112.3	1366
	112.1	1440
111.7	1391	
Mean ± SD		1436±56
CV		3.91%
J.S.	111.4	1234
	110.7	1243
	110.6	1207
	111.8	1259
	110.8	1290
Mean ± SD		1247±31
CV		2.45%
G.M.	112.1	1475
	112.2	1535
	112.3	1575
	113.1	1503
	112.1	1468
	112.9	1528
	111.6	1358
	111.2	1463
Mean ± SD		1488±65
CV		4.37%

Table 4 Maximal peak power during repeated tests performed at a preset crank velocity of 110 rev min⁻¹. The individual and mean values attained by each subject are shown. CV denotes the coefficient of variation.

Force-Velocity Relationship.

In the present investigation the isokinetic technique described above was used to characterise the force-velocity and power-velocity relationship of human muscle and to determine the optimum velocity for maximal power output. An important observation made in the course of these experiments was the close inverse linear relationship between maximum peak force and crank velocity. This is consistent with previous data obtained from experiments on young male adults (Sargeant et al, 1981).

As early as 1922 Hill reported such a linear relationship between maximum force development and contraction velocity in human muscle although these results were not consistent with the classical in vitro studies where an exponential relationship was observed (Gasser and Hill, 1924; Fenn and Marsh, 1935; Hill, 1938). In these classic studies however the force-velocity relationship was obtained from experiments on isolated muscle, free of the joint, in which the force measured was therefore directly related to the tension developed within the muscle. In vivo however the torque or force generated may not always reflect actual muscular tension since the load placed on a muscle is modified by the limb and lever system.

The force exerted on the cranks during cycling is the result of a complex action involving several muscle groups operating across two or more joints. This is somewhat different from the situation in vitro and it is perhaps not too surprising to find differences in the muscles force-velocity characteristics under the two sets of conditions. It should be pointed out however that a number of workers

have observed a curvilinear relationship between force and contraction speed under in vivo conditions similar to that found in vitro.

Wilkie (1950) reported such a finding in the human elbow flexors while other investigators (Thorstensson, Grimby and Karlsson, 1976; Coyle, Costill and Lesmes, 1979) obtained a similar relationship during maximal knee extensions performed on an isokinetic dynamometer. The discrepancy between these results and those obtained in the present investigation may reflect differences between the muscle groups involved or alternatively it may be due to the testing procedure employed.

In vivo a muscle will register its peak force when its joint angle has an optimal mechanical advantage (Thorstensson et al, 1976) however it also requires a finite time to develop this tension which will depend upon its fibre type composition (Buchthal and Schmalbruch, 1970; Sica and McComas, 1971; Milner-Brown, Stein and Yemm, 1973; Burke and Edgerton, 1975). In the present study peak force during each revolution was consistently attained at approximately 90° past TDC this being the optimum position mechanically to apply tangential force. Hence the mechanical advantage remained the same throughout the range of velocities studied.

During isokinetic knee extensions performed on the Cybex dynamometer however, Thorstensson et al (1976) found that the angle at which peak torque was attained increased progressively as the angular velocity increased which may reflect an inability at the faster speeds to develop maximal tension before passing the optimal joint angle. This would place the muscle under a progressively greater

mechanical disadvantage as the velocity increased which could contribute to a disproportionate loss of force at the higher velocities producing the curvilinear relationship observed. It is interesting to note that McCartney, Heigenhauser, Sargeant and Jones (1983) using an isokinetic cycling technique similar to the present one observed a linear relationship between maximum force and crank velocity over the range studied (60-160 rev.min⁻¹) which is in agreement with the present findings.

It is of course possible that a curvilinear relationship would be found using the present technique if slower velocities could be studied. However where there was any deviation from the straight line relationship found in the majority of subjects this was towards a levelling-off of force at the lower velocities and not towards the even sharper increases observed in isolated muscle (Gasser and Hill, 1924; Fenn and Marsh, 1935; Hill, 1938). Such a loss of force at low contraction speeds has also been reported during knee extensions performed on the Cybex apparatus (Perrine and Edgerton, 1978; Ingemann-Hansen and Halkjaer-Kristensen, 1979) and it has been suggested that this divergence from the classical force-velocity curve in human muscle may be due to some neural mechanism which acts to restrict maximal force development in vivo, particularly at the lower velocities where force is highest.

The present results although not directly comparable with those obtained in vitro should be considered to represent a functional relationship between force and contraction velocity in intact human muscle. As a consequence of this relationship maximal peak power was found to describe a parabolic relationship with crank velocity where

the apex represented the optimum velocity for maximal power output. In the present group of subjects this optimum velocity was predicted to lie between 83.1- 170.5 rev.min⁻¹. However despite these intersubject differences mean values were not significantly different in the 3 groups of subjects studied. The overall mean value of 117.8 rev.min⁻¹ obtained for the group as a whole corresponded to a speed of movement at the knee of ~ 250° sec⁻¹ which is consistent with earlier observations using the Cybex dynamometer where a plateauing of power output was reported at similar speeds (Thorstensson et al, 1976; Coyle et al, 1979; Gregor, Edgerton, Perrine, Campion and De Bus, 1979).

Standardisation for Muscle Size

In the present investigation maximal peak power calculated at optimum velocity showed considerable variation in the subjects studied. In the male adults the mean value was 1239W which was equivalent to 156 and 151% of the mean values attained by the children and female adults respectively.

Some of this variation could be accounted for by expressing power output in terms of the upper leg muscle (plus bone) volume. Standardising the results in this way reduced the coefficient of variation for this measurement from 18.3 and 19.2% to 13.5 and 16.7% in the male adults and children respectively. In the female adults however differences in power output were not reduced by this standardisation procedure. This may reflect the difficulties involved in obtaining accurate skinfold measurements on the upper leg in

females with a relatively thick layer of subcutaneous fat. This in turn may have lead to some inaccuracies in the upper leg muscle (plus bone) volume in these subjects.

Comparing the mean standardised values of maximal short-term power output in the three groups of subjects showed this to be significantly lower in the children than in both the male and female adults ($p < 0.05$). This implies that the differences in power output between these groups were not due to muscle size alone. It may be that there is some fundamental change in muscle composition during growth which is responsible for the variation in power output. Since muscle grows by an increase in fibre size rather than fibre number, the overall number of fibres within a muscle aswell as the ratio of Type I to Type II fibres remains unaltered. However a relatively greater increase in cross-sectional area of the more powerful Type II fibres during growth could contribute to the greater power output of adult muscle when compared to that of the children.

Muscle Fibre Type

The effect of muscle fibre type composition upon maximal short-term power output was investigated in a subgroup of the adult subjects. The linear relationship observed in this group between maximal power output (standardised for the upper leg muscle (plus bone) volume) and the relative proportion of type II fibres is consistent with previous data obtained using the Cybex apparatus (Thorstensson et al, 1976; Coyle et al, 1979). In a recent investigation Bar-Or et al (1980) also reported a greater capacity for power output during brief maximal cycling exercise in subjects

with a higher preponderance of type II fibres while Nygaard, Houston, Suzuki, Jorgensen and Saltin (1981) observed a similar relationship in the human elbow flexors. These results support empirical observations made on athletes which showed that those involved in events such as sprinting or weight-lifting where powerful, fast contractions were required had a greater proportion of type II fibre area than those involved in endurance events.

This enhanced force production of type II fibres has been attributed to their higher intrinsic rate of shortening as well as to a more rapid rate of tension development when compared to type I fibres (Close, 1972; Thorstensson et al, 1976). Higher activities of creatine phosphokinase (CPK) and myokinase (MK) have also been found in type II as compared to type I fibres (Thorstensson, 1976; Costill, Fink and Pollock, 1976; Thorstensson, Sjodin, Tesch and Karlsson, 1977) which may be indicative of type II fibres being better adjusted to replenishing the ATP stores during short-term high intensity exercise.

The linear relationship observed in the present subjects between optimum velocity and the % cross-sectional area (CSA) of Type II fibres is consistent with earlier observations on human muscle (Thorstensson et al, 1976) where higher maximal velocities were found in those muscles with a higher preponderance of type II fibres.

At 50% CSA of type II fibres which would be fairly typical for a normal untrained subject the present results indicate an optimum velocity of $116.1 \text{ rev.min}^{-1}$ which is in close agreement with the overall mean value of $117.8 \text{ rev.min}^{-1}$ obtained in the present group of

subjects. Thus in order to make a true assessment of maximal short-term power output during cycling these results indicate that in population studies on normal healthy individuals a pedalling speed of $\sim 117 \text{ rev. min}^{-1}$ should be employed.

The higher rate of fatigue observed in male subjects with a predominance of type II fibres confirms earlier findings obtained with human muscle during both maximal knee extensions (Coyle et al, 1979) and cycling (Bar-Or et al, 1977). Assuming that the initial levels of high-energy phosphate are the same at the start of exercise this may reflect a faster utilisation of ATP in the more powerful type II fibres when compared to the slower type I fibres. On this basis subjects with a higher proportion of type II fibres would deplete the high energy phosphate store more rapidly which could account for the greater loss of power output in these subjects.

Conclusions

The present results indicate an inverse linear relationship between maximum force and crank velocity during cycling with maximal power output being attained at a velocity of $117.8 \pm 17.1 \text{ rev. min}^{-1}$. No significant differences in optimum velocity were apparent between the 3 groups of subjects studied although both male and female adults were found to generate somewhat higher levels of maximal power output than the children when results were standardised for the active muscle mass. In adults optimum velocity and maximal power output ($W \cdot 1 \cdot U_{LV}^{-1}$) were correlated to the % CSA of type II fibres and in the males fatiguability was also greater in those subjects with a predominance

of type II fibres.

CHAPTER 3

The Effect of Prior Exercise
on Maximal Short-Term
Power Output in Man.

INTRODUCTION

The maximal capacity of human muscle to generate power during short-term dynamic exercise of a few seconds duration is dependent upon the rate of splitting of ATP and phosphorylcreatine since these represent the most immediate source of energy available to the contracting muscle. During maximal exercise however the rate at which these high energy phosphates may be broken down is faster than their rate of resynthesis. Consequently maximal power can only be sustained for a few seconds before the high energy phosphate store becomes exhausted or depleted to a critically low level resulting in a subsequent fall in power output. The maximum power attainable should therefore be related at any given time to the concentration of unsplit high energy phosphate in the active muscle fibres and any reduction in this might be expected to result in a diminished capacity for maximal short-term power output.

Previous investigations have shown decreases in the concentration of ATP and PC measured at steady state to be directly related to the exercise intensity at submaximal work loads (Hultman, Bergstrom and McLennan-Anderson, 1967; Karlsson, Diamant and Saltin, 1971). On the basis that short-term power output as measured in the present investigation should reflect the prevailing level of high energy phosphate in the fatigued muscle one might therefore expect to see a corresponding decrement in power output as a result of such exercise.

In the present study I sought to confirm this by manipulating the level of unsplit high energy phosphate experimentally by having subjects perform submaximal exercise of varying duration and intensity prior to measuring their maximal short-term power output.

METHODS

The effect of prior exercise on maximal short-term power output from human muscle was investigated in two separate studies. The first of these examined the effect of varying the prior exercise duration; the second investigated how power output was effected by different intensities of prior submaximal exercise.

In both studies maximal short-term power output was measured during 20 seconds of maximal exercise on the isokinetic ergometer as previously described in Chapter 2. Prior exercise was also performed on the isokinetic ergometer. In each case measurements were made at a standard preset velocity of 112 rev. min⁻¹.

The procedure followed in each of the two studies is described below and outlined in Figs.1 and 2. Following this is a description of the measurement techniques employed.

Protocol

STUDY I

AIM: To examine the effect of varying the duration of constant load prior exercise on subsequent maximal short-term power output.

The subjects studied were two healthy male adults who were physical education students at The Polytechnic of North London. Table I shows their physical characteristics including measurement of maximal oxygen uptake ($\dot{V}O_{2max}$) which was directly determined (see Measurement Techniques).

Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_{2\max}$ $l\ min^{-1}$
PJ	26	162.8	66.7	50.7	3.20
SM	24	177.4	88.7	72.5	4.56

Table 1 Physical characteristics of the subjects studied.

An outline of the experimental protocol is shown in Fig. 1. In the control run the 20 second maximal test was performed after rest at ambient conditions (20 - 23°C). In the experimental runs the short-term power output measurement was immediately preceded by a period of prior submaximal exercise on the isokinetic ergometer.

The work level during prior exercise which was equivalent to ~95% of the subjects $\dot{V}O_{2\max}$ was determined in the first run. Here subjects were required to exercise for 6 minutes on the isokinetic ergometer at the highest intensity they could sustain over this period. Pedalling speed was determined by the electric motor which was set to give a crank velocity of 112 rev. min⁻¹. The force exerted on the pedals during this time was visually displayed to the subject on a meter to enable them to maintain this at a constant level. If during the prior exercise period the subject failed to maintain the initial force throughout the entire 6 min then the test was repeated at a lower load which could be sustained.

In subsequent runs where the duration of prior exercise was shortened subjects were required to reach the same target forces to ensure that the relative work load remained the same. Where prior exercise was of 3 or 6 min duration measurements of $\dot{V}O_2$, $\dot{V}CO_2$ and \dot{V}_E were made during the final 2 minutes as described in the section on techniques.

Upon completing the period of prior exercise subjects were then immediately instructed to make a maximal effort for 20 sec during which their maximal short-term power output was determined. For each

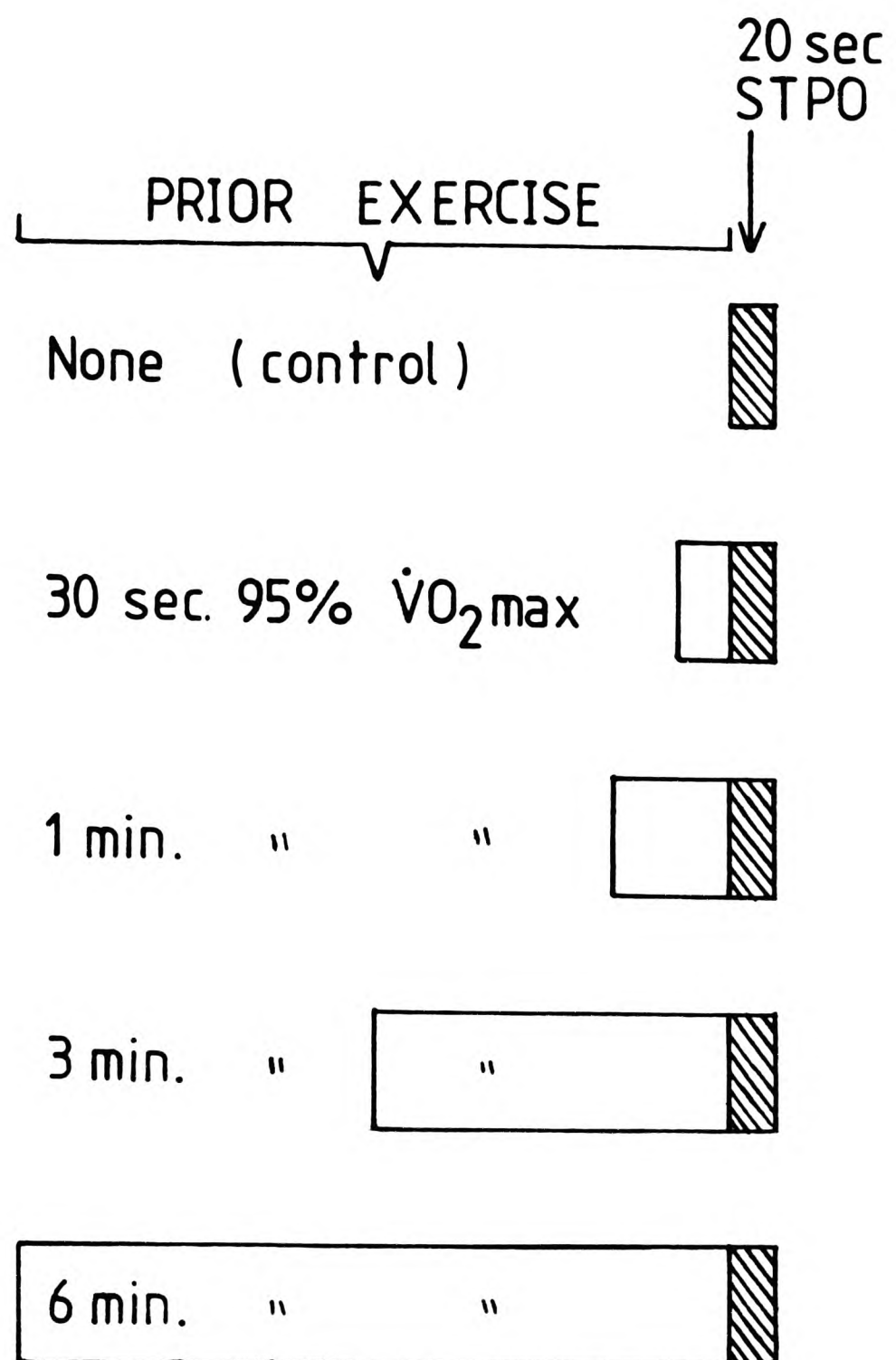


Fig.1 Protocol for Study I showing duration of prior exercise (load $\sim 95\% \dot{V}O_2\text{max}$) before measurement of short-term power output in the 20 second test.

of the short-term power output measurements peak force and peak power during each revolution were determined and maximum values were calculated by taking a mean of the three highest consecutive readings to occur throughout the 20 sec period .

STUDY II

AIM: To examine the effect upon subsequent short-term power output of performing different levels of prior submaximal exercise.

Five healthy adult male subjects whose physical characteristics are shown in Table 2 were studied. An outline of the experimental protocol is shown in Fig.2. As in the first study the short-term power output measurement was initially performed from rest without any prior exercise as a control . In the experimental runs the 20 sec test was immediately preceded by 6 minutes of prior exercise on the isokinetic ergometer at work loads which which ranged from 30-100% of the subjects previously determined $\dot{V}O_2\text{max}$.

As in Study I the force exerted on the cranks during prior exercise was visually displayed to the subjects to enable them to maintain the initial level throughout the 6 min period. Oxygen uptake, carbon dioxide production and ventilation were measured as before during the final 2 minutes of prior exercise.

Upon completing the 6 min of prior exercise subjects were immediately instructed to make an all-out effort for 20 seconds to enable their maximal short-term power output to be assessed.

During each of the 20 second tests peak force and peak power were measured during each revolution and maximum values were determined in

Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_{2\max}$ $l\ min^{-1}$
(1) AT	42	170.8	73.8	66.0	3.90
(2) KD	22	169.3	66.3	58.2	3.76
(3) AS	36	178.7	71.0	62.0	3.20
(4) VW	23	184.0	77.5	73.0	3.61
(5) SM	22	178.6	84.5	72.1	4.81

Table 2 Physical characteristics of the subjects studied.

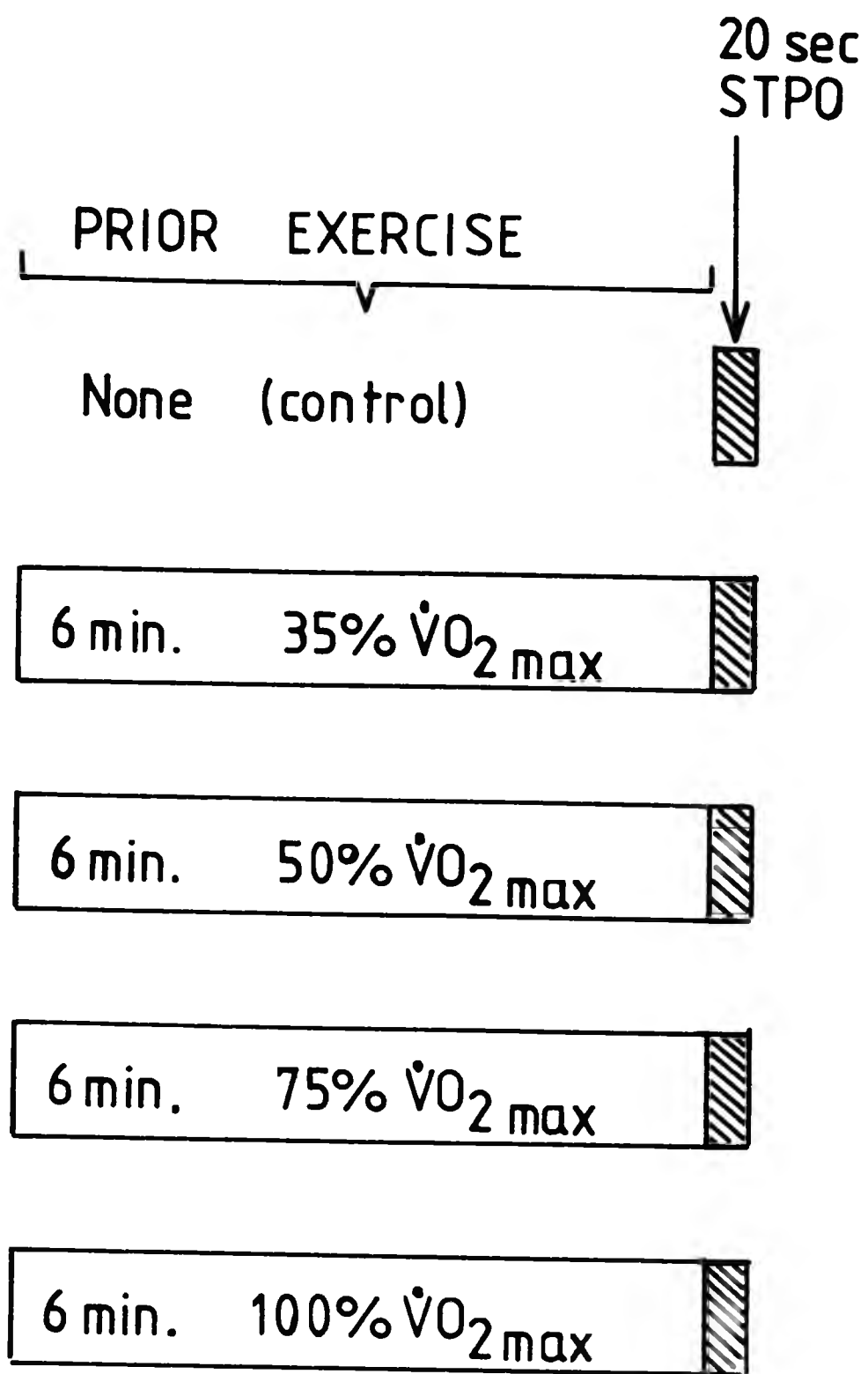


Fig.2 Protocol for Study II showing approximate levels of prior exercise (duration 6 minutes) performed before measurement of short-term power output in the 20 second test.

the same way as before. A fatigue index was also compiled in order to determine the rate of fatigue over the 20 second period. This was done according to the following equation where the 15 second value like the maximum value for peak power was calculated by taking a mean of three consecutive readings .

$$FI = \frac{PP_{max} - PP \text{ at } +15 \text{ sec}}{15} \quad W \text{ sec}^{-1}$$

Measurement Techniques

Prior Exercise

Prior exercise was performed on the isokinetic ergometer at a pre-set pedalling speed of 112 rev min⁻¹. Force exerted on the cranks during this time was measured by means of strain gauges attached to the cranks as previously described (see Chapter 2).

During the final 1-2 min of prior exercise oxygen uptake was measured using a continuous open circuit technique. This involved the subjects breathing through an Otis-McKerrow low resistance mouthpiece which was connected by wide bore tubing on the inspired side to a dry gas meter (Parkinson Cowan CD4). A photoelectric relay fitted to the dial of this meter enabled inspired volume to be read on a digital counter which could be set to count continuously or during successive 15 or 30 second intervals. On the expired side the exhaled air passed from the mouthpiece via similar wide bore tubing to a 5 litre polyethylene bottle designed to produce mixing of the tidal air. Near the outlet of this container a sample line was inserted and air was extracted at 500 ml/min over a drying agent (magnesium perchlorate) and through an infra red carbon dioxide analyser (P.K.Morgan) by an

electric pump. The sample gas was next pumped through a flow meter and finally through a paramagnetic oxygen analyser (Servomex OA 150) before exhausting to atmosphere.

Calibration of the carbon dioxide and oxygen analysers was carried out as previously described (see Chapter 2) before the start of the exercise. Cardiac frequency was measured using lightweight disposable electrodes and this was recorded on an ultraviolet oscillograph recorder (SE Labs.). Using these techniques inspired volume, heart rate, $\dot{V}O_2$ and $\dot{V}CO_2$ were each measured at 30 sec intervals during the final 1-2 minutes of prior exercise. The mean $\dot{V}O_2$ was used as an indication of the exercise intensity since it enabled the work load to be expressed relative to subjects maximal oxygen uptake ($\dot{V}O_{2max}$). The latter was measured directly in separate experiments.

Measurement of Short-Term Power Output

Maximal short-term power output in these studies was measured during 20 seconds of maximal exercise on the isokinetic ergometer as previously described in Chapter 2. All measurements were made at a standard pre-set velocity of $\sim 112 \text{ rev.min}^{-1}$. Force exerted on the cranks during the 20 sec test was measured by strain gauges enabling peak force and peak power for each revolution to be determined. Maximum values of peak force and peak power were determined by taking a mean of the three highest consecutive values to be attained during the test.

Maximal Oxygen Uptake

Maximal oxygen uptake was measured directly during progressive exercise on an electrically braked cycle ergometer as previously described (see Chapter 2). Measurements were made during the final 2 minutes at each work load using standard Douglas bag technique and as before the criterion used to confirm that maximal levels of oxygen uptake were attained was that there was no further increase in $\dot{V}O_2$ with increasing work load. If plateau levels were not achieved in the first run then subjects were brought back another day and further measurements were made but at different supramaximal work loads.

RESULTS

STUDY I

During prior exercise the mean $\dot{V}O_2$ for the 3 minute and 6 minute runs was 95.5 and 93% $\dot{V}O_{2max}$ in SM and PJ respectively. Where prior exercise was of shorter duration the work load was assessed by comparison of the mean peak force with that attained in the longer runs. Table 3 shows the mean peak force measured over 20 consecutive revolutions during each of the prior exercise periods. This proved to be fairly consistent for the different durations of prior exercise, the coefficient of variation being less than 5% in both subjects.

The effect of performing increasing durations of prior exercise upon subsequent short-term power output is shown in Table 3 and Fig.3. Results are shown separately for both subjects. Data points represent peak power for each revolution over the 20 second duration of the short-term power output measurement.

In all experiments maximal peak power during the 20 sec test was attained within a few revolutions from the start after which there was a fall in peak power as the muscle fatigued. An increasing decrement in maximal peak power was observed in both subjects as the duration of prior exercise was increased up to 6 minutes.

When maximal values of peak power were expressed as a percentage of the control value and plotted against the duration of prior exercise a curvilinear relationship was obtained as shown in Fig.4. After only 30 seconds of prior exercise at a work load equivalent to ~95% $\dot{V}O_{2max}$ a decrement in power output of ~10% was observed. (Table

Subject	<u>Prior Exercise</u>			<u>STPO Measurement</u>	
	Duration (secs)	Mean PF (N)	$\dot{V}O_2$ (% $\dot{V}O_{2max}$)	PPmax (Watts)	(% control)
SM	0	-	-	1607	100
	30	250	-	1489	93
	60	260	-	1391	87
	180	249	93	1264	79
	360	244	98	1192	74
PJ	0	-	-	1030	100
	30	193	-	892	87
	60	194	-	810	79
	180	206	88	694	67
	360	187	98	685	67

Table 3 Maximal peak power during the short-term power output measurement at 112 revmin⁻¹. Tests were performed from rest and following different durations of submaximal exercise (work load ~95% $\dot{V}O_{2max}$)

Subject	<u>Prior Exercise</u>			<u>STPO Measurement</u>	
	Duration (secs)	Mean PF (N)	$\dot{V}O_2$ (% $\dot{V}O_{2max}$)	PPmax (Watts)	(% control)
SM	0	-	-	1607	100
	30	250	-	1489	93
	60	260	-	1391	87
	180	249	93	1264	79
	360	244	98	1192	74
PJ	0	-	-	1030	100
	30	193	-	892	87
	60	194	-	810	79
	180	206	88	694	67
	360	187	98	685	67

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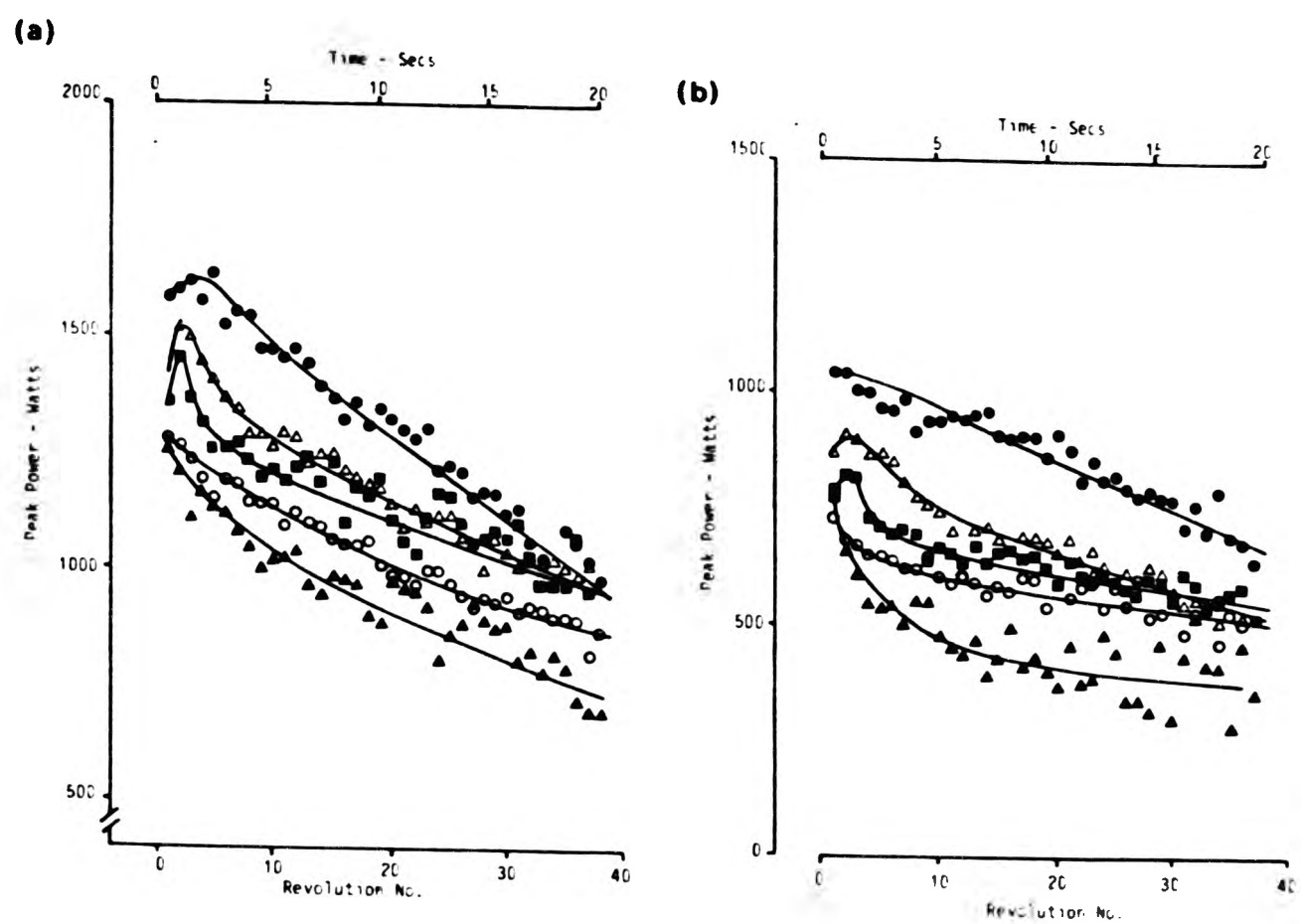


Fig.3 Peak power for each revolution during the 20 second test when performed from rest (●) and following prior exercise of 30 sec (△), 1 min (■), 3 min (○) and 6 min (▲) duration. Data are shown for both subjects (a) S.M., (b) P.J.

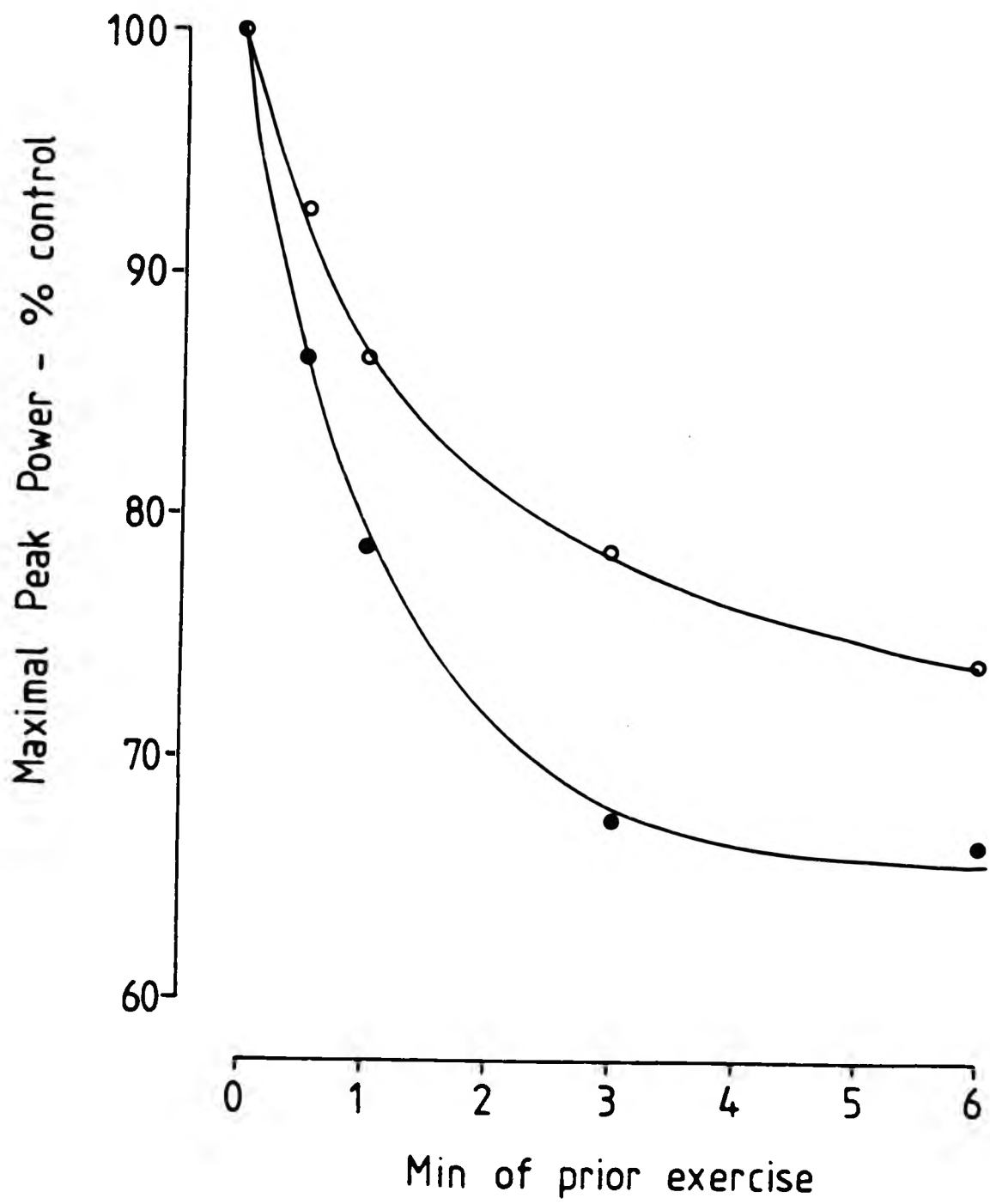


Fig.4 Maximal peak power (expressed as a percentage of the control value) in S.M. (○) and P.J. (●) following different durations of prior exercise.

3). This decrement increased substantially with the duration of prior exercise up to 3 min when maximal peak power had fallen to 67 and 79% of the control value in the two subjects. When the period of prior exercise was extended to 6 min the additional loss in power output was only slight resulting in an overall decrement of ~ 30% compared to 27% after 3 min.

STUDY II

The effect of performing different levels of prior exercise upon subsequent short-term power output is shown in Fig.5 which shows data for one subject, this pattern being typical of the group as a whole. Following prior exercise at 39 and 56% $\dot{V}O_2$ max maximal peak power was increased by 15.0% and 10.5% respectively during the subsequent 20 sec test when compared to the control value. Following higher work loads equivalent to 74 and 80% $\dot{V}O_2$ max a substantial decrement in power output was observed during the maximal test which increased with the prior exercise intensity.

When changes in maximal peak power for all 5 subjects were expressed as a percent of the control value (%PPmax) and plotted against the prior exercise intensity (% $\dot{V}O_2$ max) the results shown in Fig.6 were obtained .

Following the lower levels of prior exercise (<60% $\dot{V}O_2$ max) all 5 subjects showed an increase in PPmax when compared to control values although in one of the subjects this improvement was rather slight (<1%). The greatest increases in PPmax were observed where the prior exercise intensity was between 33-48% $\dot{V}O_2$ max when improvements of 8-15% were achieved in 4 of the 5 subjects.

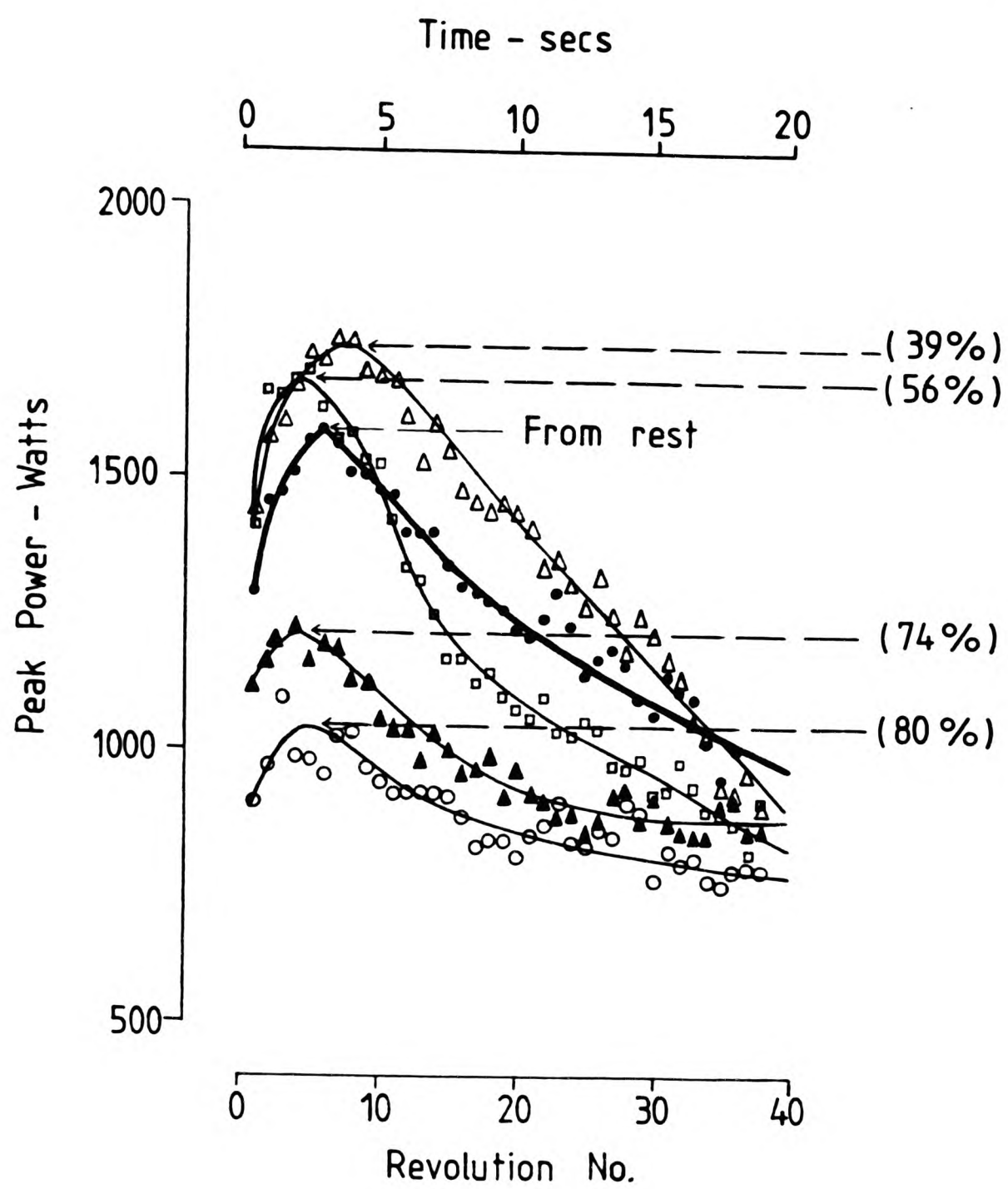


Fig.5 Peak power for each revolution during the 20 second test when performed from rest (●) and following 6 minutes prior exercise at 39 (△), 56 (■), 74 (▲) and 80 (○) % $\dot{V}O_2$ max. Data are shown for one subject (V.W.).

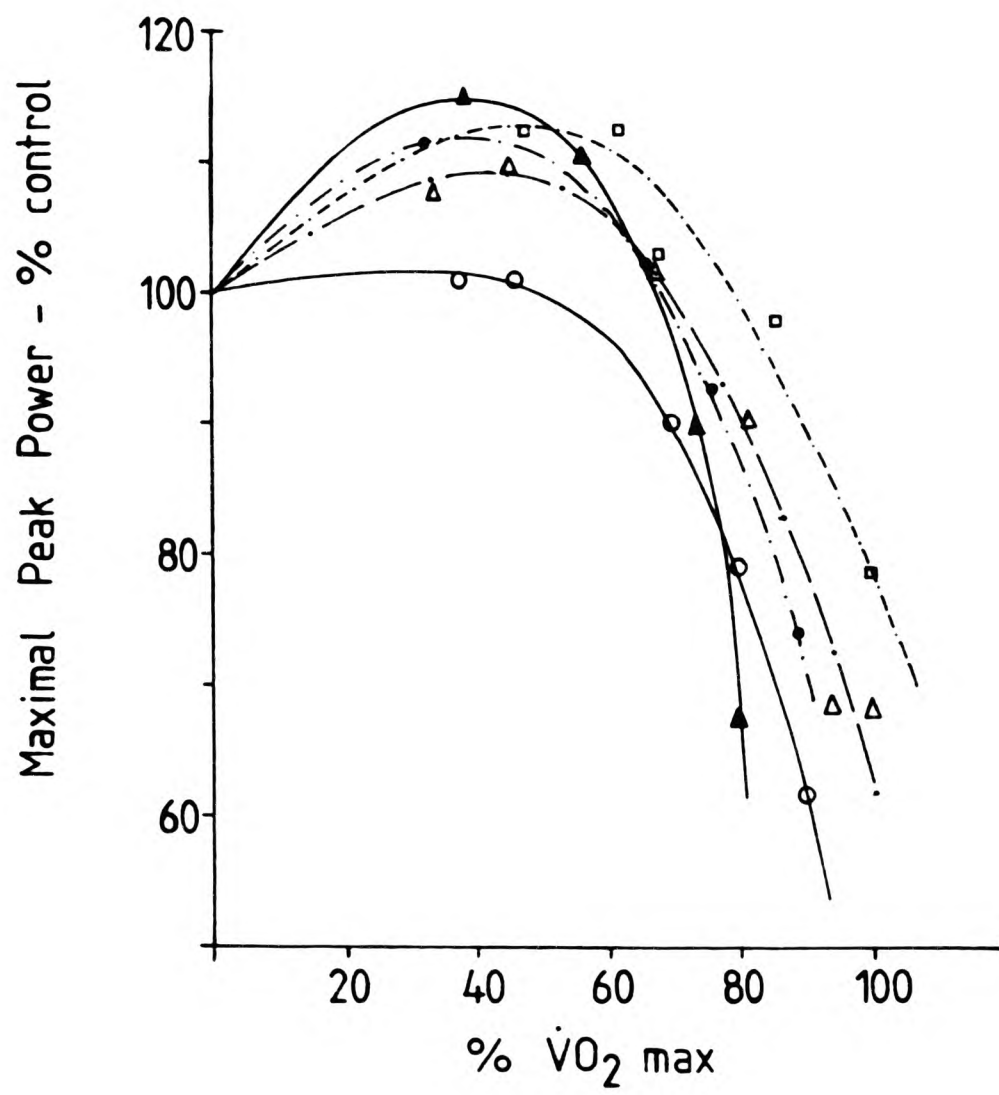


Fig.6 Maximal peak power during the 20 second test (expressed as a percentage of the control value) following different intensities of prior submaximal exercise (% $\dot{V}O_2$ max). Data are shown for 5 subjects.

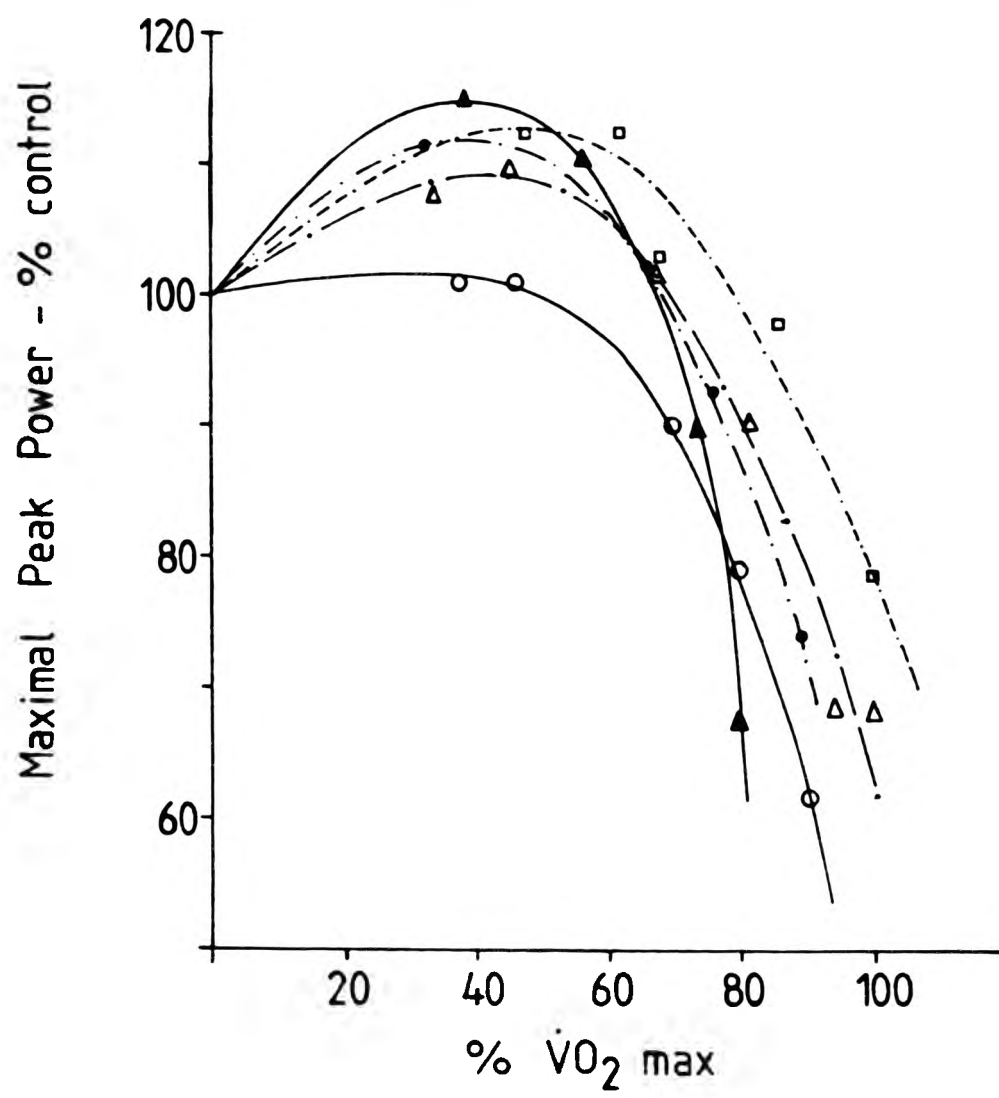


Fig.6 Maximal peak power during the 20 second test (expressed as a percentage of the control value) following different intensities of prior submaximal exercise (% $\dot{V}O_2$ max). Data are shown for 5 subjects.

Following higher work loads an inverse relationship was observed between prior exercise intensity and subsequent short-term power output resulting in a 20-40% fall in PPmax following the heaviest work loads.

The group data has been summarised in Table 4 and Fig.7. In order to calculate a mean relationship data was grouped according to prior exercise intensity in the range 30-45, 45-60, 60-75, 75-90 and 90-100% $\dot{V}O_2\text{max}$. At work loads below 60% $\dot{V}O_2\text{max}$ there was a significant increase ($P < 0.05$) in subsequent short-term power output of ~8.5% when compared to control values. Following higher levels of prior exercise the maximal power attained in the 20 sec test was inversely related to the previous work load. The relationship between the two could be described by the following regression equation;

$$y = 165.35 - 0.989x \quad (r = -0.7895, n = 16, p < 0.001)$$

where $y = \text{PPmax (\% control)}$

and $x = \text{prior exercise intensity (\% } \dot{V}O_2\text{max)}$.

This equation predicts that the greatest loss in maximal peak power following prior exercise at 100% $\dot{V}O_2\text{max}$ should be of the order of 34% in the present group of the subjects.

When a fatigue index was calculated to show the loss in power output over the duration of the 20 sec test this was found to vary with the prior exercise intensity. In general the higher the value of PPmax attained at the start of the test the greater was the subsequent rate of fatigue over the remainder of the 20 sec period (Table 4, Fig. 8).

Subject	Prior Exercise Intensity	PPmax		Fatigue Index	
	% $\dot{V}O_2$ max	Watts	% control	W.sec ⁻¹	% sec ⁻¹
KD	-	942	100	12.7	1.35
SM	-	1462	100	24.2	1.62
AS	-	1480	100	41.8	2.73
AT	-	1354	100	34.7	2.48
VW	-	1510	100	33.0	2.11
Mean				29.7±11.4	2.06±0.58
KD	33.8	1015	107.6	14.1	1.39
AS	38.1	1494	100.8	51.1	3.42
AT	32.6	1509	111.4	32.6	2.16
VW	38.8	1738	115.1	49.3	2.84
Mean	35.8±3.1		108.7±6.1	36.8±17.3	2.45±0.88
KD	45.5	1034	109.6	22.8	2.20
SM	47.8	1642	112.3	28.2	1.71
AS	46.3	1496	100.9	33.2	2.22
VW	56.2	1669	110.5	48.8	2.92
Mean	49.0±5.0		108.4±5.1	33.3±11.2	2.27±0.50
KD	67.6	928	98.4	16.6	1.78
SM	61.8	1643	112.4	29.7	1.81
SM	68.2	1505	103.0	21.4	1.42
AS	70.0	1335	90.2	40.7	3.05
AT	66.2	1386	102.3	30.4	2.20
VW	73.7	1359	90.0	32.1	2.37
Mean	67.9±4.0		99.4±8.5	28.5±8.5	2.11±0.57
KD	81.4	854	90.5	13.8	1.61
SM	86.1	1433	98.0	23.6	1.65
AS	80.0	1171	79.1	29.1	2.48
AT	76.4	1255	92.7	24.6	1.96
AT	88.7	1003	74.2	11.7	1.17
VW	79.8	1018	67.5	15.3	1.51
Mean	82.1±4.5		83.7±11.9	19.7±7.0	1.73±0.45
KD	94.2	647	68.7	10.3	1.58
KD	100.0	642	68.3	8.6	1.30
SM	100.0	1152	78.8	23.2	2.02
AS	90.3	909	61.6	25.5	2.81
Mean	96.1±4.8		69.3±7.1	16.9±8.7	1.93±0.66

Table 4 Maximal peak power (PPmax) and rate of fatigue during the 20 second maximal test. Measurements were made at a crank velocity of 112 rev min⁻¹ from rest (control) and following 6 min prior exercise at a number of different work loads.

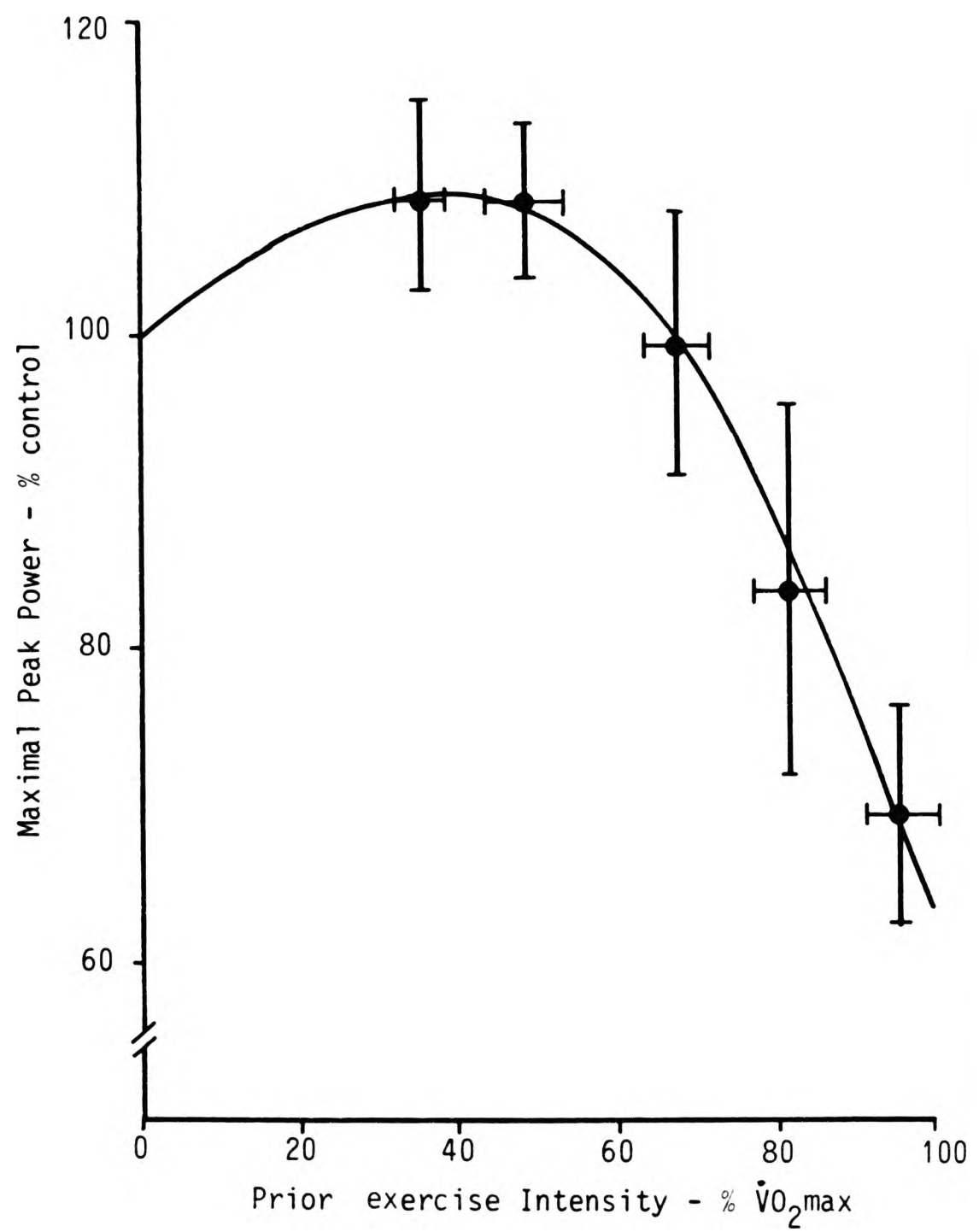


Fig.7 Maximal peak power during the 20 second test (% control) following different intensities of prior submaximal exercise (% $\dot{V}O_{2max}$). Mean values \pm one SD are shown (see Table 4).

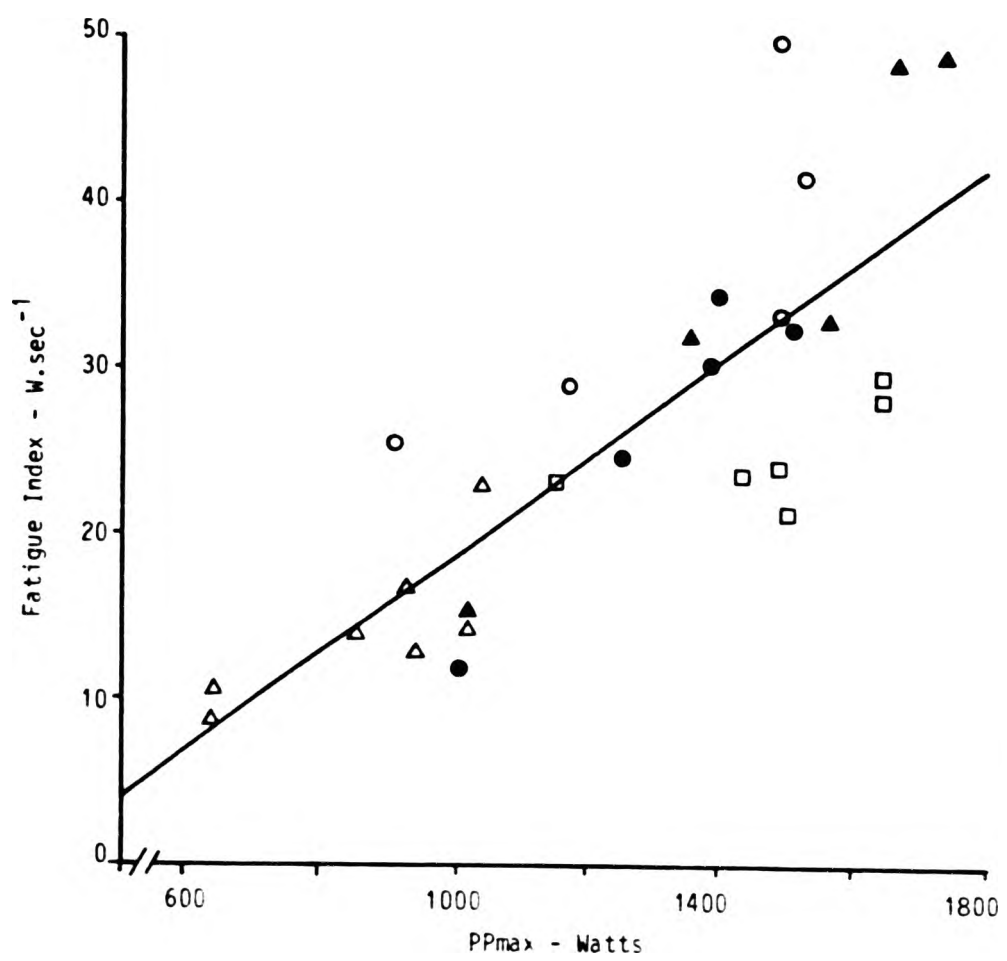


Fig.8 Linear regression line based on data from 5 subjects showing relationship between maximal peak power and the subsequent rate of fatigue during the 20 second test (n = 16; r = -0.7895; p < 0.001).

DISCUSSION

This study set out to examine the effect of prior exercise of varying duration and intensity upon subsequent short-term power output from human muscle.

In the first part of the study an increasing decrement in short-term power output was observed as the duration of prior exercise was increased from 30 seconds to 3 minutes. The additional loss of power incurred by extending the time of prior exercise from 3 to 6 minutes was only small. This suggests the attainment of a 'steady state' condition during the latter 3 minutes of prior exercise in which equilibrium is reached between the breakdown and resynthesis of metabolic substrate.

These isokinetic measurements confirm the earlier work of Margaria et al (1971) who measured maximal power output during a freely accelerating stair run. Consistent with the present results they found that the decrement in power output following submaximal exercise was little affected by increasing the duration of this exercise beyond 3 minutes.

On the basis of these results the effect of constant load prior exercise upon subsequent short-term power output from human muscle appears to be maximal after approximately 3-6 minutes of such exercise. Thus in the second study which set out to examine the effect of prior exercise intensity upon subsequent short-term power output all measurements of power output were made following prior exercise of 6 minutes duration.

In this second study an inverse relationship between prior exercise intensity and subsequent short-term power output was observed when the former exceeded 60% $\dot{V}O_2$ max. These results are in agreement with the findings of Margaria et al (1971) who measured maximal anaerobic power using a stair run technique and reported an increasing decrement in power output with increasing level of prior submaximal exercise.

When Margaria calculated the energy during the anaerobic test which was due to the splitting of high energy phosphate over and above that involved in the previous aerobic work he found that this decreased in a linear fashion with the intensity of the previous exercise.

Comparable findings have been made in biochemical studies in which the concentration of high energy phosphate in human muscle during exercise was determined by biopsy (Hultman et al, 1967; Karlsson et al, 1971; Karlsson, 1971; Karlsson and Saltin, 1970; Knuttgen and Saltin, 1972). In these studies a direct relationship was found between the extent of high energy phosphate depletion and the intensity of the previous work load where this was between 60-100% of the individuals $\dot{V}O_2$ max. The reduction in high-energy phosphate was much less or virtually nil at lower work loads. In general most of the depletion could be accounted for by a reduction in the PC concentration since appreciable decreases in ATP were usually only observed when phosphorylcreatine had become depleted to a critically low level which appeared to be between 20-40% of its resting value.

In a more recent study using ^{31}P nuclear magnetic resonance

(n.m.r.) Dawson et al (1978) also found that the decrease in force during repeated fatiguing contractions of frog gastrocnemius muscle was closely correlated with a fall in the PC concentration although the ATP concentration showed little change until fatigue was very advanced.

This depletion of phosphorylcreatine during exercise may be responsible for the decrements in power output observed in the present study. The loss of power observed in the first part of the study may reflect a progressively greater depletion of PC in the active muscle fibres as exercise continues. On this basis an equilibrium between the rate of PC breakdown and resynthesis appears to be reached after approximately 3 minutes since the effect of extending the duration of prior exercise beyond this time is rather small. Thus for a given submaximal work load the extent of PC depletion would appear to be maximal after approximately 3 minutes of exercise. Indeed Hultman et al (1967) who measured ATP and PC in human muscle during various stages of dynamic exercise observed a rapid decrease in high-energy phosphate during the first 2 min after which they reported very little change.

The inverse relationship observed in the second part of the study between prior exercise intensity (when this exceeded $60\% \dot{V}O_2\text{max}$) and subsequent short-term power output may be compared with the fall in PC which occurs at work loads above $55\% \dot{V}O_2\text{max}$ (Hultman et al, 1967; Karlsson et al, 1971).

Presumably the concentration of unsplit high energy phosphate in muscle depends at any given time upon the equilibrium of the creatine

kinase reaction between ATP and PC. On the basis of the above findings this would appear to shift towards lower levels of PC as the work load increases in order to maintain a constant level of ATP in the working muscles.

Since the creatine kinase reaction between ATP and PC represents the most rapid method of resynthesising ATP available to the contracting muscle a decrease in the concentration of PC could have a marked effect on the muscles capacity for rapid ATP regeneration. One way in which such a decrease in the rate of ATP production might effect tension development is through a decrease in the rate of cross-bridge cycling since both the formation and detachment of cross-bridges are processes which require ATP. The re-uptake of Ca^{2+} ions by the sarcoplasmic reticulum during relaxation also depends upon the splitting of ATP and a decrease in the rate of this process or in the rate of cross-bridge detachment would lead to a prolongation of the relaxation time.

A number of authors (Sjöholm, Sahlin, Edstrom and Hultman, 1983; Wiles and Edwards, 1982) have reported a pronounced prolongation of relaxation time at fatigue following electrical stimulation. The same investigators also found that the normalization of relaxation rate during recovery followed a strikingly similar time course to the resynthesis of PC. The effect of depleting the PC store may therefore be to prolong relaxation time so that the rate of cross bridge-cycling and hence the power which can be generated by the muscle are decreased.

While this would appear to be a very plausible explanation for the loss of power observed in the present study the effect of other

factors should not be ruled out. A number of workers have found a direct relationship between lactate accumulation and the decrease in PC in human muscle following both isometric and dynamic exercise (Harris, Sahlin and Hultman, 1977) and it is possible that the decreases in short-term power output reported above may be due to elevated concentrations of lactic acid as a result of the previous exercise.

Margarita, Cerretelli, Di Prampero, Massari and Torelli (1963) measured plasma lactate following exercise which lead to exhaustion in 1-10 min. On the basis of their findings they suggested that appreciable increases in lactate production did not occur at work loads below 100% $\dot{V}O_2$ max. However in a number of later studies Knuttgen and Saltin (1972) and Karlsson et al (1971) measured muscle lactate over a range of submaximal work loads between 19-100% $\dot{V}O_2$ max. Although they reported little or no increase in lactate accumulation at work loads below 60% $\dot{V}O_2$ max they found it to be considerably elevated in relation to the work level at the higher intensities with values of 15-25 mmoles kg^{-1} wet weight being obtained during maximal exercise.

At physiological pH increases in lactate are accompanied by an equivalent (equimolar) production of H^+ and the resultant fall in pH has long been proposed to limit performance during maximal exercise of brief duration.

Decreases in pH have previously been associated with a reduced rate of lactate efflux from the muscle (Mainwood and Worsley-Brown, 1975) and a decreased rate of muscle glycolysis due to inhibition of

phosphofructokinase (Trivedi and Danforth, 1966) either of which could contribute to a loss of force and hence power output during maximal exercise.

Nakamura and Schwartz (1972) and Fuchs, Reddy and Briggs (1970) found that the binding of Ca^{2+} ions to troponin during excitation-contraction coupling was also reduced in the presence of high concentrations of hydrogen ions and this too could have a marked effect on tension development.

Although pH was not determined in the present study previous investigations have shown it to be considerably reduced at exercise levels above 60% $\dot{V}O_2$ max with values falling as low as 6.4 following maximal work loads (Sahlin, Harris and Hultman, 1975). Hence in the present experiments muscle pH may have been sufficiently reduced following the higher levels of exercise to bring about such an inactivation of Ca^{2+} binding during the contraction process resulting in a reduction in cross-bridge formation and hence a loss of contractile force.

In biochemical studies changes in pH have also been shown to have an effect on the activity of creatine kinase. Noda, Kuby and Lardy (1954) and more recently Sahlin et al (1975) found that the creatine kinase equilibrium in muscle was shifted towards lower concentrations of PC as the intracellular pH decreased. This suggests that the extent of PC depletion during exercise may depend to a large extent upon changes in pH.

Since increases in lactate concentration at work loads below 50-60% $\dot{V}O_2$ max appear to be small the decrease in pH and hence the shift

in the creatine kinase equilibrium should only be slight. At higher work loads increases in muscle lactate through their effect on pH would be expected to shift the creatine kinase equilibrium towards lower levels of PC as the exercise intensity increased. This might explain an earlier observation made by Knuttgen and Saltin (1970) who found that the reduction in PC following steady state exercise below 60% $\dot{V}O_2$ max was only slight when compared to the higher work loads.

These findings could also have important implications with regard to the present results since a shift in the creatine kinase equilibrium towards increasingly lower levels of PC following the higher levels of prior exercise (> 60% $\dot{V}O_2$ max) may account for the decrease in maximal power output which resulted. However since changes in PC, pH and lactate in human muscle during exercise all appear to be correlated it is not clear which if any of these factors is mainly responsible for the loss of power.

In Study II the lower levels of prior exercise were found to produce an increase in subsequent short-term power output. This could be due to a number of factors. As mentioned earlier the energy derived from glycolysis and oxidative phosphorylation at the start of the maximal test is likely to be only small compared to that obtained from the splitting of high-energy phosphate already available in the active muscle fibres. During prior exercise however the rate of both glycolysis and oxidative phosphorylation will increase to a new steady state level above that at rest in order to meet the new energy requirement. Even at the lower work loads where there is no net

accumulation of lactate (Karlsson et al, 1971; Knuttgen and Saltin, 1972) there may still be an increased rate of glycolysis if this is accompanied by an increased rate of lactate efflux. The turnover of ATP via glycolysis and oxidative phosphorylation will therefore be higher following prior exercise than at rest. Consequently there may be an increased contribution albeit a small one from both aerobic and anaerobic metabolism to the ATP supply at the start of the 20 sec maximal test as a result of previous exercise.

Another factor which could effect the capacity to generate power during maximal exercise of brief duration is muscle temperature. A number of workers have found performance during short-term maximal exercise such as the 100m dash (Simonson, Teslenko and Gorkin, 1936), 50m swims (Schmid, 1947) and sprint tests on a cycle ergometer (Asmussen and Boje, 1945; Binkhorst, Hoofd and Vissers, 1977; Bergh and Ekblom, 1979) to be improved at elevated muscle temperatures and in some cases a direct linear relationship between performance capacity and muscle temperature was observed (Asmussen and Boje, 1945; Bergh and Ekblom, 1979).

Asmussen and Boje also found that in their subjects improvements in performance were similar independent of whether the increase in muscle temperature was achieved by active or passive methods indicating that the beneficial effect they observed was preponderantly a temperature effect and not the result of circulatory, respiratory or hormonal changes which may have occurred as a result of the previous exercise.

The improvements in power output observed in the present

investigation following the lower levels of prior exercise may be due to such an elevation of muscle temperature since significant increases in this have previously been reported even after light exercise (Asmussen and Boje, 1945; Saltin, Gagge and Stolwijk, 1968). Such increases in power output would necessarily involve an increased utilisation of ATP at the start of the maximal test which might also account for the more rapid rate of fatigue observed in these runs.

It should be pointed out however that a number of investigators have found warming-up to have little or no effect upon subsequent performance despite considerable increases in muscle temperature (Hipple, 1955; Karpovich and Hale, 1956; Skubic and Hodgkins, 1957). It may be that the effects of warming-up depend to a large extent upon both the the type of warm-up and the exercise used to assess its effects. The effect of both passive and active warm-up is therefore examined more fully in a later chapter of this thesis.

Conclusions

The present results indicate that at work loads above 60% $\dot{V}O_2$ max steady state prior exercise results in a fall in subsequent short-term power output which becomes more marked as the intensity of the previous exercise is increased . The underlying causes of the observed functional decrement may be lack of metabolic substrate (ATP and PC), changes in intramuscular pH or lactate, or other factors altered as a result of the previous exercise. The fact that a number of workers have found ATP concentrations to be virtually unchanged during fatigue where marked decrements in force were apparent (Harris et al, 1976; Wilkie, 1981) suggests that it may be the rate of ATP turnover

which limits tension development and not the prevailing concentration of ATP at any given time which appears to remain constant. Since the creatine kinase reaction is the most rapid method of resynthesing ATP the loss in power output observed in the present investigation may therefore reflect changes in the equilibrium between high energy phosphate breakdown and resynthesis which may be mediated in turn by changes in pH.

The improvements in power output which were attained following the lower levels of prior exercise ($< 60\% \dot{V}O_2\text{max}$) may be explained at least in part by changes in muscle temperature since a number of workers have reported an increased performance capacity during maximal exercise of brief duration as a result of previous warming-up.

CHAPTER 4

The Recovery in Maximal Short-Term
Power Output Following
Steady State Exercise.

INTRODUCTION

As described in the previous chapter the decrement in power output following steady state exercise appears to be related to the relative work load where this is above 60% $\dot{V}O_2$ max. This loss of power may be related to changes in muscle lactate, PC or pH as a result of the previous exercise. Information regarding which, if any of the above factors is responsible for the impaired performance might be obtained by following the recovery in short-term power output after exercise. It may be that this follows a similar time course to the resynthesis of some substrate or the removal of some metabolic end product.

In previous investigations the resynthesis of PC following exercise has been shown to be a very rapid process (Hultman, Bergstrom and McLennan-Anderson, 1967; Karlsson, Funderburk, Essen and Lind, 1975; Harris et al, 1976) while the recovery in pH and muscle lactate appear to follow a slower time course (Sahlin, Harris, Nylinde and Hultman, 1976). However the functional implications of these findings with regard to maximal performance capacity has not to the authors knowledge been previously examined.

It was the purpose of the present study to follow the recovery in maximal short-term power output after a fatiguing exercise and to determine the time-course of such recovery in human muscle.

METHODS

The subjects studied were four healthy adult males whose physical characteristics are given in Chapter 3 (Table 2 subjects 2-5).

An outline of the experimental procedure is shown in Fig.1. Short-term power output was determined at a standard velocity of ~ 112 rev.min⁻¹ during 20 sec of maximal exercise on the isokinetic ergometer as previously described. Initially this was measured from rest as a control. In subsequent runs subjects were required to perform 6 min prior exercise on the isokinetic ergometer at the highest intensity they could sustain over this period.

As in the previous study force exerted on the cranks during this time was visually displayed to the subject on a meter to help them maintain this at a constant level throughout the 6 min period. This force was recorded on an ultraviolet oscillograph as previously described to allow the average force during prior exercise to be determined.

Oxygen uptake, carbon dioxide production , ventilation and heart rate were measured during the final 2 minutes of prior exercise, all gas analyses being made using a continuous open circuit technique as described in Chapter 3.

Upon completing the period of prior exercise subject's short-term power output was measured during the 20 sec test either immediately after the prior exercise period or following rest intervals of 5 sec to 6 min duration.

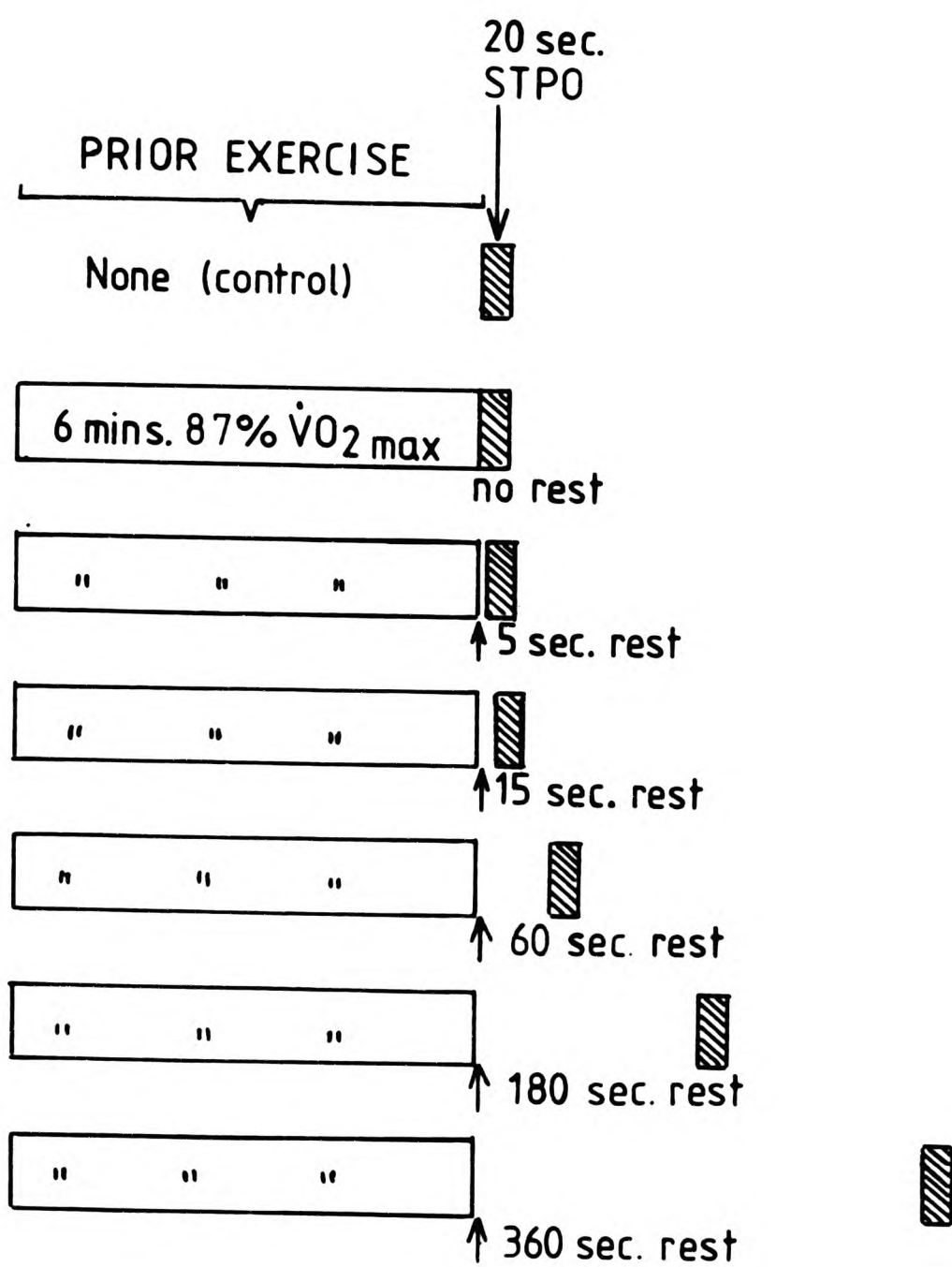


Fig.1 Protocol showing duration of recovery interval interposed between prior exercise (load $\sim 87\% \dot{V}O_2$ max) and the subsequent short-term power output measurement.

For each of the short-term power output measurements peak force and peak power during each revolution were determined and maximum values calculated as previously described. A fatigue index was also compiled using the method outlined in Chapter 3 which involved determining the mean rate of fatigue in the 15 sec period after maximal peak power was attained.

RESULTS

Maximal Short-term Power Output

The mean work load during prior exercise based on results from all four subjects over the complete series of experiments was equivalent to $87.8 \pm 8.6\% \dot{V}O_{2\max}$.

Maximal short-term power output measured immediately after prior exercise showed a marked decrement of the order of 32% (range 26 - 38%) when compared to control values (Table 1, Fig.2). In the remaining runs however maximum values of peak power in the 20 sec test increased very rapidly as the recovery period was lengthened as shown in Fig.2 where maximal peak power expressed as a percent of the control value (% PPmax) is plotted against the duration of recovery.

All 4 subjects showed an initial rapid increase in PPmax with recovery time although the rate varied somewhat between individuals. By 6 min however all 4 subjects had recovered beyond their initial control value showing improvements in PPmax of between 4.5 - 13.3%.

Mean values of maximal peak power based on results from all 4 subjects were determined for each recovery interval to give the relationship shown in Fig.3. This indicated that recovery in short-term power output approximated to an exponential process with a half-time of ~32 sec. Initially recovery was very rapid , so that control values of maximal peak power (PPmax) were attained again within one minute of terminating prior exercise. At 6 min there was a mean

Subject	Prior Exercise	Recovery Time	PPmax		Fatigue Index	
	% $\dot{V}O_2$ max	secs	Watts	% Control	W sec ⁻¹	% sec ⁻¹
KD	-	-	1036	100	25.4	2.5
AS	-	-	1481	100	41.8	2.7
AT	-	-	1357	100	34.7	2.5
VW	-	-	1507	100	33.0	2.1
Mean	-	-			33.7	2.4
					±6.7	±0.2
KD	100	0	642	68.3	8.6	1.3
AS	90	0	909	61.6	25.5	2.8
AT	89	0	1003	74.2	11.7	1.2
VW	80	0	1017	67.4	15.3	1.5
Mean	90	0	893	67.9	15.3	1.7
	±8.3			±5.2	±7.4	±0.7
KD	99	5	737	71.2	9.4	1.3
AT	67	5	1446	106.4	28.6	2.0
VW	94	5	1122	74.6	24.3	2.2
Mean	87	5	1102	84.1	20.8	1.8
	±16.9			±19.4	±10.1	±0.5
KD	96	15	886	85.6	14.2	1.7
AS	85	15	1487	100.4	54.4	3.7
AT	80	15	1186	87.5	24.6	2.1
VW	86	15	1281	85.1	30.1	2.4
Mean	87	15	1210	89.7	30.8	2.5
	±6.7			±7.3	±17.0	±0.8
KD	91	60	1001	96.5	23.0	2.3
AS	88	60	1600	108.0	48.0	3.0
AT	98	60	1548	114.2	42.1	2.7
VW	97	60	1223	81.0	31.0	2.5
Mean	94	60	1343	99.9	36.0	2.6
	±4.9			±13.1	±11.2	±0.3
AS	86	180	1575	106.2	58.9	3.7
AT	72	180	1576	116.0	36.3	2.3
Mean	79	180	1575	111.1	47.6	3.0
	±9.5			±6.9	±16.0	±1.0
KD	94	360	1129	108.8	22.5	2.0
AS	80	360	1548	104.5	37.6	2.4
AT	86	360	1513	111.6	38.8	2.6
VW	87	360	1711	113.3	51.8	3.0
Mean	87	360	1475	109.6	37.7	2.5
	±5.7			±3.8	±12.0	±0.4

Table 1 Maximal peak power (PPmax) and rate of fatigue during the 20 second maximal test. Measurements were made at 112 rev min⁻¹ during recovery from prior exercise (work load ~87% $\dot{V}O_2$ max).

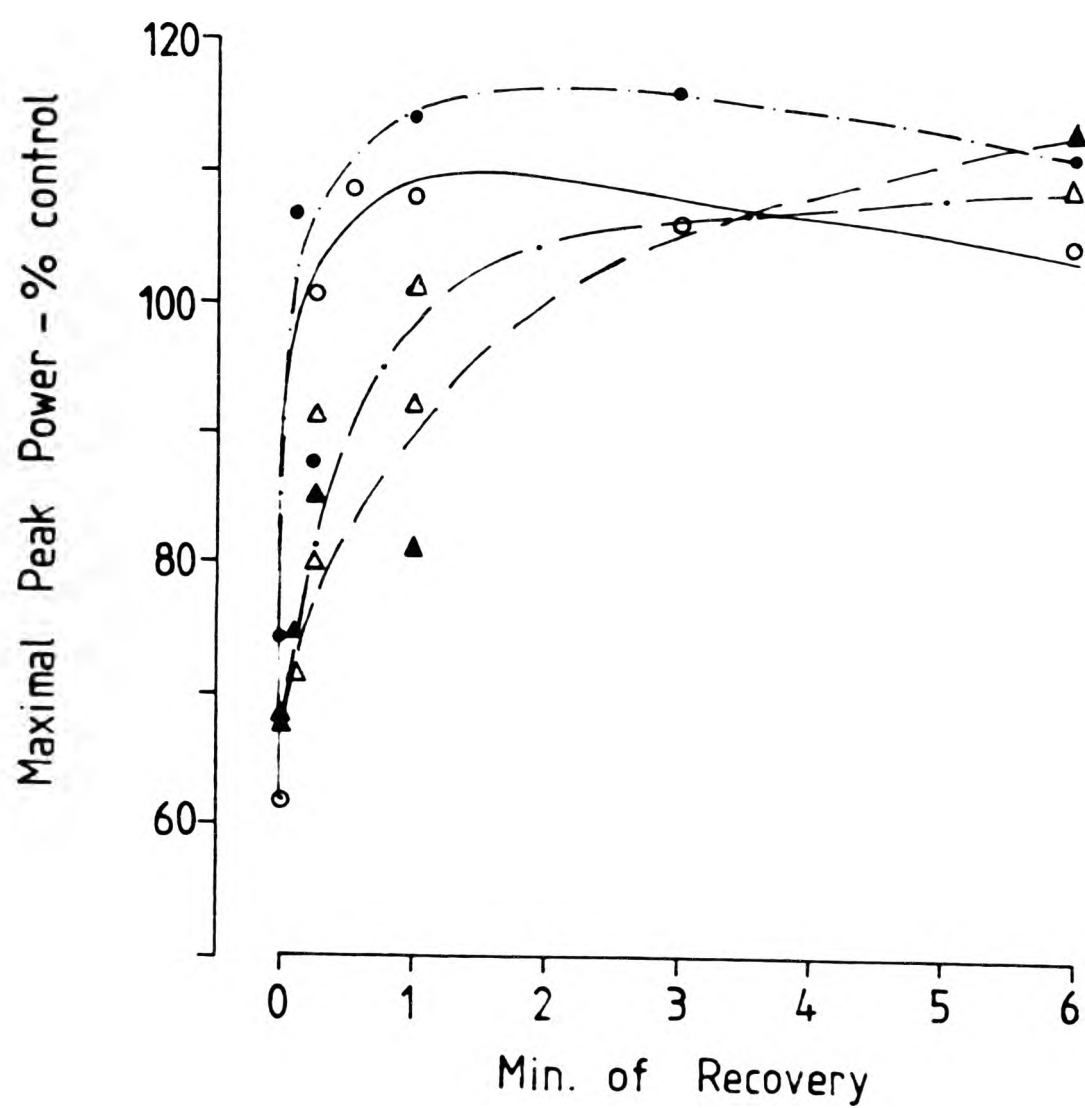


Fig.2 Time course of recovery in maximal peak power (expressed as a percentage of the control value) following 6 minutes prior exercise (load ~87 % $\dot{V}O_2$ max). Data are shown for the 4 subjects studied.

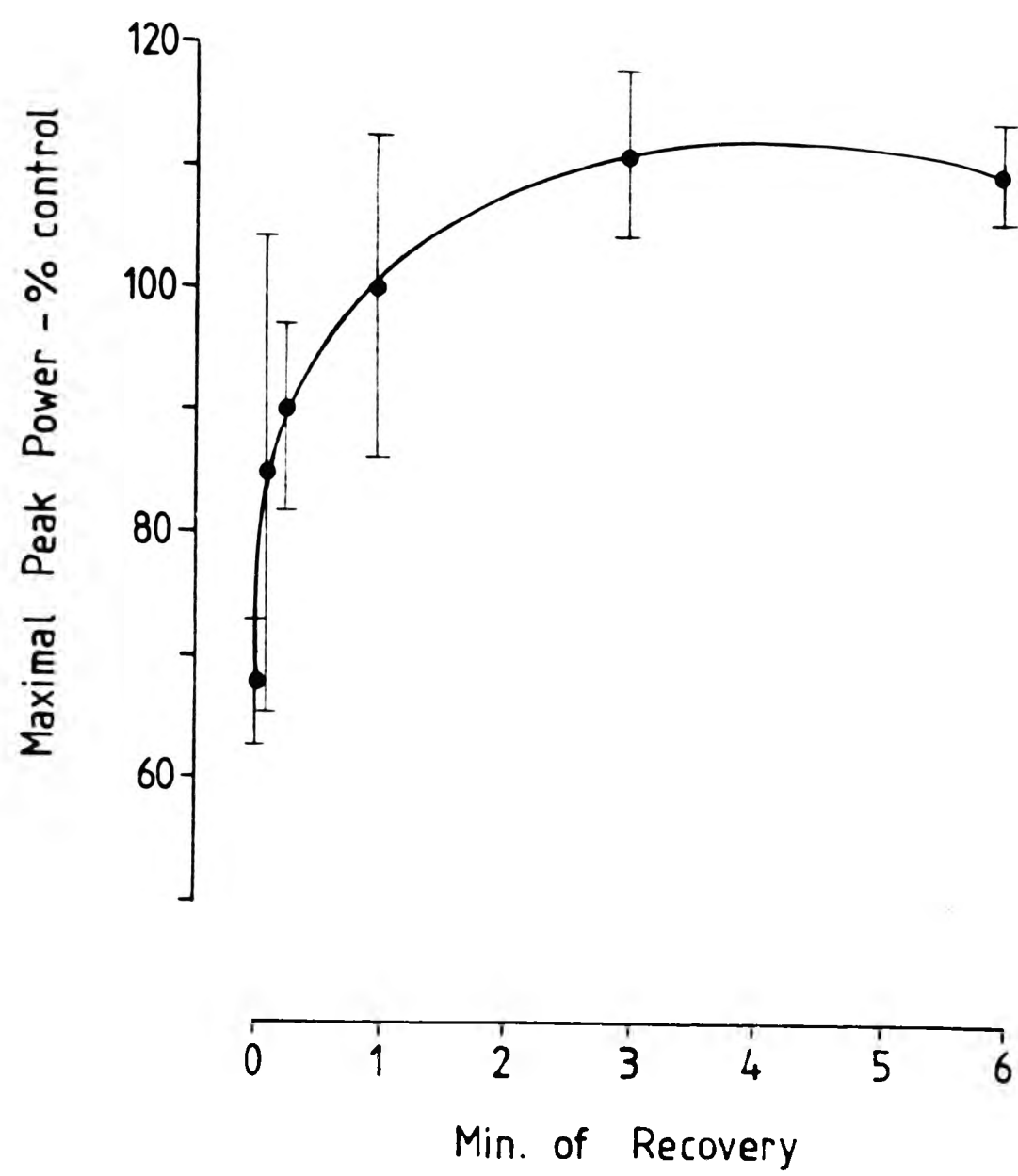


Fig.3 Time course of recovery in maximal peak power following 6 minutes prior exercise. Mean values \pm one SD are shown for the 4 subjects studied.

increase in power output of $9.6 \pm 3.8\%$ which was significant ($p < 0.05$) when compared to control values.

Rate of Fatigue

Immediately after prior exercise the fatigue index during the maximal test was substantially reduced when compared to control values. However as maximal peak power attained in the 20 sec test increased during recovery so too did the rate of fatigue over the remainder of the test as shown by an increase in the fatigue index. This was true whether the rate of fatigue was expressed in absolute terms as $W \text{ sec}^{-1}$ or as a percent of the maximum value of peak power attained at the start of the test (Table 1).

When rate of fatigue was plotted against the recovery period it appeared to follow a very similar pattern to the recovery in short-term power output following prior exercise (Fig. 4). The muscle therefore became more fatiguable as its capacity to generate power recovered. Hence the rate of fatigue during the 20 sec test increased from a mean value of $15.3 \pm 7.4 W \text{ sec}^{-1}$ ($1.7 \pm 0.7\% \text{ sec}^{-1}$) immediately after prior exercise to $37.7 \pm 12.0 W \text{ sec}^{-1}$ ($2.5 \pm 0.4\% \text{ sec}^{-1}$) after 6 minutes of recovery.

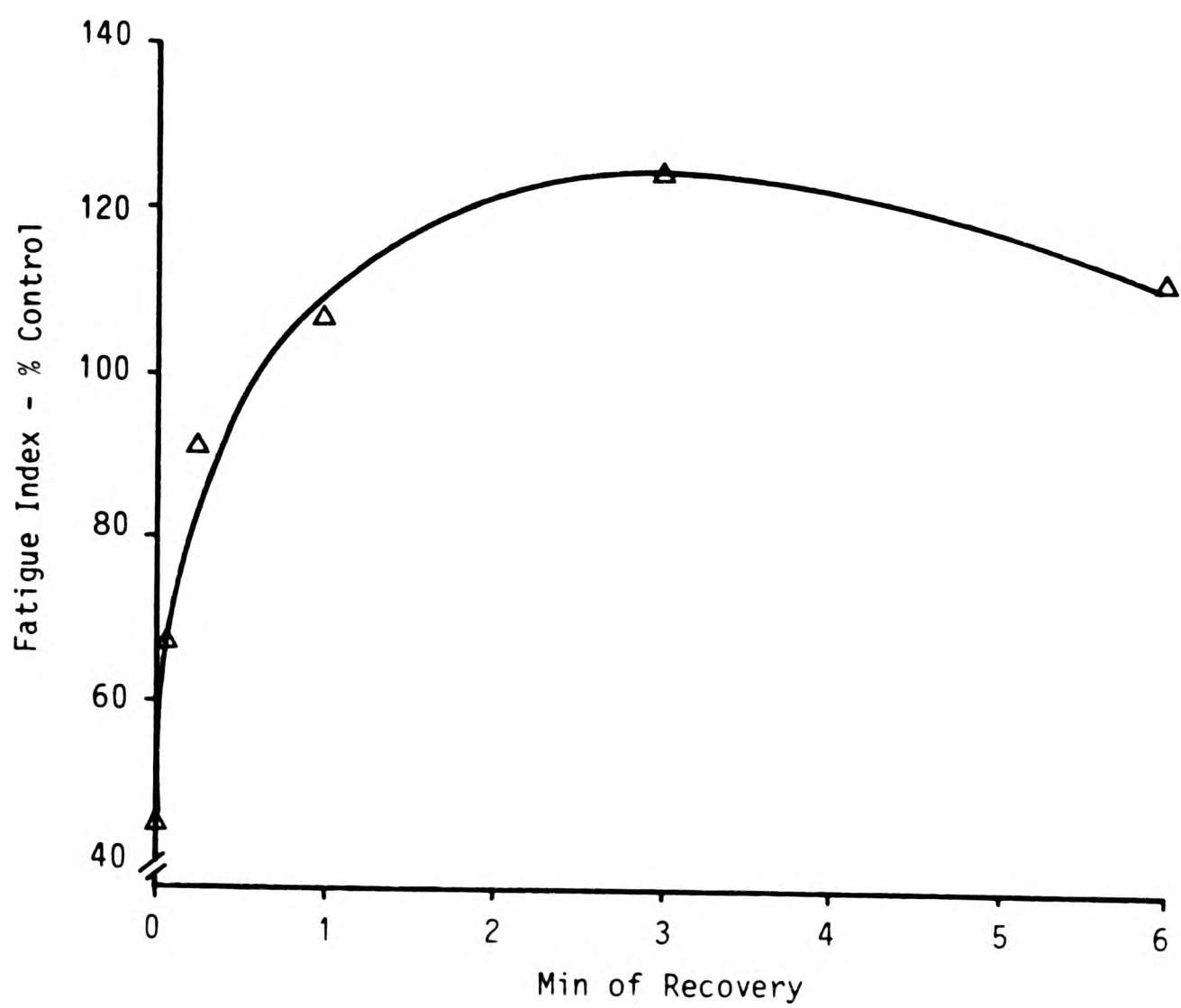


Fig.4 Relationship between rate of fatigue during the 20 second test (expressed as a percentage of the control value) and duration of recovery following 6 minutes prior exercise at $\sim 87\% \dot{V}O_{2max}$.

DISCUSSION

The recovery in dynamic performance capacity following previous steady state exercise has not to the author's knowledge been previously examined in man. In the present study recovery in maximal short-term power output was shown to be a very rapid process with 3 of the 4 subjects achieving complete recovery within approximately one minute of terminating the previous exercise. By 6 minutes of recovery all 4 subjects had improved their power output beyond the repayment of the decrement when compared to control values. The mean increase was $9.6 \pm 3.8\%$.

Individual variation in the rate of recovery during the initial stages may have been due at least in part to the fact that the subjects differed somewhat in the intensity of prior exercise that they could sustain over the 6 min period. Hence one subject over the series of experiments achieved a mean work load during prior exercise which was equivalent to ~95% of his maximal oxygen uptake ($\dot{V}O_{2\max}$) compared to a mean work load in another individual which was equivalent to only 82% of his $\dot{V}O_{2\max}$. The latter subject also showed the most rapid recovery in PPmax following exercise which might reflect the lower initial decrement observed in this subject when compared to the others.

When a mean relationship was derived to describe the pattern of recovery in maximal peak power this appeared to follow an exponential time course with a $t_{1/2}$ of approximately 32 seconds. These results may be compared with the findings of Stull and Clarke (1971) who investigated the recovery in hand-grip strength in man following

repeated maximal contractions and found that the strength score of their subjects had returned to ~98% of the initial control value after only 70 sec rest. These workers also observed a further improvement when the recovery period was extended so that after 235 sec rest subjects showed a 7% increase in hand-grip strength above the initial control value.

On closer examination of their results these workers found the recovery in maximum strength following dynamic exercise to consist of two exponential components, an initial fast component which persisted for about 35 sec followed by a slower component which controlled the remainder of recovery. Although the recovery in short-term power output in the present study appeared to describe a single exponential process there was however evidence of an initial rapid component on the semilogarithmic plot as shown by a deviation from the straight line relationship. When the present results were superimposed upon the results of Stull and Clarke (1971) the pattern of recovery proved to be very similar (Fig.5).

This rapid recovery in both isometric and dynamic strength suggests that it may be dependent upon resynthesis of some substrate or clearance of some metabolite from the muscle. The removal of lactate and the recovery in pH following dynamic exercise have been shown to proceed rather more slowly than the recovery in short-term power output observed in the present investigation ,complete recovery only occurring after 20 min or longer (Sahlin et al, 1976). However the half time of 32 sec obtained in the present investigation agrees fairly well with the half time of 25 sec which Margaria, Edwards and Dill (1933) obtained for the repayment of the alactic portion of the

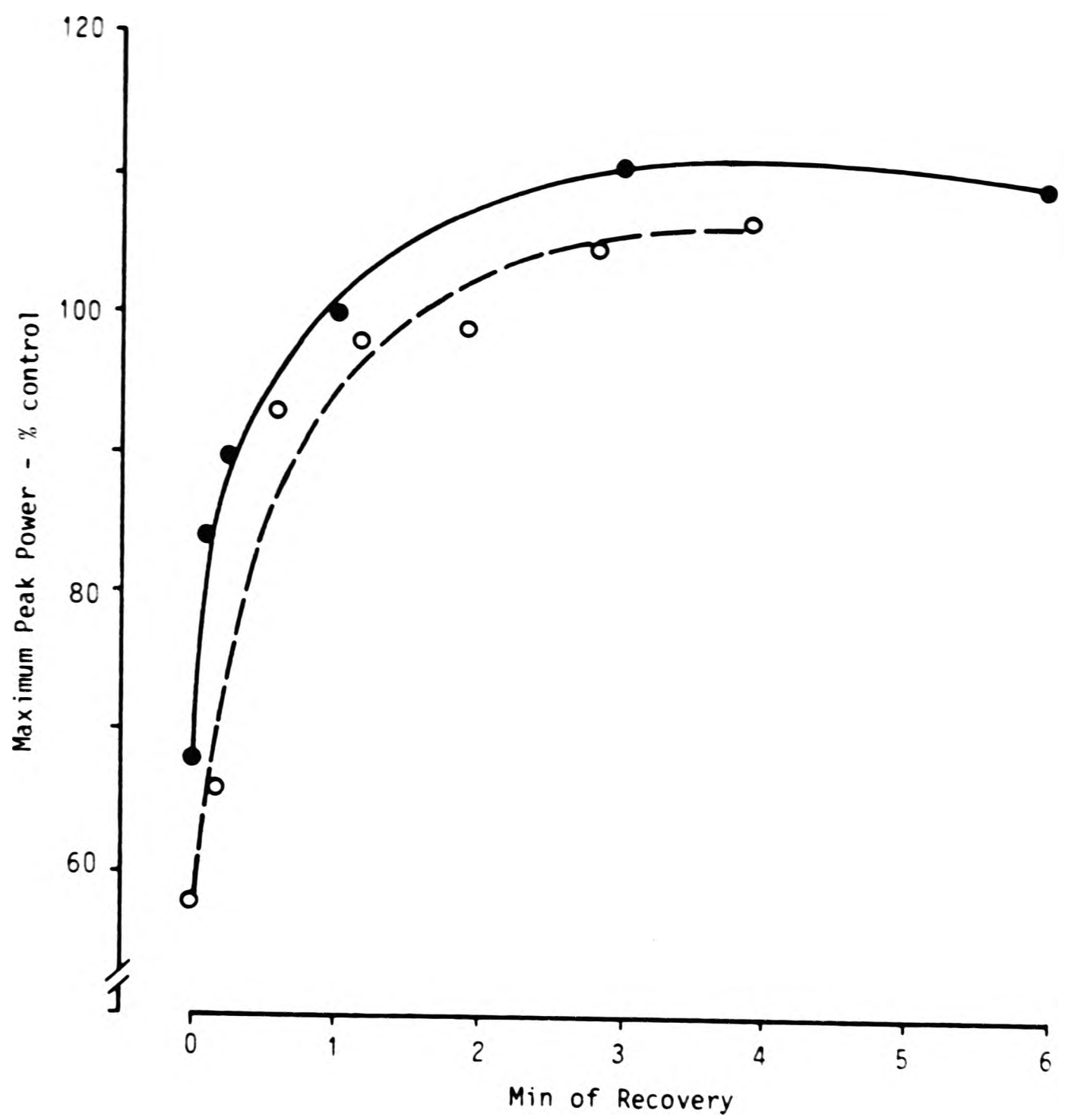


Fig.5 The time course of recovery for maximal peak power (present study) and isometric strength (from Stull and Clarke, 1971) following prior dynamic exercise. Isometric strength (broken line) is expressed as a percentage of the initial value before exercise for comparison with the present data (continuous line).

oxygen debt which suggests that recovery in power output may be related to the resynthesis of high energy phosphate in the fatigued muscle.

During exercise most of the reduction in high energy phosphate in human muscle can be attributed to a reduction in PC since the concentration of ATP only appears to fall when fatigue is very advanced (Harris et al, 1976 ; Wilkie, 1981). Harris and his associates (1976) followed the resynthesis of PC in human muscle following exhaustive cycle ergometer exercise and found this to be a biphasic process consisting of a fast and a slow recovery component. The pattern of resynthesis appeared to be very similar to the pattern of recovery observed in the present investigation although the recovery curve for short-term power output was shifted to the left of the curve for PC resynthesis indicating that functional recovery was a slightly more rapid process.

Hence after one minute of recovery the results of Harris et al showed that PC following exhaustive cycle ergometer exercise was restored to approximately 76% of its initial value. In the present investigation short-term power output had recovered to within 1% of the control value by this time implying that complete resynthesis of the PC store is not necessary in order to attain maximal levels of power output . However as maximal power output proceeded to increase during recovery so too did the rate of fatigue over the remainder of the 20 second test. Hence at 1 and 3 minutes of recovery the fatigue index although not significantly different, nevertheless tended to be higher than in the control run (Table 1).

These findings may be explained by an observation made by Edwards

and his associates (1972). They found that the utilisation of ATP and PC at the start of maximal isometric exercise was increased at elevated muscle temperatures while subsequent endurance time was reduced. In the present investigation the prevailing muscle temperature at the start of the maximal test would be elevated above the resting value as a result of the previous exercise. Thus after only one minute of recovery a low concentration of high-energy phosphate at the start of maximal exercise might be partly offset by an increase in its rate of breakdown. If this occurred then a more rapid utilisation of ATP and PC at the start of the maximal test might lead to a greater rate of fatigue over the remainder of the exercise as the contracting muscle became more dependent upon the slower processes of glycolysis and oxidation to provide the ATP for contraction. Extending the recovery period beyond a minute would enable the resynthesis of PC to proceed further towards its resting level. Since the temperature of the quadriceps muscle would very likely be elevated still at this time this could explain the improvements in power output and the increased rates of fatigue which were observed after 3 and 6 min of recovery.

However an increased muscle temperature is likely to have a number of other effects which might also influence power output. As early as 1906 Zuntz, Loewy, Muller and Caspari noted that viscous resistance was reduced at elevated muscle temperatures and they suggested that this would increase the external work produced by decreasing frictional losses during contraction. Wiles and Edwards (1982) also reported a decrease in relaxation time with increasing temperature. If this is the rate limiting factor during cross-bridge

cycling then an increase in relaxation rate might also contribute to an increased power output. The fact that relaxation time is also markedly prolonged at fatigue (Wiles and Edwards, 1982; Sjöholm et al, 1983) and that it recovers over a similar time course to that of short-term power output in the present investigation suggests that the recovery in maximal power may be related to the recovery in relaxation rate or that both processes are governed by some common factor such as the resynthesis of PC.

Another effect of prior exercise which might persist for several minutes during recovery and which could also have an effect upon subsequent short-term power output is the switching on of substrate cycling (Newsholme, 1978). Although the contribution from glycolysis to the production of ATP at the start of the maximal test is likely to be only small this may nevertheless be increased as a result of the previous exercise if the rate of substrate cycling was sufficiently increased during recovery.

There are therefore a number of possible causes for the improvements in power output observed in the present investigation. However it is interesting to note that Stull and Clarke (1971) who followed the recovery in isometric strength of the hand-grip muscles following prior exercise also found this to be improved during recovery. They attributed this to a warming-up effect of the previous exercise, however other investigators have found warm-up to have no beneficial effect upon isometric force (Binkhorst et al, 1977, Edwards et al, 1972). In these studies however passive warm-up was employed. It may be that the improvements observed by Stull and Clarke

were due to some kind of neuromuscular facilitation as a result of the previous exercise.

Conclusions

The recovery in short-term power output observed in the present investigation appears to follow a similar time course to the resynthesis of PC which Harris et al (1976) obtained following dynamic exercise. Together with the results of the previous study (see Chapter 3) these findings favour the view that maximal short-term power output is primarily dependent upon the amount of unsplit phosphorylcreatine in the active muscle fibres. Although PC as such may not directly determine power output it is likely that changes in its concentration will reflect changes in the rate of energy supply since the creatine kinase reaction between ATP and PC represents the most rapid pathway for ATP resynthesis in the contracting muscle. Other factors which affect the rate of ATP supply such as muscle temperature might also influence power output. The improvements in power output observed during recovery may therefore be due to an increase in muscle temperature as a result of the previous exercise.

CHAPTER 5

The Effect of Active and Passive
Warm-up upon Maximal
Short-term Power Output

INTRODUCTION

In the previous two chapters prior exercise was found under certain conditions to produce an increase in subsequent short-term power output when compared to control values. It was suggested that these improvements may be mediated through an increase in muscle temperature as a result of the previous exercise.

Changes in muscle temperature might affect muscle function in a number of ways. Zuntz et al in 1906 suggested that a decreased viscous resistance and an increased rate of neuromuscular transmission at elevated temperatures may increase the muscles capacity for work. A faster and more complete dissociation of oxygen from haemoglobin (Barcroft and King, 1909) and an increase in muscle blood flow (Clarke and Hellon, 1959) have also been found at elevated muscle temperatures. These changes would tend to enhance the oxygen supply to the working muscles which could also potentially improve muscle performance.

However with regard to the beneficial effect of warming-up the evidence to date is somewhat conflicting. Several workers have reported marked improvements in performance during short-term cycling (Asmussen and Bøje, 1945; Schmid , 1947; Bergh and Ekblom, 1979; De Bruyn-Prevost and Lefebvre, 1980; Sargeant, 1983), 100m runs (Simonson, Teslenko and Gorkin, 1936; Högberg and Ljunggren, 1947) and 50m swims (Schmid, 1947) while others have found warm-up to have little or no beneficial effect upon subsequent performance (Hipple, 1955; Karpovich and Hale, 1956; Skubic and Hodgkins, 1957; Davies and Young, 1981; Genovely and Stamford, 1982).

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There are several factors which could be responsible for such discrepancies. One possible explanation is the great variety of warming-up regimes employed. In previous investigations increases in muscle temperature have been achieved either by active warm-up or by passive methods such as short-wave diathermy, massage, or hot baths or showers. The effect of passive warm-up will depend upon the increase in temperature attained. The effect of active warm-up however may be influenced not only by the intensity of the warm-up but also on the duration of the recovery period allowed before the performance.

Karpovich and Hale (1956) further classified types of warm-up as being either specific or general. Specific warm-up employed the same movements as those to be used in the performance while general warm-up included limbering-up exercises such as sit-ups or running on the spot aswell as all passive methods of warm-up. It was suggested by these workers that general warm-up has its effect purely through an increase in muscle temperature while specific warm-up might also act to improve co-ordination during the performance.

Apart from differences in the warming-up procedure another factor which may have contributed to the conflicting results obtained in earlier investigations is the choice of criterion exercise employed to assess the effect of warm-up. While a number of workers examined the effect of warm-up upon the ability to perform short sprint efforts such as 50m swims (Schmid, 1947) or 100m runs (Simonson et al, 1936; Högberg and Ljunggren, 1947) aimed at taxing anaerobic capacity, others investigated its effect on exercises of longer duration (Karpovich and Hale, 1956; Högberg and Ljunggren, 1947).

The duration of performance may be critical since an increased rate of fatigue at high temperatures (Edwards et al, 1972) may mitigate against a higher initial power thus masking any warm-up effect.

This wide variation in both the method of warm-up and the criterion exercise used to assess its effects makes a comparison of the literature very difficult. It is still not clear whether the effect of warming-up is mediated mainly through an increase in muscle temperature or whether other factors are also involved.

The aim of the present study was to make a quantitative assessment of the effects of both active and passive warm-up upon subsequent short-term power output and to examine how these changes are related to the accompanying increases in muscle temperature using the two techniques.

Subjects

The subjects studied were 4 healthy male adults who had each given their informed consent to take part in the study. Physical characteristics of the subjects are shown in Table I.

METHODS

A study was designed to examine the effects of both active and passive warm-up upon subsequent short-term power output. Active warm-up was achieved by prior exercise of differing duration and intensity while passive warm-up was affected by hot water baths of varying temperature. The experimental procedure is outlined below. This is followed by a description of the techniques employed in the study.

Protocol

Maximal short-term power output was measured from rest and following different intensities of both active and passive warm-up.

The experimental procedure is shown in Fig.1. On each testing day the short-term power output measurement was initially performed from rest without any prior warm-up as a control. In each case resting muscle temperature was determined immediately beforehand.

In 1 of the 4 subjects (AS) blood samples were taken immediately before a number of the control runs and then again at approximately 2, 4, 6, 8 and 10 min of recovery for determination of plasma lactate.

Subject	Age yrs	Height cm	Weight kg	LBM kg	$\dot{V}O_{2\max}$ $l\ min^{-1}$
M.N.	27	170.3	59.20	53.85	3.40
J.S.	21	169.2	63.95	54.59	3.39
G.M.	24	192.7	76.10	67.74	4.29
A.S.	39	179.0	68.70	60.25	3.20

Table 1 Physical characteristics of the four subjects studied.

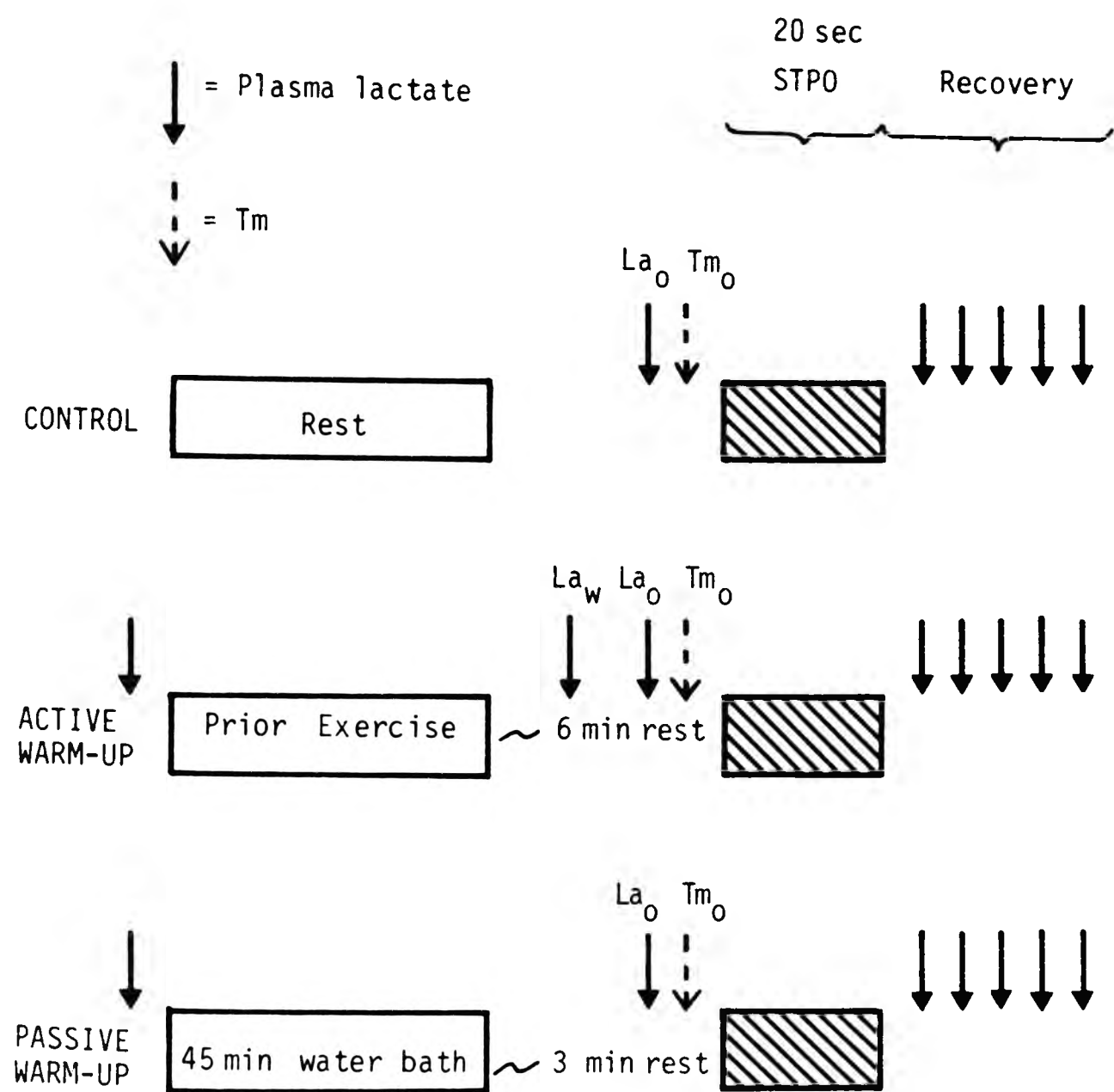


Fig.1 Protocol showing the experimental procedure employed. Maximal short-term power output was measured from rest and following different intensities of active and passive warm-up. The times at which plasma lactate and muscle temperature were determined for each run are indicated by arrows. La_w represents the plasma lactate concentration at the end of prior exercise; La_0 is the prevailing concentration at the start of the 20 sec. test and La_p (not shown) is the peak value attained from the 5 measurements made during recovery.

In the experimental runs the effects of both active and passive warm-up upon subsequent short-term power output were investigated.

Active Warm-up

Active warm-up was affected by having subjects perform different levels of submaximal exercise prior to the performance of the 20 sec test. This involved the subjects cycling an electrically braked (Lode) cycle ergometer for between 6-20 min at constant load. Oxygen uptake was determined during the final two minutes of exercise using a continuous open-circuit technique (see Chapter 3) to enable the relative work load to be determined.

At the end of prior exercise subjects were allowed a recovery period of 6 minutes duration before measurement of their short-term power output. This recovery interval was chosen on the basis of previous results (see Chapter 4) where marked improvements in power output were observed 6 minutes after prior exercise.

In an attempt to reduce temperature gradients in the muscle subjects were required to wear insulating trousers during the prior exercise period in order to prevent surface heat loss. The upper part of the body was kept cool by an electric fan during this time to prevent large increases in core temperature. At the end of prior exercise the insulating trousers were removed during the fifth minute of the recovery interval to allow measurement of muscle temperature prior to the performance of the 20 sec test.

In one subject (A.S.) blood samples for determination of plasma lactate were taken immediately before the start of prior exercise and

then again in the recovery interval to allow the prevailing plasma lactate concentration at the start of the maximal test to be determined. Further samples were then taken approximately 2, 4, 6, 8 and 10 min after completing the 20 sec test so that peak plasma lactate could be determined.

Passive Warm-up

The effect of passive warm-up upon subsequent short-term power output was examined using water baths of varying temperature (36-44°C) to warm the subjects leg muscles prior to the performance of the 20 sec test.

This involved the subjects standing in the water bath for approximately 45 min with their legs fully immersed as far as the gluteal fold. During this time the upper part of the body was kept cool by a large electric fan in order to prevent excessive increases in core temperature. Subjectively the subjects also felt more comfortable if the body was kept cool and were therefore more likely to endure the full 45 min in the bath.

At the end of the warm-up period muscle temperature was measured in the lateral part of the quadriceps muscle after which short-term power output was measured during the 20 sec maximal test.

In the run where the hottest water bath was employed blood samples for plasma lactate determination were taken in one of the subjects (AS) both before and after the warm-up period. Further samples were taken approximately 2, 4, 6, 8 and 10 min after

completing the subsequent 20 sec test to enable peak plasma lactate to be determined.

Measurement Techniques

Short-Term Power Output Measurement

Short-term power output was measured during 20 seconds of maximal exercise on the isokinetic ergometer as previously described. All measurements were made at a standard preset velocity of 110 rev.min⁻¹.

During the 20 second test force exerted on the pedals was measured by way of strain gauges attached to the cranks so that peak force and peak power during each revolution could be determined (for full description of method see Chapter 2).

As in earlier studies maximum values of peak force (PFmax) and peak power (PPmax) were determined by taking a mean of the three highest consecutive readings to be attained throughout the 20 second period. A fatigue index was compiled over the following 15 seconds of the test as previously described (see Chapter 2).

Muscle Temperature Determination.

Intramuscular temperature was measured in the lateral part of the quadriceps muscle approximately midway between the gluteal fold and the knee joint space. Measurements were made using a 5cm long copper-constantan needle thermocouple which was sensitive to changes in temperature of 0.01°C .

The needle was introduced into the muscle as deeply as possible and then withdrawn 1 cm at a time while the output which was displayed on a digital voltmeter was recorded.

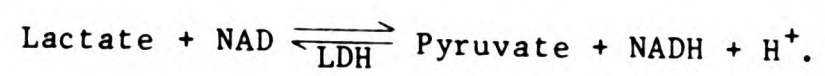
The lateral part of the quadriceps muscle was chosen for the temperature measurements since it was an area free of any major nerves or blood vessels. Saltin, Gagge and Stolwijk (1968) also demonstrated that this part of the muscle was actively engaged during cycling as indicated by the relative increase in muscle temperature with increasing work load.

In large limb muscles such as the quadriceps muscle however considerable gradients have been found to exist both at rest and during exercise (Clark, Hellon and Lind, 1958; Asmussen, Bonde-Petersen and Jorgensen, 1976; Wiles, 1980) with the highest temperature usually being found in the deepest part of the muscle. In the present study measurements of muscle temperature were therefore made at 4, 3, and 2cm depths in order to obtain a measure of the temperature gradient.

Plasma Lactate Determination

Plasma lactate was determined in one of the four subjects studied (A.S) at the times indicated in the protocol (see Fig. 1). Venous blood samples were drawn from an antecubital vein with the subject at rest. These were immediately dispensed into preweighed chilled plastic tubes containing ethylenediaminetetracetic acid and stored at 0°C. Samples were centrifuged within 24 hours of being taken and the plasma was seperated off for subsequent analysis. Lactate was determined

using a fluorimetric enzyme technique which was based on the following reaction ;



The NADH_2^+ concentration was determined by reading the absorbance at 340 nm and from this the initial lactate concentration was determined.

RESULTS

Muscle Temperature.

In the present investigation muscle temperature (T_m) was measured at three depths in order to take account of temperature gradients in the muscle which were in some cases quite considerable.

This was demonstrated in a preliminary series of experiments where muscle temperature was measured in the lateral part of the quadriceps both at rest and following different levels of submaximal exercise. Measurements were made in 10 subjects at 4, 3, 2 and 1 cm beneath the skin surface.

Measurements made at a depth of 1cm were frequently in the subcutaneous fat layer and for this reason all 1cm readings were disregarded. However considerable gradients up to 4.3°C were observed in resting muscle between the temperature measured at 4cm depth and that measured at 2cm. In all cases the highest temperature was found in the deeper part of the muscle.

Following exercise this gradient was reduced although in some cases it was still quite marked with differences in temperature up to 2.9°C being observed between the 4cm and 2 cm readings.

On the basis of these observations it was decided that a value representative of the working muscle temperature would best be attained by making measurements at a number of depths. In the present study muscle temperature was therefore measured at 4, 3 and 2cm depths and the mean value determined.

When expressed as a mean of the three measured values resting muscle temperature in the present subjects ranged from 34.13 - 36.70°C. The mean gradient between the 4 and 2cm readings was 2.08±1.02°C with gradients up to 4.2°C being observed between the two depths .

Following warm-up the gradient was reduced below that found at rest (Fig.2) and averaged 1.03±0.48°C and 0.66±0.53°C following active and passive warm-up respectively.

The range of muscle temperatures achieved by warm-up were between 35.8 - 39.0°C for active compared with 36.9 - 39.3°C for passive. The ranges were therefore very similar using either method of warm-up allowing for a direct comparison of the Q_{10} values.

Maximal Short-term Power Output

Mean values of resting muscle temperature and maximal peak power based on data from the control runs were determined for each subject and are given in Table 2.

Following warm-up all 4 subjects showed an improvement in maximal peak power (PPmax) during the subsequent 20 sec test when compared to their mean control value (Table 2). There was no significant difference in the increases obtained by active as opposed to passive methods of warm-up.

When maximal peak power expressed as a percent of the mean control value was plotted against the prevailing muscle temperature it was found to increase linearly with the latter following both active

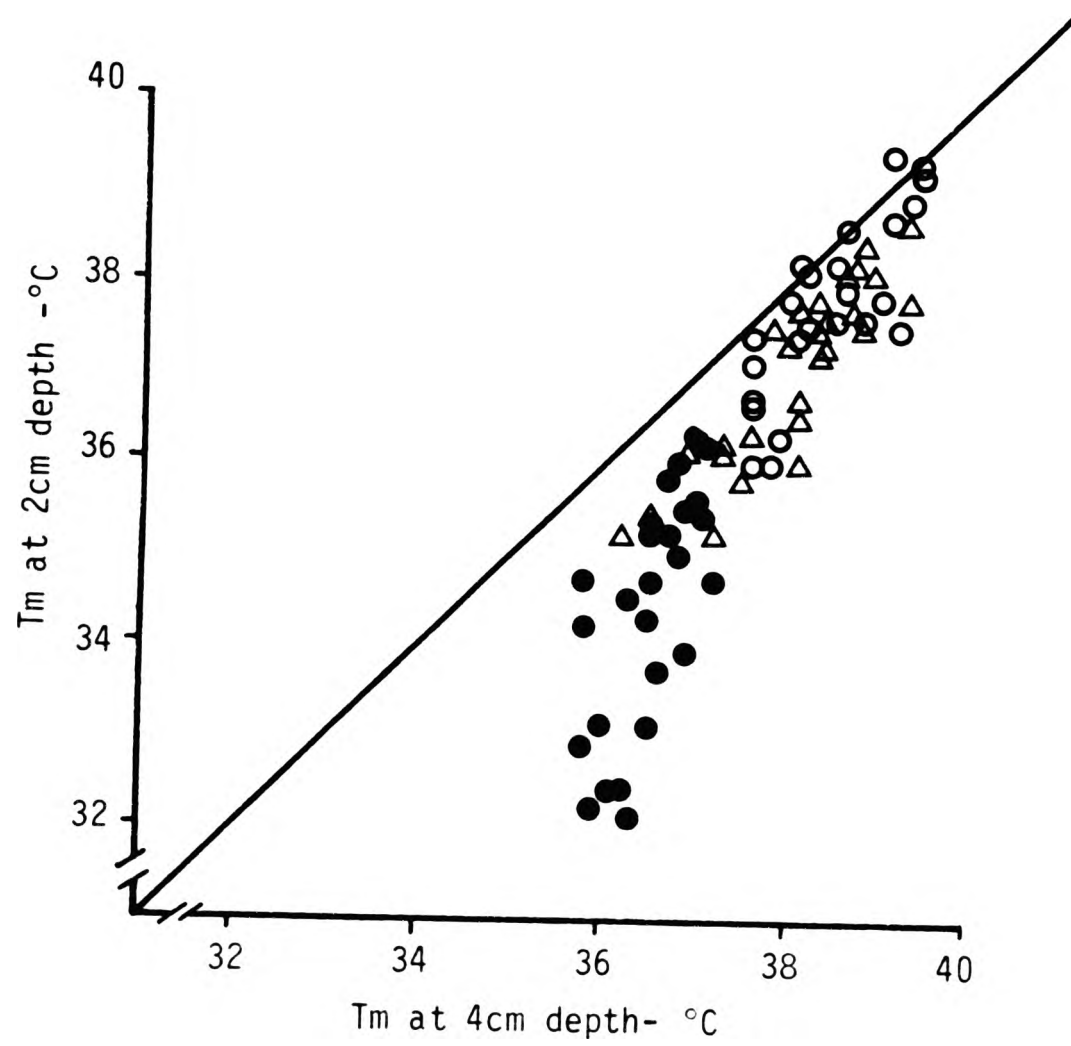


Fig.2 Relationship between temperature measurements made at 4cm and 2cm depths in the lateral part of the quadriceps muscle. Closed symbols represent resting measurements; open symbols represent measurements made following either active (Δ) or passive (\circ) warm-up. The line shown represents the line of identity.

Subject	Controls			Following Active Warm-up			Following Passive Warm-up		
	T _m °C	PPmax Watts	FI W. sec ⁻¹	T _m °C	PPmax % control	FI W. sec ⁻¹	T _m °C	PPmax % control	FI W. sec ⁻¹
MN	35.91 ±0.60	1084 ±60	24.6 ±4.8	37.98 ±0.96	108.0 ±4.4	25.7 ±4.8	38.46 ±0.65	107.7 ±5.7	25.0 ±2.7
AS	36.17 ±0.39	1436 ±56	39.7 ±5.2	37.56 ±0.97	105.1 ±4.3	43.1 ±4.9	38.36 ±0.73	105.1 ±5.9	42.1 ±4.6
JS	35.04 ±0.79	1247 ±31	26.0 ±5.9	37.71 ±0.67	107.4 ±0.9	26.3 ±4.2	38.01 ±0.53	106.8 ±6.5	28.8 ±9.3
GM	34.93 ±0.58	1488 ±65	22.7 ±3.9	37.38 ±0.85	106.9 ±6.7	20.8 ±3.7	37.86 ±0.69	107.9 ±3.9	18.9 ±3.4

Table 2 Mean values of muscle temperature (T_m), maximal peak power (PPmax) and rate of fatigue (FI) are shown for each subject under each of the three experimental conditions. Values of PPmax in the runs following warm-up are expressed as a percent of the mean control value.

Subject	Controls			Following Active Warm-up			Following Passive Warm-up		
	T _m °C	PPmax Watts	FI W. sec ⁻¹	T _m °C	PPmax % control	FI W. sec ⁻¹	T _m °C	PPmax % control	FI W. sec ⁻¹
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and passive warm-up. Based on data from all 4 subjects these relationships could be described by the following equations (Fig.3);

Following passive warm-up: $PP_{max} (\% \text{ control}) = 19.53 + 2.27 \times T_m (^{\circ}\text{C})$
($n=54$; $r=0.605$; $p<0.001$)

Following active warm-up: $PP_{max} (\% \text{ control}) = 4.46 + 2.70 \times T_m (^{\circ}\text{C})$
($n=53$; $r=0.684$; $p<0.001$)

These regression equations indicated an increase in PP_{max} of 2.7 and 2.3% per $^{\circ}\text{C}$ rise in muscle temperature following active and passive warm-up respectively, the equivalent Q_{10} values being 1.30 and 1.25 over the range of muscle temperatures studied (34-40 $^{\circ}\text{C}$).

Rate of Fatigue

In the control runs the decline in peak power during the 20 sec test was between 15.8 and 46.5 W sec^{-1} in the four subjects studied. Expressed in terms of the maximal peak power attained at the start of the test these values were between 1.16 - 3.12% sec^{-1} . Following warm-up this fatigue index was slightly greater than the control value in three of the four subjects studied (Table 2) although this difference was not significant and individual values showed no correlation to muscle temperature in any of the four subjects studied.

Plasma Lactate.

Resting levels of plasma lactate in the subject studied (A.S) showed little day to day variation and averaged 0.70 ± 0.19 mmol. litre^{-1} .

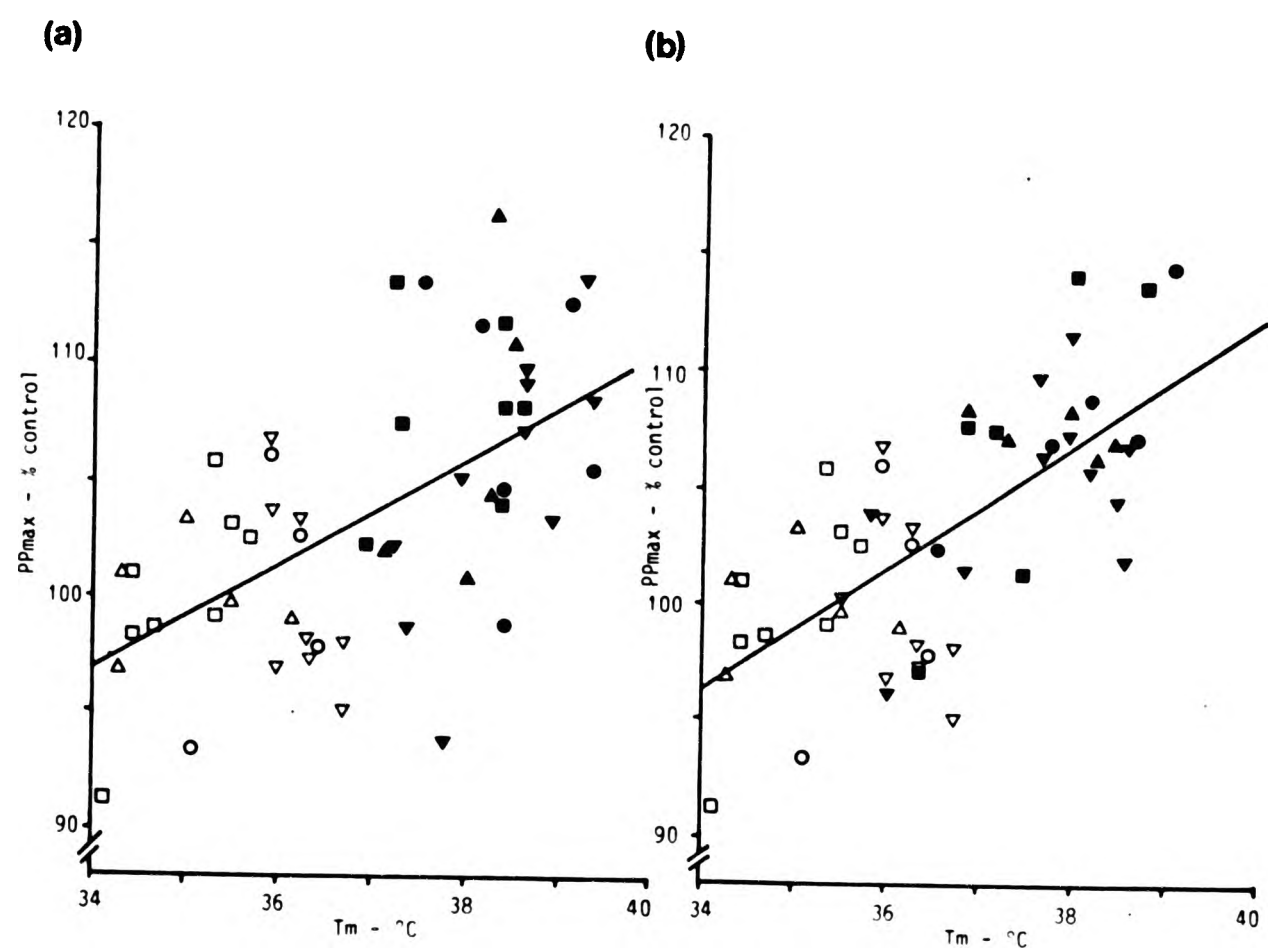


Fig.3 Relationship between maximal peak power (expressed as a percentage of the control value) and mean muscle temperature following (a) passive and (b) active warm-up. Open symbols represent control measurements made from rest; closed symbols represent measurements made following warm-up. Lines indicate linear regression based on data from all 4 subjects.

In the control runs plasma lactate increased from the resting level at the start of the 20 sec test to reach a peak value 4-6 min after completing the exercise (Fig 4a). Peak plasma lactate was almost exactly the same in the two control runs where values of 8.49 and 8.54 mmol l⁻¹ were obtained (Table 3).

Passive warm-up had very little effect on plasma lactate concentration which was very similar to the resting value at the end of the warm-up period. Following the subsequent maximal test peak plasma lactate was attained after 4 min of recovery (Fig. 4b). This peak value was almost identical to that obtained in the control runs (8.29 compared to 8.51±0.04 mmol litre⁻¹ respectively).

Following active warm-up there was little change in plasma lactate as a result of the lower levels of prior exercise. However when the intensity of prior exercise exceeded 65% of the subjects $\dot{V}O_2$ max substantial increases in plasma lactate were observed 4-5 min afterwards which were related to the relative work load (Table 3, Fig. 4c). Following the highest work loads plasma lactate concentrations as high as 10.7 mmol l⁻¹ were recorded. In these cases plasma lactate was substantially elevated still at ~8.5 mmol l⁻¹ at the start of the subsequent maximal test (Table 3, Fig. 4c). This was reflected in the peak concentrations of plasma lactate attained after performance of the 20 sec test which also tended to be higher the greater the intensity of the preceding exercise (Fig.5).

In order to estimate the net lactate produced during the 20 sec maximal test independent of that produced as a result of the previous submaximal exercise several basic assumptions had to be made. First of

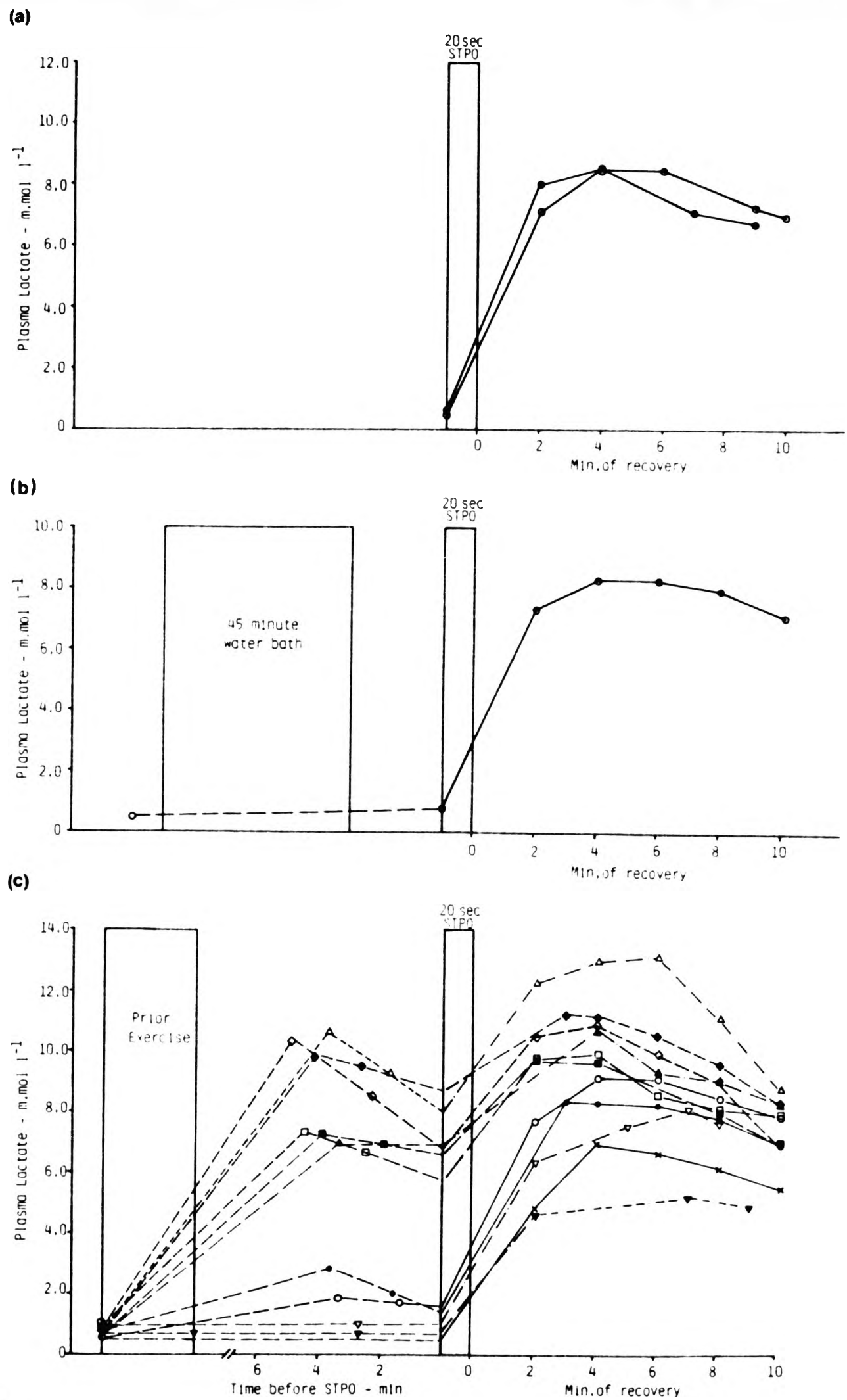


Fig.4 Plasma lactate concentration measured prior to, and during recovery from the 20 sec. maximal test when performed (a) from rest, and following (b) passive and (c) active warm-up at 17 (▼), 20 (▽), 25 (x), 48 (○), 65 (●), 75 (□), 80 (■), 83 (◇), 87 (△◆), 92 (▲) % $\dot{V}O_2$ max. Data are shown for one subject (A.S.)

Prior Exercise % VO ₂ max	T _m °C	La _o mmol.l ⁻¹	PPmax % control	La _p mmol.l ⁻¹	La _t (La _p -La _o) x TBW mmol
Control	35.90	0.42	106.9	8.49	324.7
Control	36.23	0.62	103.4	8.54	318.4
(PW)	39.33	0.77	108.4	8.29	314.4
19.9	36.00	1.05	96.1	8.11	283.8
25.2	36.77	0.57	101.4	6.98	257.7
47.8	37.87	1.76	107.4	9.15	297.1
65.2	37.63	2.03	106.5	8.38	255.3
74.5	38.43	6.70	104.4	9.96	131.1
80.1	38.13	6.96	105.9	9.72	111.0
83.2	38.53	8.55	106.9	0.89	94.1
86.7	37.90	9.37	111.6	13.20	154.0
87.3	38.53	9.53	101.9	11.20	71.2
91.6	37.57	6.99	109.8	10.67	147.9

Table 3 Maximal peak power (PPmax) and net lactate production (La_t) during the 20 sec test when performed from rest (control) and following passive (PW) and active warm-up. T_m represents the mean muscle temperature before the start of the 20 sec test; La_t is determined by subtracting the prevailing plasma lactate concentration at the start of the 20 sec test (La_o) from the peak value measured at the end of exercise (La_p) and multiplying by the total body water (TBW).

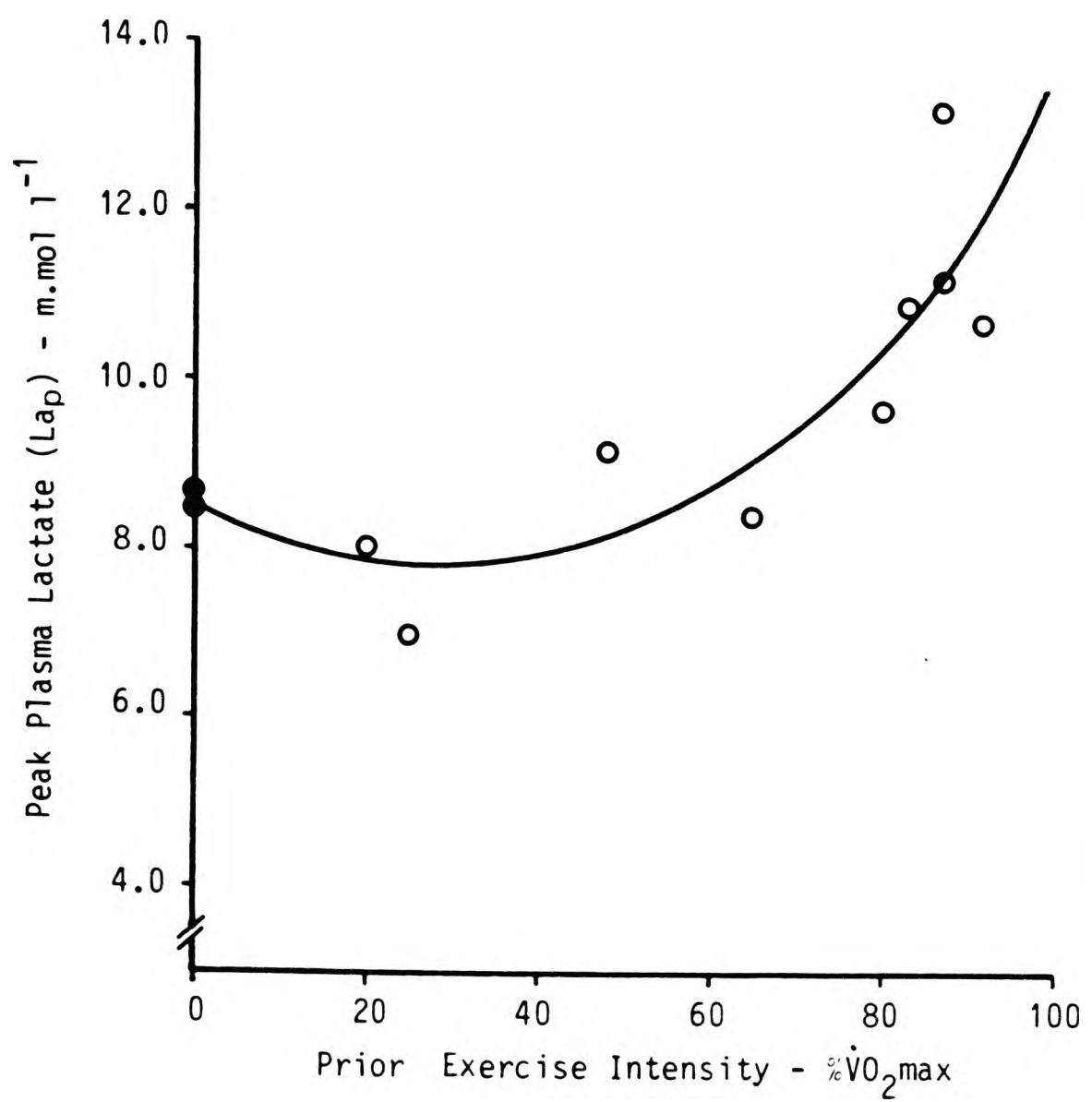


Fig.5 Peak plasma lactate (L_{ap}) following the 20 sec. maximal test when performed from rest (closed circles) and following different intensities of prior exercise (open circles). Data are shown for one subject (A.S.).

all the plasma lactate concentration measured towards the end of the recovery period following prior exercise was assumed to be the prevailing concentration at the start of the subsequent maximal test (La_0). Secondly it was assumed that there was no removal of lactate from the total pool in the time taken for peak plasma lactate levels to be reached following the maximal test and finally in order to calculate total lactate production it was assumed that lactate was evenly distributed throughout the total body water (TBW).

On the basis of these assumptions an estimate of the net lactate produced during the 20 sec test (La_t) was made according to the following equation

$$La_t \text{ (mmol)} = (La_p - La_0) \times TBW$$

where La_p = peak plasma lactate concentration following the 20 sec test
(mmol l^{-1})

La_0 = the prevailing plasma lactate concentration at the start
of the 20 sec test (mmol l^{-1})

and $TBW \text{ (l)} = 0.6 \times \text{body weight (kg)}$

The results of this calculation showed that net lactate production during the 20 sec test (La_t) decreased in relation to the prior exercise intensity (Table 3). The relationship was curvilinear with the decrease in La_t becoming more marked as the prior exercise intensity exceeded $65\% \dot{V}O_{2\text{max}}$ (Fig. 6).

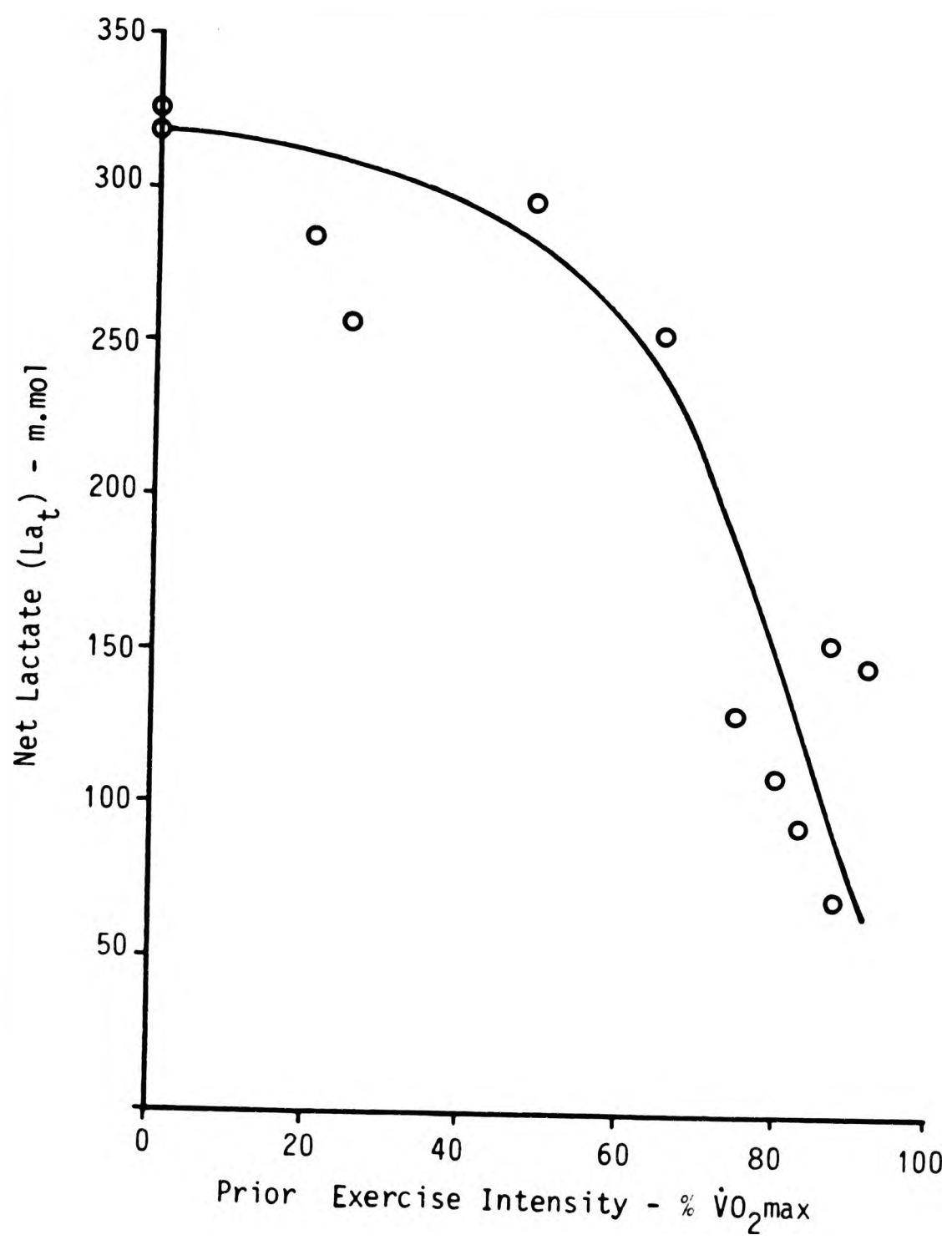


Fig.6 Net lactate production during the 20 sec. maximal test when performed from rest and following different intensities of prior exercise. Data are shown for one subject (A.S.). Symbols as for Fig.5

Discussion

Muscle Temperature Measurement

In any investigation which seeks to assess the effect of warm-up upon performance it is important that the muscle temperature (T_m) measurement accurately reflects the temperature of the active muscles.

In the present investigation the lateral part of the quadriceps muscle was chosen for temperature measurements since this muscle had previously been shown to be actively engaged in cycling exercise as indicated by its rapid increase in temperature to levels above the rectal value (Saltin, Gagge and Stolwijk, 1968). The lateral part of the quadriceps muscle was also an area free of any major nerves or blood vessels which made it possible to attain temperature readings at considerable depths under the skin surface.

The main problem with making the temperature measurements in this muscle were the marked gradients in temperature which existed. Previous workers found such gradients to be quite pronounced (Saltin et al, 1968; Asmussen, Bonde-Petersen and Jorgensen, 1976; Wiles, 1980) and in the present study differences in temperature of 2 or more degrees in resting muscle were not uncommon (Fig. 2). In addition Asmussen et al (1976) found that such gradients were not constant but varied depending upon the limb temperature.

In order to account for such gradients in the present study muscle temperature was measured at three depths and the average value was taken to represent the mean muscle temperature. Steps were also taken

to reduce the gradients during active warm-up by having the subjects exercise with their legs covered in order to prevent evaporative heat loss through the skin. This appeared to be quite effective since the mean temperature gradient across the muscle following active warm-up was reduced below that at rest and was more comparable to that obtained with passive warming where an almost uniform muscle temperature was achieved (Fig.2).

Maximal Short-term Power Output

When mean muscle temperature was plotted against the maximal peak power attained in the 20 second test all subjects showed an increase in power output with increasing T_m . In the present study these improvements were equivalent to 2.7 and 2.3% per $^{\circ}\text{C}$ following active and passive warm-up respectively. The corresponding Q_{10} values were 1.30 and 1.25 which are consistent with those previously attained for the handgrip muscles (Binkhorst et al, 1977). Since velocity was held constant these improvements were due to an increase in maximal peak force indicating a shift in the force-velocity curve (cf. Sargeant, 1983; Binkhorst et al, 1977).

The present results may be compared with those of Asmussen and Boje (1945) and Bergh and Ekblom (1979) who found that active warm-up improved the time to perform a short maximal cycling exercise by 3.3 and 4.4% per $^{\circ}\text{C}$ respectively (T_m range 36-40 $^{\circ}\text{C}$). Asmussen and Boje reported similar improvements in performance when they used passive methods to warm the muscle.

In a more recent study Davies and Young (1983) examined the

effect of passive heating on both jumping and cycling performance. The improvements in power output which they observed during cycling were rather slight and amounted to only 1.6% per °C. However jumping performance was improved by ~3.8% per °C which was consistent with values previously obtained using passive warm-up (Asmussen and Boje, 1945).

In the present study where both active and passive warm-up were employed , the improvements in power output during cycling were of the order of 2.5% for each °C rise in muscle temperature and no significant difference was observed between the two methods of warm-up.

These results suggest that the beneficial effect of warming-up is mediated almost entirely through an effect upon muscle temperature and that any other physiological changes as a result of prior exercise have little or no influence upon the muscle's capacity for maximal power output.

The precise nature of this temperature effect in improving the muscles capacity to generate power is open to discussion. One possible mechanism was proposed by Zuntz et al in 1906. They suggested that a reduction in muscular viscosity at elevated temperatures might improve performance by reducing frictional losses during contraction.

Another way in which the muscles capacity for power output may be improved is through an increase in its rate of energy supply. The delivery of oxygen to the active muscle fibres is improved following warm-up due to an increase in muscle blood flow (Clarke and Hellon, 1959) and a greater and more rapid dissociation of oxygen from

haemoglobin in the muscle's capillaries (Barcroft and King, 1909). The muscle's capacity for ATP production via aerobic pathways may therefore be enhanced at higher temperatures however the effect of this during brief intense exercise of the type studied in the present investigation is likely to be only small.

Elevated temperatures in the muscle may also increase the rate of anaerobic metabolism through an effect on creatine kinase and the enzymes of glycolysis. In a previous investigation Edwards et al (1972) found increased levels of several glycolytic intermediates in heated muscle at rest which suggests that the rate of glycolysis may be increased at elevated temperatures.

In the present study attempts were made to assess the contribution from glycolysis to the performance of the 20 second test by measuring changes in the plasma lactate concentration. In order to determine lactate production in the muscle from plasma lactate values however a number of assumptions regarding lactate production and removal had to be made. It was first of all assumed that the lactate produced in the muscle during exercise eventually appeared in the plasma and secondly it was assumed that this lactate was evenly distributed throughout the total body water and that there was no removal from this pool in the time taken for peak values to be attained.

Values estimated in this way will almost certainly be an underestimation of the total lactate produced during exercise since some of this lactate will be reutilised locally within the muscle and will not appear in the plasma. However Sahlin et al (1976) calculated that the rate of lactate disappearance from muscle

following exhaustive exercise was approximately the same as its rate of uptake by the blood implying that little was reutilised by the muscle once the exercise was over.

If it may be assumed that the lactate which appears in the plasma is at least proportional to the lactate produced in the muscle then the values obtained in the present study although not representative of total lactate production during the maximal test may at least provide an indication of whether this is altered by warm-up relative to control values.

Working on this assumption passive warm-up had little effect upon net lactate production during the maximal test (La_t) which was similar to that attained in the control runs. Following active warm-up however La_t decreased with the intensity of the previous exercise when this exceeded 60% of the subject's $\dot{V}O_{2max}$ although it was little affected following lighter work loads.

These findings suggest that the rate of glycolysis during the maximal test is unaffected by passive warm-up although it is reduced in relation to the prior exercise intensity following active warm-up. The marked reductions in La_t following the higher levels of prior exercise ($> 60\% \dot{V}O_{2max}$) suggest that glycolysis may have been inhibited in these runs due to a fall in muscle pH. Considerable reductions in intramuscular pH which prevail for some time afterward have been reported following brief intense exercise (Hermansen and Osnes, 1972; Furusawa and Kerridge, 1977; Sahlin, 1978) and a number of studies have shown that phosphorylase and phosphofructokinase (PFK) which play an important role in regulating the rate of

glycolysis are also extremely sensitive to changes in pH (Danforth, 1965; Gevers and Dowdle, 1963; Trivedi and Danforth, 1966).

If a fall in muscle pH as a result of heavy prior exercise produced such an inhibition of these regulatory enzymes in the present study then it had little or no effect upon performance. The present group of subjects showed substantial improvements in maximal power output following the higher levels of active warm-up which were comparable to those attained by passive warm-up.

These findings indicate that the maximal power attained during the first few seconds of dynamic exercise is largely independent of the contribution from glycolysis. This implies that the energy for contraction at the start of exercise is derived mainly from the splitting of high energy phosphate already present in the active muscle fibres. There is no evidence to suggest that the concentration of ATP and PC within the muscle will be affected by changes in temperature, however the rate at which they are broken down may be increased due to an increase in the rate of myosin ATPase and creatine kinase.

In an earlier investigation Edwards et al (1972) obtained results which indicated that the rate of ATP and PC utilisation was increased at elevated muscle temperatures even though the isometric tension developed by the muscle remained the same. Studies on frog muscle (Larson, 1970) suggest that this might reflect an increased tendency for the actin and myosin filaments to slide apart at higher muscle temperatures. This would be consistent with the increased rates of relaxation observed in heated muscle (Asmussen et al, 1976; Wiles

and Edwards, 1982; Davies and Young, 1983). During isometric contractions the additional energy provided by the increased rate of ATP turnover would therefore appear to be lost at the expense of maintaining isometric tension. The situation may be somewhat different during dynamic exercise however where a reduction in relaxation time might potentially improve performance through an increase in the rate of cross-bridge cycling.

In a recent study Davies and Young (1983) measured changes in the muscle's contractile properties following passive heating and cooling in order to determine their effect on maximal power output which was also measured. They found that the changes in power output during dynamic exercise reflected changes in the rate of tension development and relaxation. Hence time to peak tension (TPT) and half-relaxation time (1/2 RT) were reduced at elevated muscle temperatures while maximal power was increased. However the increases in power output which were equivalent to 3.8 and 1.6% per^oC for jumping and cycling respectively were rather less than would be expected from the changes in TPT and 1/2 RT which were reduced by approximately 12% per^oC. Nevertheless these changes in the muscle's contractile properties at elevated temperatures may play a part in improving dynamic performance capacity following warm-up.

It is evident from the above discussion that there are several factors which could contribute to the improvements in power output observed in the present study. However it would appear that any detrimental effects of prior exercise on, for example, muscle pH are more than compensated for by the warming-up effect.

Rate of Fatigue

In the present study fatiguability was assessed by measuring the loss of power over the 20 sec test when this was performed from rest and following warm-up. Three of the four subjects studied showed an increased rate of fatigue when the maximal test was preceded by warm-up, however the extent of this increase was rather small and did not prove to be significant. In an earlier investigation based on the present isokinetic technique Sargeant (1983) determined the rate of fatigue over the 20 sec test following both warming and cooling of the leg muscles and found this to be temperature dependent. It may be that the greatest changes in the muscles fatiguability come about as a result of cooling the muscle below its normal temperature. Much greater changes in muscle temperature can be achieved by cooling as opposed to heating the muscle where the increase in muscle temperature may not be sufficient to demonstrate any significant changes in the rate of fatigue.

Conclusions

In conclusion the present results indicate a beneficial effect of both active and passive warm-up upon subsequent short-term power output. Improvements in power output were similar independent of the method of warm-up and were equivalent to ~2.5% per °C rise in muscle temperature. These results suggest that the effect of active warm-up is almost entirely attributable to an increase in muscle temperature. Elevated levels of plasma lactate appeared to have no detrimental effect upon subsequent performance despite the fact that glycolysis was

apparently inhibited following the higher levels of prior exercise. However the results of Chapter 4 indicate that following high levels of active warm-up the interval between prior exercise and the performance task should be sufficient to allow concentrations of PC in the muscle to recover to within a few percent of their resting values in order to achieve the desired increase in maximal power output.

CHAPTER 6

Maximal Short-term Power Output of
Human Muscle Following Prolonged
Concentric and Eccentric Work

INTRODUCTION

In an earlier part of this thesis the effect of prior exercise of up to 6 minutes duration upon subsequent short-term power output was examined and it was found that recovery from such exercise was complete within 1-6 minutes. However previous studies have shown that more prolonged exercise, especially where this has a large eccentric component, even at submaximal work loads results in long term muscular fatigue which persists for several hours or even days (Edwards, Hill, Jones and Merton, 1977; Davies and White, 1981; Edwards, Mills and Newham, 1981; Newham, Mills, Quigley and Edwards, 1983c; Friden, Sjostrom and Ekblom, 1983).

The physiological basis of this long-lasting element of fatigue is still not entirely understood. A number of workers have examined the effects of largely eccentric or concentric exercise upon a number of factors such as the development of muscular soreness (Friden, Sjostrom and Ekblom, 1981; Newham et al, 1983c; Wattrous, Armstrong and Schwane, 1983; Schwane, Johnson, Vandenakker and Armstrong, 1983), the appearance of intramuscular enzymes in the plasma (Schwane et al, 1983; Newham, Jones and Edwards, 1983a) or the contractile properties of the muscles themselves (Newham et al, 1983c; Edwards et al, 1981; Davies and White, 1981 and 1982).

In general it appears that exercises with a large eccentric component such as downhill walking or box-stepping have a more marked and long-lasting effect than concentrically biased exercises such as level or uphill walking or running. However the effects of such exercise on muscle function have generally been assessed by examining

changes in isometric force during either electrically stimulated or voluntary contractions while very little attention has been paid to the effect upon the muscle's dynamic properties.

Komi and Rusko (1974) examined force production during repeated concentric and eccentric contractions and found that the loss of force was greatest during eccentric work. However Friden and his colleagues (1983) appear to be the only workers who have looked at the effect of prior eccentric exercise upon the ability to perform dynamic muscular contractions.

It was therefore the purpose of the present investigation to assess the effects of prolonged concentric and eccentric exercise upon both isometric force and the maximal power attained during short-term cycling.

SUBJECTS

Four healthy adult subjects (one male, three female) who had each given their informed consent took part in the investigation. Physical characteristics including measurements of $\dot{V}O_2\text{max}$ are shown in Table I.

METHODS

In this study the effects of prolonged eccentric and concentric exercise upon subsequent short-term power output from human muscle were investigated. This involved measurement of subject's maximal power output at various stages during recovery from prolonged uphill and downhill walking. The techniques employed together with details of the protocol employed are described below.

Measurement Techniques.

Maximal Short-term Power Output.

Determination of maximal short-term power output was carried out during 20 seconds of maximal exercise on the isokinetic ergometer as previously described (see Chapter 2 for full description of method).

Tests were performed at four crank speeds which approximated to 50, 80, 110 and 125 rev.min⁻¹ and during each test peak force and peak power during each revolution were measured for each leg and the mean values determined. The maximum power attained in the test was then calculated by taking a mean of the three highest consecutive readings during the 20 second period as previously described (Chapter 2).

Subject	Age (yrs)	Height (cm)	Weight (kg)	$\dot{V}O_2\text{max}$ (l min ⁻¹)
PD	27	164.6	55.1	2.76
CG	23	166.9	54.4	2.41
OR	23	160.7	69.4	2.37
AS	39	177.7	67.0	3.22

Table 1 Physical characteristics of the subjects studied.

Maximal Voluntary Contraction

Maximal isometric voluntary contractions of the quadriceps muscle were measured according to the method described by Edwards et al (1981). Subjects were seated in an upright position in an adjustable straight-backed chair so that the angle of flexion at the knee was $\sim 90^\circ$ with the lower leg hanging free (Fig. 1). A belt fastened around the pelvis was used to secure the subject in the seat and a further strap which was connected to a strain gauge was looped around the ankle at the level of the lateral malleolus. The position of the strain gauge was then adjusted so that this strap was at right-angles to the lower leg running parallel to the ground. Output from the strain gauge was amplified and the signal recorded on an ultraviolet oscillograph (S.E. Labs.).

When instructed to do so subjects by pulling against the strap made a maximal voluntary contraction (MVC). On each occasion three measurements were made an additional one being made if the highest value was widely different from the other two or if it was thought that a maximal effort had not been made. This procedure was then repeated for the other leg and the highest value for each leg was used to determine the mean MVC for both legs.

Contractile Properties.

Electrically stimulated contractions of the quadriceps muscle were measured using the same apparatus as that used to measure the MVC. In this case however two large saline soaked aluminium foil electrodes $\sim 13 \text{ cm}^2$ in size were applied to the proximal and distal parts of the

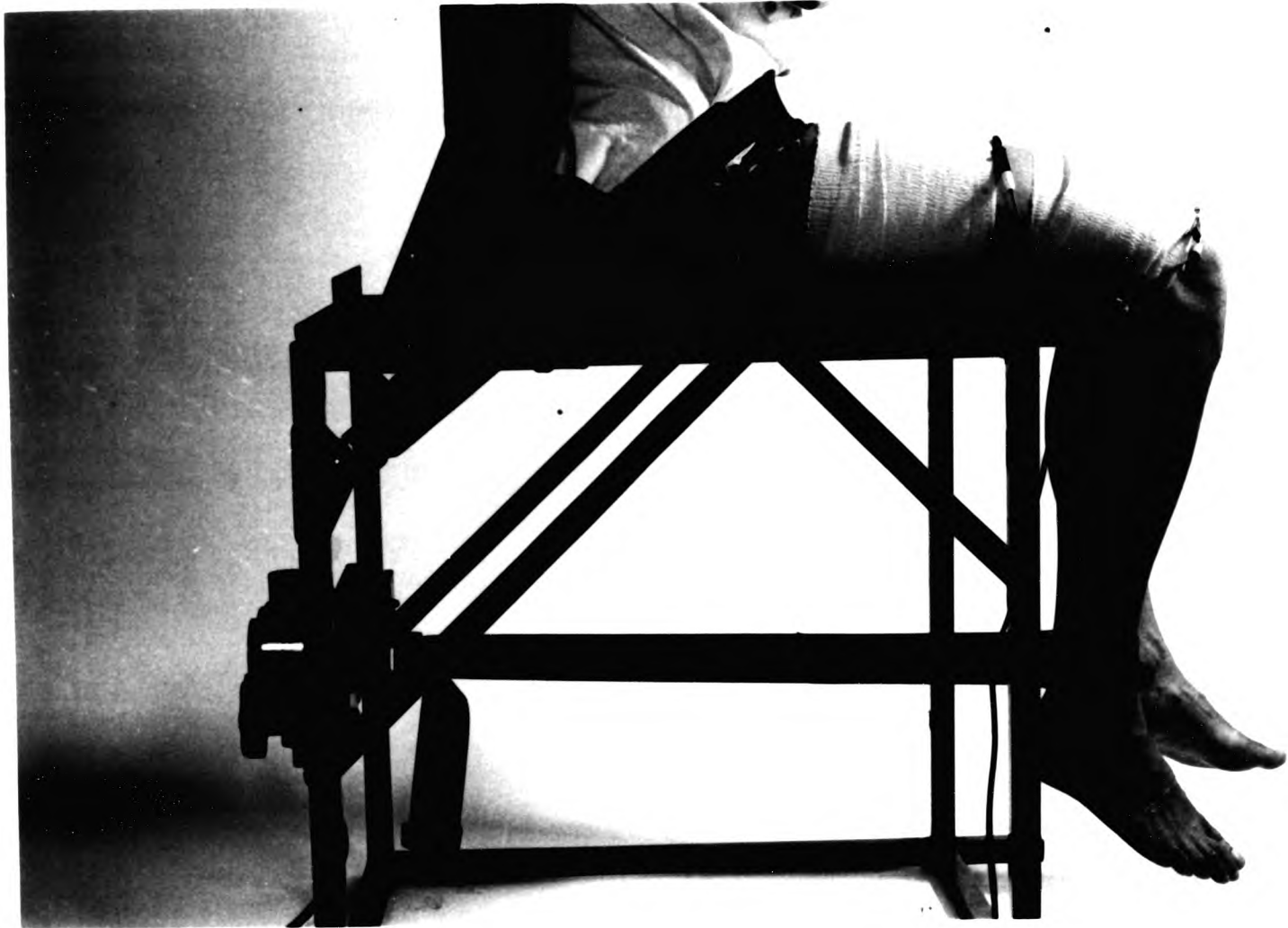


Fig.1 The chair used to measure maximum voluntary and stimulated contractions of the quadriceps muscle. Force exerted is measured by means of a strap looped around the ankle which is attached at its other end to a strain-gauged load cell.

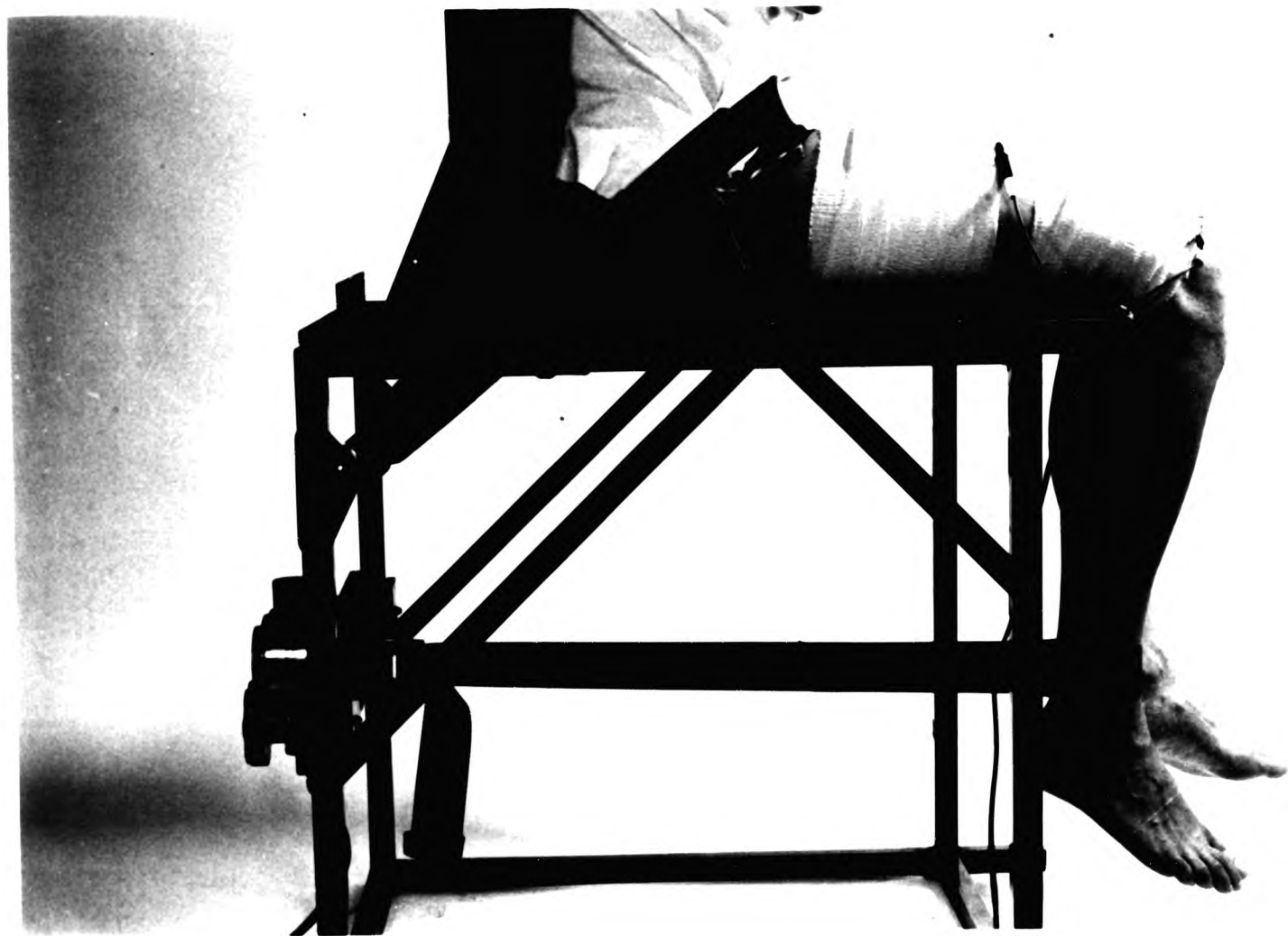


Fig.1 The chair used to measure maximum voluntary and stimulated contractions of the quadriceps muscle. Force exerted is measured by means of a strap looped around the ankle which is attached at its other end to a strain-gauged load cell.

thigh in order to electrically stimulate the muscle percutaneously.

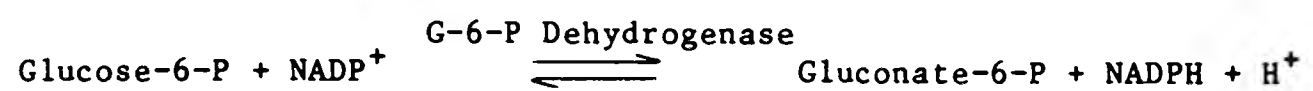
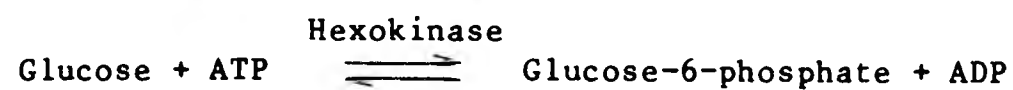
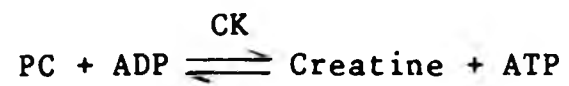
Stimulation was with unidirectional square wave pulses of 50 microseconds duration and the stimulation voltage was chosen so as to activate at least 30-35% of the muscle. This was determined by intermittent stimulation of the muscle at a physiological frequency of 30 Hz during which the stimulation voltage was gradually increased until a force equivalent to ~33% of the MVC was attained. This level of electrical stimulation was generally well tolerated by the subjects and the discomfort experienced was usually rather mild. Using this stimulation voltage the force-frequency characteristics of the muscle were then monitored by stimulating at 1 Hz for 5 seconds and at 10, 20, 50 and 100 Hz for three seconds each in succession.

For the purpose of the present investigation force attained at a stimulation frequency of 20 Hz was expressed as a percentage of that achieved at 50 Hz ($T_{20/50} \%$). This enabled any relative loss of force at low as compared to high frequencies of stimulation to be determined.

Plasma Creatine Kinase.

At each sampling time ~ 5 ml of venous arterialised blood was taken from the antecubital vein and dispensed into heparinised tubes. Samples were centrifuged at 2000-3000G within 24 hours of being taken and the plasma pipetted off for subsequent analysis.

Creatine kinase activity was determined using an enzymatic technique (Forster et al, 1974) based on the following series of reactions;



The activity of creatine kinase was assessed spectrophotometrically by measuring the change in absorbance at 340 nm due to the appearance of NADPH.

Experimental Procedure

Each subject performed three treadmill tests which were carried out at least seven days apart. The first of these was a progressive exercise test which was employed to determine maximal oxygen uptake, the remaining two tests represented the experimental runs and consisted of prolonged uphill or downhill walking.

During the progressive test which started on the level, subjects were required to walk at a speed of either 5 or 6.4 km hr⁻¹ for 5 min at each of a number of progressively increasing gradients until exhaustion. During the final two minutes at each work load $\dot{V}O_2$ was measured using a continuous open circuit technique as previously described (see Chapter 3) and subjects were assumed to have reached $\dot{V}O_{2\text{max}}$ when there was no further increase in $\dot{V}O_2$ with increasing work load.

From this data it was possible to predict an incline which at the same walking speed would require an oxygen uptake equivalent to ~80% of the subjects $\dot{V}O_{2\text{max}}$. In the uphill test subjects were required to walk at this speed and incline on the treadmill for a period of one

hour during which their oxygen uptake was measured a number of times as previously described. During the downhill run subjects walked at a speed of 6.4 km hr^{-1} with the treadmill at its steepest gradient (16.5°) until exhaustion which occurred somewhere between 29-40 min in the present group of subjects.

Oxygen uptake measurements were made at various stages during exercise using the open circuit technique described earlier.

On the day preceding both the uphill and the downhill tests a control blood sample was taken for the determination of creatine kinase activity and the quadriceps muscle was tested for the following;

- 1) Maximal short-term power output at pedalling speeds of $\sim 50, 80, 110$ and 125 rev min^{-1} .
- 2) Maximal voluntary isometric force
- 3) Electrically stimulated contractions at frequencies of 1, 10, 20, 50 and 100 Hz.

At the end of the treadmill exercise muscle temperature was measured in the lateral part of the quadriceps muscle at three depths as previously described (Chapter 5) and the force and power measurements described above were then repeated at $\sim 1, 4, 8, 12$ and 24 hours of recovery and thereafter at 24 hour intervals until recovery was complete. Blood samples for the determination of plasma creatine kinase were taken once a day until pre-exercise values were reattained.

RESULTS

Maximal oxygen uptake for the four subjects was 2.69 ± 0.39 litres min^{-1} (mean \pm SD) or 44.2 ± 7.1 ml $\text{kg}^{-1} \text{min}^{-1}$ when standardised for body weight.

During uphill walking the work load was equivalent to 79.1 ± 1.9 % $\dot{V}O_2$ max which was significantly higher ($p < 0.05$) than that attained during the downhill test (Table 2). However while the oxygen uptake remained fairly constant during the uphill test, in downhill walking it tended to increase as the exercise proceeded so that it was considerably higher at the end of the test than during the initial few minutes of exercise (Fig. 2). However despite the fact that the downhill exercise was metabolically less taxing than the uphill test subjects were only able to continue the former for between 29-40 min (mean \pm SD = 34.6 ± 5.4 min) at which time they were at the point of physical collapse.

At the end of exercise muscle temperature was measured in the lateral part of the quadriceps muscle at three depths and in all four subjects both deep (at 4cm) and mean muscle temperature were found to be higher following downhill as opposed to uphill walking (Table 2).

During recovery all the parameters measured showed greater changes following downhill as opposed to uphill walking. After the uphill test the maximum voluntary contraction (MVC) was only significantly reduced in measurements made approximately 24 hours post-exercise when a mean decrement of 5.3% was observed (Table 3, Fig. 3a). Following the downhill test the loss of force was much

Subject	UPHILL WALKING					DOWNHILL WALKING				
	Speed (km hr ⁻¹)	Incline (%)	Duration (min)	Mean VO ₂ (% VO _{2max})	T _m (°C)	Speed (km hr ⁻¹)	Incline (%)	Duration (min)	Mean VO ₂ (% VO _{2max})	T _m (°C)
PD	6.4	9	60	80.6	37.47	6.4	29	40	43.8	38.97
CG	5.0	12	60	79.3	37.60	6.4	29	29	57.5	38.13
OR	5.0	12	60	76.4	35.97	6.4	29	32	63.7	39.43
AS	6.4	12	60	80.1	39.53	6.4	29	38	53.7	40.33

Table 2 Experimental data for uphill and downhill walking. Muscle temperature (T_m) is expressed as a mean of the 4, 3 and 2 cm values.

Subject	UPHILL WALKING					DOWNHILL WALKING				
	Speed (km hr ⁻¹)	Incline (%)	Duration (min)	Mean $\dot{V}O_2$ (% $\dot{V}O_{2max}$)	T_m (°C)	Speed (km hr ⁻¹)	Incline (%)	Duration (min)	Mean $\dot{V}O_2$ (% $\dot{V}O_{2max}$)	T_m (°C)
PD	6.4	9	60	80.6	37.47	6.4	29	40	43.8	38.97
CG	5.0	12	60	79.3	37.60	6.4	29	29	57.5	38.13
OR	5.0	12	60	76.4	35.97	6.4	29	32	63.7	39.43
AS	6.4	12	60	80.1	39.53	6.4	29	38	53.7	40.33

Table 2 Experimental data for uphill and downhill walking. Muscle temperature (T_m) is expressed as a mean of the 4, 3 and 2 cm values.

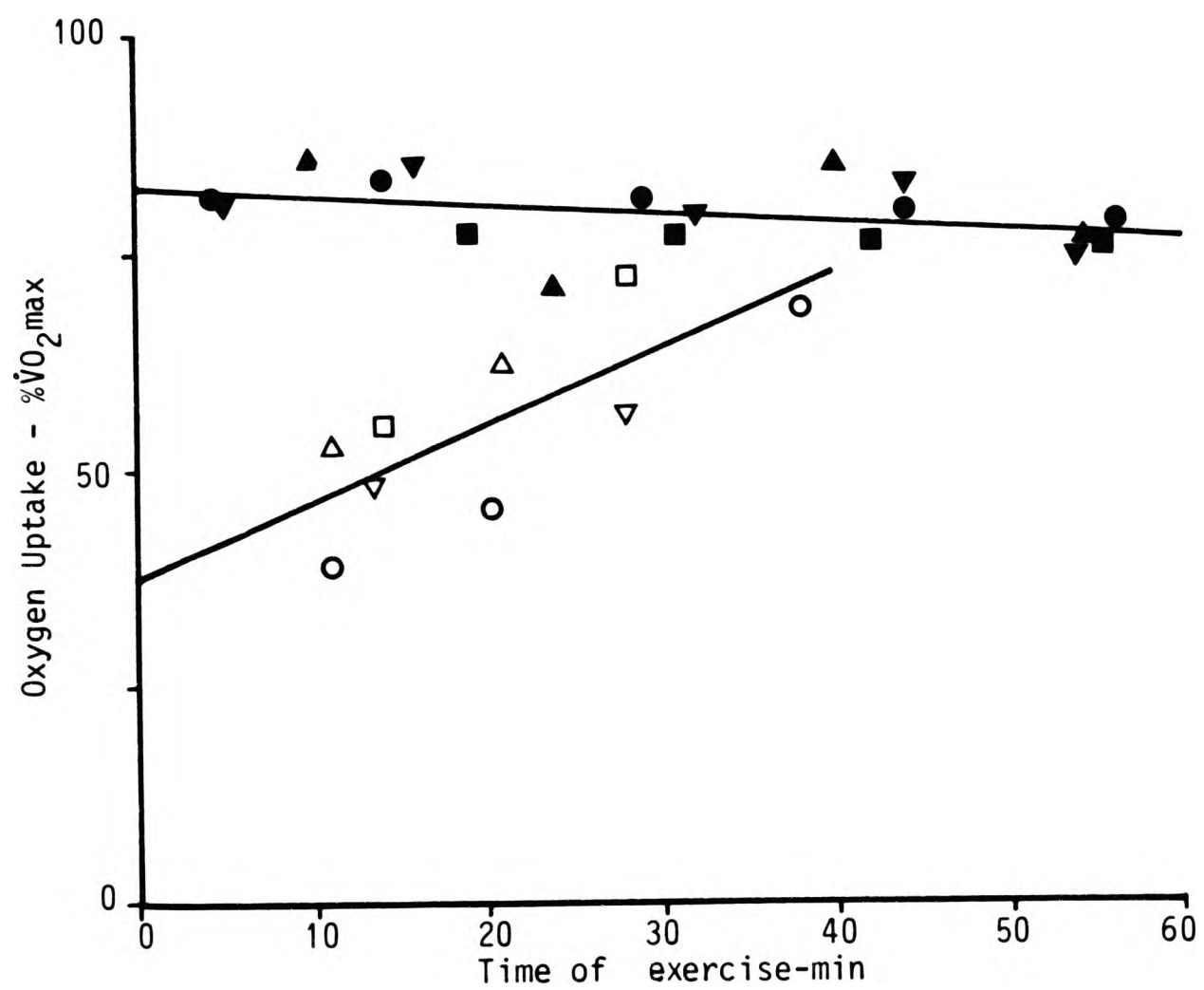


Fig.2 Oxygen uptake (expressed as % $\dot{V}O_2$ max) measured during prolonged uphill (closed symbols) and downhill (open symbols) treadmill walking. Individual data are shown for the 4 subjects studied.

greater and the MVC remained significantly lower than the control value ($p < 0.05$) until 4 days post-exercise (Table 3, Fig.3b). The greatest loss of force was observed ~24 hours after exercise when the mean decrement was $45.3 \pm 11.7\%$.

Following both types of exercise electrical stimulation of the quadriceps muscle showed that the force generated at 20 Hz was considerably reduced when compared to that attained at 50 Hz. After uphill walking the greatest decrement in low frequency force was observed approximately 12 hours later when the $T_{20/50} \%$ ratio had fallen to $61.66 \pm 5.75 \%$ compared to a control value of $81.08 \pm 2.83 \%$ (Table 3, Fig.4a). Following the downhill test the decrement was even greater with the $T_{20/50} \%$ ratio falling to $36.61 \pm 7.26 \%$ against a control value of $80.51 \pm 2.55 \%$ (Fig.4b).

Maximal short-term power output measured on the isokinetic ergometer was depressed at all four speeds following uphill walking although at no time during recovery were the values significantly reduced below the controls (Table 3, Figs.5a-d). The only exception to this occurred in the measurement made approximately 24 hours post-exercise at $\sim 110 \text{ rev min}^{-1}$ where the decrement was found to be significant ($p < 0.01$). Once again following downhill walking the fall in maximal short-term power output was much more marked with the greatest decrements occurring 4-48 hours post-exercise (Table 3, Figs.5e-h). The maximal mean decrements in this group of subjects expressed as a percent of the control value were 14.84, 16.0, 22.89 and 17.25 at 50, 80, 110 and 125 rev min^{-1} respectively. Following both types of exercise the greatest decrement was therefore observed at a crank velocity of 110 rev min^{-1} and after downhill walking the

Time post exercise (hrs)	UPHILL WALKING						DOWNHILL WALKING					
	MVC	T _{20/50}	PPmax 50	PPmax 80	PPmax 110	PPmax 125	MVC	T _{20/50}	PPmax 50	PPmax 80	PPmax 110	PPmax 125
1	91.5	98.4	94.1 ±4.1	98.5 ±1.8	96.5 ±4.3	99.7 ±4.4	-	-	89.4 ±11.3	**86.6 ±2.9	**85.3 ±2.6	91.8 ±7.7
4.5	96.9 ±7.1	93.9 ±8.4	91.3 ±6.5	94.1 ±7.4	93.5 ±7.5	97.9 ±12.0	**73.5 ±6.3	***58.7 ±4.8	87.8 ±8.4	**90.1 ±2.5	**86.7 ±3.6	85.8 ±10.8
7.5	101.0 ±6.1	**81.1 ±2.8	100.0 ±5.5	93.8 ±8.7	91.9 ±9.9	101.7 ±9.2	**72.7 ±8.0	**48.6 ±10.8	88.9 ±10.6	*89.2 ±5.7	***84.1 ±2.2	88.8 ±7.8
11	93.1 ±7.2	*76.0 ±4.8	94.3 ±11.6	93.9 ±12.5	92.9 ±8.5	97.0 ±13.2	**67.1 ±9.0	**45.4 ±8.8	88.4 ±9.3	*91.8 ±4.0	***84.3 ±0.9	90.0 ±10.3
24	*94.7 ±2.1	*89.7 ±5.2	95.7 ±7.6	95.9 ±4.7	**95.3 ±1.6	96.9 ±2.6	**54.7 ±11.7	*69.1 ±16.6	85.2 ±19.8	84.0 ±15.6	*77.1 ±10.0	88.2 ±11.2
48	94.5 ±10.7	96.2 ±9.3	94.8 ±6.5	93.9 ±13.9	97.0 ±6.7	96.0 ±8.4	*57.8 ±16.2	*88.4 ±6.2	90.2 ±21.5	*85.2 ±8.2	*77.7 ±15.9	82.8 ±17.6
72	104.1 ±1.0	94.4 ±3.6	100.3 ±3.7	96.6 ±12.3	98.2 ±7.1	97.2 ±12.0	*69.9 ±18.1	*89.2 ±5.4	89.0 ±15.4	89.5 ±10.1	*85.3 ±6.5	95.3 ±15.4
96	96.6 ±2.0	101.5 ±14.8	101.3 ±5.4	99.1 ±6.6	96.2 ±8.0	100.9 ±4.3	89.7 ±8.4	90.1 ±12.3	97.6 ±10.0	100.5 ±5.4	*92.4 ±3.7	99.9 ±8.6
192	-	-	-	-	-	-	90.3 ±1.5	91.9 ±1.2	98.0 ±8.5	100.0 ±1.5	93.1 ±5.1	102.8 ±10.7

Table 3 Maximum voluntary force (MVC), T_{20/50} ratio and maximal peak power (PPmax) at 50, 80, 110 and 125 rev min⁻¹ following prolonged uphill and downhill walking. All values are expressed as a percent of the initial value determined the day before testing. The significance of change is denoted as follows; * p < 0.05; ** p < 0.01; *** p < 0.001.

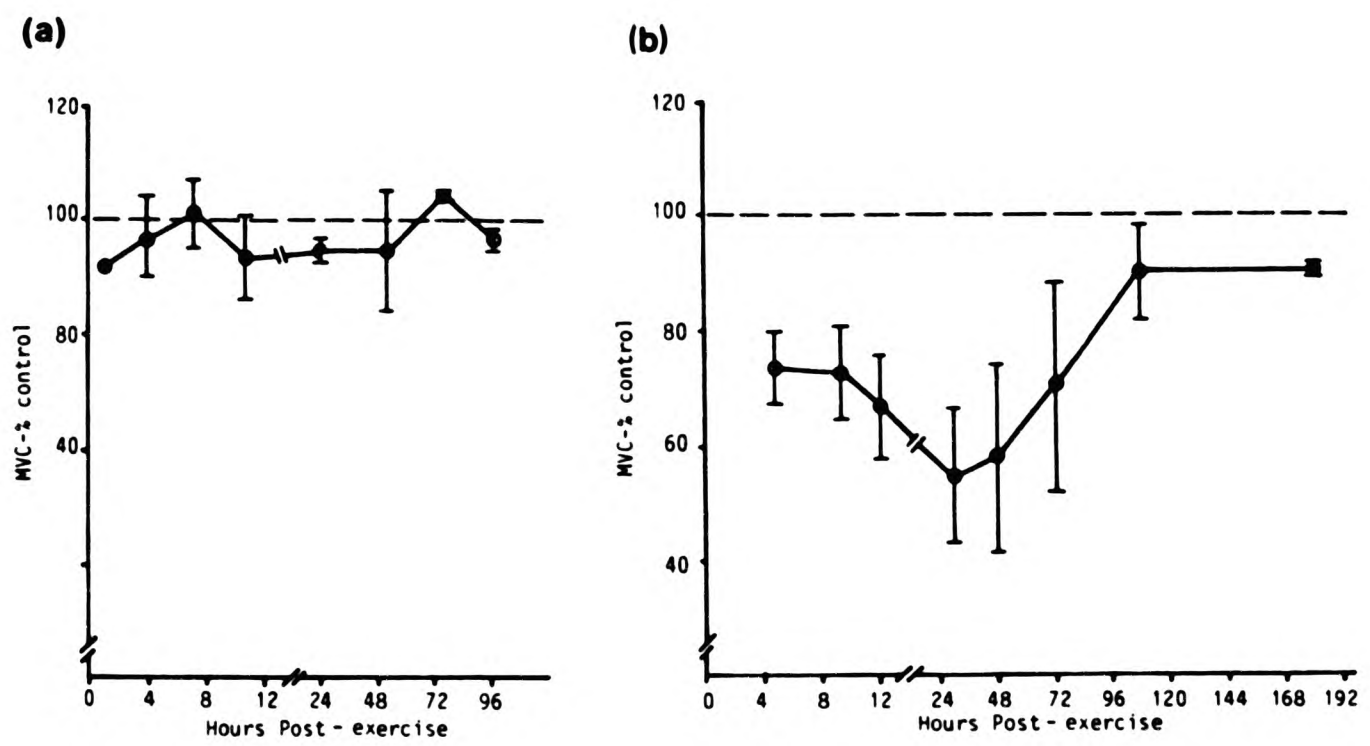


Fig.3 The force exerted by maximal voluntary contraction of the quadriceps muscle (% control) during recovery from prolonged (a) uphill and (b) downhill walking. Mean values \pm one SD are shown (n=4).

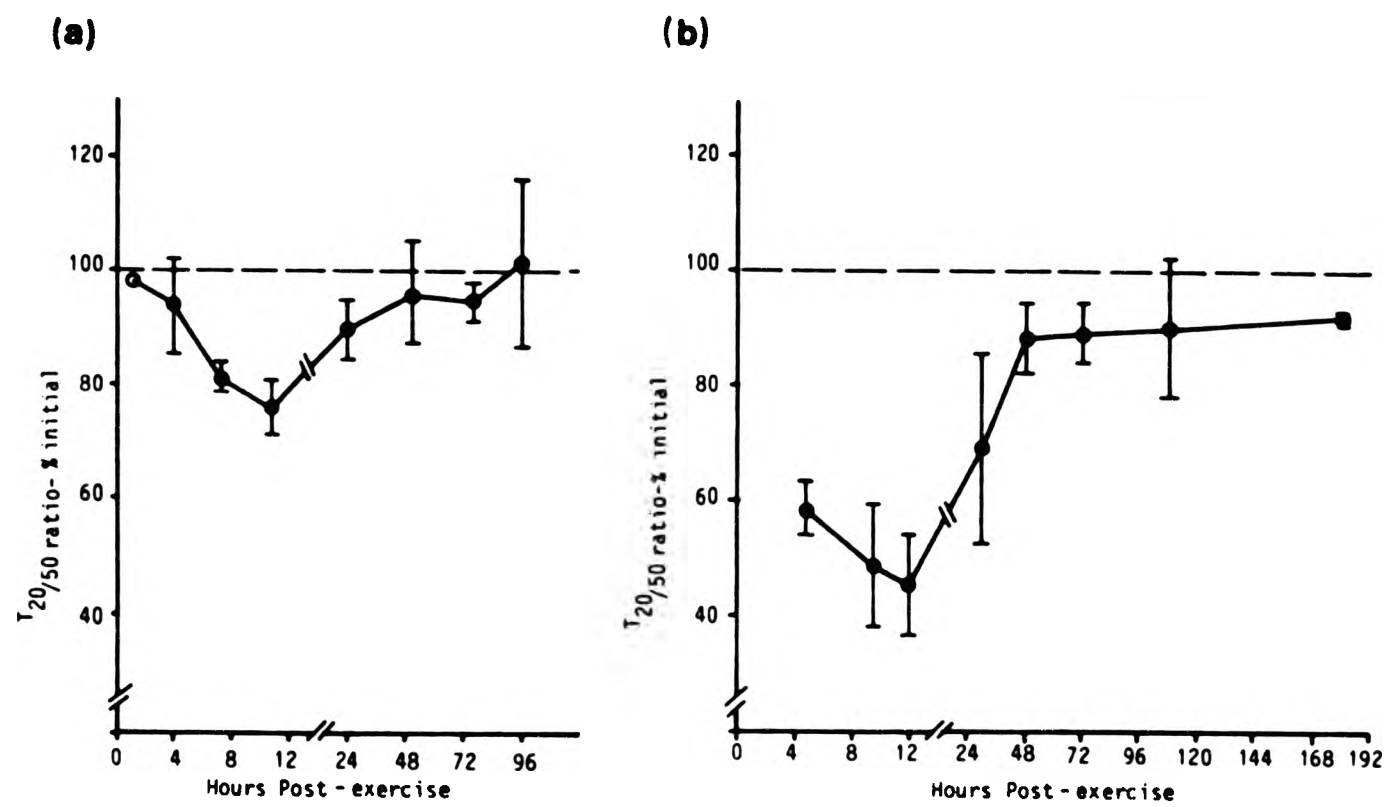


Fig.4 The force generated by electrical stimulation of the quadriceps muscle during recovery from (a) uphill and (b) downhill walking. The force exerted at 20 Hz is expressed relative to that at 50 Hz and values are given as a percentage of the initial control ratio. Values shown are the mean \pm SD (n=4).

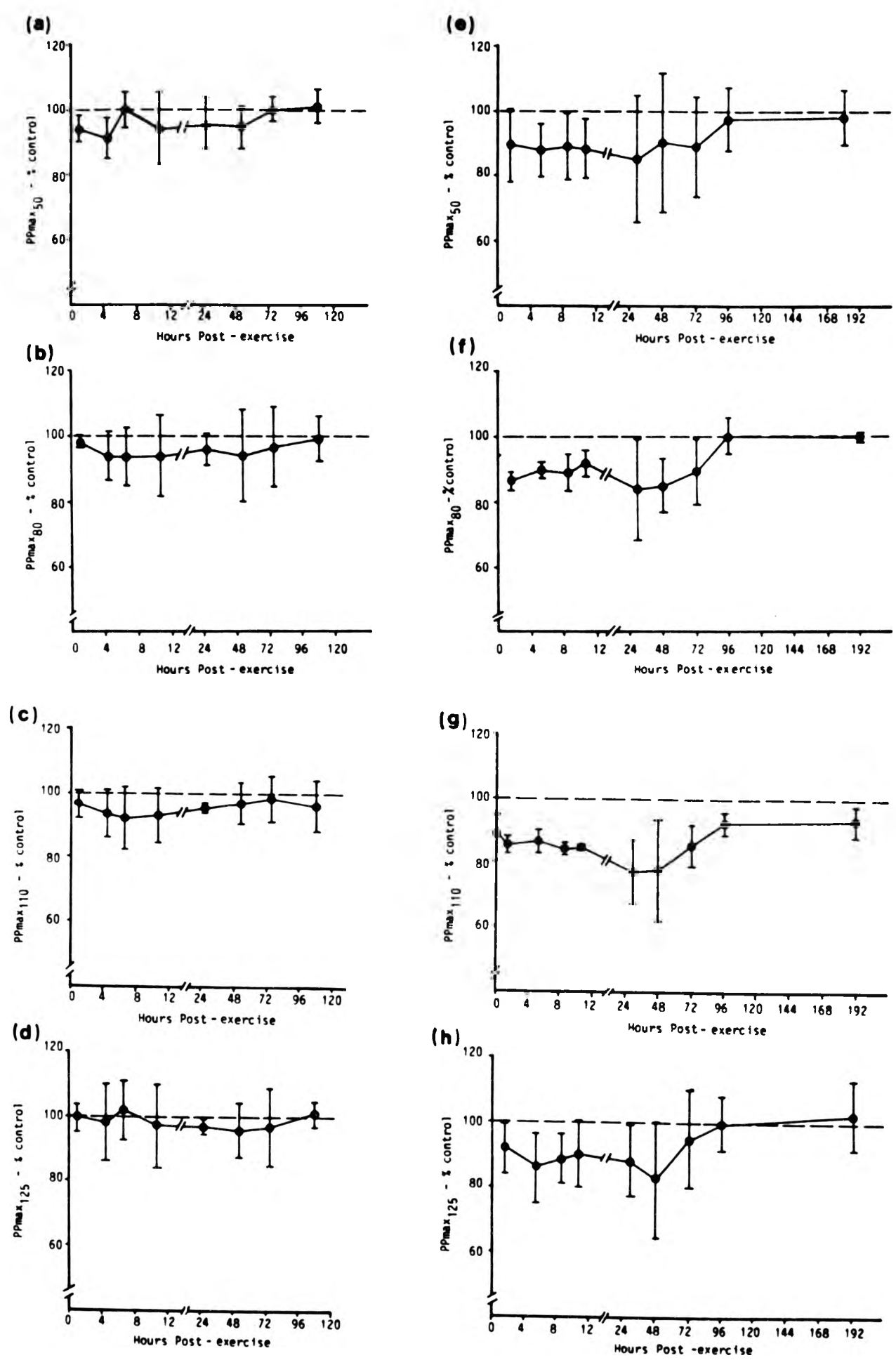


Fig.5 Maximal peak power attained in the 20 sec maximal cycling test during recovery from prolonged uphill (a-d) and downhill (e-h) walking. Maximal peak power (PPmax) at each speed (50, 80, 110 and 125 rev, min⁻¹) is expressed as a percentage of the control value. Mean values \pm one SD are shown (n=4).

recovery in maximal short-term power output was also more prolonged at this speed where it was incomplete still 7 days after exercise.

Plasma creatine kinase activity was elevated in all subjects 4 hours post-exercise and continued to rise over the following 24 hours to values between 2-7 times the normal figure (Fig.6). In 3 of the 4 subjects CK activity then began to decline. The remaining subject however showed a dramatic and delayed rise in plasma CK activity which was increased 100-fold by 4 days after exercise and was considerably elevated still at 9 days. Despite this drastic increase in plasma creatine kinase activity the decrements in force and power output in this subject were no greater than those observed in the other 3 subjects.

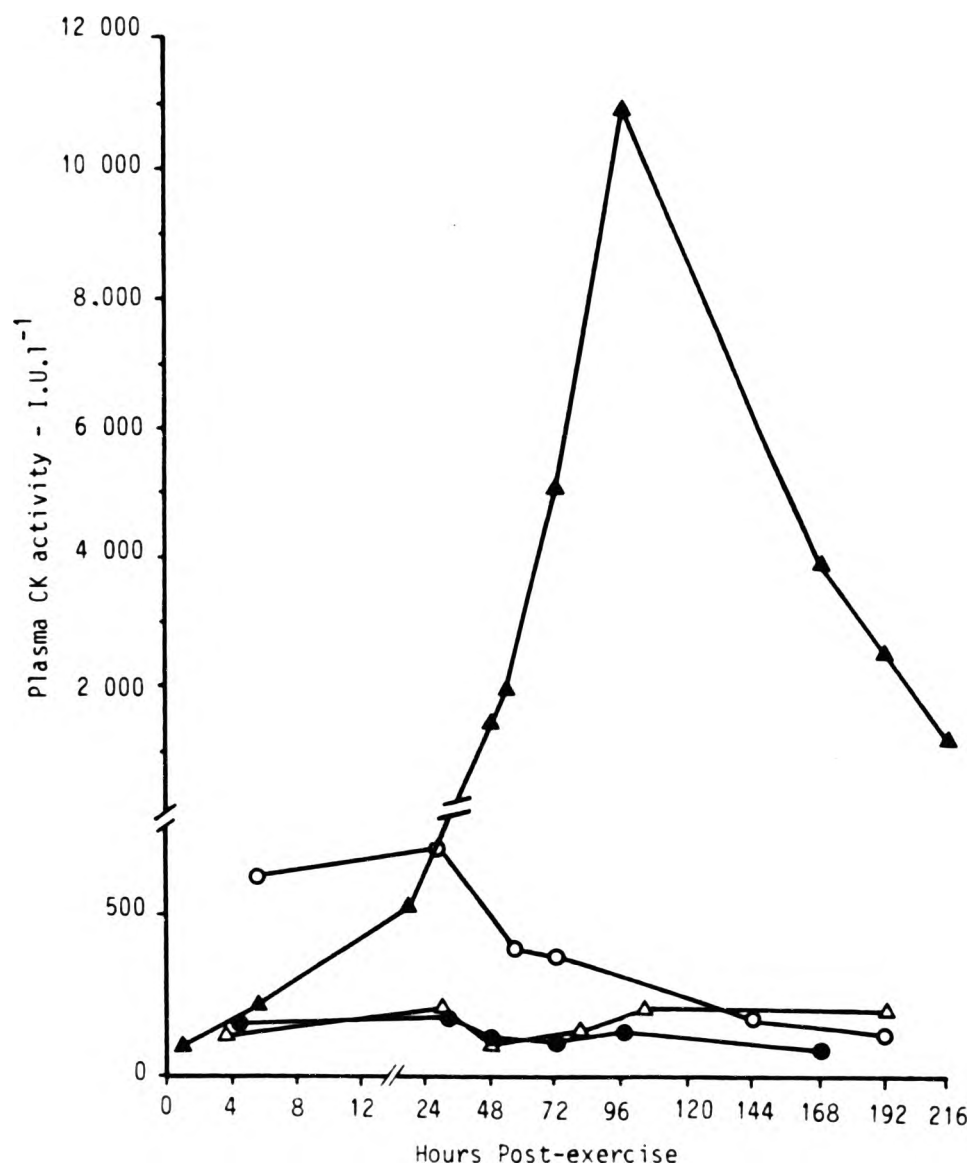


Fig.7 Plasma creatine kinase activity (I.U.l⁻¹) during recovery from prolonged downhill walking. Data are shown for the 4 subjects studied.

Discussion

In the present study changes in all the parameters studied were more marked following downhill as opposed to uphill walking despite the fact that the metabolic cost was relatively less in the downhill test. This argues against a metabolic basis for the observed changes in force and power output following exercise and suggests that some factor related more to the eccentric component of the exercise was responsible.

The greater decreases in maximum voluntary force and in the force generated at low frequencies of electrical stimulation following eccentric work are consistent with the findings of previous investigations (Edwards, Mills and Newham, 1981; Davies and White, 1981, 1982; Friden, Sjöholm and Ekblom, 1983; Newham, Mills, Quigley and Edwards, 1983c). However in contrast to the findings of Edwards and his colleagues (Edwards et al, 1981, Newham et al, 1983c) who observed the greatest decrement in force immediately following exercise, in the present study the force generated by the muscle continued to decrease for up to 12 hours after terminating exercise in the case of low frequency stimulated contractions and for up to 24 hours in the case of the MVC with complete recovery only occurring 5-8 days post-exercise.

These differences may be due to the extent of eccentric work involved in the exercise. In the aforementioned studies the eccentric exercise consisted of box-stepping for 15-20 min where negative work by the quadriceps muscle (stepping down) was consistently performed by the same leg. In the present study all subjects were required to

walk downhill at a steep gradient and fast walking speed to the point of physical collapse which was between 29-40 minutes. Deterioration towards the end of exercise was sudden and very rapid and was characterised by a feeling of profound weakness until eventually the subject was unable to continue. The severity of the exercise employed in this study might be responsible for the slower recovery in contractile force when compared to box-stepping.

The decreases in short-term power output observed in the present study were also greater following downhill as opposed to uphill walking . When expressed relative to pre-exercise values these were of approximately the same magnitude at all the speeds studied although there was consistently a slightly greater decrement at 110 rev min⁻¹ throughout the recovery period. The greatest loss of power as with maximal isometric force was observed 1-2 days after exercise with pre-exercise values being attained 4-8 days afterwards at all speeds but 110 rev min⁻¹ where recovery was still incomplete at eight days.

The greater loss of power at 110 rev min⁻¹ as compared to the other speeds may be related to the fact that this velocity is very near to the optimum velocity for power output (cf. Chapter 2) where any detrimental effects of the previous exercise might be more pronounced.

The decrements in maximal short-term power output observed in the present study may be compared with the findings of Friden et al (1983) who employed an isokinetic (Cybex) dynamometer to measure the peak torque attained at different angular velocities during single isolated contractions of the knee-extensor muscles. These workers

found considerable reductions in peak torque following eccentric cycling exercise and with the exception of the forces attained at zero speed (isometric force) they found this decrement to be greater the higher the angular velocity with the greatest observed changes occurring approximately 20 minutes after exercise. It should be pointed out however that these workers made no further measurements after the 20 minute tests until 3 days post-exercise so that any continued deterioration in the 48 hours following exercise would have gone unnoticed. The time course of recovery appeared however to be similar to that observed in the present study with the force at all but the highest angular velocity of $300^{\circ} \text{ sec}^{-1}$ reaching its pre-exercise value by the 6th day. This may be compared with the more prolonged recovery in power output observed in the present study at 110 rev min^{-1} where the speed of movement at the knee is generally of the order of $250\text{-}270^{\circ} \text{ sec}^{-1}$.

The progressive loss of force and power output which was observed in the present study during the first 1-2 days after downhill walking has not to the author's knowledge been previously reported. As mentioned earlier the observed changes in force and power output cannot be explained simply in terms of the metabolic cost of the prolonged exercise since the energy cost of the downhill test was relatively less than that of the uphill test and yet the former type of exercise produced more profound changes in muscle function than the latter.

It is possible that the greater detrimental effect of downhill exercise upon muscle function is related to the higher tensions developed in the muscle during eccentric as compared to concentric

contractions (Katz, 1939; Asmussen, 1956; Curtin and Davies, 1973). Studies using integrated electromyography (IEMG) have shown that during eccentric contractions IEMG activity at a given tension is approximately half that observed during concentric contractions (Bigland, Ritchie and Woods, 1976). This suggests that the tension per active muscle fibre is effectively doubled if EMG activity is assumed to be proportional to the total fibre activity. These high tensions per unit cross-sectional area during eccentric contractions will put great mechanical stress on the muscle and its attachments and it is possible that this could lead to structural damage either to the contractile elements or to the membrane systems running between the fibres.

In recent studies evidence of structural changes in muscle following prolonged exercise has been found both in laboratory animals (van Linge, 1962; Hecht, Schumann and Kunde, 1975; Armstrong, Ogilvie and Schwane, 1983) and in man (Newham, McPhail, Mills and Edwards, 1983b; Friden, Sjostrom and Ekblom, 1981, 1983; Schwane, Johnson, Vandenakker and Armstrong, 1983) and a number of workers reported such changes to be associated more with the eccentric component of the exercise (Newham et al, 1982; 1983b; 1983c; Armstrong et al, 1983). Such disturbances to the myofibrillar structure included broadening and streaming of the Z-lines and in some cases total disruption of the sarcomeres (Friden et al, 1981; Newham et al, 1983b). Newham et al found that immediately following eccentric exercise such damage was often concentrated in small areas affecting only one or two adjacent myofibrils while in biopsies taken approximately 30 hours post-exercise the damage although essentially the same as that observed

earlier, had developed to involve a greater number of sarcomeres and also tended to be more widespread rather than focal. Armstong et al (1983) made a similar observation in rat muscle where they found the amount of necrosis following eccentric exercise to be greater two days after exercise than in the immediate post-exercise period.

If such damage occurred only during the exercise period then one would expect the overall trend in the time following exercise to be towards recovery. However the results of the aforementioned studies seem to indicate that considerable damage develops in the post-exercise period resulting in an increased severity of structural lesions 1-2 days post-exercise. It may be that the initial exercise causes microscopic damage which appears as focal lesions and that these become progressively more extensive during the following few days. Why the damage should progress in this way during recovery is not clear. Newham et al (1983b) suggested that the apparent increase in damage may be part of the inevitable process of repair or alternatively that continued use of the muscle following the initial changes served to exacerbate the damage.

This continued deterioration of muscle structure following eccentric exercise may explain the results of the present study where the greatest loss of force and power output was observed sometime after terminating the exercise. The initial cause of such damage however is open to discussion. One mechanism that may be involved is an inability to maintain calcium homeostasis due to damage to the sarcoplasmic membrane (Wrogemann and Pena, 1976). It was suggested by Jones (1981) that such damage may result in a lowered release of calcium for each excitatory action potential which could explain the

loss of force observed at low stimulation frequencies.

Friden et al (1983) suggested that the resultant flooding of calcium into the muscle cell as a result of damage to the sarcoplasmic reticulum could cause a calcium-induced weakening of the Z-band which might eventually result in sarcomere disruption.

Another possibility is that disruption of the protein elements in the myofibrils during exercise may expose the structural components of the muscle to hydrolysis by proteases thought to be located in the sarcoplasmic reticulum. In animal skeletal muscle a calcium-activated protease enzyme has been found which causes specific removal of Z-bands and is thought to be involved in myofibrillar protein turnover (Busch, Stromer, Goll and Suzuki, 1972; Reveille, Goll, Stromer, Robson and Dayton, 1976). If such an enzyme existed in human skeletal muscle then it is possible that high levels of intracellular calcium following eccentric exercise may activate the enzyme to increase the damage which could explain why the condition of the muscle initially deteriorates in the first 1-2 days after exercise.

Another factor which has not yet been mentioned which could conceivably contribute to the eccentric exercise-induced damage is muscle temperature since in the present study this was considerably higher following downhill as opposed to uphill walking. This might be expected since muscle blood flow may be related more to the metabolic needs of the muscle rather than heat transport. Thus the heat produced during eccentric work may to some extent be contained within the muscle. If high muscle temperatures in conjunction with the high forces attained during negative work were a factor involved in the

disruption of the muscle's contractile elements then this could explain why eccentric contractions have such an adverse effect. Whatever the ultimate cause of the damage however it is likely to have a marked effect on the contractile properties of the muscle concerned resulting in a loss of muscle function as observed in the present study.

Further support for the hypothesis that some form of structural damage is responsible for the decrements in force and power output following eccentric exercise is obtained from the CK measurements which were made after the downhill test. Elevated levels of this enzyme in the plasma are generally taken to be an indicator of muscle damage (Altland and Highman, 1961; Halonen and Konttinen, 1962) and a number of workers have found such increases to be related more with eccentric than concentric contractions (Armstrong et al, 1983; Newham et al 1983a; Schwane et al, 1983). In the present study plasma CK activity was substantially elevated in all four subjects following the downhill test although it should be pointed out that the extent of this increase tended to vary somewhat between individuals. In 3 of the subjects peak values of plasma CK were attained approximately 24 hours post-exercise where the increase was approximately twice the resting value in 2 of the subjects and about seven times the resting value in the other. The fourth subject (AS) however showed a sudden and marked increase in CK activity ~24 hours post-exercise although the peak value which was approximately two orders of magnitude greater than the control value was not attained until the fourth day after exercise. These differences in CK activity were not however reflected in the force and power measurements since the subject who showed this great increase in plasma CK showed similar decrements in power output,

maximum voluntary force (MVC) and the $T_{20/50}$ % ratio to the remaining subjects.

Large delayed increases in plasma CK such as that observed in AS in the present study have previously been reported both in rat muscle (Armstrong et al, 1983) and in human muscle (Newham et al, 1983a) following eccentric exercise. However the mechanisms involved in the efflux of intramuscular enzymes into the plasma during and following exercise have yet to be elucidated. Some investigators have suggested that changes in membrane permeability induced by contractile activity are responsible for such increases in plasma CK (Altland et al, 1961; Halonen et al, 1962) while Rowland (1980) proposed that actual damage to the muscle cell membrane is the cause of this efflux. It may be that the small increase in CK activity immediately following eccentric exercise is due to a change in membrane permeability as a result of small localised lesions in the myofibrils as observed by Newham et al (1983b) but that as the damage becomes more extensive this initiates in some subjects a process which results in large quantities of enzyme being released into the plasma. Why some subjects should be more susceptible to this effect than others is not yet known. Armstrong et al (1983) suggested that perhaps all the fibres recruited sustained sublethal injury and that the necrotic proportion which may be responsible for most of the enzyme efflux simply represented random events where the process passed a point of no return. A further possibility may be that the necrotic fibres merely represent a population of susceptible cells which may have been weakened by some other condition unrelated to the exercise but which manifested itself under the exercise stress.

Conclusions

The results of the present study indicate that exercise with a large eccentric component produces greater changes in maximal short-term power output and in voluntary and stimulated isometric contractions than does concentric work of a considerably higher metabolic cost. It is therefore proposed that these changes and the increased activity of plasma CK which was observed after eccentric exercise are related to disturbances of the myofibrillar structure and possibly also to damage to the muscle cell membrane as a result of mechanical overload.

CHAPTER 7

Summary
and
Conclusions

The aims of this thesis were to assess maximal power output from human muscle during short-term dynamic exercise and to investigate the factors which might influence it.

In order to determine the maximal power which could be generated in this type of exercise it was first necessary to assess the force-velocity and power-velocity characteristics of the muscles to be studied to enable the optimum velocity for power output to be identified. This was done by measuring the force exerted and power generated during an isokinetic cycling test which was performed at a number of different pedalling speeds. Maximum force was shown to be inversely and linearly related to velocity in the majority of subjects studied, in contrast to previous *in vitro* findings. These results were thought to reflect the modifying influence of the limb and lever system on the force being generated. It was also proposed that the development of very high forces at slow contraction speeds may be inhibited by some central control mechanism 'in vivo' in order to prevent muscle damage.

As a mathematical consequence of this linear correlation between force and velocity the relationship between maximal peak power and crank velocity described a parabola the apex of which represented the optimum velocity for power output. At this optimum velocity maximal short-term power output was shown to be related to the upper leg muscle (plus bone) volume and also in a subgroup of the adult subjects to the percent cross-sectional area of type II fibres (%CSA type II). In this same subgroup there was also a positive correlation between optimum velocity and the %CSA type II although there was no

correlation between optimum velocity and either the age or sex of the subject. Thus for subjects with an equal distribution of Type I and Type II fibres an optimum velocity of $117 \text{ rev. min}^{-1}$ was predicted. This indicates that in population studies on normal untrained subjects the load on an ordinary cycle ergometer should be chosen so that subjects achieve approximately this speed if a measurement of their maximal power output is to be made.

During the course of the above experiments it was noted that the majority of subjects attained maximal power within a few seconds from the start of exercise when the ATP used during contraction would be derived mainly from the splitting of phosphorylcreatine (PC). It was proposed that the muscles' capacity for power output during brief maximal exercise would therefore be dependent upon the prevailing concentration of PC within the active muscle fibres. In the next part of this thesis this was investigated by having subjects exercise at different submaximal work loads prior to the performance of the maximal test in order to previously deplete the muscles' high energy phosphate. Where the previous work load was above 60% of the subjects' $\dot{V}O_2\text{max}$ there was a corresponding decrease in subsequent short-term power output with increasing prior exercise intensity which was thought to reflect the extent of PC depletion caused by the previous exercise. Later experiments on the recovery from such exercise appeared to confirm this view since the pattern and time course of recovery in power output were remarkably similar to those observed for PC resynthesis following heavy dynamic exercise.

When the short-term power output measurement was preceded by lower

levels of prior exercise substantial improvements in power output were observed which were attributed to the concomitant increase in muscle temperature. The effect of warm-up was examined in more detail in a further set of experiments to determine to what extent this might improve subsequent performance. Both active and passive warm-up of varying intensity were investigated and the improvements in power output were found to be similar independent of the method of warm-up. These improvements were of the order of 2.5% per °C rise in muscle temperature which were consistent with previous observations.

With active warm-up the greatest improvements in power output were therefore observed following the highest levels of prior exercise providing the interval between warm-up and the subsequent performance was long enough to allow for phosphorylcreatine resynthesis. However these improvements were of the same order as those achieved in earlier experiments when the maximal test was immediately preceded by low levels of prior exercise ($< 60\% \dot{V}O_2\text{max}$).

These findings could have important implications for the athlete to whom the attainment of a maximal performance is paramount. A moderate warm-up immediately before the start of the event may be sufficient to produce a maximal improvement in power output where a heavier warm-up would result in high-energy phosphate depletion and a reduction in subsequent power. However where a delay of several minutes is expected before the start of an event then the present results suggest that a heavier warm-up would be more beneficial. The optimum warm-up will therefore depend upon the interim period between the warm-up and the start of the event and the intensity of the warm-up should therefore be chosen with this in mind.

In the final part of this thesis the effect of prolonged concentric and eccentric exercise upon subsequent performance was examined. It was found that the eccentric exercise had a more marked and long-lasting effect upon performance during both dynamic and isometric exercise than concentric work even though the metabolic cost of the latter type of exercise was much greater. Following eccentric exercise the greatest decrements in force and power output were observed 12-48 hours afterward with recovery occurring over the next few days. This loss of muscle function was attributed to mechanical damage to the contractile elements as a result of the high tensions developed during eccentric as opposed to concentric contractions.

The loss of force following downhill walking was much less for dynamic contractions than for isometric contractions which could reflect differences in recruitment pattern in the two types of contraction. Electrical stimulation of the quadriceps muscle demonstrated that there was a profound loss of force at low stimulation frequencies when compared to the higher ones. It is possible that higher stimulation frequencies where the force is comparatively well preserved are employed more during rapid dynamic contractions resulting in a smaller decrement in force in this type of exercise.

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APPENDIX I

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Isokinetic Measurement of Maximal Leg Force and Anaerobic Power Output in Children

A.J. Sargeant, P. Dolan, and A. Thorne

Introduction

The assessment of maximal anaerobic power output has usually involved the measurement of external work performed during a brief 'sprint' effort either cycling or stair-climbing (e.g., Asmussen and Boje 1945; Margaria et al. 1966). It is, however, important that if a measurement of true maximal power output is to be made then the active muscles must be operating at optimum velocity for power generation as defined by the force-velocity relationship (Wilkie 1960). It is difficult to guarantee this condition during a freely accelerating sprint effort against a constant load. We have, therefore, developed a technique based on cycling whereby both maximal leg force and power output can be measured under isokinetic conditions. The present paper reports the application of this technique to a group of 13-year-old boys.

Subjects and Methods

Eight active healthy boys (mean age 13.7 years) were studied. The physical characteristics of the boys are given in Table 1. Lean Body Mass (LBM) was estimated from the

Table 1. Physical characteristics of the eight boys studied (mean \pm SD)

Age (years)	Height (cm)	Weight (kg)	LBM (kg)	ULV (l)
13.7 ± 0.3	162.6 ± 13.3	51.2 ± 11.2	42.9 ± 8.9	3.94 ± 0.95

^a Abbreviations: LBM, lean body mass, ULV, upper leg muscle plus bone volume (ULV = mean of right and left legs)

sum of four skinfold measurements (Durnin and Rahaman 1967) and upper leg muscle plus bone volume was measured anthropometrically (Jones and Pearson 1969; Sargeant and Davies 1977b). Informed consent was obtained from the boys, their parents, and the school authorities.

Leg force and power output were measured using a modified stationary cycle ergometer as previously described (Sargeant et al, 1981). Briefly, the modification

consisted of the addition of a 3-hp electric motor, which was used to drive the pedal cranks through a variable reduction gearbox (Fig. 1). The speed of crank rotation could be set in the range 20–200 rpm. Once set, the subject was asked to make a maximal

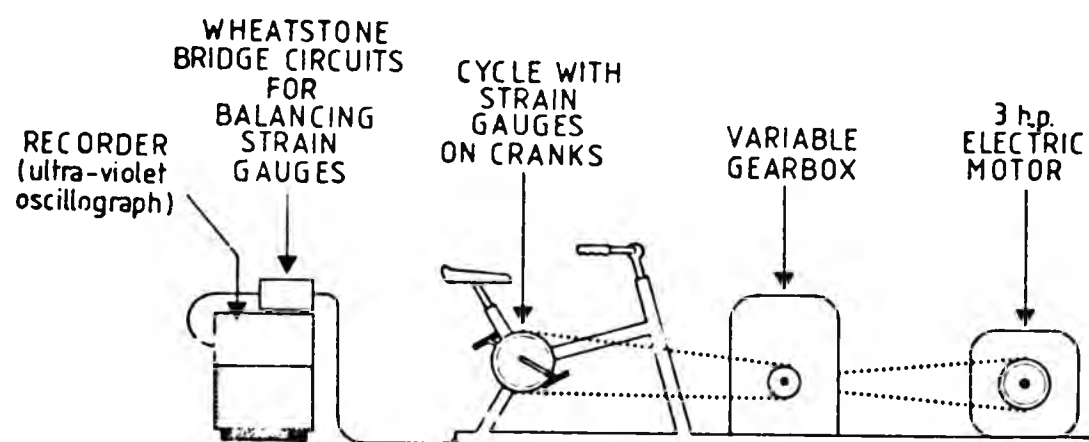


Fig. 1. Schematic diagram of the isokinetic cycle ergometer

effort lasting for 20 s in an attempt to increase the crank velocity. However, due to the characteristics of the motor-gear system, the velocity remained constant at the set level despite maximal effort on the part of the subject. Throughout the 20 s, the forces exerted and, by integration, the power generated were independently and continuously monitored by means of strain gauges bonded to both cranks as previously described (Hoes et al. 1968; Sargeant and Davies 1977a, 1977c). In the present study, measurements were made on each subject at crank velocities of 50, 75, 110 and 120 rpm. Maximal oxygen uptake ($\text{VO}_{2\text{max}}$) was directly measured in separate experiments by standard techniques when the subjects exercised on a conventionally braked cycle ergometer (Mark CFK). Group values are given as mean \pm SD throughout the text.

Results

Figure 2 shows a section of a force recording made during a maximal effort at a pedal frequency of 110 rpm. Peak force was exerted at $\sim 90^\circ$ past the top dead centre in each revolution. During the course of each 20-s effort, the peak force declined from the maximal level (PFmax) attained within the first few seconds (Fig. 3).

The level of PFmax attained by our subjects was inversely and linearly related to crank velocity over the range studied (Fig. 4). As a mathematical consequence, peak power output (PPmax) calculated at the instant of PFmax was a parabolic function of crank velocity and the optimum crank velocity for maximal power output was identified as 111 ± 14 rpm (Fig. 4). At optimal velocity, PPmax for our subjects was 785 ± 164 W (Table 2). This latter value is the product of a single leg at the instant of gener-

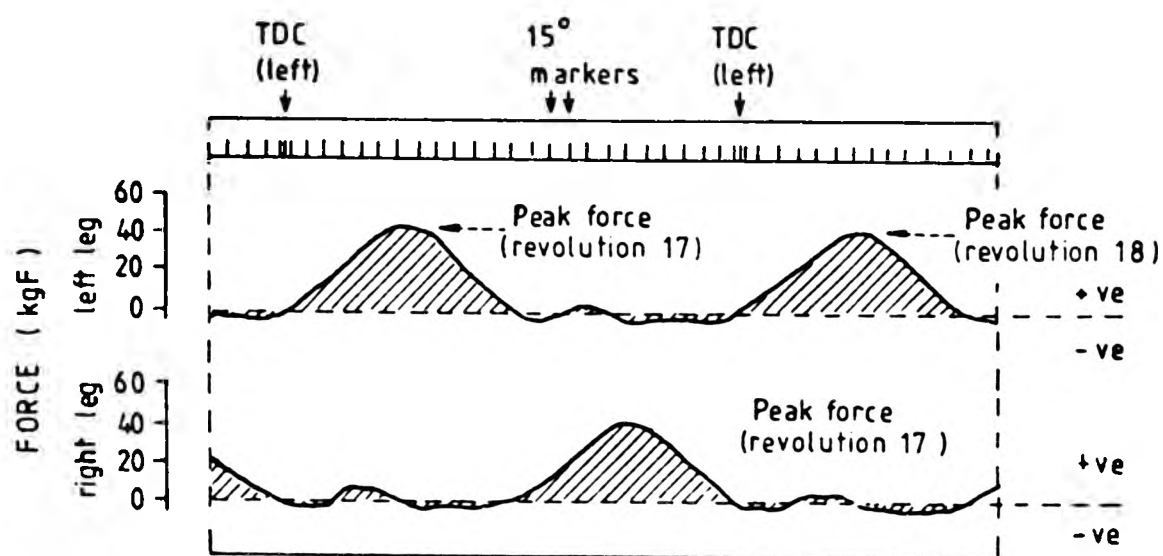


Fig. 2. Section of force recording made during 20-s maximal effort. Positive force (+ve) is applied to the cranks in the direction of rotation; negative force (-ve) counteracts the forward rotation of the cranks and is seen in the recovery phase of each cycle. The 15° interval markers appear along the top of the record. Left hand crank top dead centre (TDC) is given by a triple marker. (Subject TK, crank velocity 110 rpm)

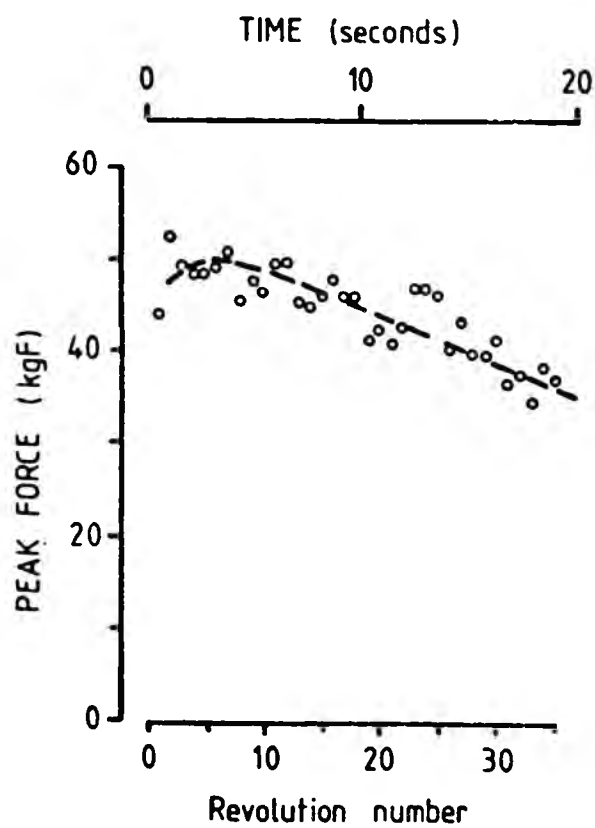


Fig. 3. Peak force (mean right and left legs) for each revolution during 20-s maximal effort. Maximal peak force (PFmax) is achieved within a few seconds and thereafter peak force declines over the 20 s. (Subject TK, crank velocity 110 rpm)

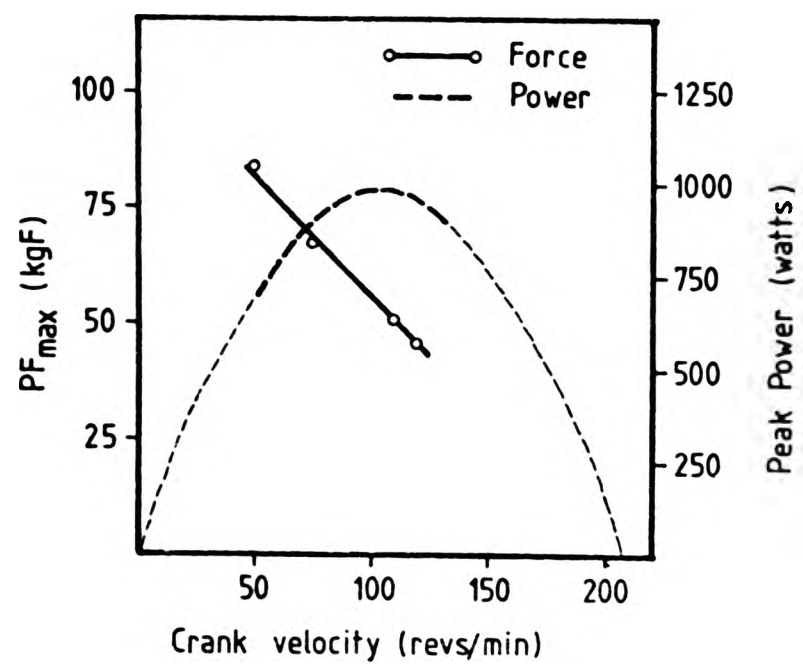


Fig. 4. Relationship between maximal peak force (PF_{max}) and crank velocity. Maximal peak power (PP_{max}) was calculated from the inverse linear relationship of $PF_{max}/velocity$. The heavy dashed line indicates $PP_{max}/velocity$ relationship within experimental limits, the lighter dashed line the theoretical extrapolation. (Subject TK)

Table 2. Maximal power output (\bar{v}) for the eight 13-year-old boys studied (mean \pm SD)

Maximal peak power output (PP_{max}) (one leg)	Maximal mean power - complete revolution (PCR_{max}) (two legs)	Maximal mechanical power available from aerobic sources ^a (two legs)
785 ± 164	395 ± 89	185 ± 38

^a Calculated from net VO_2 max assuming mechanical efficiency of 0.25

ating peak force. By comparison, the maximal mean power output for a complete revolution calculated from the integration of the force records for both legs and including the active and recovery phases of each revolution was 395 ± 89 W at optimum velocity (Table 2).

Discussion

This paper describes the application of an isokinetic technique for the measurement of short-term (anaerobic) power output in children and confirms and extends previous data obtained from experiments on young adults (Sargeant et al., 1981). Consistent with this previous data is the observation of linear force-velocity relationship over the range of crank velocities studied (Fig. 4). The mathematical consequence of such a linear force-velocity relationship is that the power-velocity relationship is of parabolic form reaching an apex at ~ 110 rpm, which is the optimum for maximal power generation and seems to agree with empirical observations on the speed of movement in sprinters and cyclists.

In comparing the present data with that of other groups, it is important to make standardizations with respect to the size of the active muscle. When this is done by expressing power in relation to the upper leg muscle (plus bone) volume (ULV), the present group of 13-year-old boys are seen to generate somewhat lower levels of maximal peak power than the young adults previously studied (204 ± 36 compared with 286 ± 12 W/l ULV; Sargeant et al., 1981). We would speculate that this could be partly due to the latter group having (at 50% of the total cross-sectional area) a greater proportion of type II fibers than the present group, although we have no data on this specific point for the boys (for discussion of this point, see Coyle et al. 1979; Gregor et al. 1979; Thorstensson et al. 1976). Other authors have also observed that even when standardized for body or muscle size, the anaerobic power output of children is low when compared to adult values (see, e.g., Bar-Or in this volume).

The maximal mean power output for a complete revolution (P_{CRmax}) is considerably less than the PP_{max} due to the presence of a recovery phase for each leg (Fig. 2, Table 2). In our subjects, P_{CRmax} at optimal velocity was 395 ± 89 W. This contrasts with the calculation of maximal mechanical power output available from aerobic sources, which was 185 ± 38 W (Table 2).

In conclusion, the present data clearly demonstrates the importance of controlling or assessing velocity in measurements of short-term power output and indicates that the optimal pedaling rate for maximal power output in short-term anaerobic exercise is approximately 110 rpm, which is in agreement with previous findings.

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APPENDIX II

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Illinois.

EFFECTS OF SUPPLEMENTAL PHYSICAL ACTIVITY ON BODY COMPOSITION
AEROBIC AND ANAEROBIC POWER IN 13-YEAR OLD BODY.

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INTRODUCTION

It is an interesting question as to how much of an effect physical education programmes might reasonably be expected to have upon the short term physical fitness of children (Kemper et al 1978; Duquet and Gregoire 1978; Rutenfranz and Singer 1980).

In an attempt to provide some insight into this question we have studied the effect of supplementing the normal school physical education programme with additional periods specifically devoted to fitness training.

METHODS

Twenty eight 13 year old boys attending a secondary school in North London were studied over an eight week period in the Christmas term (September - December). The school authorities, parents, and boys, gave their informed consent to participation in the study. The children were divided into a control and a training group. Both groups followed the normal curriculum nominally consisting of 150 minutes of sports activities per week, but the training group had in addition two further periods, total time 2.5 hours, which were specifically devoted to fitness training. The content of the supplementary periods was designed to include a balanced mixture of short term power and endurance training as well as some weight training.

Maximum oxygen uptake was directly determined using standard techniques as previously described by our laboratory (see Sargeant, Dolan, and Thorne 1983).

Short-term (anaerobic) power was assessed using the isokinetic cycle ergometer developed in our laboratory (Sargeant, Hoinville, and Young 1981). The data reported here is maximal peak power (PPmax) that is, the power generated at the instant of maximal peak force, measured at a pedalling speed of 108 revs/min. PPmax is expressed as the sum of both legs. For further discussion of these techniques readers are referred to Sargeant et al 1981.

Lean body mass was estimated from the sum of four skinfolds (Durnin and Rahaman 1967). Leg muscle (plus bone) volumes were estimated by anthropometry (Sargeant and Davies 1977).

RESULTS

At the start of the study there were no significant differences in age, height, weight, lean body mass or leg(muscle plus bone) volume, although it might be noted that the training group were slightly taller and heavier than the control group (Table 1). Following the 8 week period of study both groups were taller and heavier than at the start ($p > 0.001$). The control group showed an increase of lean body mass which was proportionate (2.5% : $p > 0.01$) to the overall increase in body size, while in the training group there was an increase in lean

body mass of nearly twice that level (+4.8% : $p > 0.001$). The increases in lean body mass were associated with larger leg muscle volumes . For example, in the control group upper leg muscle (plus bone) volume increased, although not significantly, by 3%, while the training group showed a much more dramatic and highly significant increase of 9.7% ($p > 0.01$). It is only in this last characteristic that there is a significant difference between the groups at the end of the study period ($p > 0.05$).

Table 1 near here

Maximum oxygen uptake was not significantly different between the groups before training , a slightly higher absolute value for the training group reflecting the tendency for this group to be somewhat larger and heavier than the controls: this was largely accounted for when standardised for body weight (Table 2). During the study period the VO_2 max of the training group increased in direct proportion to body size , hence when expressed standardised for body weight there was no change from the initial value of 53 ml/kg/min. In marked contrast the control group showed a significant decrease of 6% in absolute terms and this difference was exacerbated when account was taken of the growth related increase in body weight so that there was a 9% reduction in VO_2 max when expressed in ml/kg/min ($p > 0.01$). As a consequence of the deterioration in the control group there was a significant difference ($p > 0.05$) between the groups at the end of the study in absolute values of VO_2 max and when

TABLE 1. Mean(SD) data for age, height, weight, lean body mass(LBM), leg muscle plus bone volume(LV), and upper leg muscle plus bone volume(ULV).

Significance: Between groups, $p > 0.05$

: Within groups, * $p > 0.05$; ** $p > 0.01$; *** $p > 0.001$

PHYSICAL CHARACTERISTICS

	Age yrs	Height cm	Weight kg	LBM kg	LV l	ULV l	
Control (n = 13)	Before	13.6(0.27)	158.3(8.5)	48.9(6.1)	40.6(5.1)	5.11(0.87)	3.38(0.66)
	After	13.8(0.27)	159.8(8.7)	50.0(6.2)	41.6(5.3)	5.24(0.82)	3.48(0.58)
	% Change	+1.5% ***	+0.9% ***	+2.2% **	+2.5% **	+2.5	+3%
Training (n = 15)	Before	13.7(0.28)	164.8(10.6)	52.5(9.7)	43.7(7.1)	5.78(1.07)	3.82(0.75)
	After	13.8(0.28)	166.6(11)	54.1(9.8)	45.8(7.5)	6.24(1.23)	4.19(0.85)
	% Change	+1.5% ***	+1.1% ***	+3% ***	+4.8% ***	+8% ***	+9.7% **

standardised for body weight.

Table 2 near here

Maximum peak power output (PPmax) was slightly, though not significantly, greater in the training group compared to the control this difference again reflecting the slight size difference between the groups. Hence when expressed in terms of body weight PPmax is almost identical for the two groups (30.9 and 31.2 watts/kg. for the control and training groups respectively : Table 3). During the study the control group increased their PPmax from 1522 to 1578 watts ($p > 0.05$), but since this was associated with a concomitant increase in body size there was no significant change in the PPmax expressed as watts/kg body weight. The training group showed a larger increase from 1622 to 1760 watts (8.5% : $p > 0.01$). Thus when expressed in terms of body weight there was a 5% increase in PPmax from 31.2 to 32.6 watts/kg although this does not reach conventional levels of significance.

Table 3 near here

DISCUSSION

The normal curriculum followed by the children in this study consisted of three hours of sports related activities per week, which included only a relatively small proportion of time devoted

TABLE 2. Mean(SD) of directly measured maximal oxygen uptake in absolute terms (l/min) and standardised for body weight (ml/kg/min)

MAXIMUM OXYGEN UPTAKE				
	Control group (n=8)		Training group (n=8)	
	l/min	ml/kg/min	l/min	ml/kg/min
Before	2.41 (0.39)	50 (6)	2.67 (0.33)	53 (8)
After	2.27 (0.35)	45 (3)	2.73 (0.29)	53 (8)
% Change	-6% *	-9% **	+2%	0%

TABLE 3. Mean(SD) data for maximal peak (anaerobic) power given in absolute terms (watts) and standardised for body weight (watts/kg). Significance as for table 1.

MAXIMAL PEAK POWER (2-legs)				
	Control group (n=11)		Training group (n=15)	
	watts	watts/kg	watts	watts/kg
Before	1522(292)	30.9(3.6)	1622(298)	31.2(5.2)
After	1578(342)	31.3(4.8)	1760(312)	32.6(3.2)
% Change	+3.7% *	+1.2%	+8.5% **	+5%

specifically to elements of fitness training. In contrast the supplementary periods were devoted entirely to fitness training organised on a group basis and using standard gymnasium equipment and activities : these included, interval sprints, 4 minute pursuit races, weight training, etc.. Clearly some care is needed before the results of the present study can be extrapolated to other situations.

It should also be noted that the study was carried out during the Christmas term between September and December, immediately following the long summer vacation. It seems reasonable to suppose that in boys of this age their habitual physical activity level would be much higher during the summer vacation than on return to school when most of the time will be spent in sedentary classroom based activities. At the same time there will be an additional factor, the effect of shorter daylight hours progressively curtailing outdoor activities over this period.

In the light of these observations it is perhaps not surprising that the normal p.e. curriculum of only 3 hours per week fails to compensate for the enforced overall reduction in physical activity, and that as a consequence the control group shows a significant deterioration in maximum oxygen uptake from 50 to 45 ml/kg/min (-9% ; $p > 0.05$). Even with the two period supplement the training group only manage to maintain maximum oxygen uptake at the initial value of 53 ml/kg/min but do not improve it.

In contrast short-term (anaerobic) power output, presented here as maximal peak power (PPmax) increases significantly in both groups. In the control group this increase is by 3.7% ($p > 0.05$) which can be seen as an accurate reflection and consequence of physical growth over the study period. Total body weight increasing by 2.2% and upper leg muscle mass increasing by 3% (Table 1). The training group also show an increase in PPmax expressed in absolute terms, from 1622 to 1760 watts. Although this change of 8.5% is over twice that of the control group it is associated with a similarly large increase in upper leg muscle volume (+9.7% : $p > 0.01$).

In conclusion it would seem that either directly or indirectly it is possible to at least compensate for the enforced reduction in physical activity on returning to school after the summer vacation when considered in terms of maximum oxygen uptake, by supplementing the normal school p.e. programme. It also appears that changes in maximal short-term power are closely related to the size of the active muscle in these groups and that it is possible to increase this by means of a training programme occupying relatively little time.

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APPENDIX III

Reprint of: Sargeant, A.J., Dolan, P. and Young, A.
(1984) Optimal velocity for maximal short-term
(anaerobic) power output in cycling. Int. J. Sports
Med. 5:124-125 Suppl.

Optimal Velocity for Maximal Short-term (Anaerobic) Power Output in Cycling

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Human Physiology Laboratory, Polytechnic of North London, and
* Nuffield Department of Orthopaedic Surgery and Clinical Medicine, Oxford University**Introduction**

In recent years there has been an upsurge of interest in the measurement of short-term anaerobic power, although in fact many investigators have, over the years, made measurements of a brief maximal sprint effort performed pedalling a cycle ergometer or running up a flight of stairs (see e.g. Asmussen and Boje 1945; Margaria, Aghemo, and Rovelli 1966). However, as Wilkie (1960) has pointed out it is crucial when measuring power output from muscle that the external load is closely matched to the capacity of the active muscles so that they operate at the optimum velocity for maximal power output as defined by the force velocity relationship. Clearly this is a difficult condition to fulfill or assess using a technique such as stair climbing or an accelerating sprint effort performed on a cycle ergometer, since velocity and hence power output will be changing throughout the measurement period, and these changes will be independent of any other factors which may be the subject of investigation. In order to overcome this problem we described in an earlier paper a technique utilising an isokinetic cycle ergometer which allowed measurements of muscle force and power output to be made under constant velocity conditions (Sargeant, Hoinville, and Young, 1981). Using this technique we were able to identify the optimal velocity for true maximal short-term power output measurement in this form of cycling exercise.

Methods

To measure leg force and power output a cycle ergometer was modified as previously described (Sargeant, Hoinville, and Young 1981). Briefly this involved the addition of a 3 hp electric motor driving the cranks through a variable speed gearbox which allowed the pedal crank speed to be set in the range 23 - 180 revs/min. After the required speed was set subjects were asked to make a maximal 20 sec effort in which they attempted to speed up the ergometer, but due to the characteristics of the motor-gear system this was not possible.

The forces exerted on the cranks, and by integration the power generated, were continuously monitored during each 20 sec maximal effort by means of strain gauges bonded to the cranks (Sargeant and Davies (1977). With this technique we are able to calculate peak force in each revolution. Maximal peak force could thus be calculated for each velocity. The maximal peak force usually occurring within the first few seconds of exercise before the onset of fatigue.

Results and Discussion

We have now studied 31 adults and 24 children (13 year old boys). Subjects were usually measured at 4 or more velocities in the range 23 to 171 crank revolutions/min. All subjects showed a linear relationship between maximal peak force and crank velocity ($r > 0.99$) as previously reported (Sargeant et al 1981). Power calculated from the linear force-velocity regression was a parabolic function of velocity. The apex of the parabola occurring at the optimal velocity for true maximal power generation.

The mean (SE) optimal velocity for our adult subjects was 111 (2.34) and for the children 112 (3) revs/min. There was no significant difference between the two groups. Thus for population studies using normal cycle ergometers where it is not possible to hold velocity constant these data indicate that the loading should be chosen in order to achieve a pedalling speed of approximately 111 revs/min if a true measurement of maximal short-term power is required.

In a smaller sub-group we have examined the effect of muscle fibre type on the optimal velocity. The mean optimal velocity for the whole of this sub group was not significantly different to the rest of the group. If however the sub-group is divided into two further groups, those with < 50% cross-sectional fibre area of type II fibres ($n = 10$), and those with > 50% ($n = 6$), then there is a significant difference between the groups ($p > 0.02$). The optimal velocity for the group with predominantly slow twitch fibres was 104 (3.5) revs/min compared with 119 (4) revs/min for the group with a predominance of fast twitch fibres. The finding for cycling is analogous to those findings of previous investigators studying leg extension force measured on the Cybex dynamometer (Coyle, Costill, and Lesmes 1979; Gregor et al 1979; and Thortensson, Grimby, and Karlsson 1976).

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Int. J. Sports Med. 5 (1984) Supplement

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APPENDIX IV

Reprint of: Dolan, P. and Sargeant, A.J. (1984)
Maximal short-term (anaerobic) power output
following submaximal exercise. Int.J. Sports Med.
5:133-134 Suppl.

APPENDIX IV

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Maximal short-term (anaerobic) power output
following submaximal exercise. Int.J. Sports Med.
5:133-134 Suppl.

Maximal Short-term (Anaerobic) Power Output Following Submaximal Exercise

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Introduction

The maximal capacity of human muscle to generate power during short-term dynamic exercise of a few seconds duration is dependent upon the rate of splitting of the high energy phosphate compounds ATP and phosphorylcreatine (PC) since these represent the most immediate source of energy available to the contracting muscle. During maximal exercise however the rate at which these high energy phosphates may be broken down is faster than their rate of resynthesis and maximum power output can therefore only be sustained for a few seconds before the high energy phosphate store becomes exhausted or depleted to a critically low level resulting in a subsequent fall in power output. Presumably then the maximum power attainable will be related at any one time to the concentration of unsplit high energy phosphate in the active muscle fibres and any reduction in this might therefore be expected to result in a corresponding decrement in maximum short-term power output. It was the aim of the present study to investigate this by attempting to manipulate the level of unsplit high energy phosphate in the exercising muscle by having subjects perform submaximal exercise of differing duration and intensity prior to measuring their maximum short-term power output.

Methods

The subjects studied were two healthy adult males whose physical characteristics are shown in Table 1.

Table 1 Physical characteristics for the 2 subjects studied

Subject	Age (yrs)	Weight (kg)	Height (cm)	LBM (kg)	VO _{2max} (l/min)
PJ	26	66.7	162.8	50.65	3.20
SM	24	88.7	177.4	72.54	4.56

Maximal short-term power output was assessed during the performance of a short (20 second) dynamic exercise which was carried out at a constant velocity of 110 revolutions/min on an isokinetic cycle ergometer (Sargeant, Hoinville and Young, 1981). The cranks of this ergometer were instrumented with strain gauges thereby enabling the forces exerted on the pedals to be measured throughout the exercise period. During each revolution peak power was determined and the maximum peak power was calculated by taking the mean of the three highest consecutive readings to occur during the 20 second exercise.

Each subject performed a series of these 20 second runs

on the isokinetic ergometer starting either from rest or following prior exercise of varying duration (30 sec – 6 min) at a load which represented ~ 95% of their previously determined maximum oxygen uptake (VO_{2max}). Actual determination of oxygen uptake was carried out during the final minute of prior exercise, where this was of 3 or 6 min duration, using a continuous open-circuit technique for gas analysis. The forces exerted on the cranks during this time were also recorded to enable the average power output during prior exercise to be determined. By exerting the same average force during the shorter prior exercise runs subjects were therefore able to exercise at the same relative work load.

In a further part of the investigation we went on to examine in one subject the effect upon maximum short-term power output of varying the intensity of prior exercise. In this case the subject performed 6 min prior exercise at work loads ranging from 40–100% VO_{2max} before short-term power output was measured during the 20 second maximal test.

Results and Discussion

The effect upon maximal short-term power output of increasing the duration of prior exercise is shown in Fig. 1 below. Results are expressed as a percentage of the control

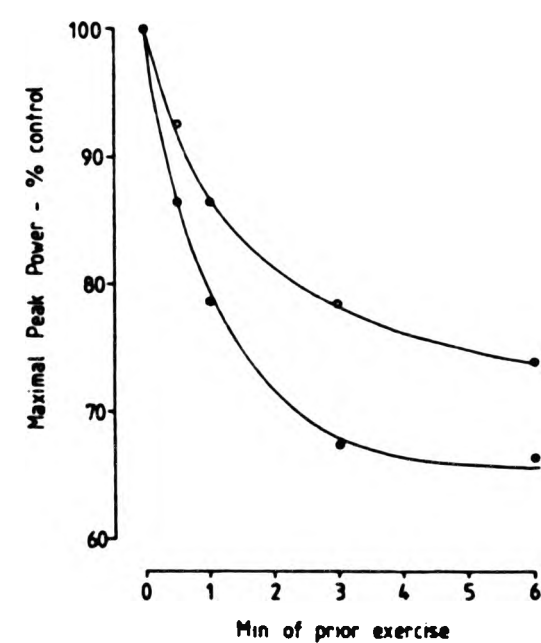


Fig. 1 Maximal peak power expressed as a percentage of the control value in 2 subjects following different durations of heavy (~ 95% VO_{2max}) exercise.

value measured from rest. After only 30 sec prior exercise at a work load corresponding to $\sim 95\% \text{VO}_{2\text{max}}$ a marked decrement in power output of the order of 10% was observed in both subjects. This decrement increased substantially with increasing duration of prior exercise up to 3 minutes, the additional loss in power output being slight when the period of prior exercise was extended to 6 min. These results would seem to indicate that during high levels of submaximal exercise there is a progressively greater depletion of high energy phosphate in the active muscle as exercise continues up to 6 min duration as indicated by the increasing decrement in power output. However, since the additional loss of power incurred by extending the prior exercise period from 3 min to 6 min is only small this would seem to suggest that during this time a steady state condition is attained in which the rate of high energy phosphate resynthesis is equivalent to the rate of breakdown. There is therefore no further net fall in high energy phosphate concentration and hence in short-term power output when the period of prior exercise is extended beyond this time. These findings may be compared with the results of Margaria, di Prampero, Aghemo, Derevenco and Mariani who found that the decrease in maximal anaerobic power following submaximal exercise was unaffected by increasing the duration of prior exercise beyond 3 min.

In Fig. 2 the effect of varying the intensity of prior exercise upon subsequent short-term power output is shown. Short-term power output is expressed as the percentage change in maximal peak power from the control value ($\Delta\% \text{PP}_{\text{max}}$) and this is plotted against the prior exercise intensity ($\% \text{VO}_{2\text{max}}$). Where the previous work load was below 62% of the subject's $\text{VO}_{2\text{max}}$ a substantial increase in maximal power output of 12% was observed during the subsequent 20 second test. This improvement may be due at least in part to the increase in muscle temperature since passive warm-up has previously been shown to produce an improvement in maximal short-term power output which would be of the order of 6% for each $^{\circ}\text{C}$ rise in muscle temperature at a crank velocity of 110 rev/min (Sargeant, 1983). Following higher levels of prior exercise an inverse relationship was observed between prior exercise intensity and subsequent short-term power output resulting in a decrement in power output of 21% following the heaviest work load (100% $\text{VO}_{2\text{max}}$). The concentration of high

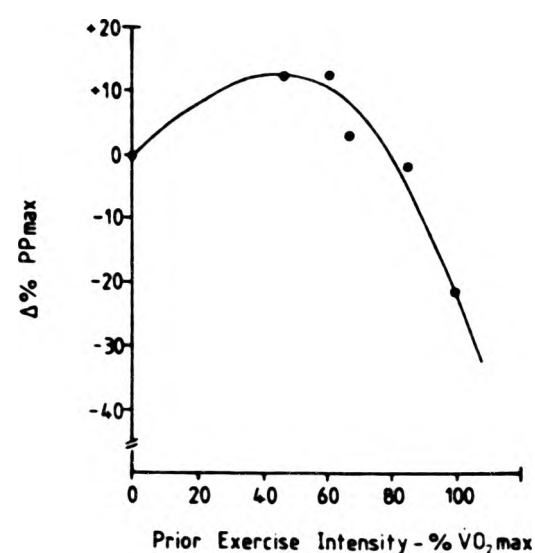


Fig. 2 Changes in maximal peak power expressed as a percentage of the control value ($\Delta\% \text{PP}_{\text{max}}$) in 1 subject (SM) following different levels of submaximal exercise.

energy phosphate and more particularly of PC in muscle biopsy samples taken after submaximal exercise has also been shown to decrease with increasing work loads above 60% $\text{VO}_{2\text{max}}$ (Karlsson, Diamant and Saltin, 1971; Knuttgen and Saltin, 1972). Since the power developed during the first few seconds of maximal exercise is presumably related to the amount of ATP and PC within the active muscle fibres the present results may therefore be interpreted to reflect the prevailing level of high energy phosphate following different durations and intensities of submaximal exercise.

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APPENDIX V

Reprint of: Dolan, P. and Sargeant, A.J. (1983)
Effect of prior exercise on maximal short-term power
output of human muscle. J. Physiol. 343:107-108P.

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Journal of Physiology, 343, 107-108P]

Effect of prior exercise on maximal short-term power output of human muscle

BY PATRICIA DOLAN and A. J. SARGEANT, *Human Physiology Laboratory, Polytechnic of North London, Kentish Town, London NW5 3LB*

Maximal power output of human muscle during short-term dynamic exercise lasting for a few seconds is dependent upon an immediate supply of high energy phosphate (ATP and PC) within the active muscle fibres. We have examined the effect of manipulating the level of this by having subjects exercise submaximally prior to measurement of maximal short-term power output. Five healthy subjects were studied and power output was measured during a 20 sec maximal effort performed on an isokinetic cycle ergometer at a constant velocity of 110 crank rev. min⁻¹ (Sargeant, Hoinville & Young, 1981). Results are expressed as the percent change in maximal peak power ($\Delta\% PP_{max}$) from control values measured from rest (Fig. 1). Following moderate levels of prior exercise ($< 60\% \dot{V}_{O_{2max}}$) improvements in power output of 8-15% were observed in four of the five subjects studied (cf. Margaria, di Prampero, Aghemo, Derevenco & Mariani, 1971). At higher work loads there was an inverse relationship between prior exercise intensity and subsequent short-term power output resulting in a 20-40% loss in power following the heaviest work loads.

We subsequently examined the effect of interposing a variable recovery interval (5 sec to 6 min) between prior exercise at $\sim 87\% \dot{V}_{O_{2max}}$ and the short-term power output measurement (Fig. 1*b*). Short-term power output returned to control values

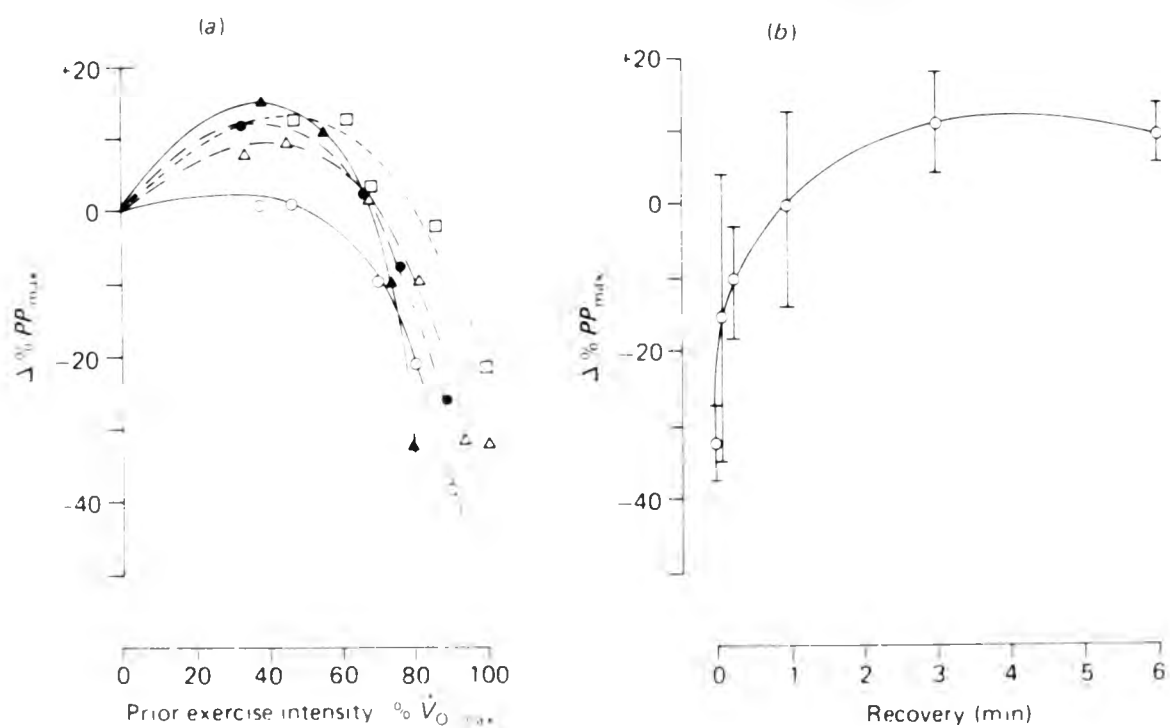


Fig. 1. (a) Changes in short-term power output after different levels of prior exercise (individual values for 5 subjects). (b) Time course of recovery in short-term power output following 6 min dynamic exercise at $\sim 87\% \dot{V}_{O_{2max}}$ (Mean \pm s.d., $n = 4$)

within ~ 1 min, although a further increase of $9.6 \pm 3.8\%$ (mean \pm s.d.) was observed after 6 min of recovery. These observations on functional recovery are comparable with the direct determinations of PC made by Harris, Edwards, Hultman, Nordesjo, Ny Lind & Sahlin (1976) who observed a similar time course for resynthesis during recovery from exhaustive dynamic exercise.

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APPENDIX VI

Reprint of: Dolan, P., Greig, C. and Sargeant, A.J.
(1985) Effect of active and passive warm-up on
maximal short-term power output of human muscle.
J. Physiol. In Press.

within ~ 1 min, although a further increase of $9.6 \pm 3.8\%$ (mean \pm s.d.) was observed after 6 min of recovery. These observations on functional recovery are comparable with the direct determinations of PC made by Harris, Edwards, Hultman, Nordesjo, Nylinde & Sahlin (1976) who observed a similar time course for resynthesis during recovery from exhaustive dynamic exercise.

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SARGEANT, A. J., HOINVILLE, E. & YOUNG, A. (1981). *J. appl. Physiol.* **51**, 1175-1182.

APPENDIX VI

Reprint of: Dolan, P., Greig, C. and Sargeant, A.J. (1985) Effect of active and passive warm-up on maximal short-term power output of human muscle. *J. Physiol.* In Press.

C. 60

Effect of active and passive warm-up on maximal short-term power output of human muscle

By P. DOLAN, C. GREIG and A. J. SARGEANT. *Human Physiology Laboratory, Polytechnic of North London, Kentish Town, London NW5 3LB*

Changes in muscle temperature have long been proposed to influence human performance during maximal exercise of brief duration, although the evidence to date is somewhat conflicting. In contrast to earlier work (Asmussen & Boje, 1945) some investigators have found that 'warm-up' had very little, or no, beneficial effect on subsequent performance (Karpovich & Hale, 1956; Davies & Young, 1983). Such discrepancies may reflect differences in the type of warm-up employed or in the exercise used to assess its effect. In the present study we have examined the effect of both active and passive warm-up on maximal short-term power output of human muscle during cycling. The subjects studied were four healthy male adults. Short-term power output was measured at a constant pedalling rate of 110 crank rev. min⁻¹ on an isokinetic cycle ergometer (Sargeant, Hoinville & Young, 1981) from rest, and following passive and active warm-up. Passive warm-up was effected by 45 min immersion of the legs in water baths at temperatures between 37 and 44 °C. Active warm-up consisted of pedalling an electrically braked cycle ergometer at work loads which ranged from 17–92% $V_{O_{2,max}}$. Following warm-up there was a rest period of 6 min before the determination of maximal power output (Dolan & Sargeant, 1983). In all experiments muscle temperature was measured in the quadriceps immediately prior to the maximal test using a needle thermocouple. Measurements were made at three depths (4, 3 and 2 cm) and the mean value determined.

Following both active and passive warm-up all subjects showed a significant increase in maximal peak power ($P < 0.01$). Expressing maximal peak power (PP_{max}) as a percentage of the control value the relationships to muscle temperature (T_m) for all subjects combined were described by the following regression equations:

$$\text{active warm-up: } PP_{max}(\%) = 4.46 + 2.70 T_m \quad (n = 53; r = 0.68; P < 0.001).$$

$$\text{passive warm-up: } PP_{max}(\%) = 19.53 + 2.27 T_m \quad (n = 54; r = 0.61; P < 0.001).$$

These regression equations indicate that over the range of muscle temperature studied (34–40 °C) there was a 2.7 and 2.3% increase in maximal power per °C rise following active and passive warm-up respectively. The difference between the two modes of warm-up was not, however, significant, and rather small, suggesting that the beneficial effect of prior exercise on maximal short-term power output is almost entirely attributable to an increase in muscle temperature (see Dolan & Sargeant, 1983).

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APPENDIX VII

Reprint of: Dolan, P., Greig, C. and Sargeant, A.J.
(1985) Muscular weakness following prolonged
eccentric and concentric exercise in man. J. Physiol.
In Press

P. 37

Muscular weakness following prolonged eccentric and concentric exercise in man

BY P. DOLAN, C. GREIG and A. J. SARGEANT. *Human Physiology Laboratory, Polytechnic of North London, Kentish Town, London NW5 3LB*

Earlier studies have indicated that, following prolonged exercise, there is long-lasting muscular weakness (see, for example, Edwards, Hill, Jones & Merton, 1977; Davies & White, 1982). In general, it appears that exercise involving a large eccentric component has a more marked effect than exercise involving concentric muscle contractions. However, previous studies have concentrated on measurement of muscle function in terms of voluntary or electrically stimulated isometric contractions. We have sought to extend this work by making measurements of maximum short-term power output (PP_{\max}) under isokinetic conditions at four velocities (Sargeant, Hoinville & Young, 1981), and, in addition, measurements of maximal voluntary isometric contraction (MVC) and the force frequency response of the quadriceps muscle characterized by the $T_{20/50}$ ratio (see Edwards *et al.* 1977).

Uphill walking involving predominantly concentric contractions was performed for 1 h on a motor-driven treadmill at a level eliciting $79 \pm 2\%$ (s.d.) of the subjects maximum oxygen intake. Only at 24 h post exercise were there significant reductions, albeit rather small ones of $5 \pm 2\%$ in MVC ($P < 0.05$), and $5 \pm 2\%$ in peak power (PP_{\max}) measured at a constant velocity of 110 rev min^{-1} . $T_{20/50}$ ratio was significantly reduced at +7.5 h by $19 \pm 3\%$ ($P < 0.01$) and remained so at 24 h (by $10 \pm 5\%$; $P < 0.05$) but had recovered by 48 h.

Prolonged eccentric exercise was performed by the subjects walking downhill at the maximum negative gradient possible on our treadmill (-25%) at a speed of 6.44 km h^{-1} . None of our four healthy, but untrained, subjects were able to complete 1 h of exercise at this level due to extreme muscle weakness leading to collapse. Mean exercise time was 35 min. Although the metabolic cost rose progressively throughout the exercise period it still only approached 70% of $V_{O_2, \max}$ by the end of exercise. Despite the significantly lower metabolic cost of the eccentric exercise there was a profound and sustained loss of muscle function which persisted for 96 h post exercise. MVC fell progressively to $55 \pm 12\%$ of pre-exercise values at 24 h ($P < 0.01$) and had still only recovered to $70 \pm 18\%$ by 72 h ($P < 0.05$). There were similar dramatic falls in PP_{\max} . Measured at 110 rev min^{-1} , PP_{\max} fell to $77 \pm 10\%$ at 24 h and it was still reduced after 96 h ($92 \pm 4\%$; $P < 0.05$). $T_{20/50}$ also fell progressively to $45 \pm 9\%$ of pre-exercise levels after 11 h and remained significantly lower until +96 h. These results demonstrate clearly the considerable loss of dynamic function following prolonged eccentric exercise. It has been suggested that the relatively higher tensions developed within the muscle during eccentric exercise lead to progressive damage at the level of Z band (Newham, McPhail, Mills & Edwards, 1983) and certainly the loss of dynamic function shown by our subjects seems to follow a similar time course.

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