AN INVESTIGATION OF DYNAMIC HUMAN MUSCLE FUNCTION USING A VARIABLE INERTIAL LOADING SYSTEM

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ABSTRACT

This thesis has developed and utilised an inertial loading system to study human skeletal muscle power output. Specifically, the apparatus has been used to study the effects of different modes of exercise, muscle myosin isoform composition and the effects of ageing on the ability of the lower limb muscles to generate explosive power.

A variable inertial loading system was designed and constructed which allowed for the sensitive detection of the rotational properties of a flywheel from which the contractile characteristics of muscle could be inferred. When housed in the Nottingham Power Rig (NPR) the peak power generated by young non-trained male subjects from a single lower limb thrust ranged from 608 – 965 Watts and was found to occur at inertial loads ranging from 0.09 – 0.22 kgm^2. To investigate the low power outputs observed at the low inertial loads, where the contraction time was short, a pre release mechanism was incorporated into the flywheel assembly. Significant increases in power output of ~ 17% were achieved at the lowest inertial load ($P = 0.02$), if a prior build up of isometric torque was allowed prior to movement. This suggested that at the low inertial loads, without the pre release, insufficient time was allowed for the muscle to generate its maximum power output.

The flywheel system was incorporated into a cycle ergometer to allow power – velocity characteristics to be examined during inertial sprint cycling. Peak power obtained in young subjects ($n = 9$) was significantly higher in the cycle exercise when compared with the NPR (1620 vs. 937 Watts). In contrast to the NPR where a parabolic relationship between power and inertial load was observed, during sprint cycling power plateaued above a 'critical' load. It was concluded that the repetitive acceleration of inertial loads, above this critical threshold, will always allow the expression of peak power during cycling as ultimately a velocity will be achieved which corresponds to that required for peak power generation.

An analysis of the myosin heavy chain (MHC) isoform composition of the vastus lateralis muscle was performed in young and elderly male subjects ($n = 14$, mean age 29.4 and 73.8). The percentage MHC-II isoform composition was significantly lower in the older subjects as was the velocity at which peak power occurred (V_{opt}). Overall the V_{opt} during sprint cycling was found to be related to the percentage MHC-II composition of the vastus lateralis ($R = 0.82$, $P<0.001$).

Finally, muscle power was examined in Elite level master Olympic weightlifters ($n = 54$, aged 40 – 87 years) and aged matched controls. On average the weightlifters generated ~ 32% more peak power than their aged matched counterparts and required significantly higher inertial loads to express their peak power output. In spite of 'load optimisation', power declined at twice the rate of strength. The levels of power suggest a 20 year advantage for the weightlifters.

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LIST OF ABBREVIATIONS

NPR – Nottingham Power Rig

MNPR – Modified Nottingham Power Rig

n1 – number of flywheel turns during fall of known mass

n2 – number of flywheel turns after known mass reaches floor

PP_k – Peak power

Tm_{ppk} – Time to peak power

Tq_{ppk} – Torque generated at peak power

V_{ppk} – Velocity at peak power

Tq_{pk} – Peak torque

MP – Mean power

V_{opt} – Optimal velocity at peak power during cycling

T_{opt} Torque generated at peak power during cycling

LLV – Lean limb volume (muscle + bone)

MPP – maximum peak power

AP_{mpp} – Average power at the load at which peak power occurred

TO_{mpp} – Torque generated at maximum peak power

V_{mpp} – Velocity generated at maximal peak power

T_{mpp} – Time to maximal peak power

INTRODUCTION

Muscle power is the rate of doing work, or the product of force of contraction and speed of movement. The ability of the muscular system to generate power ultimately determines the ability to perform physical tasks, whether this is for athletic performance, or in the case of the frail elderly person in rising from a chair. Over the years a number of different methods have been developed for measuring muscle power in man. These have ranged from simple tests such as the Sargent jump (1921) to the Margaria test (1966) both of which utilise the body as a method of loading the muscles. The advent of isokinetic devices in the late 1960's has seen their extensive use in studies of human muscle function. Whilst these tools, which allow joints to rotate at constant angular velocities, are useful, they do not reflect the way muscles behave in the environment, which is against inertial loads and against gravity.

With a view to having a method of testing muscle power which reflected the way that muscles behave and in a manner which was practical and safe for elderly people, Bassey and Short (1990) developed an inertial system, the Nottingham Power Rig (NPR). This comprised of a fixed inertial flywheel system which allowed the average power during a single lower limb thrust lasting less than 1 second to be calculated. Studies of muscle power using this fixed inertial loading device have shown lower limb power to be closely associated with functional ability in elderly people (Bassey et al. 1992, Skelton et al. 1994). The NPR had the advantage of practicality in that a simple pin and optoswitch system could be used to time the rotation of the flywheel. However, as will be discussed later in this thesis, this method does not give an accurate measure of muscle power. This method had two possible limitations which were; 1) the fixed inertial load, which may not represent the optimum for all subjects, and 2) the timing of the contraction phase, which relied on assumptions of constant acceleration during the exertion period. More accurate measures of muscle power have been obtained by others using inertial loading, where strain gauge systems have been placed between the limb and the inertial load and where goniometers have been placed on the limb in order to measure velocity of joint rotation (Aagaard et al. 1994). The work presented in this thesis focuses on the design of a variable inertial testing system which is both practical and capable of making valid, objective measures of muscle function in man without the need for other force transducers or goniometers. This system was based on the accurate timing of the contraction phase via a high resolution rotary encoder and on allowing the inertial load to be varied so it could be 'optimised' in terms of power output for different individuals.

Single fibre studies have shown that muscle myosin composition determines the ability to generate power (Bottinelli et al. 1996). At the *in vivo* level this has been shown to some extent using isokinetic apparatus (Andersen and Aagaard 1998, Harridge et al. 1995). However the role of muscle composition in terms of the MHC isoform composition with respect to power output under inertial loading conditions is not known. Part of the work undertaken in this thesis uses the inertial system to study the relationship between muscle MHC isoform composition and power output under different exercise conditions in young and old people.

Previous studies of muscle power output in man such as with sprint cycling have reported values of power output up to ~2000 Watts (Davies et al. 1984, Sargeant et al. 1981), considerably greater than that reported for the NPR (Bassey et al. 1990). The design of the inertial system allowed muscle power during these two different tasks to be compared.

When tested using inertial loading, muscle power in older people has been shown to decline at a greater rate than isometric strength (Davies et al. 1983, Skelton et al. 1994). This suggested that the older muscle, as well as being weaker was also slower. However, in these studies a single inertial load (either body weight or a heavy flywheel) was used, therefore it is also possible that the inertial load was too high for weaker people to express their maximal power. By optimising the inertial load, the question as to whether the rate of decline in power output with increasing age is greater than that of isometric strength is re-examined in this thesis.

Much work has also been carried out with regard to possible interventions, to reduce or ameliorate the effects of ageing on the muscular system. It has been reported that resistance training in the elderly shows similar improvements to that in younger individuals (Evans 1995, McCartney et al. 1996, Fiatarone et al. 1994, Skelton et al. 1995, Harridge et al. 1998). However, the effects of long term resistance training in elite weightlifting athletes have not been studied extensively. The study of such athletes is important as it may aid our understanding of the limits to which older muscles can potentially function. The apparatus developed as part of this thesis has been applied to studying such athletes.

PART I

LITERATURE REVIEW

The review of literature aims to provide a background to the work in the study. This is firstly, by outlining the structure of skeletal muscle and the basic mechanisms involved in the contraction of muscle. This includes a brief review of the theories of muscle contraction and the mechanical properties of muscle. Details are given regarding the classification of muscle fibres and myosin heavy chain isoform composition, and their relevance with respect to power output. An overview of the different methods of testing muscle output characteristics along with the relative merits of each is given. Finally to explain the specific use in this thesis of subjects who might be expected to differ in their ability to generate power, a section is included which examines both the effects of age on the ability to generate both power and strength, and also the effects of resistance training on these abilities.

1) MUSCLE PROPERTIES

Movement occurs as a result of the muscles developing force and pulling on the skeletal framework via the tendons. The force is generated by the interaction of the contractile proteins, actin and myosin more specifically the interaction of the myosin heavy chain heads with active sites on the actin; these proteins form the molecular motors of skeletal muscle. The following section examines the factors involved in the generation of force and power by the muscular system.

i) MUSCLE STRUCTURE

Skeletal muscle consists of many bundles of fibres, each fibre being approximately 50μm in diameter. A muscle fibre itself is composed of many myofibrils, within the myofibril; myosin and actin filaments form the basis of the fundamental contractile unit of skeletal muscle, which is the sarcomere. Actin, or the thin filament, consists of a double helix arrangement. Myosin or the thick filament consists of a large asymmetrical molecule. This consists of two heads, which are about 165 Å (1.65×10^{-8} m) long and 65 Å (6.5×10^{-9} m) wide at one end which continues into a tail which is about 1500 Å (1.5×10^{-7} m) long and 20 Å (2×10^{-9} m) wide. The myosin molecule consists of two heavy chains, which weigh about 200 kDa, these form the helical tail structure and the two heads, which form the cross bridges towards the actin filament and four light chains, which range from 15 kDa to 25 kDa in weight. The total length of the myosin molecule is approximately 150 nm whereas the cross bridges are approximately 13 nm in length and lie in six rows along the myosin filament. Two of the light chains are classified as regulatory and two as alkali, one of each type is attached to the myosin head. The myosin molecule can also be determined in terms of its meromyosin structure, the heavy chain being split into light meromyosin (LMM) and heavy meromyosin (HMM), with the HMM being further subdivided into a S1 region which consists of the head region and the S2 region, which consists of a rod like section and has no ATPase activity (for review see Schiaffino and Reggiani 1996).

ii) MECHANISM OF MUSCLE CONTRACTION

For muscle contraction to take place the myosin cross bridges must make contact with the actin filament. For this to occur the tropomyosin, which is bound to the actin filament, must move in order to expose the active sites on the actin. On activation calcium is released and binds to the subunit of troponin, TN-C, this causes a conformational change in the tropomyosin, X-ray diffraction studies (Rayment et al. 1993) have suggested that during muscle activation tropomyosin moves from it's lateral position on the actin filament by about 15 Å (1.5 x 10^{-9} m) towards the centre of the actin helical groove this has the effect of uncovering the active sites on the actin. What follows next is termed the cross bridge cycle.

The myosin head, which in its resting state has stored energy by the splitting of ATP into ADP and P_i , undergoes a conformational change, which can be termed the repriming of the power stroke. The myosin head then attaches itself to an exposed site on the actin filament. This actomyosin connection triggers the sequential release of P_i and ADP from the myosin head. On releasing P_i the myosin undergoes a conformational change which is termed the power stroke, here the converter region is thought to act as a hinge, and the result is a movement of the actin past the myosin by approximately 11 ± 2.4 nm (Finer et al. 1994). Immediately following the movement ADP is released. The release of ADP is thought to be strain related, if the filaments are under load the release of ADP is slower hence aiding the release of ADP once the movement is over. Once ADP is released ATP rapidly replaces it causing the disassociation of myosin from actin, in order to repeat the cycle. The total time for this complete cycle is of the order of 50 ms, with the power stroke conformational change taking approximately 2 ms. The force generated by the power stroke is being approximately 3.4 ± 1.2 pN (Finer et al. 1994). However, Molloy et al. (1995) using double optical trap techniques to overcome the limitations of the study performed by Finer et al.

(1994), have found smaller values of movement per cross bridge stroke (4 nm) and less force per cross bridge stroke (1.7 pN).

Cross bridge theory

Based on studies using electron microscopy, H. E. Huxley (1954) proposed that muscles shorten by a process of two sets of interdigitating filaments sliding past each other. Later A. F. Huxley (Huxley, 1957) proposed that these filaments slide past each other as a result of the action of independent force generators or cross-bridges – the so called 'cross bridge theory'. He suggested that the thick filaments had side attachments (cross bridges), which contained an elastic element and allowed connection of the thick filament to the thin filament at specialised sites. The 'cross bridges' which were continuously moving whilst unattached due to Brownian motion, were thought to act as independent force generators and were thought to attach spontaneously. The attachments were constrained to occur asymmetrically in order to generate force, which tended to shorten the sarcomere. The rates of attachment and detachment were controlled by constants f and g respectively. These constants were linear functions of the distance from the active site on actin to the equilibrium position (x) of the cross bridge. When the mathematical model was compared with the experimental results of Hill (1938) a good agreement was found in terms of the force – velocity relationship during concentric contraction. During eccentric work the predictive model overestimated the rate of energy liberated as heat, which is supposedly linearly related to the speed of lengthening. This was not found to be the case experimentally (Abbot 1953). However Huxley (1957) suggested that this insufficiency of the predictive model could be overcome by assuming that during eccentric work the cross bridges did not require any energy to disassociate rather they were released by mechanical means. Later in 1971 in light of the experimental evidence of fast and slow force transients from an isometric stepwise length change, Huxley revised the original theory (Huxley and Simmons 1971). The revised theory allowed the cross bridge to exist in different states whilst attached, thereby allowing work to be performed in small steps. To allow this conformational change without relative displacement of the thick and thin filaments, an undamped elastic element is present within each cross bridge. The elastic element was therefore responsible for the fast, force drop transient during the length step and the cross bridge conformational change being responsible for the fast recovery section of the force during recovery.

The original theory of The 'swinging cross bridge' (Huxley 1969), has now been modified in light of spectroscopic and structural observations to allow for the very small movement of the cross bridge head seen, whereby the distal portion (C-terminal), of the cross bridge was suggested to act as a lever arm (Cooke, 1986), this has been termed the 'swinging lever arm hypothesis'. Later Rayment, (1993) also suggested a model determined from the combination of the molecular structure of the actin myosin complex and that obtained by cryo-electron microscopy, which agreed with that suggested by Cooke (1986).

iii) BASIC MECHANICAL PROPERTIES

The study of muscle mechanics enables a quantitative description of the physiological aspects of muscular contraction. The use of mathematical models to help understand and describe these physiological events has made use of the mechanics of muscular contraction. The methods employed are involved even at the level of the single fibre or filament interaction, but are complicated further at the level of the whole muscle or *in situ*. There are many factors to consider when concerned with the mechanics of muscular contraction, but properties of muscle contraction that are of particular importance from a mechanical viewpoint are those of force and velocity and how these factors are effected by the length of the muscle. Investigation of these factors began many years ago. Since the early work of Hill (1938) who proposed a hyperbolic force - velocity relationship for muscle $\mathbf{V = b(F_o\text{-}F)/(F+a)}$ from his empirical work on the heat production and shortening of muscle, many studies have examined the relationship using *in vitro* preparations of both animal and human muscle and by making *in vivo* measurements on both animal and human subjects. The relationship still remains practical today. The interactions of muscle fibre mechanical properties and the relationship to the structural properties of muscle have been further elucidated by single fibre experiments.

Length – tension relationship

Ramsey and Street (1940) were among the first to demonstrate that force developed by a muscle fibre changed as a factor of its length. They found that force was optimal when a fibre was close to its slack length of 2 μ m, and that the force declined on either side of this fibre length.

The work of Gordon, Huxley and Julian (1966), on frog muscle, determined that the force generated by muscle filaments could be altered by the degree of overlap of the filaments and was proportional to the number of active cross bridges. Here when the sarcomere - force relationship is plotted there is a characteristic ascending limb, a plateau region and a descending limb. This relationship between force and length is known as the length tension relationship. Maximum force development in frog muscle is achieved at a sarcomere length of 2.0 – 2.2 μ m, at shorter and longer sarcomere lengths the force drops in a linear fashion. The reasons for the decrease in force as the sarcomere shortens are thought to be a combination of the mechanical interference caused by the thin filament overlap and at shorter lengths, deformation of the thick filament. This idea has been challenged by the observation that Ca^{2+} release from the sarcoplasmic reticulum was impaired at the shorter sarcomere lengths (Rudel and Taylor 1971). When caffeine was administered, increasing Ca^{2+} release, the force generated by the fibre increased, suggesting incomplete activation may also play a key role in the force decrement on the ascending portion of the length – tension curve. At the longer sarcomere lengths (descending limb of the length – tension curve) a decrease in the number of active cross bridges brings about the linear reduction in tension. Keurs et al. (1978) showed non-linearity during this descending limb phase. This observation was due to their method of holding the fibre at a set length and therefore allowing the sarcomeres to vary within the fibre. This effect of non-sarcomere linearity is probably more physiologically representative of the whole muscle *in vivo*.

Force – velocity/power relationship

In order to determine the force velocity relationship using *in vitro* muscle preparations either the force (isotonic) or the velocity (isovelocity) is held at a constant value (independent variable). In order to ascertain the maximum shortening velocity, which relates to the maximum rate of cross bridge cycling, a series of unloaded shortening contractions at different lengths are carried out (quick release method or slack test – Edman et al. 1979). Here the distance moved by the slack fibre and the time taken to initiate tension redevelopment is noted, thus allowing the velocity of shortening to be determined. These experiments are carried out under conditions of full activation.

Most *in vitro* force – velocity characterisations have been carried out on animal preparations. Fenn and Marsh (1935) were the first to carry out experiments relating the force – velocity properties of muscle. Later in 1938, Hill, building on the earlier work of Fenn (1923), whilst determining the heat production of frog muscles under shortening conditions, found that the heat produced was proportional to the amount of muscle shortening. From this observation the classic hyperbolic equation was formulated $\mathbf{V = b(F_o - F)/(F + a)}$, where $\mathbf{V}$ is the velocity of shortening, $\mathbf{F_o}$ is the isometric force at optimal sarcomere length, $\mathbf{F}$ is the instantaneous force and $\mathbf{a}$ and $\mathbf{b}$ are constants with a having units of force and b units of velocity. The relationship of force to velocity can be related to the kinetics of cross bridge interaction and the heterogeneity of myosin heavy chain

(MHC) isoforms. As the speed of contraction increases the probability of cross bridge attachment by the filaments with 'slower' MHC isoforms decreases, less attached cross bridges will result in a lower force generation. Also the time of cross bridge attachment is shorter thereby having less time to exert a positive force. Negative forces at very high speeds of contraction are also possible, if the detachment of cross bridges cannot keep pace with the shortening, thus allowing 'compressive forces' to be developed.

In studies using frog muscle, Edman (1993) suggested that the hyperbolic relationship described by Hill (1938), deviated from the predicted curvature at loads in excess of 80% of maximal isometric force or 1/10th of the maximal shortening velocity (V_0). This persisted even after administration of dantrolene, which reduces Ca^{2+} release from the sarcoplasmic reticulum. He concluded that the deviation seen in the force – velocity relationship as the shortening speed went below 1/10th of V_0 was due to a change in the kinetics of cross bridge function whereby less force than expected from the Hill equation is produced. Gordon et al. (1986) showed differences in mechanical properties between skinned and living frog fibres, the mechanical data from the skinned fibres better fitted the hyperbolic curve suggested by Hill than the intact living fibres. Edman, (1988) also showed that on whole frog muscle fibres a double hyperbolic relationship existed when force – velocity data was fitted. In contrast other studies have shown good agreement with the force – velocity relationship described by Hill. Close (1964) and Close and Hoh (1967) showed that using slow and fast rat muscles and new born kitten muscles respectively, a good general agreement was found with that of Hill (1938), also the curvature of the relationship delineated by (a/P_0) was on average 0.25 suggesting the muscles examined had similar properties to the frog muscles used by Hill.

Studies involving human muscle fibre have generally shown an agreement with those studies carried out on animal preparations. For obvious reasons most human studies have involved whole muscle *in vivo* tests. However, single muscle fibre preparations (Bottinelli et al. 1996) or more recently isolated single actin – myosin interactions have been possible (Finer et al. 1994). The power – velocity relationship follows a parabolic course with the power being zero at both maximum velocity of shortening (V_{max}) and where velocity is zero. It can be shown mathematically that maximal power occurs at approximately 30% of the maximum speed of shortening.

In order to gain further insight into the functional characteristics of human muscle, studies have been carried out which aim to determine the mechanical properties of different muscle fibre types based on their MHC classification. These studies have identified the differences in the force – velocity, and power output of fibres comprising of different MHC isoforms. It has been reported that the power, maximum shortening velocity and specific force was dependant upon the MHC composition of the fibres with the fibres composed of MHC-II possessing the higher mechanical values (Bottinelli et al. 1999). For expansion of this point see Chapter 4.

iv) MUSCLE FIBRE TYPES

Historically fibre type classification has been carried out using either histochemical techniques to identify the ATPase activity of fibres (Padykula and

Herman, 1955), or alternatively, based on the mitochondrial enzyme content of fibres (Ogata and Mori 1964). Within the context of histochemical classification, fibres can be described as type I type IIA or type IIB. In contrast to ATPase histochemistry which identifies fibres on the basis of the susceptibility of this ATPase enzyme to different levels of pH, more recent methods have used sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE) and immunohistochemistry, (Biral et al. 1988, Danieli-Betto et al. 1986) to identify fibres on the basis of their MHC isoform protein composition.

In human muscle 3 different isoforms of MHC have been identified, one slow isoform MHC-I and two fast, MHC-IIA and MHC-IIX. The latter previously termed MHC-IIB (see Ennion et al. 1995). However, fibres may comprise more than one MHC isoform allowing five possible classifications, MHC-I, I-IA, IIA, IIA-IIX and MHC IIX; these isoforms differ in content between muscle fibre and muscles dependant upon the requirements of the muscle. Figure 1 shows the force – velocity and power velocity relationship of a fibre consisting of slow MHC isoforms and a fibre consisting of fast MHC isoforms.

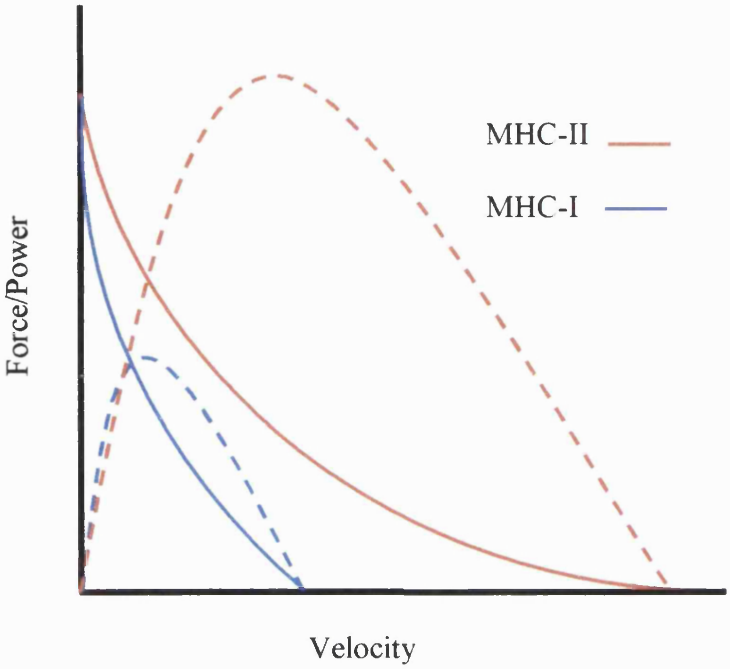


Figure 1.1 An example of typical force (continuous line) and power (dotted line) velocity relationship of skeletal muscle consisting of slow (MHC-I) and fast (MHC-II) fibres.

The myosin light chains also have different isoforms both slow regulatory (MLC2s) and fast regulatory (MLC2f), slow alkali (MLC1sa) and fast alkali (MLC1f, MLC3f) (see Schiaffino and Reggiani 1996 for review). It has been shown in human studies that it is possible for slow light chain isoforms to be associated with fast heavy chain isoforms and visa versa (Larsson and Moss 1993).

Barany, (1967) and Close (1972) showed an association between muscle fibre mechanical properties, in particular shortening velocity and fibre type based on myofibrillar ATPase as determined by histochemistry. Later Burke et al. (1971) and Kugelberg (1973) related the fatigability twitch times and tension development of motor units to fibres within the motor units classified by their mitochondrial enzyme content. This led to the classification based on the mechanical properties of the fibres, slow, relatively low tension, fatigue resistant fibres (SO), fibres which were faster but more readily fatigued and developed moderate tension (FOG), and fibres which were faster still, developed higher tension but were more readily fatigued (FF). The characteristics of twitch and tension development can overlap between motor units of different classification, whereas the properties of fatigability and the presence or absence of the 'sag' phenomenon in short unfused tetani do not overlap.

MHC composition - single fibre studies

Larsson and Moss (1993) were among the first to demonstrate a relationship between the MHC isoform content of skeletal muscle and the mechanical properties. It was shown that the maximum shortening velocity (V_{max}) was highly dependant on the MHC isoform composition. More recently Harridge et al. (1996) demonstrated that muscle fibres classified by the MHC isoform content closely related to the *in vitro* muscle properties. Fibres containing MHC-I isoforms had markedly lower values for maximum speed of shortening, rate of tension development and specific tension than fibres containing MHC–IIA isoforms, whilst fibres containing both MHC–IIA and IIX had higher values for maximum speed of shortening, rate of tension development and specific tension than those containing MHC-IIA. Bottinelli et al. (1997) reported that in skinned single fibres classified according to their MHC isoform composition, the V_{max}, W_{max}, V_{opt}, P_{opt}, V_{opt}/V_{max} ratio, Po/CSA and Vo were all significantly lower in MHC–I fibres compared to MHC-II fibres. Hilber and Galler, (1997) reported that in human fibres the "kinetics of force generating power strokes" was MHC isoform specific.

MHC composition - whole muscle studies

It has been shown in human studies that the twitch properties of the whole muscle reflects the fibre composition of the whole muscle, the triceps surae for example which has a predominantly slow fibre composition, has been recorded as having a time to peak tension of 112 ms ± 11.1 (Sale et al. 1982), this is in good agreement with that of Davies, (1982) who reported values of 111 ± 20 ms for time to peak tension for the triceps surae muscle in young men (mean age 22 years). Muscles of the eye (orbicularis oculi) which contain predominantly fast fibres, have been recorded as having twitch times of 43 ms ± 4.3 (McComas and Thomas, 1968). In contrast to single fibre studies, whole muscle studies are complicated by the arrangement of fibres, joint mechanics and activation of fibres. Aagaard and Andersen (1998) have reported that the MHC-II isoform composition of the human quadriceps femoris muscle is correlated to the peak torque generated during isokinetic contractions at medium (120^0 sec^{-1}) and high (240^0 sec^{-1}) contraction velocities. Jurimae et al. (1997) showed that in bodybuilders, endurance rowers, and controls, the percentage MHC – II isoform content of the triceps brachii was associated with measures of strength using isoinertial, isokinetic and isometric loading protocols. In elderly subjects where the level of activation was standardised using an electrical stimulation procedure, the percentage of fast myosin of the triceps surae was significantly associated with the normalised torque at 5 rads sec^{-1} (Harridge et al. 1995).

v) ACTIVATION OF MUSCLE

The basic unit of contraction is the motor unit, which consists of an innervating neuron and associated muscle fibres (Sherrington 1929). Henneman, (1965) first proposed the size principle whereby the motor units consisting of smaller diameter axons and slower fibres would be activated first, the larger motor units being activated as the requirement increased towards maximum.

The number of motor units in a given muscle will depend upon the function of the muscle. A muscle that is required to carry out fine tasks involving small forces will have a low innervation ratio i.e. will preferentially contain a small number of muscle fibres per motor units, in contrast to muscle which is required to carry out gross tasks involving large forces. In a given muscle the number (Feinstein et al. 1955) and size (Edstrom and Kugelberg, 1968), of motor units varies considerably. The motor unit territory has been shown to be in the range of 5 – 10mm, and within these regions between 10 –25 units overlap (Stalberg and Antoni, 1980). In long muscles such as the hamstrings where the muscle length is divided into more than one compartment of muscle fibres, the motor unit may consist of fibres in more than one compartment (Manzano and McComas, 1988).

Gradation of force

The voluntary force generated by a muscle can be graded by a combination of factors. A muscle can increase the amount of force generated by recruiting more fibres, or by increasing the rate of firing of the neural impulse. Early experiments by Adrian and Bronk (1929) showed that subjects were able to increase the force of contraction by a combination of increased recruitment of motor units and increased rate of firing. Experiments conducted using the spike triggered averaging technique (Milner-Brown et al. 1973), Milner-Brown et al. (1973), show that increases in motor unit recruitment in order to increase levels of force occur at the lower force levels. However, the increases in firing rate to enable increases in force levels, become more important at the higher forces. This finding by Milner Brown and co-workers has been suggested by others to depend upon the muscle under investigation. Kukulka and Clamann (1981) reported that individual muscles utilised different strategies in order to develop force. The firing rates of motor units depend upon their type and function, but an individual motor unit has the ability to increase its rate of firing approximately four fold, although it has been shown that very high rates of motor neuron discharge can be observed if force is developed quickly (Tanji and Kato, 1972). It has also been reported that the lower threshold motor units modulate their firing frequency over a relatively larger range of forces than the higher threshold units (Monster and Chan 1977). The threshold for the motor unit recruitment levels for all the motor units has been shown to decrease as the velocity of contraction increases (Desmedt andGodaux, 1977, Desmedt and Godaux 1977). This apparent lowering of the recruitment threshold could lead to an apparent reversal of motor unit recruitment at the very highest velocities of contraction. Freund, (1983) suggested that this is not what actually happens, as high velocities usually require high transient forces and that the force level would have reached the same level as that normally seen for the recruitment of a particular motor unit.

2) *IN VIVO* MUSCLE TESTING METHODS

Historical perspective

Tests of human muscle dynamic strength and/or power have taken many different forms over the years. Hill in the 1920's was one of the earliest investigators to examine *in vivo* human muscle output. When muscle output is measured *in vivo* it is the torque around the joint which is measured, this is not necessarily reflective of the forces generated by the muscle fibres or their velocity of shortening. Factors which make difficult the interpretation of *in vivo* measurements to that of *in vitro* measurements include the pennation angle of the fibres to the line of pull (Maganaris et al. 1998), and the lever arms arrangement both of which may change during a dynamic movement. Other factors include the length – tension relationship which may be affected by the compliance of the muscle – tendon complex. With these limitations in mind the *in vivo* properties of muscle have been examined using different modalities including inertial devices (Wilkie 1950) and isokinetic dynamometry (Perrine and Edgerton 1978). Wilkie in the 1950's used inertial loading to determine the contractile properties of the elbow flexors under shortening conditions, he showed that the force – velocity relationship was in agreement with that described by Hill (1938) although in order to arrive at a hyperbolic fit he had to convert all rotational values to linear values. In the 1960's tests were developed which involved using body weight as the load during whole body dynamic actions to determine characteristics such as power output e.g. Margaria – Kalamen power test (Margaria et al. 1966), this represents an inertial loading protocol with the addition of gravity, the power output being measured by the time it takes for the subject to move their body weight a set distance vertically. Earlier, Sargent (1921) developed the Sargent jump test which related the jump height achieved to the power generated. Power output has also been determined using force platforms (Zamparo et al. 1997) here the force reactions to a movement are measured by the force plate and along with simultaneous measurements of velocity, are used to calculate power output. Other methods employed to assess the strength of human muscle include, measures of the maximum amount of weight that is possible to be lifted for 1 repetition (1RM) or a set number of repetitions. Static measures of muscle force or strength have utilised strain gauge systems or calibrated spring gauges in order to determine the force generation capacity of muscle. Some modern devices such as the isokinetic dynamometer can test power and strength under a variety of contraction conditions.

With the advent of computers and improvements in electronic technology in the 1970's came the development of isovelocity testing apparatus. Among the first to utilise these devices were Perrine and Edgerton (1978). These tools were quite unique in that they allowed the velocity of movement to be controlled at a preset level. This also allowed for a relatively easy method for the determination of *in vivo* force-velocity properties of muscle. There has been some discrepancy over the hyperbolic nature of the torque – velocity relationship utilising isokinetic dynamometry under voluntary contraction conditions. Wickiewicz et al. (1984) reported a lower than expected value of torque at the lower velocities of contraction, this was suggested to be caused by a neural inhibitory mechanism. However, James et al. (1994) using femoral nerve stimulation with a release component examined the force – velocity characteristics of the human quadriceps and found a good agreement with the hyperbolic curve of Hill (1938).

vi) TEST CHARACTERISTICS

Dynamic testing systems can be differentiated on their mechanical characteristics. The intrinsic mechanical design or physical properties of the different systems identify the testing devices by the extrinsic loading or control of velocity. Some of the devices used to dynamically test muscle can incorporate a manipulation of both loading and velocity parameters.

Mechanical loading can be either constant (isotonic), of a viscous nature, or inertial. Most devices will have a combination of the above factors, but will have a predominance of one. The time history of the loading characteristics of the device can also be manipulated; it is possible to control the loading to enable preloading or afterloading. A device that allows the muscle to generate a pre set level of force prior to movement identifies preloading, whereas an after loading device is one which allows for manipulation of the loading during the movement period. Both these conditions can be provided by modern isokinetic apparatus. Speed of movement can either be constant (isokinetic devices), or if acceleration is present as with inertial loading devices, the speed will be proportional to the applied force. Each particular method of testing has its own advantage and disadvantage. With regard to the functional testing of muscle, the specificity of the test will reflect its functional significance. Inertial loading for instance has the advantage of closely reflecting the muscle loading seen in everyday use. Functional test results obtained using one particular method cannot usually be compared easily with those using an alternative method due to the individual way in which each device loads a muscle.

Muscle functional testing

Each method of either isokinetic, isoinertial or isoviscous tests of dynamic muscle function, present a unique mechanical challenge to the muscle under test. The time history of both torque and velocity are different dependant upon which test modality is used. Figure 1.2 shows typical force and angular velocity time traces using each of the three modalities.

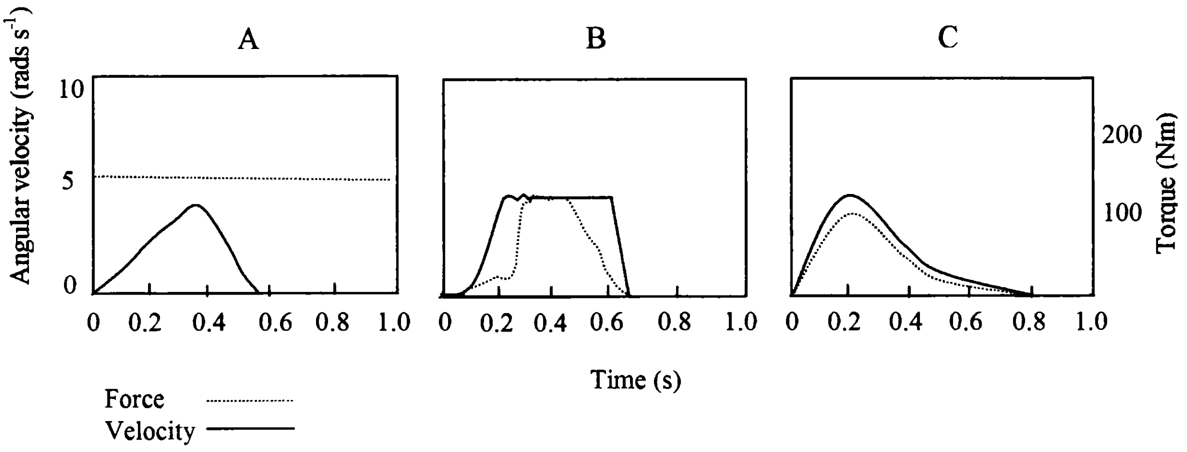


Figure 1.2. Typical traces of dynamic muscle contractions against isotonic (A), isokinetic (B) and isoviscous (C) test modalities. From Itoh et al. (1997)

Each method has its advantages and disadvantages in terms of its utility for functional testing. The method that has been most broadly employed is isometric testing; this is generally thought of as the gold standard for muscle strength testing, as generally static measures of strength are more controllable than dynamic measures. However the external validity in terms of functional relevance is questionable for measures of static strength.

Isometric testing

Muscle strength can be defined as the ability to produce maximum force. Strength can be defined in both dynamic and static terms. Both force and torque, which takes account of the lever arm characteristics, can be thought of as reflecting the strength of a muscle. The force producing capability of a muscle is a product of the muscle cross sectional area and also the fibre type and architecture. Isometric strength has been defined by Sale (1991) as, "The peak force or torque developed during a maximal voluntary contraction under a given set of conditions". The use of isometric dynamometry has been shown to be reliable in use when recording maximal voluntary contractions of elderly population groups (Davies and White 1983, McDonagh et al. 1984, Hunter et al. 1998, Winegard et al. 1996).

In terms of the validity of the measure to assess the physiology/morphology of the muscle under test, the general consensus is that tests of isometric strength give reasonable estimates of the muscle morphology in terms of its cross sectional area. Hakkinen and Keskinen (1989) report a correlation of $R = 0.7$ between the isometric strength and muscle cross sectional area for the quadriceps muscle. Fiatarone et al. (1990) reported that the 1RM of the quadriceps was significantly associated with the mid thigh muscle area ($R = 0.7$) in elderly subjects (mean age 90 ± 1 year). In agreement, Harridge et al. (1999) showed that the lean cross sectional area of the mid thigh was significantly associated with the maximum isometric force in elderly men and women.

With regard to the association of isometric strength and muscle fibre type, some ambiguity is seen in the literature. Tesch and Carlsson (1978) looked at the relationship between muscle fibre type and isometric strength in physical education students, the correlation found was $R = 0.5$, from this they concluded that fibre type distribution was an important factor in isometric strength output. Young (1984) reported a 'weak' correlation (R = 0.55) between the ratio of the quadriceps strength to the cross sectional area and percentage of type II fibres contained in a biopsy sample. Maughan and Nimmo (1984) reported that a relationship existed between the isometric strength and cross sectional area of the quadriceps in young males (R = 0.7), however they reported no relationship between the relative fibre composition and isometric strength. Similarly, Thorstensson et al. (1976) reported no significant relationship between muscle fibre composition and isometric strength.

Several researchers, again with a varying degree of association, have investigated the relationship between isometric strength and functional tasks. Hakkinen (1991) examined the isometric strength of the leg extensors, and its relationship to jump height in male and female basketball players, a correlation of 0.8 was reported. In contrast Considine and Sullivan (1973) examined the relationship between a number of isometric strength tests and tests of jump and sprint performance. They concluded that there was no relationship between isometric strength and dynamic performance from the correlation coefficients of –0.19 – 0.36. It could be said that one reason for the poor correlations may be that the experiments did not take in to account the angle at which peak dynamic strength is expressed as per the suggestion of Wilson and Murphy, (1995). However Strass (1991) utilised measures of isometric strength at the angles of significance for the dynamic action, although the correlations were better than those proposed by Considine and Sullivan (1973), they were still only average between the two measures ($r = 0.49 - 0.51$).

Isokinetic testing

Isokinetic methods of determining muscle function have been in general use for a number of years. A modern isokinetic dynamometer consists of a head unit, to which the lever arm and load cell are attached. A servomotor is contained in the head unit in order to control the movement velocity.

The basis of isokinetic dynamometry is that the velocity of movement is controlled at a preset level. The modern isokinetic dynamometers can be operated in a number of different modes and are generally described as accommodating resistance devices. In concentric mode an isokinetic dynamometer is a passive afterloading device, it can also be used in active modes to measure eccentric torques, or the lever arm fixed in order to measure isometric torque.

Most isokinetic dynamometers operate over a range of preset velocities up to approximately 5.1 rads s^{-1}, although it has been shown that valid measures are difficult above 2 rads s^{-1}. Some researchers have questioned whether in fact there is a need to test at such high levels of velocity, questioning the physiological validity. However this will depend upon the population group selected for testing, athletes for instance may require a larger range of test velocities than untrained individuals. Ghena andKurth, (1991) tested subjects at velocities up to >8 rads s^{-1}. James et al. (1994) reported that it was not routinely possible to record forces above 2.1 rads s^{-1} due to the small forces and large mechanical artefacts, these mechanical artefacts are present due to the deceleration of the lever arm and limb inertia. This inertial spike is one source of potential error which is present when using isokinetic testing devices, the other being errors due to gravity.

On the early isokinetic devices corrections for the effects of gravity were not made. This has been shown to affect the validity of results obtained where the movements were made in the vertical plane. Winter et al. (1981) reported that errors in the range of 26-43% and 55-510% were apparent in flexion and extension work measurements respectively. Erroneous results were also found in the angles at which peak torque occurred, with angle error of up to 15 degrees. The order of the error incurred is said to be larger as the loading decreases and hence the velocity increases. Modern isokinetic devices correct for the effects of gravity by utilising the method described by Nelson and Duncan (1983). Here the passive moment of the combined lever arm and limb under test is determined within the range of movement, for example during knee extensions the values at each joint angle are added to the moment determined under concentric actions at the same angle and subtracted from those eccentric actions.

Sapega et al. (1982) noted that during isokinetic movements an 'inertial spike' occurred in the torque record, which could be followed by oscillations of decreasing amplitude. This event took place at the beginning of the movement and was termed the 'torque overshoot'. During the initial movement phase, before the preset level of velocity has been reached, the limb and lever arm are free to accelerate. This overshoot phenomenon appeared due to the torque required by the system to decelerate the limb and lever arm to the preset level of velocity. Sapega et al. (1982) showed that during the free acceleration period during a hip abduction test the velocity reached 200% of the preset level prior to the control system compensating for the overshoot. As the torque of a moving rotational system is proportional to the acceleration and moment of inertia of the system, therefore the greater the mass and/or the further from the centre of rotation, the greater the torque potential and hence the torque overshoot.

Various methods have been employed in order to reduce the torque overshoot. Damping of the lever arm is one method that has been used in order to alleviate the torque overshoot. The damping effect however results in changes to the torque output record. Sinacore et al. (1983) reported that by damping the lever arm, the torque record throughout the movement was reduced and shifted in time. Therefore various researchers have suggested that damping is not a suitable method of reducing the torque overshoot (Murray and Harrison, 1986). Controlling the initial acceleration period was another method used in order to reduce the initial overshoot. Gransberg and Knutsson (1983) using a control mechanism to apply a resistive force during the initial acceleration phase found that the torque overshoot and subsequent oscillations were reduced, allowing a smooth transition from rest to the predetermined level of velocity. However the time required to reach the preset velocity was increased, thereby introducing a phase shift. It seems that the approach used by Perrine and Edgerton (1978), which involves using only the section of the torque record that occurs during a constant velocity phase of the movement, represents an empirical compromise, and this approach is one that is mostly used to date.

Other approaches using isokinetic loading include cycling (Sargeant et al. 1977, Sargeant et al. 1981, Davies et al. 1984). The use of isokinetic loading in this protocol allows the peak power output to be determined and the associated velocity at which peak power occurs (V_{opt}). The V_{opt} and maximal power output obtained during isokinetic cycling has been suggested to be representative of the general fibre population of the active muscles (Sargeant 1994). For expansion on this point see Chapter 4.

Viscous testing

The property of these devices can be thought of as having qualities of resistance to flow in a fluid or semi fluid, the coefficient of viscosity being the ratio of the tangential frictional force per unit area to the velocity gradient perpendicular to the direction of flow of a liquid. A viscous system can take different forms; it can be presented as an electromagnetic braked device (e.g. electrically braked cycle ergometer), air filled or a fluid filled resistive device. Grieve and Linden (1986) used a fluid filled device to examine the characteristics of horizontal pulling. The resistance to movement was described as being proportional to the square of the velocity of movement. Here it can be said that the resistance to movement is proportional to the applied force, the system acting as an accommodating resistance device. However unlike isokinetic devices, hydrodynamic devices allow for constantly changing force and velocity. Therefore viscous systems can be thought of as having some of the characteristics of both inertial and isokinetic systems.

Inertial testing

An inertia can be defined as, "that which resists change", and is a function of an objects mass. The properties of inertia are described by Newton's first law which states that, "Every object persists in a state of rest or uniform motion in a straight line unless compelled by an external force to change that state". The characteristics of accelerating inertias are explained using Newton's second law which states that whenever an object accelerates, the acceleration is: -

a) directly proportional to the NET force acting on the object;

b) pointing in the same direction as the net force; and

c) inversely proportional to the mass of the object.

Testing muscle function using inertial loading has certain advantages, these being that an inertial load best represents the type of challenge that muscles face in the physical environment, and also an inertial loading device is relatively cheap. However, a disadvantage from an experimental point of view is that it is not possible to control either force or velocity when the muscle contracts under inertial loading conditions. Inertial devices have been in use for many years, Best and Partridge (1928) were one of the first to use a system to examine *in vivo* human muscle using a combination of inertia and friction. Most free weight systems can be thought of as pseudo inertial systems in that they usually consist of an inertial and gravity component. The system which closest mimics a pure inertia by nullifying the effects of gravity (assuming minimal friction) is that of a wheel.

Wilkie (1950) used a pseudo inertial loading system to determine the force velocity properties of the elbow flexors. He concluded that after corrections to allow the rotational components to be expressed as horizontal components that the *in vivo* force velocity characteristics of the elbow flexors agreed with Hill's hyperbolic relationship. Murphy et al. (1994), used a combination of isoinertial and isometric testing apparatus on a group of weight trained individuals. The scores were compared to their ability to carry out a series of dynamic tests; it was found that in all cases, the isoinertial testing modality gave the highest correlations when compared with the dynamic task.

Bassey and Short (1990) devised an apparatus comprising of a fixed inertial flywheel to determine the average power output of the lower limbs during a single lower limb thrust. This was designed to provide a safe convenient and valid method of testing 'explosive' power in frail elderly people where performing other tasks would be either impossible to perform or have a high risk of injury. However the method used to determine the exertion time which is used in the calculation of average power, was an estimate rather than a direct measure. This estimation method in order to be valid relied on certain assumptions with respect to the acceleration profile during the exertion. See Chapter 1 for further discussion of this point.

Inertial cycling

More recently investigators have used cycling, as this incorporates a spinning wheel which allows for the repeated acceleration of the load in contrast to a single exertion, to examine the power component characteristics of human muscle output. Lakomy (1986) criticised the standard friction braked cycle tests for not taking account of the inertial component. He suggested that during cycling, the work done in transferring energy to an inertial flywheel whilst accelerating would be of significance. He found that by including an inertial component, if the frictional load was added after the wheel was up to speed the measured power was approximately 6% less than without the inertial component. Whereas if the subject started from rest with the load applied, the corrected values were 32% higher than those not taking the inertial component into account. This can be explained by the fact that a large energy component is required to accelerate the inertial load, and as torque is proportional to acceleration, the larger the acceleration the greater the torque component.

Lakomy (1986) had suggested that in contrast to friction braked cycling ergometry; inertial loaded cycling power output was independent of the inertial load (See Chapter 3 for expansion of this point). Martin et al. (1997) used a purely inertial flywheel to examine the power output characteristics during short term maximal cycling. In order to monitor the motion of the flywheel an optical sensor and slotted disk system was used, this allowed for a resolution of 3^0 of flywheel movement. The timing accuracy was quoted as 1 micro second; however, due to the gearing system used this resolution was reported to introduce random errors due to the changes in delta time between consecutive slots being less than the timing resolution. Using a single, relatively high, flywheel inertia (0.396 kgm^2), they concluded that a fixed inertial load is able to provide a valid measure of power output during cycling.

3) SKELETAL MUSCLE AND AGEING

vii) SKELETAL MUSCLE OUTPUT PROPERTIES AND AGEING

Strength

It has been shown that strength begins to decline from as early as fifty years of age in healthy normals. The ability to develop force peaks between the second and third decade (Hurley et al. 1995), and thereafter decreases with age. The rate of decline in muscle strength has been reported as 12 – 15% per decade in men aged between fifty and eighty years old (Hurley et al. 1995). Frontera et al. (1991) reported a strength decrement of between 15 – 26% to that seen in subjects aged 45 – 54 years old in subjects aged 65 – 78 years old. Cross sectional studies reporting the levels of isometric strength recorded in young and older men (age range 11- 70 years), indicate that younger men have in the order of 30 – 47% higher values than that of older men (Larsson et al. 1979). Differences in the rates of strength loss between the upper and lower limbs has been reported (Bemben et al. 1991), although the relative changes in strength for any given muscle group has been shown to be similar between young and older groups (Era et al. 1992). Of the longitudinal studies carried out, Bassey and Harries (1993) in a study on 620 men over a four-year period reported a 12% loss of handgrip strength in men over 65 years old. Aniansson (1986) reported that over a seven-year period, strength losses in men aged 73 - 83 were in the order of 10 – 22%, it was shown that a 14 and 25% reduction in type IIA and IIB (now designated IIX) fibres accompanied this strength loss respectively. Of interest Kallman et al. (1990) reported that not all individuals showed a decline in muscle strength with age, with 48% of those under 40 years, 29% of those 40 –59 years of age and 15% of those older than 60 years of age showing no decline in muscle strength over a nine year period. It was also observed that the higher the initial strength of an individual the greater the decline in strength with age. Greig et al. (1993) in a follow up study remeasured the isometric strength of men (median age 81 years) and women (median age 82 years) they reported that quadriceps strength was well preserved with a reduction of only 0.3% per annum.

There are inherent differences with cross sectional studies and longitudinal studies, each having particular advantages and disadvantages. The advantages of a longitudinal design are that the cohort group is consistent, hence there is no cohort effect, this effect can lead one to erroneously conclude that there is an age effect when it is in fact due to differences in the group cohort. The disadvantages of a longitudinal study are that they are generally more time consuming and therefore more expensive, if repeated testing is applied this can threaten the study validity, also dependant on the length of the study, subject attrition can be a problem. In contrast cross sectional studies are usually more efficient to carry out, are not threatened by history or testing effects but can suffer from cohort effects.

Specific strength

In order to assess the qualitative changes in muscle with age, the measure of maximal force per unit cross sectional area is used (N cm^{-2}). Factors that may be responsible for any given changes in specific strength between older and young individuals include differences in myofibrillar packing, intrinsic changes in the myosin cross bridges and changes in the tendon stiffness (Jones et al. 1989). The changes in tendon stiffness could cause changes in the forces generated by the muscle myofilaments due to the sarcomeres generating force on different portions of the length tension curve. Jubrias et al. (1997) measured the specific force in men aged 23 – 80 years old and found reductions in the specific strength of older men when compared to younger men. Contractile cross sectional area was determined using magnetic resonance imaging; therefore it is unlikely that these differences could be due to the difficulty in measuring the muscle cross-section. Alway et al. (1996) has suggested that when using less sensitive methods of assessing cross sectional area, it is possible to overestimate the contractile cross sectional area due to the intramuscular fat. In contrast to the data of Jubrias et al. (1997), Alway et al. (1996) reported that when the maximal force output was related to the muscle volume determined using MRI, no differences were found between young and older individuals, suggesting that specific tension is unchanged with age. This method averages all the fibres within a muscle and therefore may not be sensitive enough to detect individual fibre changes in specific force output. There are also inherent difficulties when associating anatomical cross sectional area and muscle strength. The strength generated is more accurately a factor of the physiological cross sectional area and this may change as a different ratio to the changes in anatomical cross sectional area as a consequence of the muscle fibre pennation angle. In a recent single fibre study Larsson et al. (1997) have reported that in fully activated skinned fibres *in vitro* produce 28% less force if the fibres originate from the muscles of older men aged

73 – 81 years compared with young men aged 25 - 31 years. These finding indicate a reduction in the specific force of fibres originating from older men. The possible contrast between the findings of Larsson et al. (1997) and those of Alway et al. (1996) could in part be due to the method used by Alway et al. (1996) being less sensitive to the individual fibre changes due to the averaging procedure.

Power

Muscle power is required for any dynamic task that requires a very high exertion over a short period of time. In terms of power to body weight ratio, Levy et al. (1994) reported that there exists a value below which performing certain functional tasks are impossible. Values of 1.5 W kg^{-1} and 2.5 W kg^{-1} being the values required to mount a step height of 30 and 50cm respectively. Skelton et al. (1998) reviewed the National Fitness Survey data (1994), and reported that 15% of men aged 70 – 74 years had a power to weight ratio below 1.5 W kg^{-1}. See Chapter 5. The ability of a muscle to develop power is determined in the main, by a combination of its volume, composition and temperature.

Using the Nottingham Power rig (NPR), it has been shown that lower limb muscle power is lost with age at approximately twice the rate of isometric knee extensor strength in men aged 65 – 89 years old (3-4% vs. 1-2% 0f a 77 year olds value) (Skelton et al. 1994). However, this method involves making measures of muscle power utilising only one fixed inertial load which may not represent the optimal load for all subjects. This can be explained in terms of the torque – velocity relationship of skeletal muscle; against a fixed load, a weaker muscle is forced to utilise a greater proportion of its maximal torque generating capacity, this may result in a contraction velocity which is less than optimal for power generation in contrast to a stronger muscle.

The greater declines in power than strength are further exacerbated by the additional slowing of ageing muscle. This is due in part to 1) a lower MHC-II proportion in ageing muscle (Klitgaard et al. 1990, Harridge et al. 1995), 2) An intrinsic slowing of ageing fibres, Hook et al. (2001) reported an 18 – 25% reduction in the speed of actin filaments from fibres expressing the slow type I beta MHC isoform. The mechanisms underlying these changes are unknown but it was suggested that postranslocational transformation of myosin by oxidative stress or glycation may play a part, 3) possibly to the lower working temperatures of older muscle (Davies and Young 1983, Young 1992). Therefore, not only does the intrinsic force velocity characteristics of the muscle change with age, but the power output available to move the body (which approximates a fixed load), is reduced possibly to some portion along the ascending limb of the power velocity curve.

viii) CHANGES IN SKELETAL MUSCLE WITH AGE

Investigations into muscle composition and size with age have revealed that there are declines in the muscle cross sectional area (CSA) and qualitative changes to the fibres themselves. The CSA of the quadriceps as examined by ultrasonography (Young et al. 1984, 1985), has been reported to decline in older men and women by 25 –33% compared to that in young subjects. When other muscles have been examined using computerized tomography similar values for the decline in muscle CSA have been reported (Rice et al. 1989, Klitgaard et al. 1990). Lexell et al. (1983) directly measured by post mortem methods the vastus lateralis of 'normal' men between the ages of 30 and 72 years old. They reported that the average reduction in muscle CSA was 18%, and the total number of muscle fibres was reduced by 25%. Work done on the quadriceps muscles by various groups (Lexell et al. 1988, Larsson et al. 1978, Aniansson et al. 1981, Aniansson et al. 1986, Grimby et al. 1982) suggest that there is a consistent loss of fibre size, this is selective in terms of the type II fibres, the type I fibres being much less affected. Grimby (1982) reported that in the vastus lateralis, the reduction in fibre size up to the mean age of 65 was equivalent in both fibre types, thereafter, the area of the type II fibres decreased by 15%, although the total mean fibre area only decreased by 5 – 10%. When the fibre composition of the limbs of the upper body (biceps brachii), has been compared to that of fibres from the lower limbs in men and women aged 78 – 81 years old, it was found that the type II fibre areas were smaller in the lower limbs than the arms (Grimby et al. 1982). It has been suggested that this observation could be due to a difference in activity pattern between the upper and lower limbs. Grimby (1983) suggested that the reduction in fibre size alone could not explain the apparent reduction in muscle volume. Lexell et al. (1982) directly examined the number of muscle fibres present in the midsection of the vastus lateralis of young men (mean age 27) and older men (mean age 72). They reported that there were approximately 110000 fewer fibres in the older men when compared to the younger men. Lexell et al. (1982) also reported the unpublished findings of Colling – Saltin who showed that infants have a higher proportion of muscle fibres than young adults (510000 vs. 395000), suggesting that muscle fibres are lost from birth. Lexell et al. (1988), in contrast to the findings of Colling –Saltin reported that the loss of muscle fibres begins at age 25 thereafter the loss accelerates. They also concluded that both a loss of muscle fibres and a reduction in the fibre CSA caused the decline in muscle CSA. The general consensus is that fibre loss affects both type I and type II fibres equally. Multi single unit EMG techniques were used by Stalberg and Fawcept (1982, 1989) in an attempt to determine whether the loss of muscle fibres resulted in motor units that contained fewer fibres or whether complete motor units were lost. They concluded that elderly motor units contained more fibres due to the increase in motor unit action potential (MUAP). This would suggest the possibility that the number of motor units decreased with age. Various workers (Tomlinson and Irving 1977, Brown et al. 1972, Doherty and Brown, 1993) have reported that the number of alpha motor neurons in the spinal cord decreased with age. Tomlinson and Irving (1977) reported that above the age of 60, some individuals exhibited only half of the number of motor neurons found in the young. Kawamura et al. (1977) also reported that the number and size of the motor axons in the ventral root degenerate and decline with age. The remaining motor units also exhibit different mechanical and histochemical properties to those seen in younger individuals. With age, a prevalence of fibre type grouping is seen; this remodelling of muscle is thought to be due to the dennervation reinnervation process that is undergone by the fibres in response to injury. Brooks and Faulkner (1995) reported that the injury to muscle fibres could be caused by contraction and that once injured the fibres undergo a repair process which is less effective in older individuals. Lieber et al. (1991) suggested that fast fibres were more susceptible to injury than slow fibres, also when denervated the fast fibres were less successful at reinnervation than the slow fibres (Desypris and Parry 1990). After dennervation it has been reported that the faster fibres are also more likely to be reinnervated by axonal sprouting from smaller slow muscle fibres (Kanda and Hashizume 1989).

Muscle fibres in everyday use are constantly undergoing damage and repair. With ageing it is thought that there is a reduced ability for the muscle fibres to repair themselves. Within a muscle cell there are many nuclei. These nuclei are post mitotic and are thought to exert influence over a number of cellular proteins within a certain domain. Within the basal lamina of the muscle cell there are also a number of satellite cells that are undifferentiated myogenic cells. The satellite cells when activated are incorporated into the muscle cell as nuclei. Therefore if the proliferation of the satellite cells is reduced the ability of the muscle fibre to recover or grow is diminished (Rosenblatt et al. 1994). In humans a cross sectional study by Hikida et al. (2000) suggested that there was no difference in the cytoplasm to satellite cell ratio between the muscle fibres of young and elderly men, even when the elderly muscle was hypertrophied by resistance training. No account was given of the possible changes in neucleo-cytoplasmic ratios between the fibre types in the elderly muscle and how these may differ from the young. This may be of interest as it has been shown that there is a preferential atrophy of type II fibres with ageing, therefore a relative reduction in fibres containing type IIA and IIX myosins.

ix) RESISTANCE TRAINING, SKELETAL MUSCLE AND AGEING

Muscle power is a major requirement for a person's ability to carry out physical tasks (Bassey et al. 1992). In a simple model to improve power output one can either increase the dynamic force generating capability of the muscle, the velocity of movement, or both.

Cross sectional studies of master athletes

Olympic weightlifters who are required to develop high levels of power, and who carry out high intensity resistance training, still show a decline in performance (i.e. in the clean and jerk and snatch) with age of approximately 1 – 1.5 % per year (Meltzer 1994). This decline in performance of master athletes is well documented and suggests an obligatory decline with age regardless of the level of physical activity. However, little is known regarding the power generating capabilities of men who have been strength training for many years and who represent the elite with respect to physical performance, with respect to those who are untrained. This aspect is examined in detail (see Chapter 5).

Sipliä et al. (1991) in a cross sectional study, reported that in subjects who were either untrained, strength trained, endurance trained or speed trained (aged 70 – 81 years), that the strength trained group showed better preservation of isometric strength in terms of grip strength, arm flexion, knee extension, trunk flexion/extension characteristics than those of the untrained group. In contrast to strength training, Harridge et al. (1997) reported that men aged 70 – 100 years who were long term endurance trained, showed similar muscle strength to untrained age matched controls. Grassi et al. (1991) showed that the lean lower limb volumes (LMV) in young endurance and power athletes (17 – 26 years) were approximately the same, whereas in older athletes (40 -78 years) the (LMV) was higher in power athletes than endurance athletes, also that power output (as assessed by a vertical jump off both feet), showed a linear decline with age in both endurance and power athletes. However, apart from the oldest age group, peak power was higher in the power athletes.

Longitudinal strength training studies in elderly people

In recent years a number of studies have shown that in older individuals muscle strength can be increased as a result of high resistance training (Hikida et al. 2000, Fiatarone et al. 1990, Frontera et al. 1988). The amount of strength increase in response to resistance training has been suggested to depend upon the muscle group trained (Hopp 1993). The physiological responses to resistance training, which result in increases in strength in older individuals, have been reported as showing a different time course. Moritani and DeVries (1980) in a study involving young and older subjects reported that despite similar increases in strength the older group increased strength predominantly by increased neural activation. The younger group, however, increased strength initially by way of increased neural efficiency and thereafter by muscle hypertrophy. The study by Moritani and DeVries was carried out over 8 weeks and on only 5 subjects per group, the time allowed may not have been sufficient to allow the muscle of the older group to undergo hypertrophy. This is not to say that older individuals cannot increase muscle size with resistance training, Roman et al. (1993) showed that both strength and size increased in the elbow flexors of elderly males following a 12 week resistance training programme. Lexell et al. (1995) looked at the short and long term effects of resistance training of the elbow flexors and leg extensors of Scandinavian men aged 70 –77 years and reported strength increases and muscle fibre hypertrophy in both muscle groups after the first 12 weeks of training. Muscle strength was found to continue to increase up to the end of one year.

Morphological adaptations to resistance training

Skeletal muscle lean anatomical cross sectional area has been shown to be associated with maximum voluntary isometric strength (Harridge et al. 1999). However, Fiatarone et al. (1990) using computerised tomography (CT) scanning on a group of 90 year old males and females reported that although muscle size had increased over the training period, no association was found between the gains in muscle size and strength as measured using 1RM. This in part could be due to the fact that the testing and training protocol were similar hence any skill related factor would be transferred. In support of this the strength gains also appear to be greater than that reported by Harridge et al. (1999), and the training period shorter, however the subjects studied by Harridge et al. were older (up to 97 years old). Studies involving magnetic resonance imaging (MRI) and CT, to examine the changes in whole muscle cross sectional area have shown that both muscle cross section and thereby volume increase in older individuals as a result of high resistance training. The reported increases in muscle cross sectional area vary dependant upon the training protocol, measurement method and the muscle group examined. Roman et al. (1993) in a study involving males of mean age 67 years examined the changes in elbow flexor cross-section. Increases in muscle volume and cross-section as measured using MRI after 12 weeks of heavy resistance training were 13.9 and 22.6% respectively. In a study by Ivey et al. (2000) the hypertrophic response of skeletal muscle to resistance training has been shown to be similar in older men (mean age 69 years) to that of younger men (mean age 25 years). They reported that after nine weeks of strength training there was no significant difference in the increases of muscle volume as determined by MRI, between the young and old subjects. See Table 1 for summary data of elderly strength training studies.

Table 1. Summary of strength training studies in the elderly. Training column - weeks/sessions per week (sets x reps x % 1RM)

Group	**N° of subjects/sex**	**Age (y)**	**Training**	**Muscle group**	**Mean strength increases %**
Harridge et al. (1999)	11/M+F	85-97	2/3 (3x8x80%)	quadriceps	138 1RM 17 isometric
Frontera et al. (1988)	12/M	60-72	12/3 (3x8x80%)	knee extensors	107 1RM
Brown et al. (1990)	14/M	60-70	12/3 (2-4x15x50-90%)	elbow flexors	48 1RM
Fiatarone et al. (1990)	10/M+F	86-96	8/3 (3x8x80%)	knee extensors	174 1RM
Grimby et al. (1992)	9/M	78-84	8-12/3 (3x8) isokinetic	knee extensors	10% at 30^0 sec^{-1}
Charette et al. (1991)	13/F	64-86	12 (6x6)	knee flexion	115% 1RM
Fiatarone et al. (1994)	100/M+F	72-98	10/3 (3x8x50-80%)	knee extensors	113% 1RM
Judge et al. (1993)	18/M+F	71-97	12 (3x8-10)	knee flexors	32 % 1RM
Anniansson et al. (1984)	26/M+F	63-86	26/3 (elastic bands)	quadriceps	6-13 % isokinetic
Skelton and McLaughlin (1996)	10/F	74-79	8/3 (3x4-8 elastic tubing)	knee extensors	20 % isometric
Sipila and Suominen (1995)	16/F	76-78	16/3 (4x8-10x60-75%)	knee extensors	14 % 1RM 14 % isometric
Skelton et al. (1995)	20/F	76-93	12/3 (3x4-8 elastic tubing)	knee extensors	27 % isometric
McMurdo and Rennie (1994)	55/M+F	83	26/3 (seated group exercise)	knee extensors	18 % isometric

Even in individuals of 90 years old increases in muscle size with resistance training can be detected, Fiatarone et al. (1990) reported mean gains in thigh cross sectional area of 9%. Harridge et al. (1999) using MRI measured the changes in anatomical lean cross sectional area of the quadriceps and reported a 9.8% increase over the same training period as Roman et al. (1993). The differences in cross sectional area increases between the studies could be in part due to the muscle groups studied. Also no mention of a lean measure of cross sectional area is given in the Roman et al. study, if non contractile tissue was included in the measure this could also account for the larger increase noted. The effects of a long-term (42 weeks) resistance-training programme were examined on the cross sectional area of the quadriceps in two groups of men aged 60-70 years and 70-80 years. An increase of 5.5% in the total cross sectional area (muscle + bone + fat) was reported; there was no effect of age (McCartney et al. 1996).

Muscle fibre size increases result from the hypertrophic responses to resistance training. Frontera et al. (1988), in a study involving 12 males of mean age 66 years, showed that following 12 weeks of high resistance training (80% 1RM) of the lower limb muscles, the quadriceps femoris increased in area by 9% as determined by CT scans. This increase in muscle cross sectional area was accompanied by a 34% and 28% increase in type I and II fibre cross sectional area respectively. Similarly Lexell et al. (1995) reported a significant increase in both type I and type II fibre area in the biceps brachii muscle and a positive correlation between the type II fibre area and strength in the vastus lateralis muscle after 12 weeks of high resistance training (85% 1RM). In contrast Hakkinen et al. (2001)

reported that men (young-mean age 42 years and older-mean age 72 years) who had undergone a 6-month resistance-training programme showed increases in strength but no significant increases in fibre area. The increases in strength were associated with a reduction in antagonistic co activation and increased activation of the agonists. The insignificant increases in fibre size recorded were attributed to the training protocol, which contained a low intensity component. It was also due to the small number of subjects per group.

Cellular and molecular adaptations to resistance training in elderly people

It has been reported that the transcription levels of specific MHC are reduced with ageing. Balagopal et al. (2001) reported that transcription levels of MHC-I isoforms are not altered by ageing but those of the MHC-IIA isoforms are decreased by 38% from young (20-27 years) to middle age (47-60 years) and a further 50% from middle to old age (>65 years). MHC-IIX decreased 84% from young to middle age and a further 48% from middle age to old age. They further suggested that although resistance training increased the fractional synthesis rate of MHC and mixed muscle protein by 47 and 56% respectively, transcript levels of MHC-I increased by 85% but that of MHC-IIA and IIX was lowered.

It has been reported that the ability of aged muscle to increase the protein synthesis rate as a result of resistance exercise is similar to that of younger muscle. Hasten et al. (2000) reported that in response to a 2-week high intensity resistance-training programme, men aged 78-84 years old increased their mixed muscle protein and myosin heavy chain protein synthesis rates to levels similar to that of a group aged 23-32 years old. Goldspink (1992) suggests that fast fibres undergo hypertrophy by increasing the protein synthesis rate, whereas slow fibres by decreasing the degradation of protein.

Resistance training in elderly men has been shown to preferentially increase the size of type II fibres. Roman et al. (1993) showed that type II fibres, MHC-II isoforms, underwent preferential hypertrophy in a group of men of mean age 67 years, who had undergone high intensity resistance training. Such an increase should in theory increase the power generating capability of the muscle as this would result in a greater proportion of the muscle being occupied by fast contracting MHC-II isoforms.

A number of studies have shown that muscle fibre transformation (in the form of the fast fibre subtypes) is evident after a period of high intensity resistance training. For example, Kraemer et al. (1995), and Hagerman et al. (2000), reported that after a period of strength training, the proportion of type IIB (IIX) fibres decreased, whilst type IIA increased. With ageing the number of fibres co expressing MHC isoforms of different types increases (Andersen et al. 1999). The effect of high intensity resistance training has been shown to decrease the co expression of MHC isoforms in hybrid fibres. Williamson et al. (2000) reported that progressive resistance training in older men (mean age 74 years), over a period of 12 weeks, reduced the expression of MHC I/IIA, I/IIA/IIX, IIA/IIX isoforms whilst no change was noted in the expression of IIA, IIX isoforms.

Whereas the myosin light chain (MLC) ratio, MLC_{3f} to MLC_2, was not changed due to resistance training.

4) SUMMARY

The dynamic contractile properties of human skeletal muscle have been studied using a variety of experimental approaches at both the *in vitro* and *in vivo* level. At the *in* vivo level the experimental work has been largely conducted using isokinetic apparatus which whilst providing important information regarding the torque and velocity properties of a muscle do not reflect the muscle use in real life situations, where muscles overcome inertial loads with the addition of gravity is usual. The central aim of this thesis is therefore to expand on the characteristics of the mechanical properties of skeletal muscle undergoing dynamic, inertial loaded contractions. It is clear that muscle composition plays a role in determining the power output of individual muscle fibres and to some extent this has also been shown with whole muscle experiments using isokinetic contractions. However, its role in inertial contractions is not known. In this regard the reduction in muscle power output with ageing could be due to a combination of weaker muscles and a composition of muscle which is dominated by slow MHC-I isoforms. With regard to the former, the lack of optimisation of inertial loading for older muscle has not allowed this to be studied objectively. It is known that athletes who are long term resistance trained are likely to be more powerful, yet an objective comparison using inertial apparatus that allows load optimisation and is safe for non trained subjects has not been performed.

The experimental work includes the development of apparatus suitable for the study of the contractile properties of muscle. The subsequent chapters investigate the effects of contraction time on muscle power output, the effect of variable inertial loading on power output and the differences in power output using two different test modalities but using the same limbs. An examination is made of the effect of the MHC-II isoform composition on power output. Finally elite level Olympic weightlifters are utilised in order to examine the differences between untrained and trained subjects under inertial loading conditions.

AIMS OF THESIS

1) To design and construct an inertial testing system to allow the accurate and optimal determination of muscle power output which does not require the use of goniometry or strain gauge systems.

2) To examine the effect of time allowed to develop power output

3) To compare the power output under different exercise conditions (sprint cycling vs. the Nottingham Power Rig)

4) To examine the relationship between power output, age and muscle MHC isoform composition

5) To examine muscle power and isometric strength characteristics in elite master weightlifters of increasing age in comparison with similar aged subjects who are healthy but who do not undertake any exercise.

PART II

CHAPTER 1

INERTIAL TESTING SYSTEM DEVELOPMENT

1.1 INTRODUCTION AND RATIONALE

Over recent decades, isotonic, isokinetic, hydrodynamic and other systems have been designed and used to measure the dynamic contractile properties of human muscle. However, it is the acceleration of inertial loads, which most closely resemble the demands placed upon skeletal muscles in everyday exertions. A.V. Hill was one of the first to realise the potential use of inertia in the form of a flywheel to study muscle in the 1920s (Hill 1920). More recently others (Aagaard et al. 1994, Berg and Tesch 1994) have also described systems for studying human muscular exertions against inertial loads, which involve the use of strain gauges to directly measure applied torque and goniometers to measure joint rotation.

In a purely rotational inertial system, such as a flywheel, the angular acceleration of the wheel is directly proportional to the applied torque. From determination of the inertia and rotational movement of the flywheel alone it should therefore be possible to infer the mechanical properties of the muscular exertions accelerating the flywheel, assuming that the body and the machine are tightly coupled.

Recently Bassey and Short (1990) developed a tool for measuring the average muscle power developed during a lower limb extensor thrust, this is known as the Nottingham Power Rig (NPR). Here a heavy flywheel of fixed inertia represented the load. The inferred value of average power applied was determined by calculating the fixed value of the flywheel inertia and by determining the motion of the flywheel. The movement time during the exertion was usually less than 1 second. The method employed involved the calculation of the terminal velocity of the flywheel (thereby obtaining its kinetic energy). Average power output was estimated on the assumption that the wheel's acceleration (and therefore the applied torque) was constant throughout the exertion. The calculation of average power is simply then the kinetic energy divided by the exertion time. The kinetic energy being derived from the equation $KE = \frac{1}{2} \bullet I \bullet \omega^2$, where I is the flywheel inertia and ω is the final flywheel angular velocity. The exertion time being estimated from the time required for the flywheel to rotate one full turn after the exertion phase (see Appendix A).

As the assumptions of constant acceleration and hence linear velocity increase during the exertion phase were of fundamental importance to the calculation of the exertion time, these factors were examined as part of this study (see Appendix A). Furthermore, because the NPR only allowed a figure of average power to be determined, an experimental analysis of the velocity and acceleration during the exertion phase was undertaken. This experiment was undertaken using a CODA motion analysis system. The results showed that the underlying assumptions regarding the constant acceleration and linear velocity increase during the exertion phase were not true therefore a suitable method of monitoring the exertion phase was required (see section 2 - system description).

When measuring either peak or average power during an exertion, the values obtained will depend upon the test protocol used. However in a test where the time of exertion is very short (typically less than 0.5 seconds) the use of a fixed inertial load as in the NPR, may not be optimal for all individuals in terms of maximising the power output; this can be illustrated best by examination of the hypothetical force-velocity characteristics of two skeletal muscles which differ in their force producing capabilities (see Figure 1.3).

In Figure 1.3 a simple example is shown where it can be seen that the possible force generated at a fixed load as represented by the arrow, allows a certain level of velocity to be generated corresponding to the particular characteristics of each force-velocity curve. For a weaker person, the level of force will represent a relatively higher proportion of their maximum force generating ability. In terms of power output this translates into disproportionately lower power for the weaker muscle, because a slower velocity of contraction is required to generate the required force. Therefore using this rationale it is hypothesised that a system, which uses a fixed inertial load as a resistance, may not allow the optimal generation of power for all individuals.

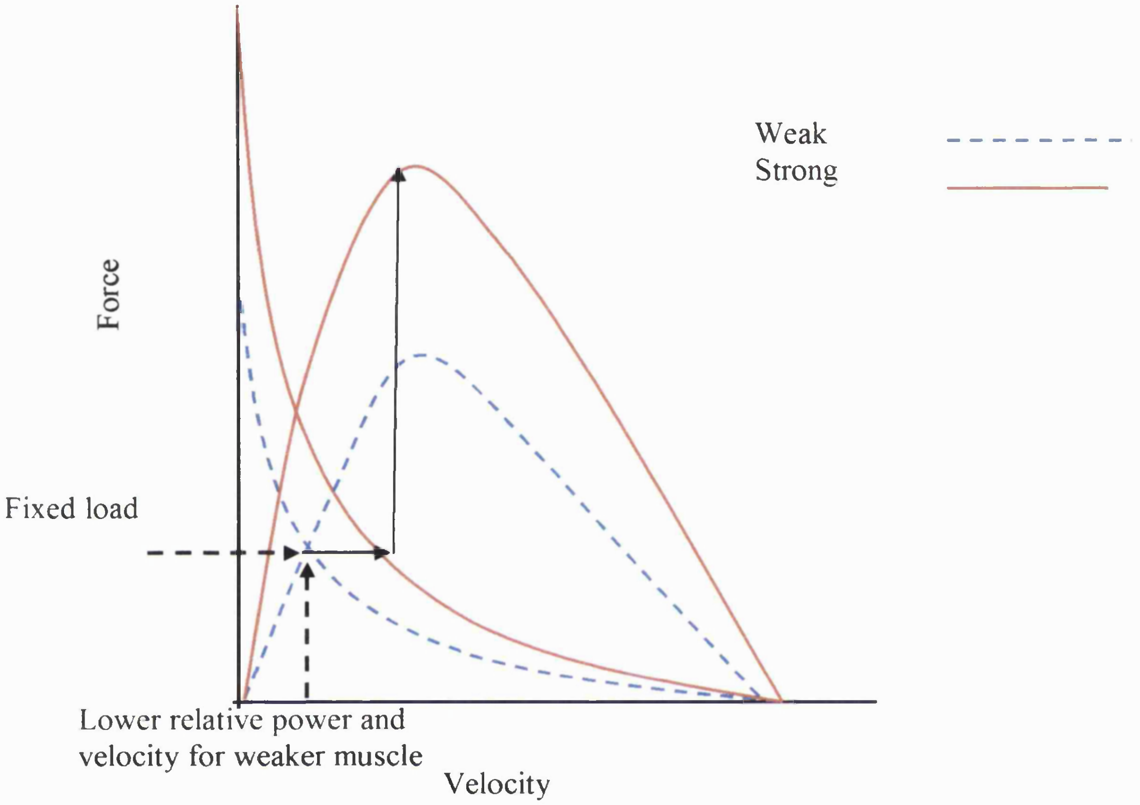


Figure 1.3 Hypothetical force-velocity curves and power characteristics for a relatively weak muscle and a stronger muscle. The arrows indicate a slower velocity of movement required by a weaker muscle to shorten at a fixed absolute load resulting in a relatively lower power output.

In light of the experimental observations of the non-linearity of velocity during the exertion phase (see Appendix A), a suitable method of monitoring the exertion was needed in order to obtain accurate measures of muscle output values actually during an exertion itself. Furthermore, it is clear that an inertial system whose inertial load could be varied in order for power output to be optimised for each individual was also needed if an objective measure of maximal muscle power output was to be obtained from an inertial system.

Aims

1) To develop a system which accurately monitors an exertion against an inertial load and which also allows discrete measures of power, torque, velocity, and acceleration to be made.

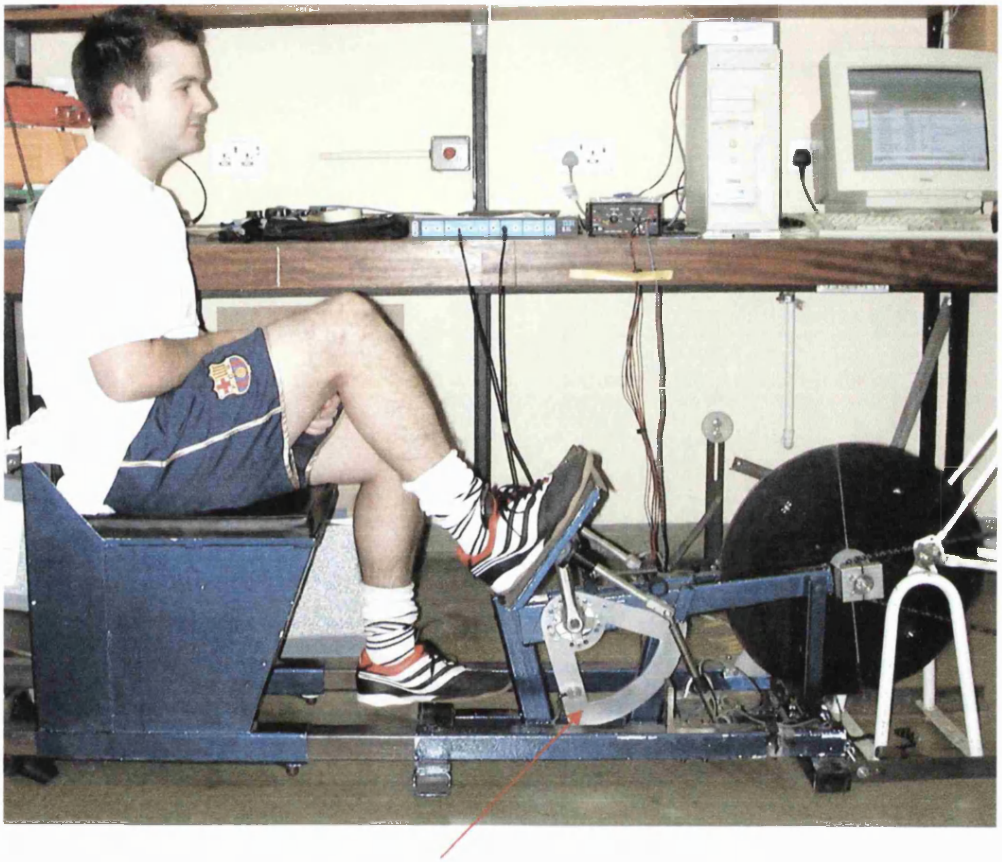
2) To design a loading system whose inertia could be varied in order to allow optimisation of power output for different individuals

1.2 NEW METHOD TO MEASURE ACCURATELY EXERTION TIME

The pilot study described in Appendix A showed the assumption of Bassey and Short (1990) that acceleration was constant and hence velocity was linear throughout the exertion phase during a thrust against an inertial load, was not true. This finding showed that if average power was to be measured accurately and if measures of other variables were to be obtained during an exertion against an inertial load, then a system was required which would allow sufficient resolution to detect, almost instantaneously, the motion of the flywheel during the exertion period.

1.2.i System description

In this section a system is described that has practical advantages over strain gauge based systems. A rotary encoder was used to monitor the rotation of a flywheel, which was sometimes accelerated to velocities in excess of 480 rpm within 200ms. From a knowledge of the rotational properties and inertia of the flywheel, several features of limb thrust may be inferred without additional strain gauge or goniometer attachments. The rotary encoder has the added advantage that it is factory calibrated and unlike custom built strain gauge systems is not subject to frequent calibration requirements and thermal drift. The system is described here installed in a modification of the muscle testing system described by Bassey,and Short (1990).



Cam with constant leverage between pedal axis and wheel axis

Figure 1.4 Subject positioning on modified power testing apparatus

The rotary encoder (Hengstler RI58 0/360AS.41RB) which gave a digital pulse for every degree of flywheel rotation was mounted on one end of the flywheel axle via a flexible coupling (Hengstler 1.076.014). The rotary encoder signals were in quadrature and had to be converted to a TTL compatible signal in order to be recognised by the A/D system. A circuit was designed to convert the rotary encoder pulses to TTL compatible signals (see Appendix B).

The rotary encoder pulses were sampled on the falling edge of each pulse by the A/D system (CED micro1401), this allowed a cumulative time to be recorded for the movement of the flywheel during the exertion period with an event resolution of $2\mu s$. The time – displacement data were collected by Spike 2 software (CED Ltd), the data were then converted to a text file by means of a purpose written script within Spike 2. The text file was then imported into a software programme (Mathcad ver 2001 Mathsoft Inc) for analysis. Details of the Spike 2 scripts and Mathcad programme used are given in Appendix C. Simply the Mathcad programme performed first and second order differentiation on the time – displacement data in order to calculate velocity and acceleration respectively. Power output was then calculated from the product of velocity and torque. Figure 1.5 shows an example of power output during the exertion phase on the NPR using the rotary encoder system (see section 1.4 – design of variable inertial system for system diagram).

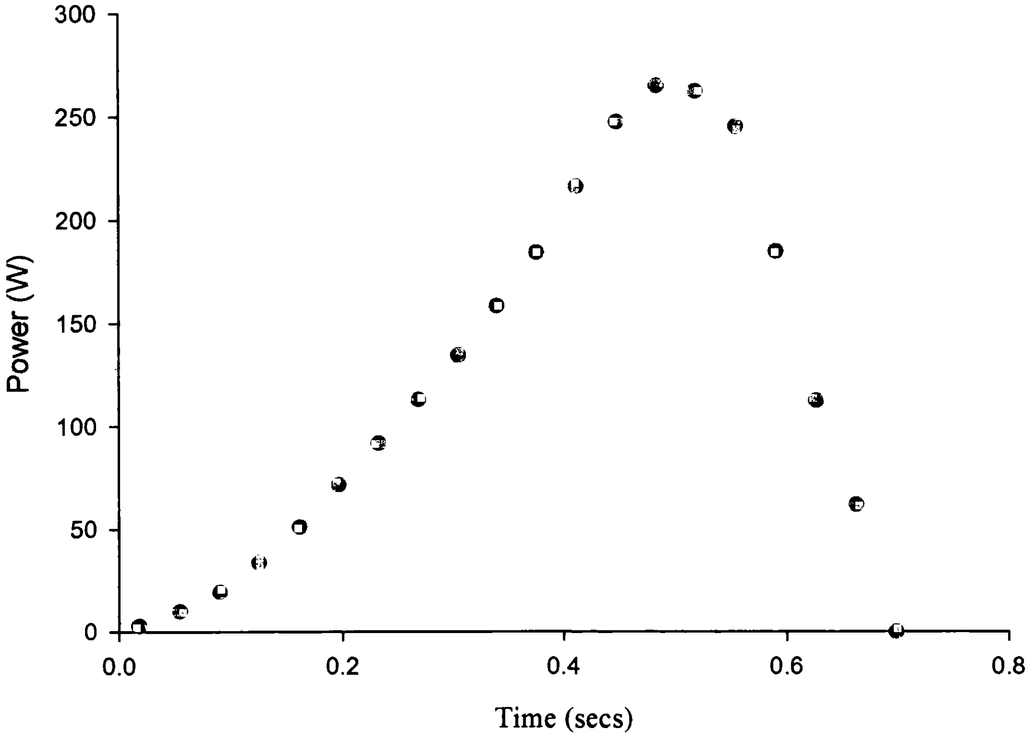


Figure 1.5 A typical trace of the power output profile during an exertion on the modified NPR using the rotary encoder system

1.3 MEASUREMENT AND VALIDATION OF TORQUE DETERMINATION METHOD

The rotary encoder gave a high resolution cumulative record of time-displacement data; hence it was possible to calculate a number of mechanical parameters as force was applied to the rotating flywheel. Therefore as opposed to the NPR (Bassey and Short 1990), whereby only a measure of average power was possible due to not having the continuous time history, the rotary encoder allowed a history of all the mechanical factors of interest throughout the exertion period.

1.3.i Treatment of time-displacement data – calculated torque

In order to determine the torque applied to the flywheel the time-displacement data from the shaft encoder was utilised. In an inertial system acceleration is proportional to the applied torque, and the calculated torque is equal to the product of angular acceleration and the flywheel inertia (for details on the calculation of the flywheel inertia see section 1.4 p 83). In order to determine the flywheel acceleration second order differentiation was applied to the time-displacement data. The use of second order differential techniques tends to amplify any noise in the signals. In order to improve the interpretation of the records a fitting technique was employed, this involved dividing the time - displacement record into a set number of intervals (usually 20) which allowed sufficient resolution to identify physiological events, whilst reducing the noise present. The intervals were then scanned for data points; these data were then fitted with a second order polynomial. The coefficients of the polynomials for each interval were first and second order differentiated which gave velocity and acceleration values respectively. These data were applied to the mid point of each interval time for subsequent plotting (see Appendix C).

Having calculated the acceleration and knowing the system inertia and losses, the torque could be calculated: -

$$\text{Torque} = (I \bullet \alpha) + \text{losses}$$

The losses include torque required to extend the return spring and the frictional losses. These are explained in section 1.4.

1.3.ii Measured torque

In order to directly measure the applied torque and compare this with the calculated method of determining torque, a strain gauge was mounted in series with the flywheel driving chain. When the system is mounted in the apparatus described by Bassey and Short (1990), the chain is connected to drive pedals via a modified cam system (see Figure 1.4) The original NPR utilised a lever system which increased the mechanical advantage throughout the exertion phase, whereas, the cam system used here was designed to maintain a constant leverage between pedal axis and flywheel axis. The strain gauge system consisted of a full Wheatstone bridge configuration to allow maximum sensitivity to loading. The four foil strain gauges (RS 632-124) were mounted on a steel ring (Figure 1.6), which had been heat-treated to improve response. A ring mounting was chosen as this design gives the least noise from lateral forces (torsion).

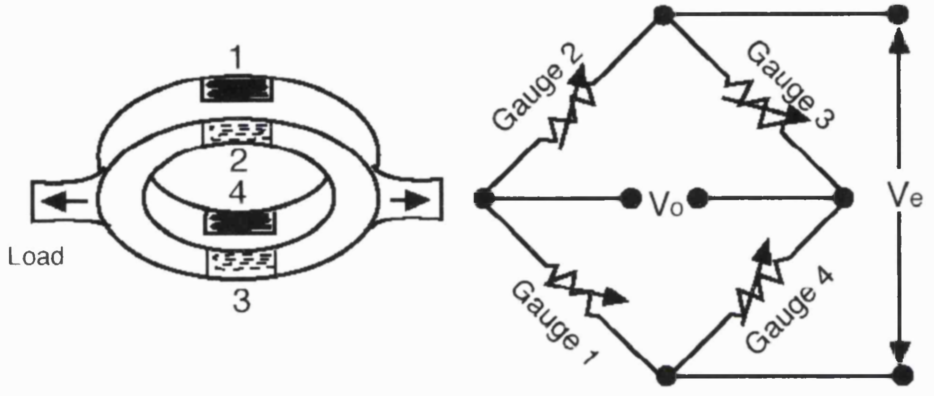


Figure 1.6 Strain gauge system utilising a full Wheatstone bridge circuit

The analogue signal from the strain gauge was amplified via a purpose built strain gauge amplifier (see appendix D). The digital signal from the rotary encoder was converted from quadrature pulses to a TTL compatible signal. Both analogue and digital signals are then sampled by an A/D converter (CED micro1401). The analogue channel was sampled at 2.5 kHz; the digital channel which is set to detect the falling edge of pulses has an event resolution of $2\mu s$. A PC, utilising Spike 2 software (CED Ltd), logged the sampled data. The analysis was carried out by utilising a mathematical software package, (Mathcad Ver 7), (see Appendix C for details of calculations). The data were fitted using first and second order polynomial techniques to allow power, velocity, acceleration, work done and torque data to be calculated. Maximal values, as well as averages, can be obtained from the data.

1.3.iii Strain gauge response

In order to examine the frequency response of the strain gauge system, the strain gauge was connected to a puller motor via a stiff spring (Ling motor ser n^0 S75 type 106 Ling dynamic systems, Royston, Herts.). In parallel with this was attached a linear potentiometer. The potentiometer allowed the displacement to be monitored and was assumed to have an instantaneous response. The puller motor on energising, gave a single small step displacement, the movement induced oscillations into the system via the spring, and these were sinusoidal in nature.

The signals from both the strain gauge and the potentiometer were fed into a PC via an A/D converter; the signals were sampled at 2.5 kHz. In order to analyse the signals a programme was written in data acquisition and analysis software (Testpoint Ver 3, Keithley Instruments Ltd). The software allowed the displacement signal to be adjusted in amplitude until it matched that of the strain gauge signal. A damped version of the displacement signal was generated by varying the frequency until it matched that of the strain gauge signal. The amount of adjustment required in order to overlay the signals gave a measure of the strain gauge signal response. Using the formula below it was found that the strain gauge had a time constant of 4.5 ms.

$$TC = \frac{1}{2 \bullet \pi \bullet F}$$

Where TC is the time constant (ms), and F is the frequency response (Hz).
By using the time constant in the analysis software (Mathcad 2001, Mathsoft) the strain gauge time lag was corrected for by using a deconvolution method, which adds to the measured strain a product of its first time derivative and the time constant (see Appendix C).

The strain gauge was calibrated for load by using a series of known masses. The masses were suspended from the strain gauge and the voltage output measured. A plot of mass against volts described the linearity of the strain gauge system (Figure 1.7).

A possible source of error inherent in strain gauge systems is thermal drift. This occurs due to a low leakage current passing through the strain gauge system whilst the system is turned on; the heating of the strain gauge system can cause a change in the resistance and hence effect the strain gauge calibration. In order to check this effect the system was switched on and calibrated at 9.00AM, left switched on and calibrated again some 10 hours later (PM). Figure 1.7 below represents both the AM calibration, and the PM calibration.

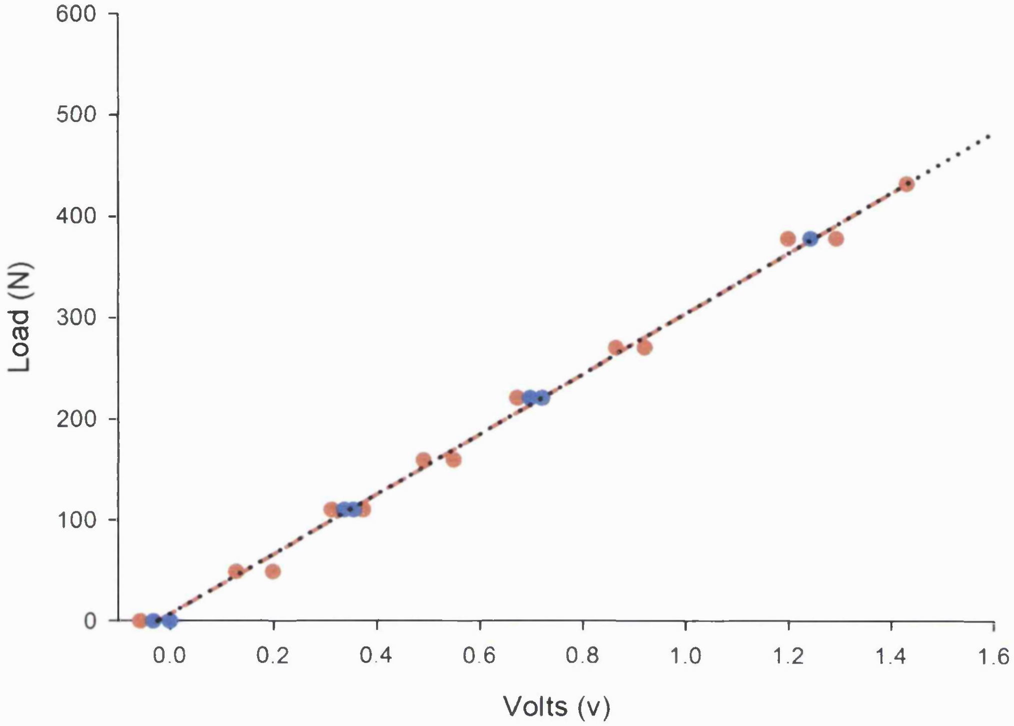


Figure 1.7. Strain gauge calibration-AM (●) and PM (●). Regression line fit AM ($y = 7 + 296x$, $r^2 = 0.994$), PM ($y = 6 + 300x$, $r^2 = 0.999$)

1.3.iv Comparison of calculated and measured torque

In order to compare the calculated torque determined from the rotational measurement of the flywheel displacement with the measured torque determined using the strain gauge system, the torque produced during an exertion was simultaneously measured and calculated. Both methods were then overlaid on the same plot. A good general agreement was found between the two methods of determining torque (see Figure 1.8 and 1.9). No statistical difference was observed between the two methods and it was decided that in all subsequent recordings the rotary encoder alone could be used to determine the mechanical characteristics of the applied exertions.

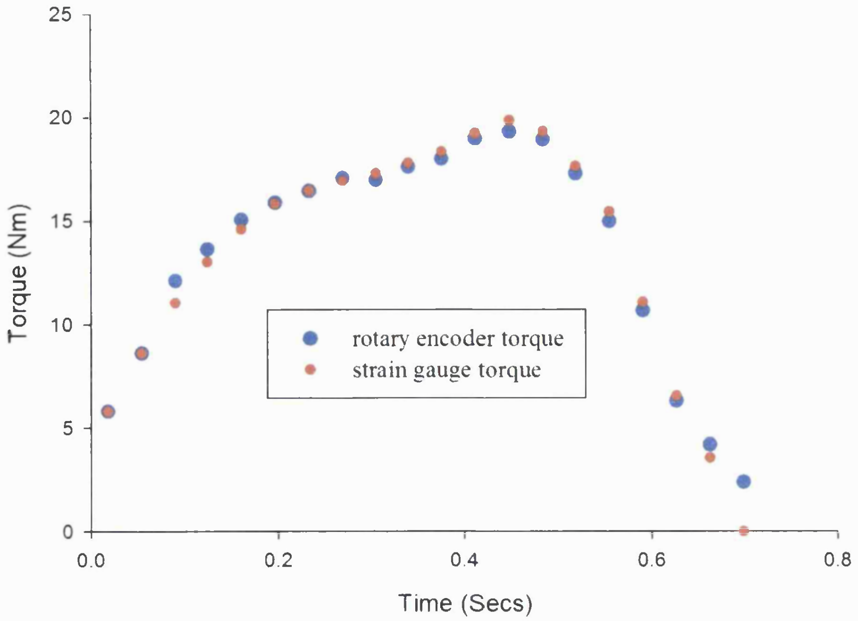


Figure 1.8. The torque applied to the flywheel during a typical exertion on the NPR using both strain gauge and rotary encoder methods.

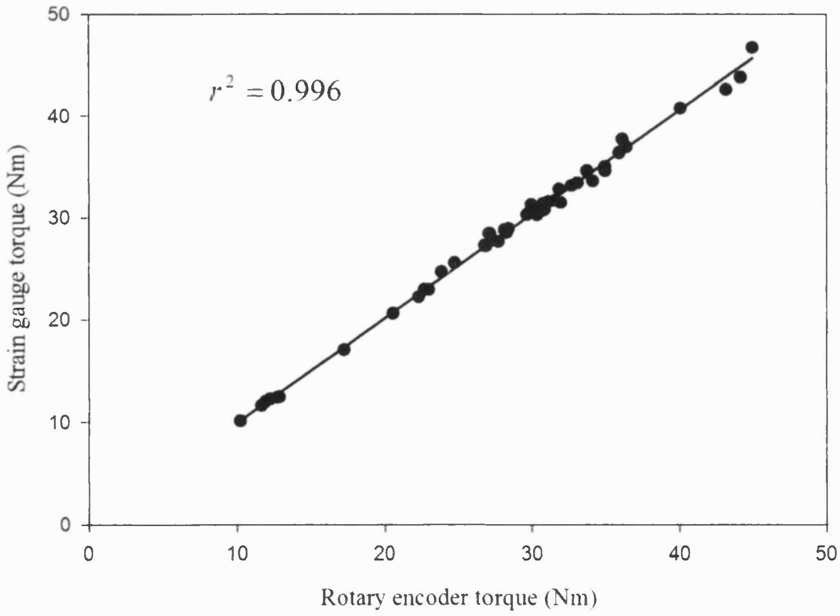
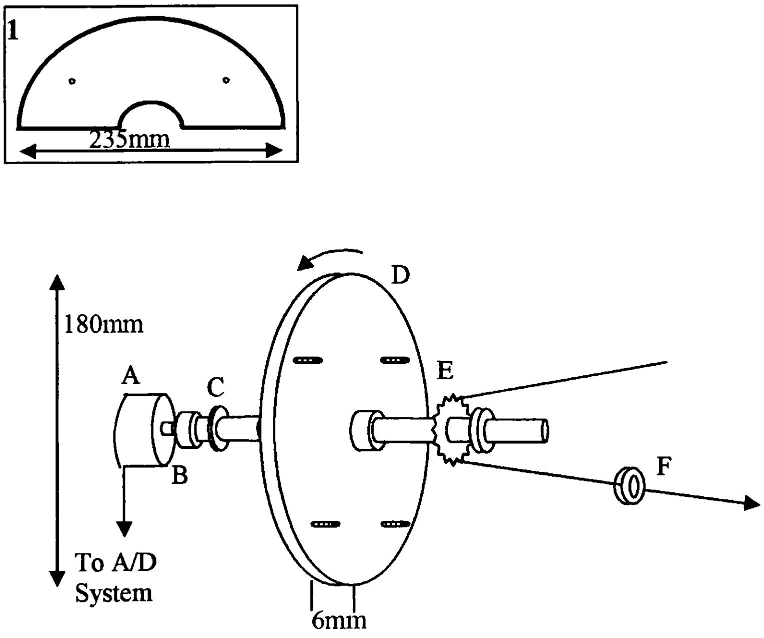


Figure 1.9. Relationship between the measured torque (strain gauge) and calculated torque (shaft encoder) for an arbitrary number of exertions made by a single subject across a range of inertias (0.024 – 0.69 Kg m^2).

1.4 DESIGN OF THE VARIABLE INERTIAL SYSTEM

As was suggested in the introduction, a fixed inertial load may not allow each subject to express their optimal power output. Therefore to test this hypothesis a variable inertial loading system was designed and constructed. The inertial flywheel system is shown in Figure 1. 10. A lightweight aluminium disc (6mm thick, 180mm diameter), with moment of inertia (MI), $0.024\,\mathrm{Kg\,m^2}$, was mounted centrally via two low friction self-aligning bearings onto a steel shaft. Semicircular steel plates (2mm thick, 235mm diameter), which could be mounted on the disc in pairs, were designed to act as inertial loading plates (Figure 1.10 inset). The shaft on one end also held a driving cog (60mm diameter) which had an inbuilt freewheeling mechanism. On the other end of the shaft a rotary encoder was mounted via a flexible coupling. The whole assembly was mounted in the NPR via specially built bearing cases which accept the self aligning bearings. The following section details the calibration procedures required in order to determine the inertia of the system. The system losses are then outlined and determined. Finally examples of exertion variables are shown using the modified system. The data on two separate occasions by 7 subjects was determined.



A. Rotary encoder

B. Flexible coupling

C. Self aligning low friction bearings

D. Aluminium flywheel

E. Freewheel mechanism

F. Strain gauge

Figure 1.10 Diagram showing variable inertial plates (1-inset) and inertial flywheel system complete with rotary encoder

1.4.i Determination of flywheel inertia – the drop test

If a mass is attached to a flywheel free to rotate and the mass is allowed to fall from a given height (Figure 1.11), the potential energy in the system, according to the conservation of energy theory, is converted into kinetic energy plus the energy due to friction in the flywheel bearings and the energy due to the translation of the mass. The potential energy is converted into translation energy during the fall of the mass and rotational kinetic energy in the form of the rotating flywheel. There is also a small amount of energy converted to heat due to friction in the system. The above statement can be summarised by the following equation: -

$$M \bullet g \bullet S = \tfrac{1}{2} \bullet I \bullet \omega^2 + W_f + \tfrac{1}{2} \bullet M \bullet v^2 \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \text{①}$$

Where M is the mass of the falling object, g is the acceleration due to gravity, S is the distance through which the mass falls, I is the flywheel inertia, ω is the angular velocity of the flywheel, W_f is the energy due to friction and v is the velocity of the falling mass.

Equation ① contains a term for the inertia of the flywheel, if the other terms are known; the equation can be rearranged to allow the inertia of the flywheel to be found. This forms the basis of the drop test.

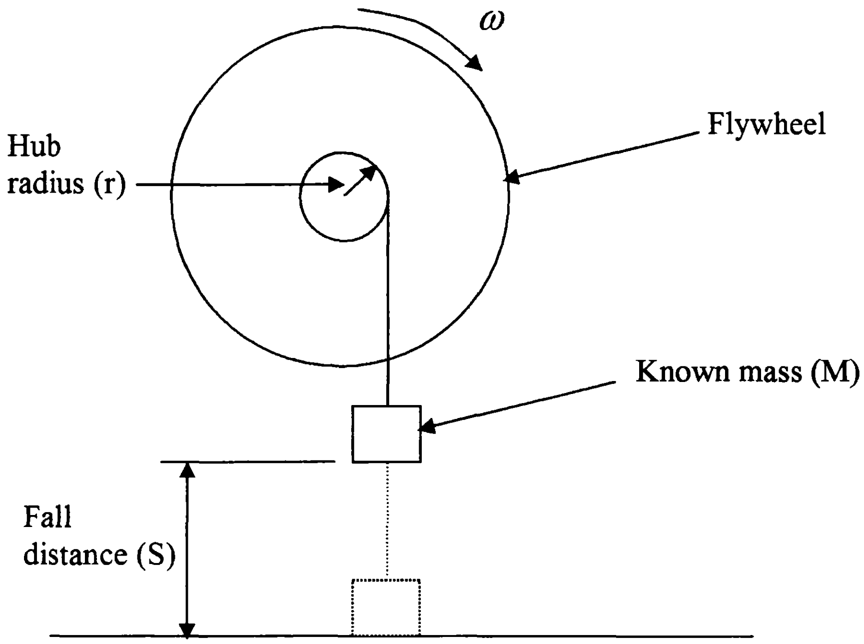


Figure 1.11 Diagrammatic representation of the drop test

The inertia of the flywheel and subsequent loadings with the semicircular steel plates was calculated by the method above (see Appendix E). The frictional component was found to have two components these were small in comparison to the inertial components. The first frictional component was due to the loading on the bearings as a product of the inertial loading, this was expressed as a constant torque opposing motion, and was calculated at the same time as the inertial component. The second frictional component was found to be a product of the torque applied to the flywheel system, which was affected by the thrust on the bearing surfaces.

Calculation of system losses

1.4.ii Frictional components

The frictional components were determined when the flywheel system was situated in the Nottingham Power Rig (Bassey and Short 1990).

1) Constant frictional component

Here the frictional loss is calculated knowing the inertia and the flywheel velocity. See Appendix E for details. The frictional loss can be determined using equation ⑦: -

$$W'f = \frac{n_1}{n_2} \bullet \frac{1}{2} \bullet I \bullet \omega^2$$

The frictional component was calculated in this way for all inertial loads

2) <u>Bearing thrust frictional component</u>

The method used in order to determine the friction as a result of bearing thrust was a scale drawing technique using force vectors (see Figure 1.12). Here all the force components applied to the flywheel were resolved with their angle of application. The force due to the mass of the flywheel and the return spring was included. The force vectors were drawn to scale and the resultant bearing thrust was calculated. This was compared with a measured value from a strain gauge mounted in the flywheel driving chain. The strain gauge was mounted so that all the applied forces, including the frictional and return spring forces would be detected. The above procedure was carried out over the range of inertial loads. A plot of bearing thrust vs. braking torque allowed a relationship to be determined (see Figure 1.13), this multiplication factor was utilised in the calculation of torque using the rotary encoder.

In an inertial system, the applied torque is proportional to the acceleration. The component of applied torque was calculated from the product of flywheel inertia and rotary acceleration. The torque value was then resolved into a force value by dividing by the effective lever arm (freewheel cog radius).

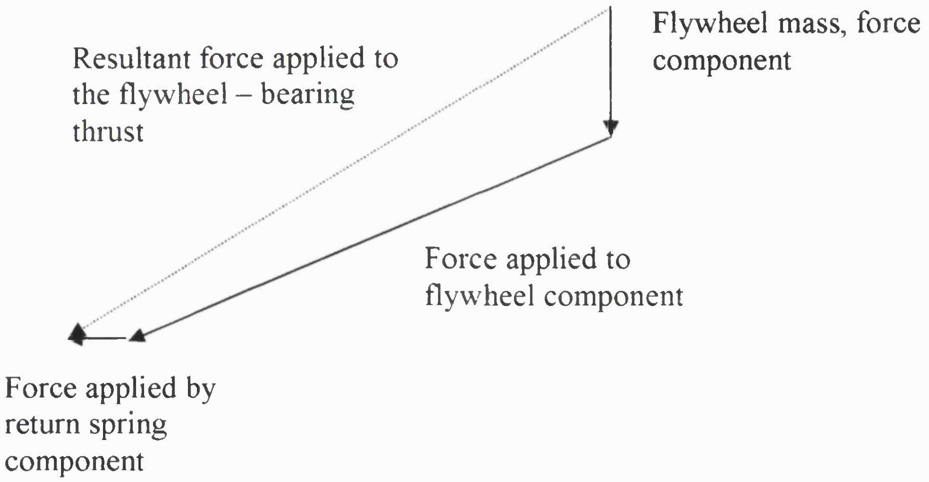


Figure 1.12. Schematic diagram showing determination of frictional force during application of force to flywheel.

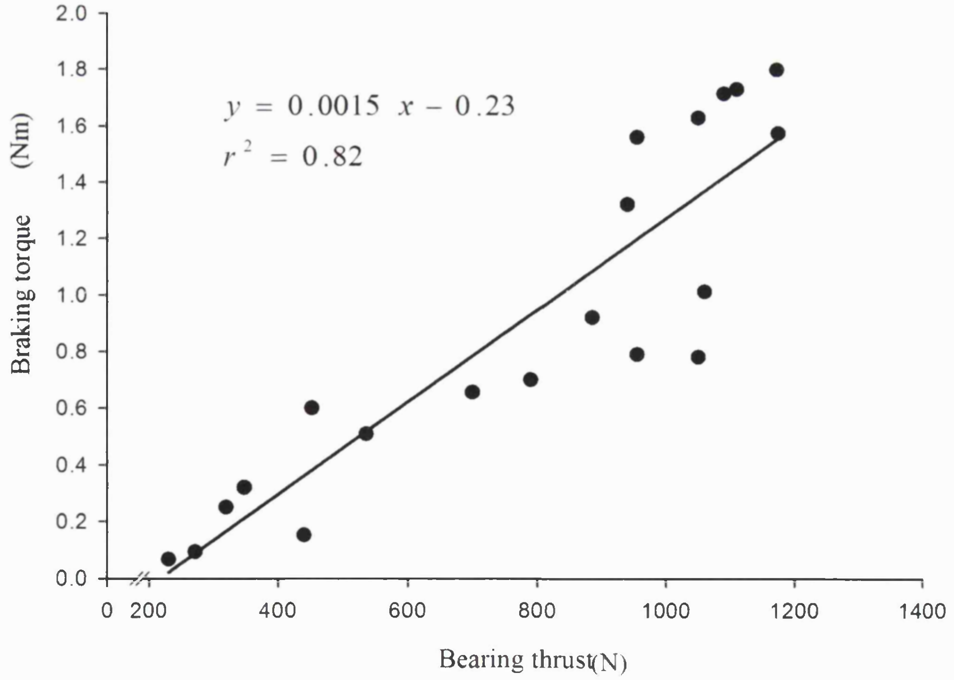


Figure 1.13 Scatter plot showing relationship between the total applied forces to the bearings (bearing thrust) and the subsequent frictional component (braking torque)

The force applied by the return spring was calculated from the product of the spring stiffness and the distance moved.

The values of bearing thrust were plotted against the braking torque (see Figure 1.13). Braking torque was determined by determining the difference between the values of torque as measured by the strain gauge and the values of calculated torque.

$$\text{Braking torque} = \text{Strain gauge torque} - (I \bullet \alpha) + (k \bullet x) + (\frac{n_1}{n_2} \bullet W' f)$$

A line was fitted in order to determine an equation which best described the relationship between the two variables (braking torque). The gradient value was used in subsequent calculations of applied torque to determine the frictional component as a result of bearing thrust.

Spring component

Calibration of the return spring was carried out by suspending the spring and attaching a series of known masses from the spring. The displacement of the spring was recorded, a plot of the spring displacement against mass allowed a line to be fitted, the gradient of which represented the spring stiffness: -

$$k = {}^{F}\!/_{x}$$

Where k is spring stiffness, F is the applied force (N) and x is the displacement (m). The typical losses for the system under conditions of both high and low inertial loading can be seen in Table 1.2

Inertia ($Kg \bullet m^2$)	Losses (J)	% Work Due to Losses	Total Work (J)
Low (0.024)	7.5	15.4	48.6
High (0.54)	16.9	9.8	173.3

Table 1.2 Typical loss values. Total work is calculated from strain gauge values i.e. inclusive of losses.

1.4.iii Exertion examples using the above system with high and low inertial loading

Typical values collected for torque, velocity, acceleration, work done and power obtained from a single subject, at a low ($0.024\ Kg\ m^2$) and a high ($0.543\ Kg\ m^2$) inertia are shown in Figure 1.14. With the larger inertia considerably greater torque is applied to the flywheel, which in this instance despite the slower angular velocity results in a greater peak and average power generation when compared with the low inertia.

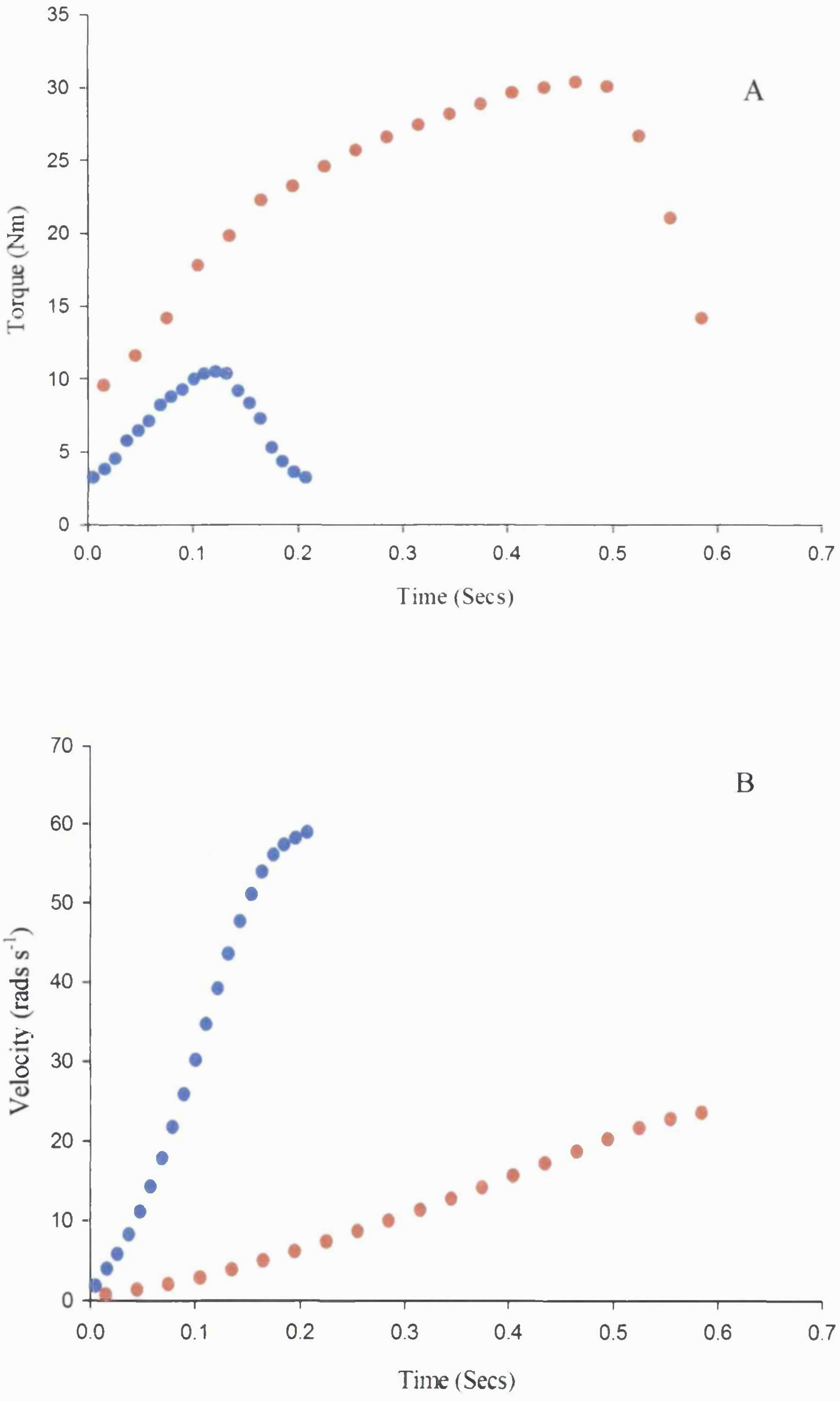


Figure 1.14 (see page 92 for legend). A) Torque B) Angular Velocity

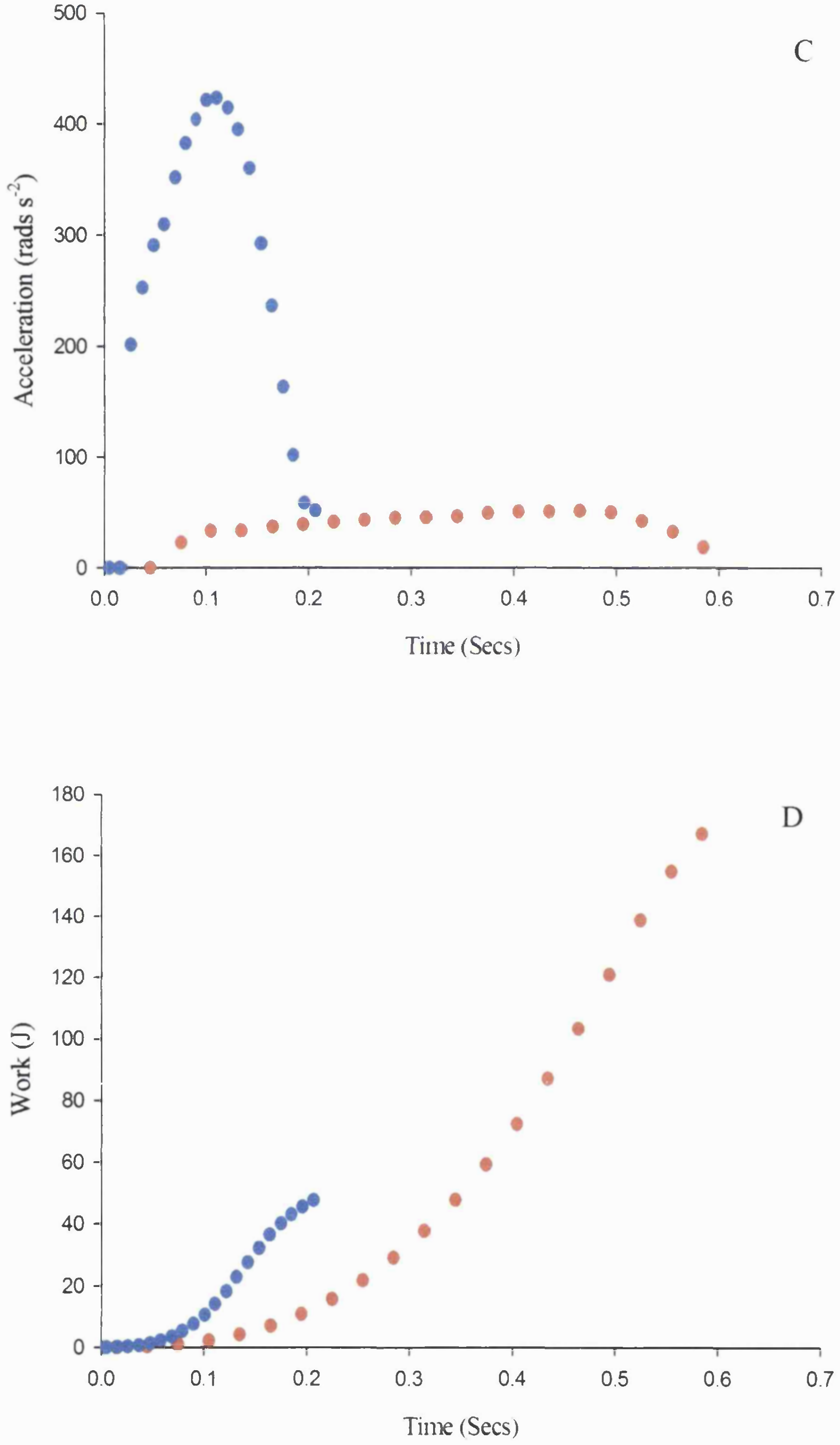


Figure 1.14 (see page 92 for legend). C) Angular Acceleration D) Work done

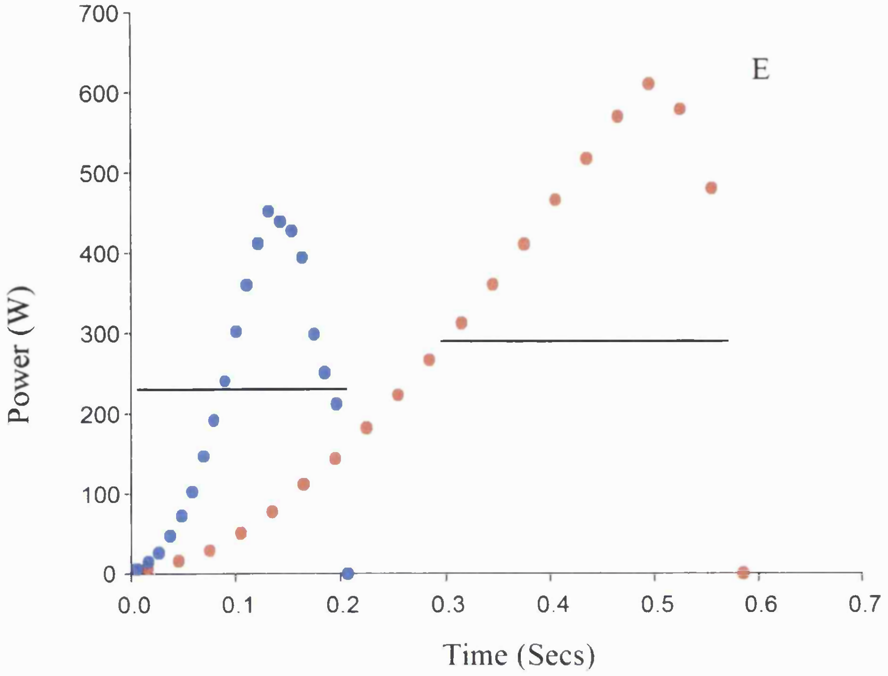


Figure 1.14 Results for one subject of a typical exertion against a high (●) and low (●) inertia. Time of exertion is 0.58 and 0.21 seconds for the high and low inertia respectively. Approximate maximal pedal velocities for these examples based on a pedal travel of ~ 85^0 for a wheel movement of 396^0 are 5.11 rads s^{-1} (49 rpm) and 13.48 rads s^{-1} (128 rpm) for the high and low inertia respectively. Example of A - E) Torque, Velocity, Acceleration, Work done and Power during a single exertion Calculated mean power is shown in graph E

(—). All values are at the flywheel.

Data collection - reproducibility

The repeatability within subjects using the device was examined on a group of seven male subjects. The physical characteristics, mean (SD), of the subjects were; age (years) 25.8 (5.8), height (m) 1.8 (0.1), weight (kg) 76.3 (6.5). The subjects each performed 4 contractions at 7 different inertial loads, range 0.024 – 0.405 Kg m^2 on two separate occasions. The best value at each inertia for each subject was used for the purposes of analysis of average and peak power. Subject testing was separated by between two and four days. A standardised 5 minute warm up (cycling 50W/60rpm) was performed before all testing procedures. The experimental data collection was carried out with the approval of the Royal Free Hospital Ethics Committee. Written informed consent was obtained from all subjects.

All exertions of the lower limb were carried out with the subjects seated in the modified Nottingham Power Rig (see Figure 1.4). Briefly the tests consisted of a single lower limb thrust with the preferred limb, the subject being seated in an upright position in the dynamometer with the knee flexed. The dynamometer has a very low backrest (10cm), to minimise the possibility of utilising the back extensor muscles in the exertion. The seat position was adjusted to ensure that the lower limb of the subject finished just short of full extension at the end of the exertion. A seat belt was attached across the hips to secure the subject into the seat, whilst performing a single maximal lower limb thrust against the pedal. The pedal was attached to the flywheel via a chain drive (see Figure 1.10). The subjects were actively encouraged to give their maximum effort.

The data obtained on seven young subjects is summarised in Figure 1.15. Maximum values for both average and peak power for this group of subjects occurred at either 0.09 or 0.22 Kg m^2 (ranging from 318 – 448Watts for average power and 608 – 965 Watts for peak power). The coefficient of variation for average power and peak power generated during the leg thrust ranged from 1.8% - 3.0% and 2.3% - 5.9% respectively. A student paired two-tailed t-test revealed no significant difference ($P=0.09$ and $P = 0.25$), between Test 1 and Test 2 for both average and peak power respectively Similar coefficients of variation were observed for the mean and peak torques to those shown for power.

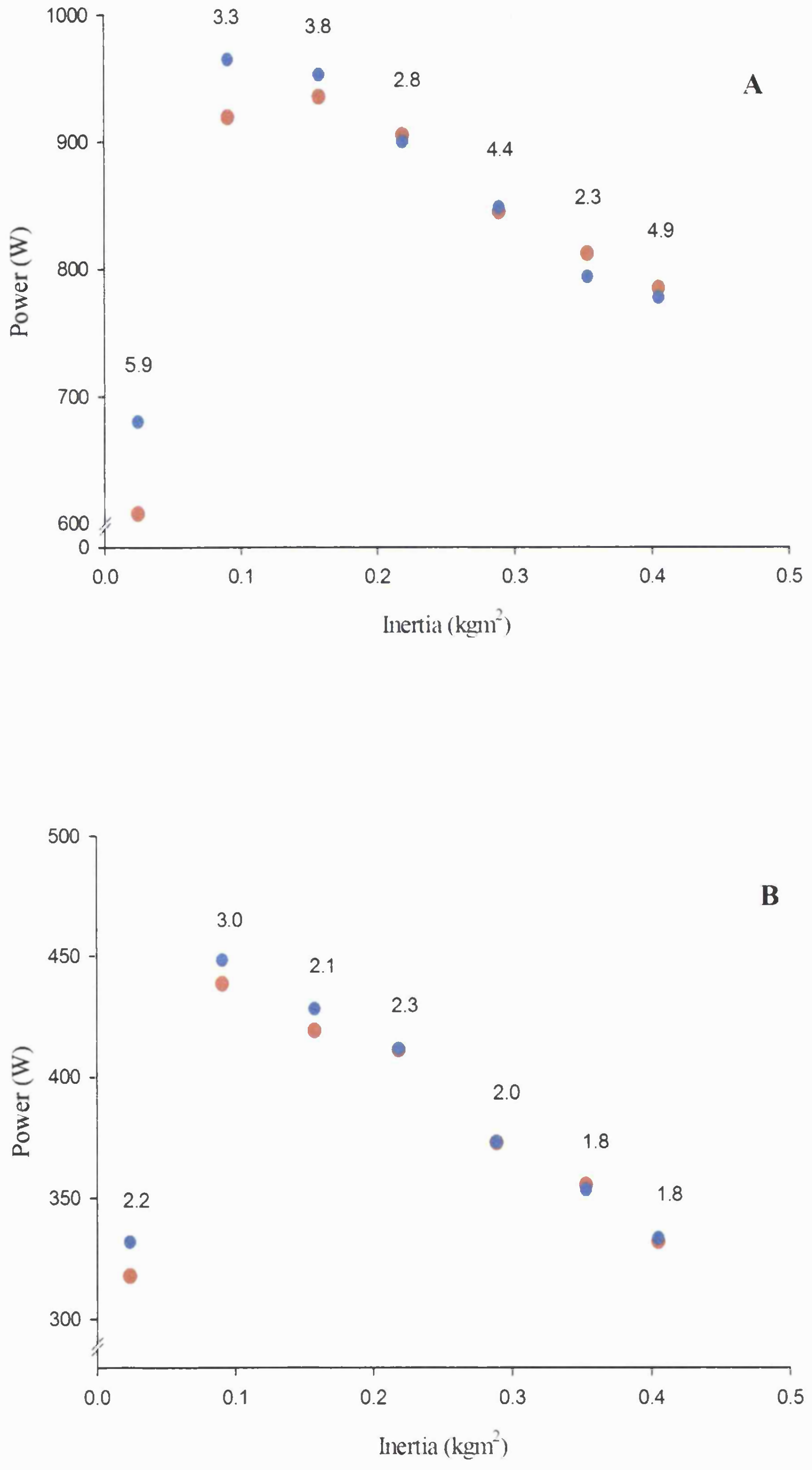


Figure 1.15 (see page 96 for legend). A) Peak power B) Average power

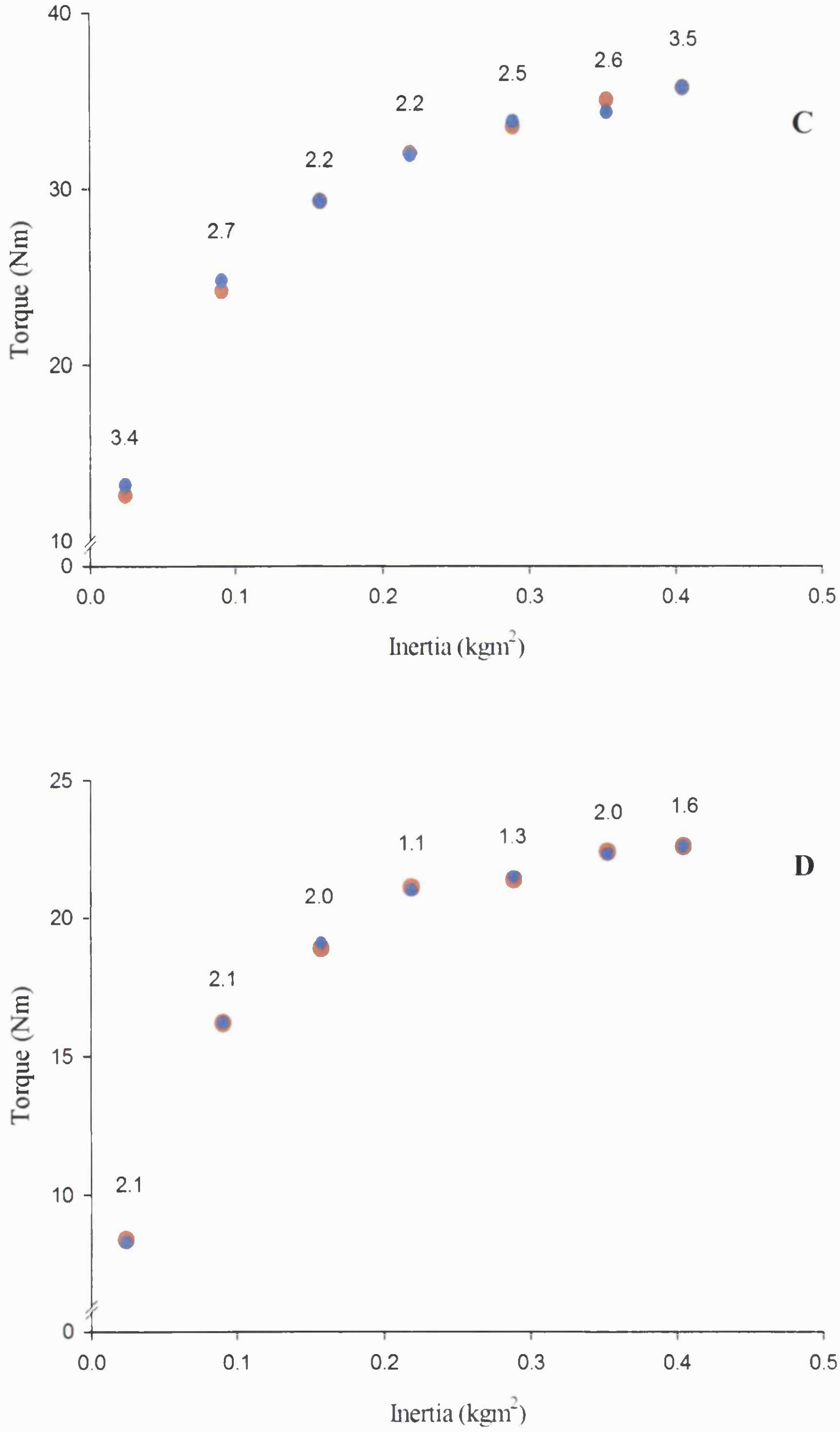


Figure 1.15 Reproducibility plots showing coefficient of variation for two separate tests at seven inertial loads (n = 7). A – D Peak power, Average power, Peak torque, Average torque. (test 1(●), test 2 (●))

1.5 SUMMARY

Developments in computing and data acquisition have allowed the development of a system with high resolution, which allows the measurement of contraction time directly, without making assumptions regarding the acceleration (see Figure 1.14c). This system allows for the accurate timing of muscle exertion by tracking the rotation of a flywheel, with a resolution of one degree. A strain gauge system was utilised in parallel with the rotary encoder in order to compare the torque applied directly to the flywheel with that calculated from the flywheels rotational properties. When using the encoder system to calculate the torque applied to the flywheel, close agreement with the strain gauge torque values over the range of inertial loads was obtained (see part 1.3, Figures 1.8 - 1.9), indicating that the strain gauge may be dispensed with in future dynamic studies.

Varying the inertia of the flywheel to allow optimisation of the load, through the addition of steel plates allows the inertia at which a subject may express their maximum power, velocity and force to be determined. In the present group of young male subjects the inertias at which maximum peak and average power occurred ranged from 0.09 - $0.22\,\mathrm{Kg\,m^2}$. However, as they were all young male subjects one might expect an even greater range in frail elderly and trained subjects due to the possible differences in the torque – velocity characteristics of their muscle.

Isokinetic devices are limited in their ability to capture data at high speeds (>5.5 rads $\mathrm{sec^{-1}}$). James et al (1994), stated that "it was not routinely possible to record forces above angular velocities of 2.1 rads s^{-1} due to the small forces and large mechanical artefacts". Whereas data collection is not impaired at high speeds of movement for the device presented where angular velocities of up to 40 rads s^{-1} were recorded. This fact allows for the detection of maximal values of power, which occur at high movement speeds. At the lowest inertia, time of contraction was typically 200ms or less (see part 1.4 Figure 1.14), which is probably insufficient for the lower limb muscles to be fully activated.

The values of average power presented in this present study are in general agreement with Bassey and Short (1990), who reported values of average power of 248 ± 67 W in young subjects. Others have reported values for peak and average power that are higher than those values shown in this present study. Martin et al (1997) reported that in young male subjects, the peak power was 2137 ± 101 W during sprint cycling, whereas Sargeant et al (1981), reported values of 840 ± 143 W per crank revolution during isokinetic cycling. These differences could be explained in part by i) the fact that the devices are different by design i.e. cycling *vs.* single leg thrusts in a leg press type action, ii) the loading protocol is different, isokinetic *vs.* inertial and/or iii) the duration of test is different; the exertion times for the device presented here are low (typically <200ms). This could have a profound effect on the power output as shown in the isometric release example (Appendix F).

In conclusion, the system developed in this chapter allows a number of mechanical properties of human muscle to be inferred from the discrete measurement of the rotating properties of a flywheel, through the use of a rotary encoder and high resolution digital data acquisition. The approach has been tested against a strain gauge system and has been shown to allow reproducible measurements of short term power output to be made.

It was observed that at the lowest inertial loads the power output declined markedly. The typical exertion times at the lowest inertial loads were < 200 ms. It might be suggested therefore that part of the reason for the observed decline in power output may be the time allowed to fully activate the muscle. In order to investigate the effect of exertion time on power output an isometric pre release device was designed and constructed. The data presented in this chapter prompt two questions; the first is why there is a significant reduction in power output at the low inertial loads? Is this due to insufficient time in which to accelerate the load or an inability to develop sufficient acceleration in order to develop optimal levels of torque? Secondly, why are the reported power output values lower than those cited in the literature for cycling? This could be in part due to cycling allowing a repeated acceleration of the inertial load. These questions are addressed in the following chapters.

PART II

CHAPTER 2

EFFECT OF ISOMETRIC PRELOADING ON POWER OUTPUT

2.1 INTRODUCTION

It has previously been shown by others using a concentric isokinetic protocol (Thomas et al. 1987, Jensen et al. 1991, Narici et al. 1991, Harridge and White. 1993) that time is a critical factor for torque generation at higher velocities of contraction. In the previous chapter it was shown that when subjects carried out voluntary maximal single leg thrusts using the modified NPR, the ability to generate power declined at both the lower and higher inertial loadings. This was characterised by a parabolic relationship between the peak power and inertial load. The factors responsible for the decline in peak power at both high and low inertias are likely to be different. Unlike isokinetic testing where velocity and hence acceleration is controlled, inertial loading allows the acceleration to vary and hence both the applied torque and the velocity of the flywheel. Under these conditions, the factors that must be considered when power output is to be maximised are those that affect the generation of both torque and velocity. For a given inertial loading, the time over which the load may be accelerated is an important factor in order to achieve an optimal velocity for power production before the end of the movement. Also, as torque is proportional to the applied acceleration, the ability to generate high torques rapidly is an important factor in the generation of power. If the inertial load is very low, high levels of acceleration are needed in order to obtain the torques required to achieve maximum peak power during a given exertion. Hence the combination of a low exertion time (typically <0.2 seconds), and high contraction velocity seen at the lowest inertial loads may have been responsible for the low values of power output. The mechanisms responsible could be i) an inability of the muscle to accelerate the load sufficiently to generate optimal torque, probably due to the inertia of the lower limb ii) insufficient time to allow for the maximal acceleration of the inertial load due to constraints of the movement and/or, iii) incomplete activation of the muscle.

In order to examine the possibility of incomplete activation a device was designed which allowed for a desired pre set isometric torque to be applied prior to release into the dynamic exertion phase. This pre - release phase would permit additional time to allow for more complete muscle activation and/or more complete stretching of the series elastic component, and therefore allow any additional torque to be developed and thus reduce the apparent decline in maximal peak power.

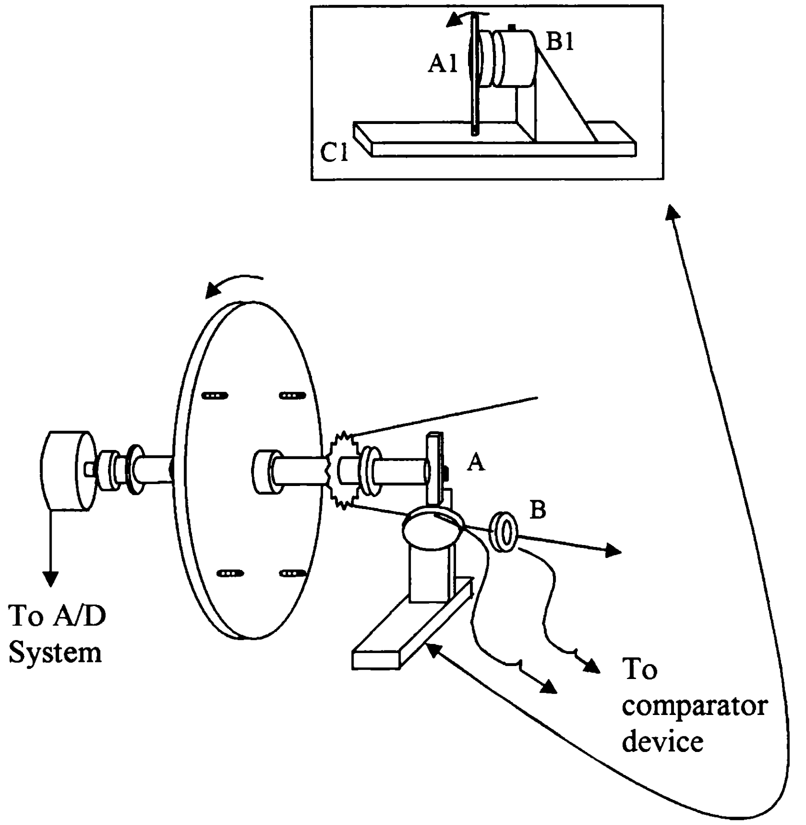
The aim of the experiment described in this chapter was thus to compare the peak power generation of voluntary exertions (no release) with exertions with set levels of pre release torque. The hypothesis being that this would enhance torque/power output at the low inertial loads, but have no effect at higher loads where sufficient time is present to allow for optimal levels of torque generation.

2.2 METHODS

Design of isometric preloading device

Preliminary tests showed that the time allowed for muscle activation during exertions at the lowest inertia loading where time of contraction is very low, typically (< 0.2 secs), may be an important factor in the generation of power output (see Appendix F). Subsequent to this investigation a more substantial experiment was carried out to further elucidate the effects of isometric preloading on power output.

In order to allow more time for complete activation of the muscle prior to shortening, a pre load device was designed which allowed a set level of force to be reached prior to release of the flywheel. The release system comprised of a locking latch mounted on one end of the flywheel shaft. The locking latch is aligned and held in place by a large electromagnet (emessem G MH X 100) which has a holding force of 3000N. This electromagnet is controlled via a comparator device (see Appendix G for circuit details). When a pre selected level of force is reached the electromagnet releases the flywheel. The force is signalled by a strain gauge signal mounted in the chain (see Figure 2.1).



Release device in situ. Is bolted in place to ensure no movement when torque is applied to solenoid

A – Locking latch
B – Strain gauge
A1 – Locking arm
B1 – Magnetic solenoid
C1 – Fixing plate

Figure 2.1 Flywheel system incorporating pre tension device – Inset magnetic solenoid and locking arm. As torque is applied to wheel axle via chain, locking latch (A) which is keyed to axle presses against locking arm (A1) which holds latch, hence wheel in place until release by deenergising magnetic solenoid.

Subjects

Five untrained healthy male subjects were tested, with the approval of the Royal Free Hospital Ethics committee. Written informed consent was obtained from all subjects. The physical characteristics, mean (SD), of the subjects were; age (years) 33.8 ± 4.8, height (cm) 180.4 ± 5.7, weight (kg) 83.4 ± 6.1.

Test protocol

Equipment

All testing was carried out on the modified NPR (see Chapter 1)

Isometric testing

Prior to the voluntary exertions a series of three isometric exertions were carried out at the starting position for the below tests. This was carried out to determine the maximum pre release torque that could be used for each individual.

Voluntary exertions – without pre release torque

The warm up and test procedure has been outlined previously in Chapter 1 (part 1.4). In order to examine the effect of pre release tension on peak power generation three inertial loads were used for these tests: - low inertial load (0.024 kg m^2), the inertial load at which maximal peak power occurred and high inertial load (0.54 kg m^2). Three exertions were carried out at each of the inertial loads with the preferred leg. The exertion at which the maximum peak power was recorded against each inertia was used for analysis.

Voluntary exertions – with pre release torque

A pre release level of torque was set which was equal to the maximal dynamic torque generated by each individual at each of the inertial loads. The rationale for this was that this level of torque would enable the maximum benefit in terms of torque generation prior to release without any further stretching of the series elastic component, as this would result in an additional advantage during the contraction due to the recoil of the series elastic component. However, at the highest inertial load where high levels of dynamic torque could be generated it was not always possible to match these levels of pre release tension, this was likely due to the muscle length tension and anatomical lever arm differences, between the start positions and those at which maximal dynamic torque occurred. In this situation, the maximum torque that the individual could generate at the start position was used. Each subject performed three pre release contractions at each inertial load; the contraction at which peak power was the greatest was used for analysis.

All exertions were separated by at least a 1-minute rest period the order of testing was always isometric, voluntary – without pre release torque and voluntary – with pre release torque. All testing was carried out on the same day. The order of inertial loading was randomised. A 5-minute warm up on an exercise cycle (50 Watts at 60 rpm) was carried out prior to testing.

Analysis of data

For all variables measured, comparisons were made between the voluntary – without pre release torque and voluntary – with pre release torque conditions at each level of inertial loading using a Student paired t test. The alpha value was set to $P = 0.05$. A Bonferroni correction was applied to the t –test alpha levels.

2.3 RESULTS

Torque settings

Table 2.1 shows the isometric release torque for each subject, the maximal dynamic torque at each inertial load and the maximal level of isometric torque possible for each subject at the pre release position. A typical trace of the applied torque with an isometric release and without an isometric release is shown for each inertial load in Figure 2.2.

Voluntary - pre release torque exertions and peak power output

The effect of an isometric pre release on peak power (PP_k) output can be seen in Figure 2.3. The approximate crank position in relation to the flywheel is calculated as, flywheel position/4.6 (similarly for Figure 2.7). In both conditions the PP_k output has a parabolic relationship with the inertial load. At the inertial load of 0.024 kg m^2 (low) the percentage increase in PP_k due to the pre release torque was on average 17%, at 0.16 kg m^2 (medium) and 0.41 kg m^2 (high) there was no significant increase. The increase in PP_k due to the isometric pre release was only significant at the lowest inertial load.

		Inertial load (kg m^{-2})					
		0.024		0.160		0.410	
Subject	Maximal isometric torque	Dynamic maximal torque	Isometric release torque	Dynamic maximal torque	Isometric release torque	Dynamic maximal torque	Isometric release torque
1	23.3	13.4	13.1	27.9	22.1	32.4	20.5
2	35.2	15.1	16.1	34.4	29.0	44.8	31.6
3	25.8	11.8	13.0	29.5	28.3	37.6	29.1
4	31.7	13.2	14.4	26.7	28.5	37.0	29.3
5	28.9	12.7	12.9	28.1	23.9	34.2	24.4

Table 2.1 Maximal isometric torque, isometric pre release values and dynamic maximal torque at each inertial load. Dynamic maximal torque values obtained from the voluntary – without pre release torque exertions. All torque values in Nm

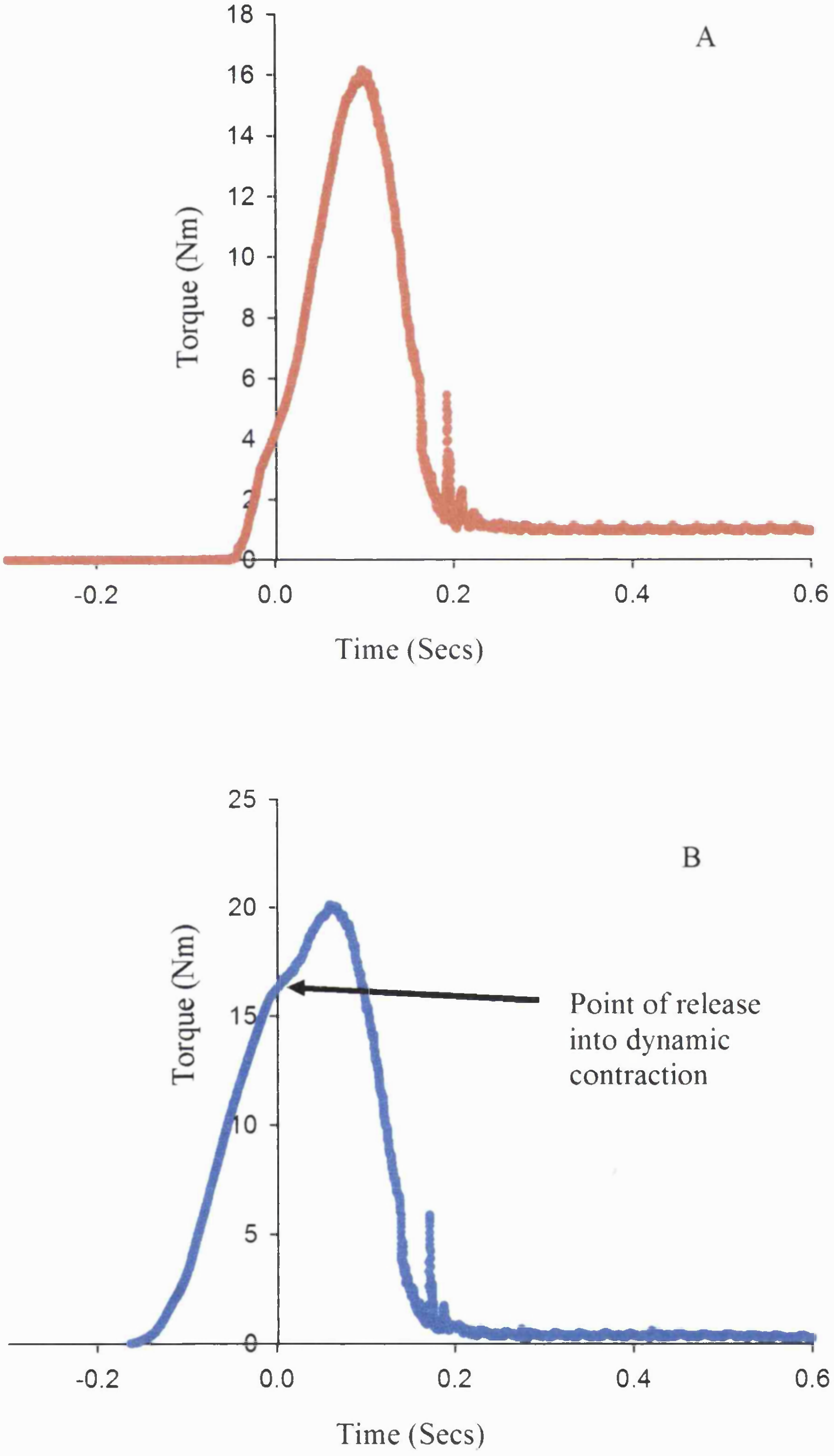


Figure 2.2 Torque generated at 0.024kgm^2 (A, B). No release (🟥) and release (🟦)

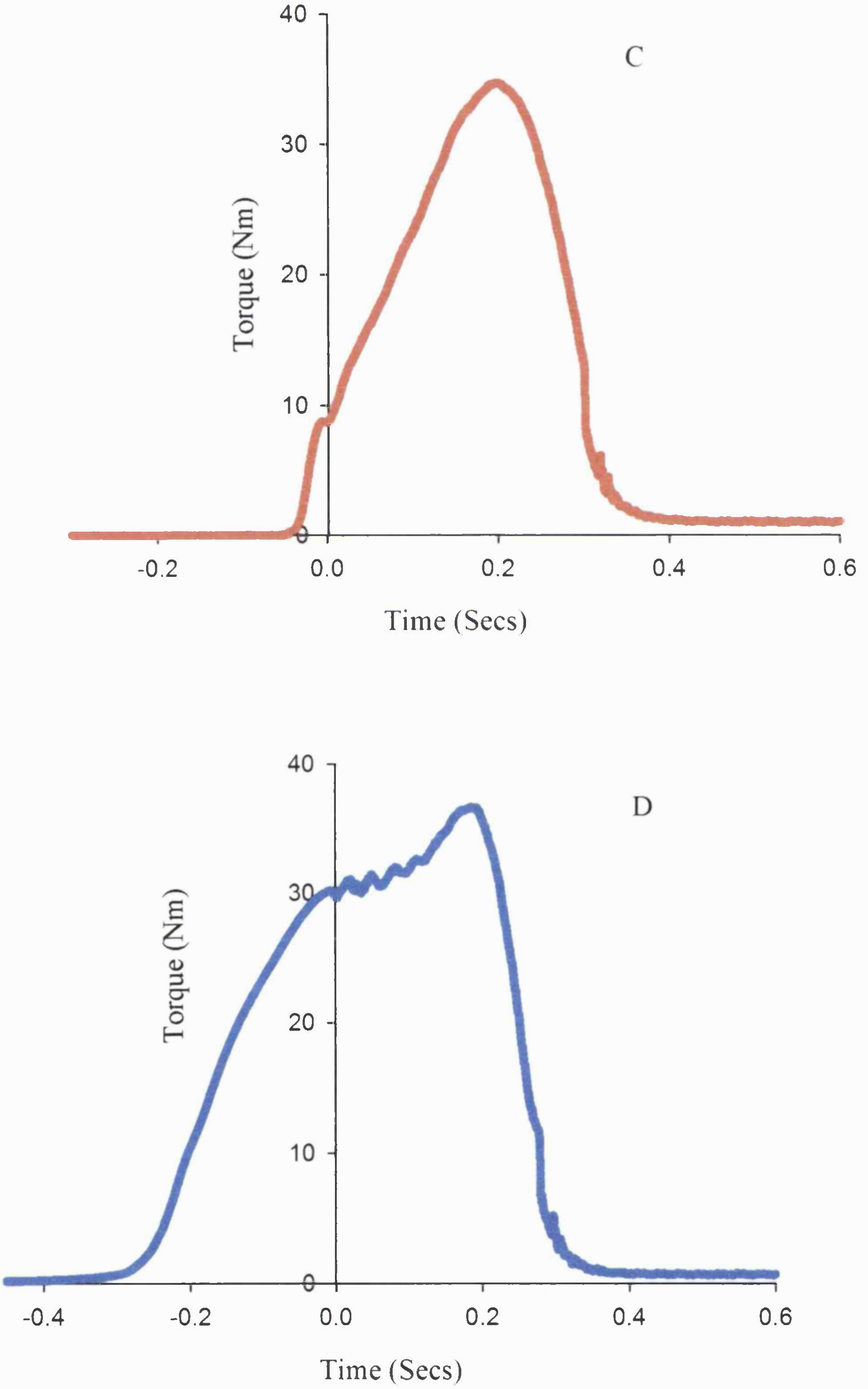


Figure 2.2 Torque generated at 0.16kgm^2 (C, D). No release (▬) and release (▬)

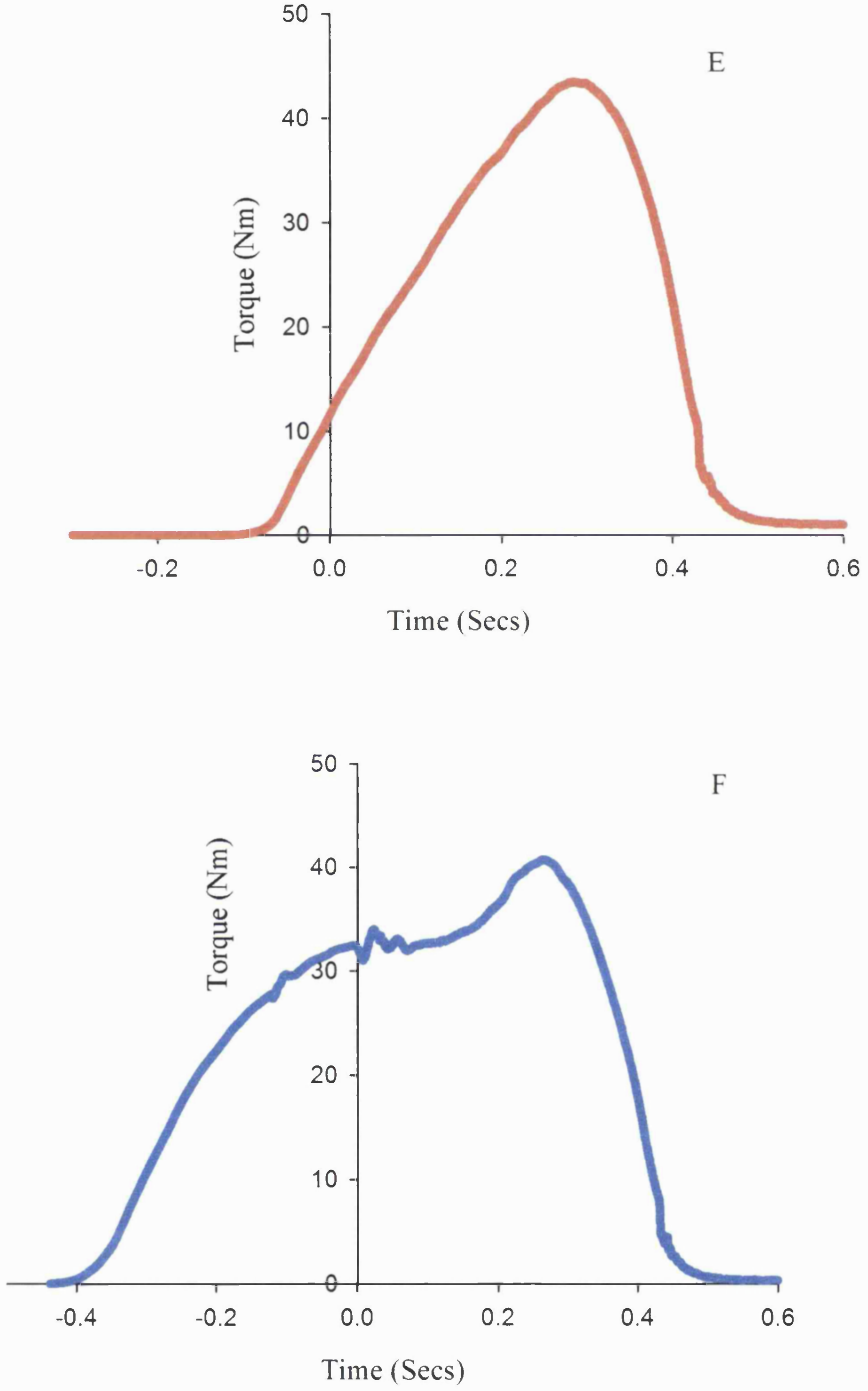


Figure 2.2 Torque generated at 0.41kgm^2 (E, F). No release (🟥) and release (🟦)

The mean pre release times, exertion times and times to generate PP_k are summarised in Table 2.2. The exertion time refers to the time taken to complete the dynamic action. The significant increase in PP_k at the lowest inertia was in spite of the time during the voluntary – pre release torque period being the lowest. The voluntary - pre release time increased with the level of pre release torque set. At the lowest inertial load the percentage increase in total contraction time (voluntary - pre release time + dynamic time period) over voluntary exertions without pre release torque was on average 145%, at the medium inertial load, the percentage increase in time was 190% and at the highest inertial load it was 175%. The time to peak power (Tm_{ppk}), was significantly lower at all inertia loads with the isometric pre release. This reduction in Tm_{ppk} was reflected in the position at which PP_k occurred. The movement of the flywheel during the exertion, which would also indicate a lesser movement of the limbs, was significantly less for both the lowest and medium inertial loads during the pre release contractions (Fig 2.4).

		Inertial load (kg m^2)		
		0.02	0.16	0.41
Pre release time		102 ± 33	336 ± 125	405 ± 146
Exertion time	Without pre release	180 ± 10	330 ± 10*	490 ± 40*
	With Pre release	160 ± 20	290 ± 20	450 ± 40
Time to peak power	Without pre release	130 ± 10†	270 ± 20†	400 ± 50†
	With Pre release	110 ± 10	210 ± 20	350 ± 40

Table 2.2 Mean values (± SD) of isometric pre release time and exertion times under both voluntary - with pre release torque and voluntary – without pre release torque conditions. * Significant difference between release and no release condition with respect to exertion time. † Significant difference in time to peak power between release and no release condition. N = 5. Time in milliseconds.

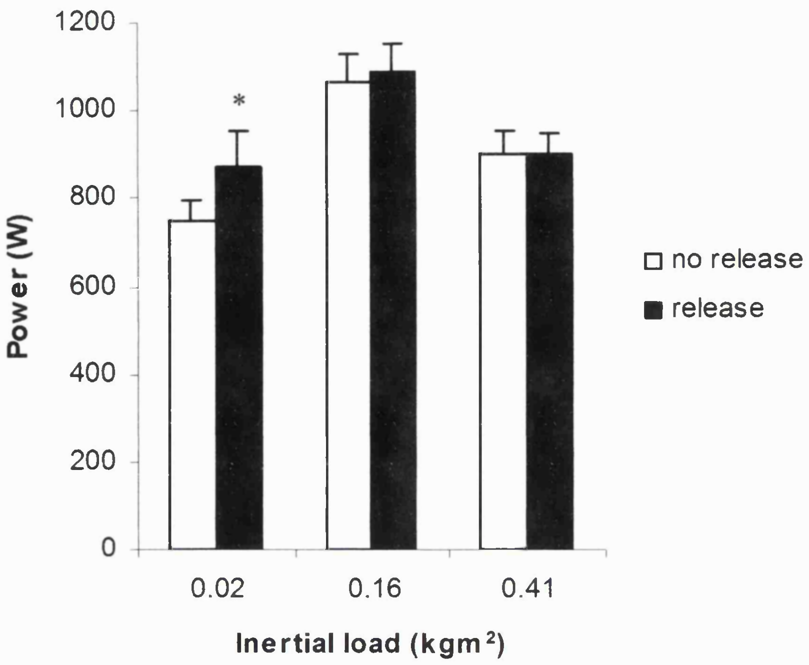


Figure 2.3 Comparison of mean values of peak power between no isometric release and isometric pre release conditions. * Significantly greater than no release condition. N = 5. Error bars ± SE

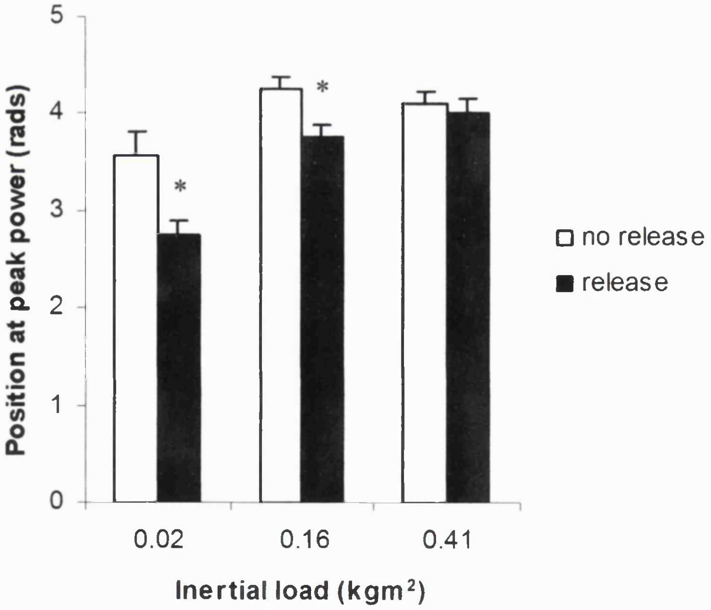


Figure 2.4 Flywheel position at peak power, relative to start. Rads of movement refer to the flywheel movement. * Significant difference between isometric pre release and without pre release conditions. N = 5. Error bars ± SE

Isometric pre release and the components of peak power (torque and velocity)

Torque at peak power

The mean values of the torque generated at peak power (Tq_{ppk}) are shown in Figure 2.5-A. There is an increase in the torque generated as the inertial load increases for both the release and no release conditions. An increase in Tq_{ppk} of 18% due to the isometric pre release is seen at the low inertial load; only at the lowest inertia is the increase in Tq_{ppk} significant ($P = 0.03$). It can also be seen that the relative position of the lower limb at Tq_{ppk} during the isometric pre release exertion is different, with the Tq_{ppk} occurring earlier in the movement at all inertial loads (Figure 2.4) and is significantly different from the no release position for the lowest and medium inertial loads.

Velocity at peak power

The mean values of instantaneous velocity at peak power (V_{ppk}) can be seen in Figure 2.5-B. The effect of the isometric pre release on velocity can be seen to be minimal; there are no significant differences between the two conditions at any inertial load. As the inertial load increases, the V_{ppk} declines.

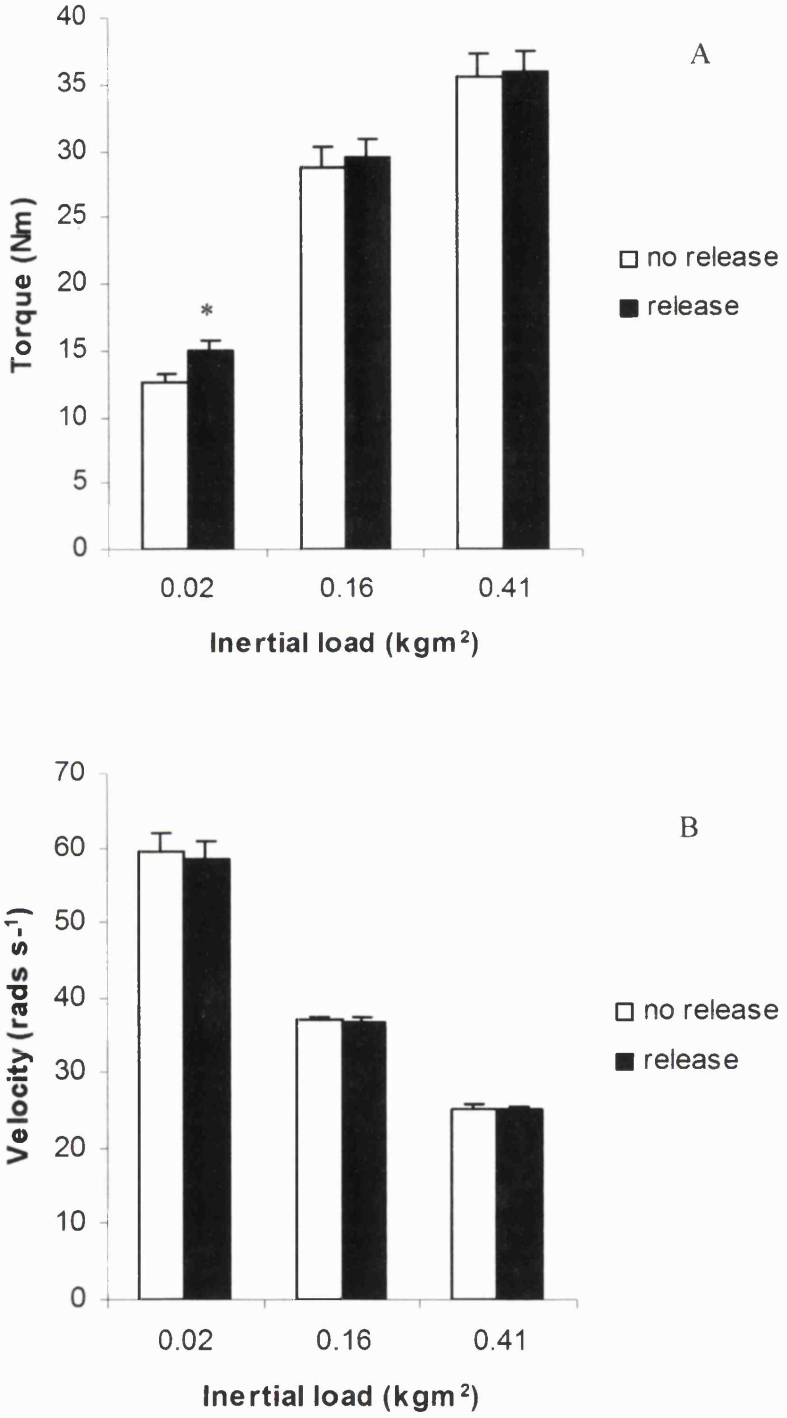


Figure 2.5 A) Mean torque at peak power. B) Mean velocity at peak power. N = 5. Error bars ± SE. * Significant difference between isometric release and no release conditions.

Effect of isometric pre release on peak torque during exertion period.

Peak torque (Tq_{pk}), can be seen to increase with increasing inertial load for both the isometric pre release and no release conditions (Figure 2.6). The Tq_{pk} is significantly greater only for the lowest inertial load with the isometric pre release but at the medium and highest inertia, there is no difference in Tq_{pk} between the two conditions. The percentage increase in Tq_{pk} due to the pre release at the lowest inertia is 18.5%.

The position at which Tq_{pk} is generated is significantly less for the release condition only at the lowest inertial load (Figure 2.7). Figure 2.8 shows the time taken to reach Tq_{pk}. There is an increase in time to Tq_{pk} with increasing inertial load for both conditions. However, the time taken to generate Tq_{pk} is significantly lower for the isometric pre release condition at all inertial loads.

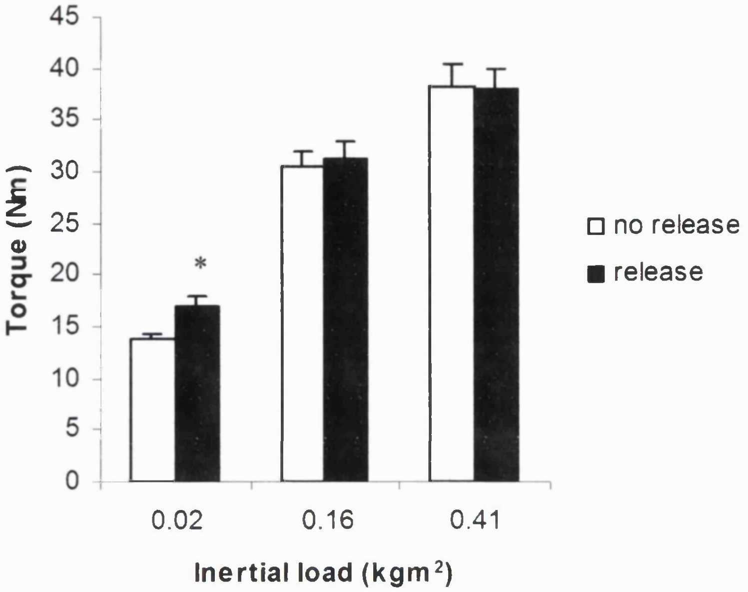


Figure 2.6 Comparison of peak torque. * Significant difference between no release vs. release condition. N = 5, Error bars ± SE

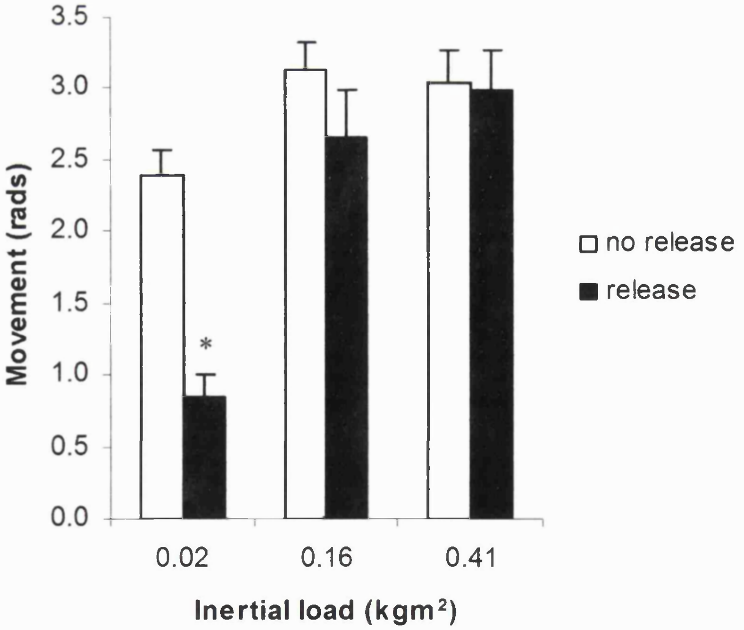


Figure 2.7 Relative position at peak torque. * Significant difference between isometric release and no release conditions. N = 5. Error bars ± SE

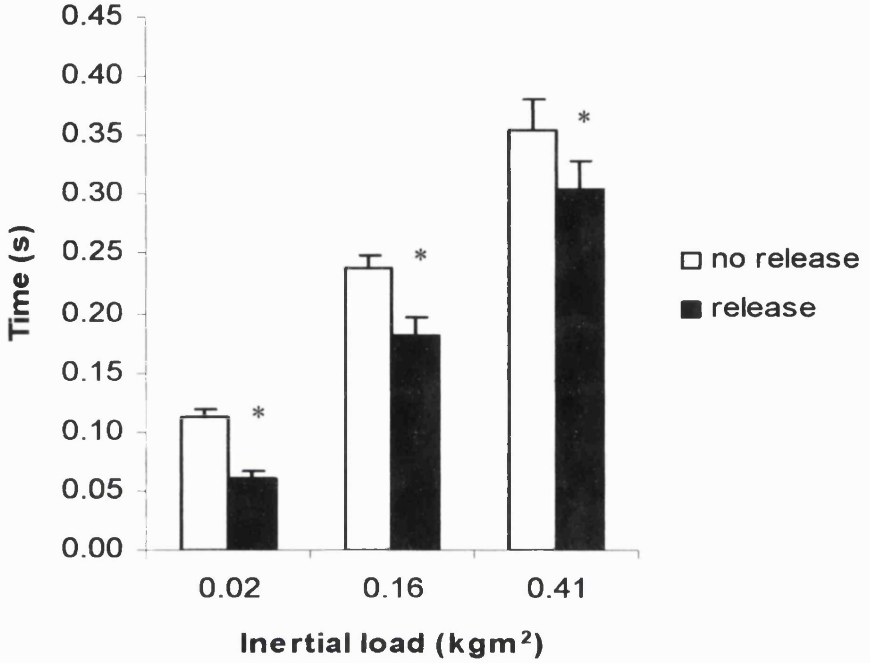


Figure 2.8 Time to reach peak torque. * Significant difference between isometric pre release and no release conditions. N = 5. Error bars ± SE

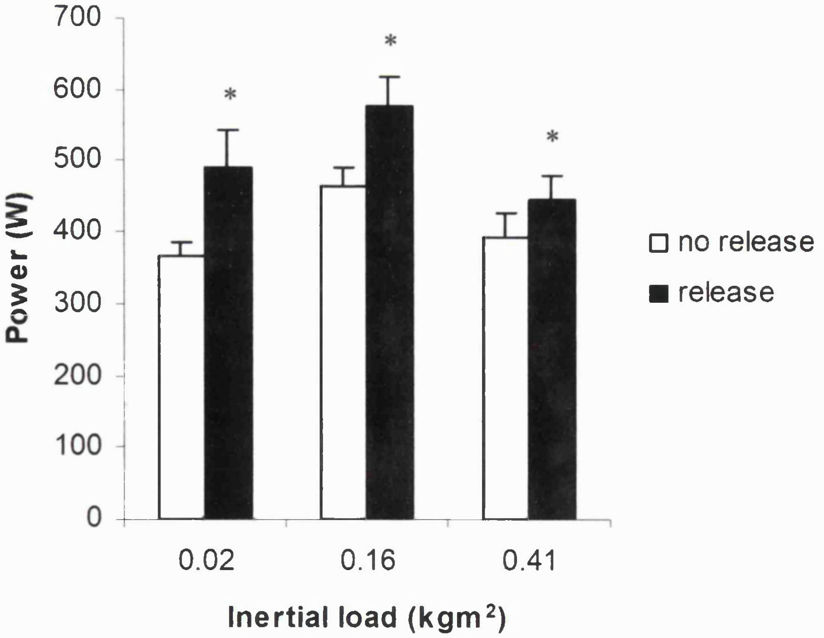


Figure 2.9 Mean power output under both no isometric release and isometric pre release conditions. * Significant difference between no release and isometric pre release. N = 5. Error bars ± SE

Isometric pre release and mean power output

The mean power (MP) generated over the whole of the exertion period was determined. The effect of the isometric pre release period was to significantly increase the MP by 25%, 19% and 11.5% at the lowest, medium and highest inertial loads respectively (Figure 2.9). A parabolic relationship between MP and inertial load was evident for both conditions. Significant increases in mean velocity and torque were also measured across all the inertial loads.

2.4 DISCUSSION

During dynamic muscular contractions, the constraints of the musculoskeletal system limit the range of movement of the limbs. At high velocities, this has the effect of limiting both the time in which the muscle can become fully activated in order to accelerate the load and the time the muscle has in order to take up the compliance of the system. This effect is perhaps most evident when using loading systems which allow acceleration to take place, as under these loading conditions the velocity is allowed to increase with increases in applied force. These factors affect the ability of the muscle to exert a force on an external load; this in turn affects the power output of the muscle.

For all subjects under both isometric pre release and no release conditions the PP_k showed a parabolic relationship with inertial load. In these experiments, it can be seen that a significant increase in PP_k generation was achieved at the lower inertial load under pre release conditions where the peak dynamic torque was set as the release tension. Here the additional time gained in order to allow for more complete muscle activation and/or a more complete stretching of the series elastic component, was on average 102 ms ± 33. This resulted in an average increase of 17% in peak power at the lowest inertial load (Figure 2.3). At the other two inertial loads, no further significant increases in PP_k output were apparent with the isometric pre release condition. At these loads, it would seem that sufficient time to allow full muscle activation is present or that the muscle can accelerate the load sufficiently to generate optimal levels of torque.

It has been shown that by allowing a pre set period of time to allow tension to be generated prior to a dynamic exertion under isokinetic conditions, dynamic torque and therefore power can be increased. Harridge et al. (1993) showed that for the plantar flexors under voluntary isokinetic shortening conditions at velocities of 4.14 rads s^{-1} the time allowed for pre shortening tension development increased the dynamic torque development. At this velocity of contraction for 0.43-0.61 rads of movement the time of a typical contraction would have been 105 - 147 ms. They found that the torque generation at this velocity without a pre release time period was less than that generated with either a 400ms or 1 s pre release period.

Similar to the high velocity isokinetic contractions examined by Harridge et al. (1993), exertions at the lower inertial loads resulted in movement times which were typically low (<0.2s), therefore it is reasonable to suggest that insufficient time is allowed for either the full activation of the musculature or complete stretching of the series elastic component. Unlike the pre release method of Harridge et al. (1993) where a pre set time period was used to allow a build up of tension, here a pre set tension was used. This method was used in order to limit the tendon stretch prior to release to a level similar to that which is experienced during the no release voluntary exertion, thereby limiting any additional storage of energy in the series elastic component.

Under isokinetic loading conditions, an increase in torque at a given velocity will have the effect of increasing power output. However, under inertial loading conditions the load at which maximal dynamic torque occurs is not necessarily the load at which peak power occurs. This can be explained in terms of the force velocity characteristics of muscle. Due to the hyperbolic nature of the force velocity curve, maximal power is achieved at approximately 1/3 of isometric force and 1/3 of maximum velocity of shortening. Therefore, under inertial loading conditions maximal power output will occur at a load where these conditions are met. However, if the inertial load is less than optimal for peak power generation, for instance, if the inertial load is very low resulting in very high contraction velocities and hence very low torques, an improvement in the peak power output may be achieved by enabling the torque level to be increased towards 1/3 of its isometric value. This in practice would require an increase in the inertial loading.

Under isometric contraction conditions, the rate of rise of tension will decrease as the tension approaches a maximum. The rate of rise is partly dependant upon the calcium concentration around the contracting filaments, which is dependant upon motor unit-firing rate. The rate of rise will also be affected in a muscle tendon unit by the compliance in the system; a stiffer system will convey the tension more rapidly than one that is very compliant. Muscles composed of faster contracting type II fibres will have a greater rate of rise of tension than muscles composed of predominantly slow type I fibres; this is due at least in part to faster fibres having faster rates of Ca^{2+} release from the sarcoplasmic reticulum and also being more sensitive to calcium than slower fibres (Bottinelli et al. 1999).

Harridge (1993) concluded that maximal activation of the plantar flexors could be achieved in approximately 484 ms. From the results of the study here it would seem that for exertions in excess of 330 ms no further significant increase in power output was evident, therefore it would thus appear that maximal activation of the active muscle mass can be achieved in this time. This study differs from that of Harridge (1993), in that the musculature involved is different, here the quadriceps are used which are composed of a larger percentage of faster fibres than the plantar flexors, and also the exertion involves a multijoint movement. As the muscle accelerates the load, the velocity increases, therefore the developed force, all things being equal, is declining in line with the force velocity characteristics of that muscle. If the velocity is such that the end of the movement is reached before the muscle is fully activated, then less than optimal forces will be generated. Conversely, if the inertial load is such that the muscle takes a considerable time to accelerate the inertia of the load, then in all probability the muscle will have sufficient time to become fully activated and will generate its full potential force during the movement. In effect by allowing more time in which to generate force prior to movement, the muscle starts further to the left along its force - velocity curve, moving down to the right as the inertial load is accelerated.

Due to the complex nature of a multijoint dynamic movement, it is not quite so clear what time would be required in order to fully activate the muscle mass. It is quite probable that due to the length tension and lever aspects of the individual muscle groups involved, a temporal aspect of muscle recruitment is used as a strategy to maximise torque production.

The relative limb position at which PP_k occurs is significantly different between the isometric release and no release conditions for both the lowest and medium inertial loads, with the position at which PP_k occurs becoming more remote from the start with increases in the inertial load. Maximal PP_k is produced at the medium inertial load and at a relative movement angle of 4.26 rads ± 0.14 for the condition without pre release torque and 3.76 rads ± 0.11 for the isometric pre release condition. This decrease in relative position at which PP_k occurs during the pre release exertion is due to the muscle developing its torque potential at this inertial load earlier in the movement. The increase in position at which PP_k is generated with inertial load increase can be explained in terms of the force - velocity relationship. It has been shown that under isokinetic loading conditions the peak torque and therefore peak power is generated more remotely with increases in velocity of movement (Osternig 1975, Kannus and Beynnon 1993). This is due to the fact that the velocity of movement is held constant; therefore, as the time of exertion gets shorter the absolute time to maximally activate the muscle causes the maximum force to be generated more remotely. Whereas under inertial loading conditions the opposite occurs due to both force and velocity being able to vary throughout the movement. At low inertial loads where the instantaneous velocity is high, the muscle will probably be on the descending limb of the power velocity curve; here PP_k will occur nearer the start of the contraction with increases in velocity. While at inertial loads where velocities are less than 1/3 V_{max} the muscle is on the ascending limb of the power velocity curve, here the PP_k will occur progressively more remotely depending on the ability of the muscle to accelerate the inertial load.

The level of Tq_{ppk} increases with the inertial load for both conditions. The increase in Tq_{ppk} with inertial load is due primarily to the increase of the inertial load as acceleration is actually reduced at the higher inertial loads. The isometric pre release enables greater levels of Tq_{ppk} to be generated at all inertial loads, but only at the lowest inertial load is it statistically significant. This would suggest that the no release condition at the medium and high inertial load permits the muscle to generate close to its full potential in terms of Tq_{ppk}.

With the release condition the significant increase in Tq_{ppk} at the lowest inertial load is a result of time which the muscle has to generate torque prior to release. With the release condition, the time at which Tq_{ppk} occurs is also significantly reduced at all inertial loads, this is because a large percentage of the maximum dynamic torque is generated prior to movement and therefore less time is required to generate Tq_{ppk} during the movement. This results in a larger acceleration phase during the earlier part of the exertion.

The Tq_{pk} increases with increasing inertial load for both release and no release exertions. The lack of any increase in Tq_{pk} with the pre release condition at the higher inertial loads suggests that these contractions were of sufficient length, to allow the muscle to generate its optimal torque at these inertial loads. At the lowest inertial load there is a significant increase in Tq_{pk} (18.5%), with the isometric release protocol. The position at which Tq_{pk} occurs is significantly less only for the release exertions at the lowest inertial load, although there is a trend at the medium inertia for the release exertions to reach Tq_{pk} nearer the start position. The difference in position at the lowest inertia is probably due to the muscle not being able to accelerate the inertial load any more from the start of movement after the isometric pre release period, and therefore the torque would decline from near the start of the exertion. However, at the higher inertial loads as sufficient time is already available to generate optimal Tq_{pk} at the optimal limb position with the no release exertions, no significant difference would be detected.

The increase in mean power over the inertial range with the pre release condition is subsequently due to an increase in the mean torque generating capacity, and at the higher inertial loads to a shorter exertion time, which translates into a higher mean velocity due to a higher level of starting torque.

In conclusion this study has shown that at the lower levels of inertial loading, power output is not optimal due in part to insufficient time to allow optimal levels of torque to be generated. It can be increased by allowing sufficient time for more complete muscle activation and/or complete stretching of the series elastic component. It was also evident at the lowest inertial loads that the power output was less than that at the higher inertial loads, even with the pre release period. This is suggested to be due to the inability of the muscle to accelerate the inertia sufficiently to generate optimal levels of torque; this is probably a consequence of the force – velocity properties of the muscles and not the activation time per se. The results of this present study show that even with the addition of the isometric pre release period, using a protocol which only allows for a single, short period of acceleration of the load the power output does not reach the levels reported in the literature for cycling. In spite of both cycling and the method used in this chapter having similarities, there are a number of factors which may be responsible for the differences in power output between the two protocols. Therefore the following chapter examines power output during cycling to investigate the effect of inertial loading on muscle function.

PART II

CHAPTER 3

POWER OUTPUT DURING INERTIAL SPRINT CYCLING

3.1 INTRODUCTION

In Chapter 1, it was shown that peak power output of the lower limb extensor muscles during a single thrust using the modified NPR, followed a parabolic relationship with inertial load. However, the values of power reported in the literature for sprint cycling are higher (McCartney et al. 1983, Martin et al. 1997, Baron et al. 1999). In the previous Chapter it was suggested that at the lower inertial loads where time of exertion was typically <0.2 second, time to fully activate the muscle mass may play a part in the decline of peak power. Whereas at the higher inertial loads insufficient time to accelerate the inertial load to a value of optimal velocity may be responsible for the decline in peak power. During sprint cycling exercise, the inertial load is repeatedly accelerated and therefore it is possible to reach a value of velocity at which peak power occurs. Under these conditions, a reduction of peak power should be seen only at the lowest inertial loads, where it may not be possible for the muscle to accelerate the inertial load sufficiently in order to generate optimal levels of force to allow maximal peak power output.

It has been suggested that during sprint cycling, peak power is independent of inertial load (Martin et al. 1997); however the time allowed to achieve peak power is an important determinant. If a protocol is used in which the time of test is very short, then a single inertial load may not be suitable for all individuals.

In vivo voluntary muscle power output has been examined in a number of different ways. Many have examined the power output under isokinetic conditions in either single joint actions (Osternig 1975, Perrine and Edgerton 1978, Patton and Duggan 1987, Kannus 1991, Madsen 1996, Taylor et al. 1991), or multi joint actions (Daly and Cavanagh 1976, Sargeant et al. 1981, Davies and Young 1983, McCartney et al. 1983, Davies et al. 1984, Sargeant et al. 1984, McCartney et al. 1985, Baron et al. 1999). The main method of testing during multi joint isokinetic experiments has been cycling. Cycling has the advantage over single joint testing in that it represents a more realistic use of the limbs, similar to that seen in everyday use. It also represents a non weight bearing method of testing in contrast to squat jumping and therefore is suitable for testing older population groups. However, cycling is a skilled motor task and it has been shown that in groups not familiar with cycling, possibly more than one testing session is required in order to elicit maximal power output (Martin et al. 2000).

The use of isokinetic methods to examine power output during cycling has an intrinsic appeal in that it allows the control of the velocity of movement. By controlling the velocity, it is possible to construct a torque-velocity relationship for the movement. Where this has been performed it has been shown by many groups that the relationship between torque and velocity during isokinetic cycling is linear in nature (Baron et al. 1999, Nakamura et al. 1985), unlike the Hill hyperbolic relationship. Davies et al. (1984) showed that in isokinetic sprint cycling (<10 seconds), the average power output in young males was 854 W. This was related to peak power measured during jumping but was 39% higher. They also stated that the traditional hyperbolic curve for the force – velocity data might be less valid when assessing *in vivo* multi joint actions such as cycling and that a straight line represented a more statistically sound fit to the force – velocity data.

The power output however still showed a parabolic relationship to the crank velocity. Utilising isokinetic loading during cycling allows the velocity at which peak power occurs to be determined easily. Others have utilised either an inertial loading method similar to that used in this thesis or friction braked loading, and also found that the power output is related to the velocity of the cycle cranks in a parabolic manner (McCartney et al. 1985, Hautier et al. 1996, Martin et al. 1997, Baron et al. 1999).

Power output during cycling exercise traditionally has been examined using either friction-braked methods as in the Wingate test (Bar-Or 1978) or isokinetic cycling protocols (Sargeant et al. 1981, Davies 1983, McCartney et al. 1983, Davies et al. 1984, Sargeant et al. 1987, Davies et al. 1989). However, the use of a purely inertial loading system to examine power output during cycling has not been extensive.

Power output during friction braked cycling is calculated from the values of flywheel or pedal speed and resistive load over a given time. It was shown by Katch and Weltman (1979) that power output using friction loaded cycling protocols was lower than that using isokinetic protocols, this was suggested to be due to "limitations in terms of maximal possible resistance". However, Lakomy (1986) noted that during friction braked cycling, velocity is not constant hence there is an accelerative component. From this he stated that when using friction cycling protocols unless the inertial component is accounted for during the accelerative phase of the load, large inaccuracies occur in the calculation of power output (~35%). He concluded that accurate measures of power output during friction braked cycling requires the calculation of the frictional load applied to the flywheel and the “acceleration balancing load”, this he defined as the load required at any instant to stop the subject from accelerating the flywheel.

There have been many different suggestions as to the optimal frictional load setting in order to maximise power output. The protocol devised by Bar Or stipulated that a loading of 75N per 1000N bodyweight, others have suggested that a fixed load is most suitable (Katch et al. 1977) whilst others have suggested that a number of resistive loads are required in order to determine maximal power (Linossier et al., 1996, Vandewalle et al. 1985). In contrast, Lakomy (1986) reported that peak power output is independent of the load used if the inertial component is accounted for in the calculations. In general agreement, Martin et al. (1997) using an inertial loading protocol reported that a single inertial load could be used to determine the peak power output.

Power output during a cycling protocol can be described differently depending on the specific interpretation of the experimenter. For instance, peak power output during a bout of cycling exercise may mean the maximal average power per pedal revolution; it could also mean the maximal power averaged over a given period of time. The period of time over which power is measured has important implications as peak power has been shown to increase with a decrease in the sample time over a given period (Lakomy 1986).

Aims

The specific aims of this Chapter were to: -

1. Determine the effect of variable inertial loading on peak power generation during a bout of maximal cycling exercise.
2. Examine the effect of inertial loading order on power output
3. To examine the development of peak power with respect to time in order to define the importance of the time variable.
4. To compare the peak power generation during cycling with that of the modified NPR.
5. To examine the peak power characteristics and velocities at which peak power occurs in relation to values cited in the literature for isokinetic cycling.

3.2 METHODS

Subjects

Nine untrained healthy young males were tested on both the modified NPR and the inertial cycle. In the randomised cycling trials seven healthy young males were tested. The Royal Free Hospital Ethics Committee approved all experimental procedures and all subjects gave written informed consent. Each test was carried out on a separate day and there was at least 2 days between subsequent test days. The physical characteristics ± SD of the subjects are age 31.6 ± 5.5 years, weight 82.6 ± 5.2 kg, height 179.9 ± 4.9 cm).

Data analysis

Repeated measures ANOVA were used to identify any significant differences in power output between inertial loads for both inertial cycling and the modified NPR tests. Where a significant difference was detected, a Tukey post hoc test was used in order to identify the specific load. All levels of significance were set to $P = 0.05$.

Equipment

The rear wheel of a standard bicycle was replaced with the inertial wheel assembly and encoder device previously described in Chapter 1. A large (80 tooth), non-standard chainwheel was used in order to allow an increase in the gear ratio and allow sufficient equivalent inertial load (resistance) at the pedals. A purpose built crank and axle assembly was constructed to allow the chainwheel to be attached to the bicycle. The cycle was modified in order to make it rigid and the whole assembly was fixed to the floor (see Figure 3.1).

The data was captured as described previously in Chapter 1. A script was written to allow the data acquisition software (Spike 2 version 2) to convert the raw data to a suitable format readable by the analysis software (Mathcad 2001). A software programme was written in a mathematical programming package (Mathcad 2001) in order to analyse the results (Appendix H).

Randomised vs. ordered inertial loading

Muscle power output has been shown to be affected by temperature (Sargeant et al 1987). Therefore, it was decided to examine if power output was affected by the order of inertial loading. As sprint cycling involved the greatest amount of muscle action of the two tests, and therefore the greatest possibility for increasing the temperature of the active muscles, the order effect of inertial loading on power output was examined using this protocol. The test sequence was first randomised followed by the ordered loading.

Figure 3.1. Inertial cycle apparatus showing inertial rear wheel and oversize front cog.

Inertial cycling test protocol (Cycling)

The testing was carried out at five inertial loads ranging from 0.024 – 0.54 kgm^2. Ordered testing was from the lowest inertial load to the highest.

Each subject carried out two maximal exercise bouts at each inertial load. Seat height was set to allow full knee extension at 180^0 of crank travel from top dead centre. All subjects started from a stationary position using their preferred leg with the pedal crank at top dead centre. Toe clips were used in order to secure the subjects onto the pedals. Subjects were instructed to pedal as hard and fast as possible until they could no longer accelerate the wheel. The test that represented the highest peak power at each inertial load was used for analysis. A rest of at least 30 seconds was given between exercise bouts. A five minute standardised warm up (50 W at 60 rpm) on an exercise cycle was carried out prior to testing.

Cycling mechanical variables - calculation of optimal velocity

Values of the peak power and associated velocity per half crank turn during a bout of sprint cycling at each of five inertial loads were used to produce individual plots for each subject. The power – velocity data was then fitted with a third order polynomial in order to determine the maximal power and associated velocity (optimal velocity – V$_{\mathrm{opt}}$). Using the equation of best fit $(P = A \times V + B \times V^2 + C \times V^3)$ V$_{\mathrm{opt}}$ was calculated as $V_{opt} = -\,{}^1\!/_3 \times \left[B + \left(B^2 + 3 \times C \times A\right)^{1/2}\right] / C$. This equation was produced from the first order differential of the polynomial equation where change in power is zero, and rearranged to allow calculation of V$_{\mathrm{opt}}$

Modified Nottingham Power Rig protocol (MNPR)

Subject positioning was as previously described in Chapter 1.Individual testing of both lower limbs was undertaken. Each subject performed two maximal exertions at each inertial load on each leg. The inertial loading and pre test warm up was as for the inertial cycling protocol.

3.3 RESULTS

Randomised vs. ordered inertial loading during sprint cycling

Figure 3.2 shows the peak power results from both the ordered and randomised inertial loading during sprint cycling tests which were performed on two separate occasions. It can be seen that no significant differences in peak power were measured at any inertial load.

Peak power output modified NPR vs. inertial sprint cycling

Comparisons of peak power (PP_k) obtained during sprint cycling (cycling) and during a maximal exertion on the modified power rig (MNPR), over a range of inertial loads are shown in Figure 3.3. The PP_k during cycling ranged from 686 ± 143 to 1620 ± 336. The PP_k during MNPR ranged from 723 ± 162 to 937 ± 246, the percentage differences between cycling and MNPR are summarised in Table 3.1. It can be seen that for cycling, the PP_k is significantly greater than that at the lowest inertial load, for all the subsequent inertial loads. The PP_k for MNPR at both the lowest and the highest inertial loads is significantly different from that at 0.158 kgm^2. The PP_k output during MNPR has a characteristic parabolic shape with the power output increasing from the lowest inertia up to a maximum at 0.158 kgm^2 and then declining as the inertial load increases. For cycling the PP_k output can be seen to increase from the lowest inertial load and at the second load (0.158 kgm^2), the PP_k plateaus showing no significant increase with increasing inertial load (see Figure 3.4). The PP_k obtained during cycling was shown to be significantly greater than that obtained for MNPR, at all inertial loads except the lowest (0.024 kg m^2).

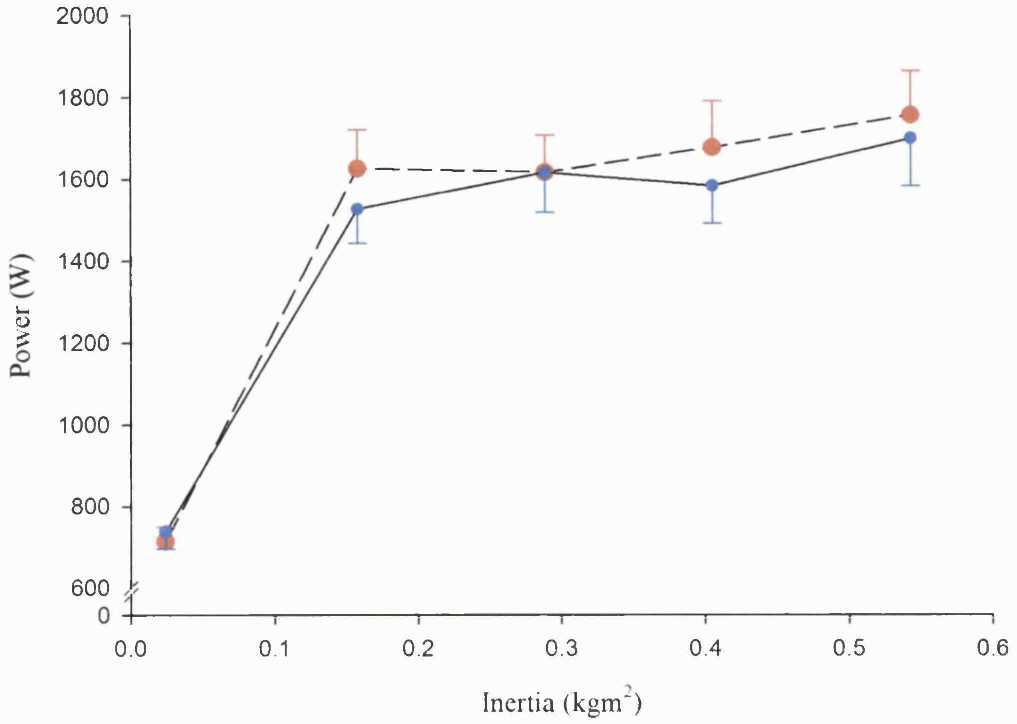


Figure 3.2 Randomised vs. ordered sprint cycling - Peak power vs. inertial load. N = 7. Error bars ± SE. Ordered (●) Randomised (●).

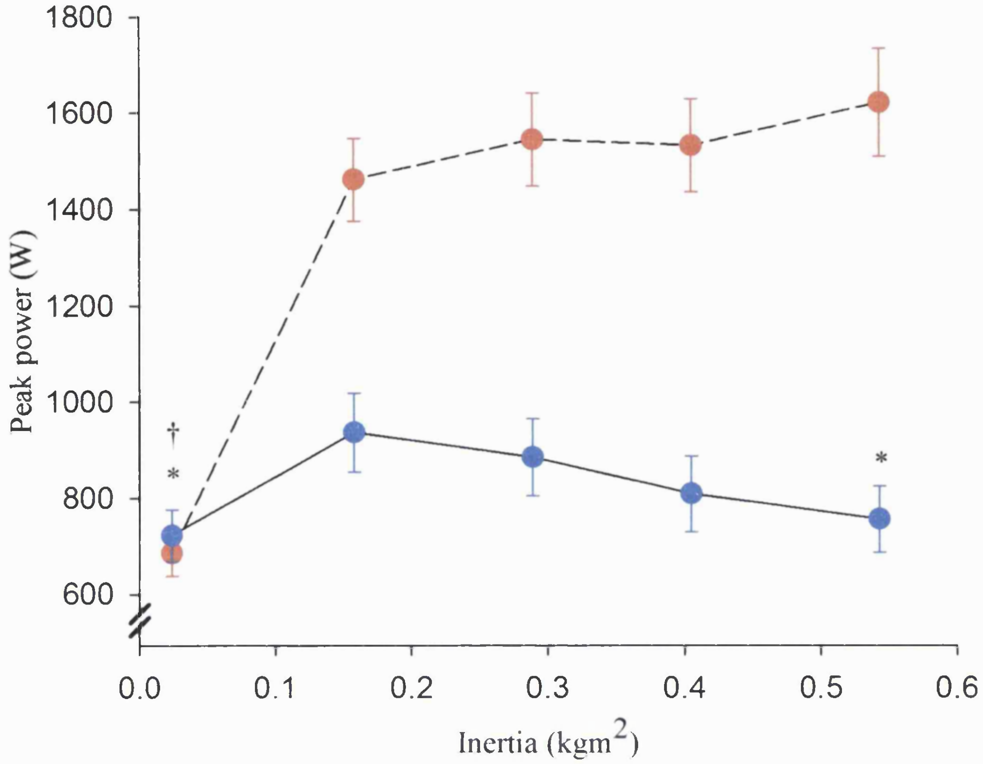


Figure 3.3 Comparison of sprint cycling (●) peak power output vs. modified NPR (●) at 5 inertial loads (0.024 – 0.54 kgm^2). † Significant difference from subsequent inertial loads (cycling). * Significant difference from 0.158 kg m^2 (MNPR). N= 9. Error bars ± SE

Table 3.1 Summary of peak power output for cycling and MNPR. Percentage differences between cycling and MNPR. * Significant difference between cycling and MNPR

	Inertial load (kgm^2)				
	0.024	0.158	0.289	0.405	0.543
Cycling	686 ± 143	1461 ± 257	1544 ± 290	1531 ± 290	1620 ± 336
MNPR	723 ± 162	937 ± 246	885 ± 239	809 ± 236	756 ± 206
% Difference	5	56*	74*	89*	114*

Peak power output characteristics during cycling

It was shown that PP_k during cycling reached a plateau after the lowest inertial load. However, the time taken to generate PP_k increased significantly with increasing inertial load (Figure 3.5).

The mean values for instantaneous torque and velocity at which peak power occurs were determined. It can be seen from Figure 3.6 that the torque - velocity relationship is almost linear ($r^2 = 0.94$). The instantaneous peak power – velocity relationship was also plotted across the range of inertial loads for the group (Figure 3.7). It can be seen from the power velocity graph in Figure 3.7 that the optimal velocity is approximately 123 rpm. The maximum extrapolated velocity from graph 3.6 is approximately 250 rpm), thus in keeping with the linear torque – velocity relationship, the optimal velocity is approximately half of this.

Figure 3.8 shows the optimal velocity power relationship for all subjects. The optimal velocity values are derived from the third order polynomial fitting procedures. The linear relationship ($r = 0.73$) between the two variables indicates that for an increase in peak power there is a concomitant increase in optimal velocity.

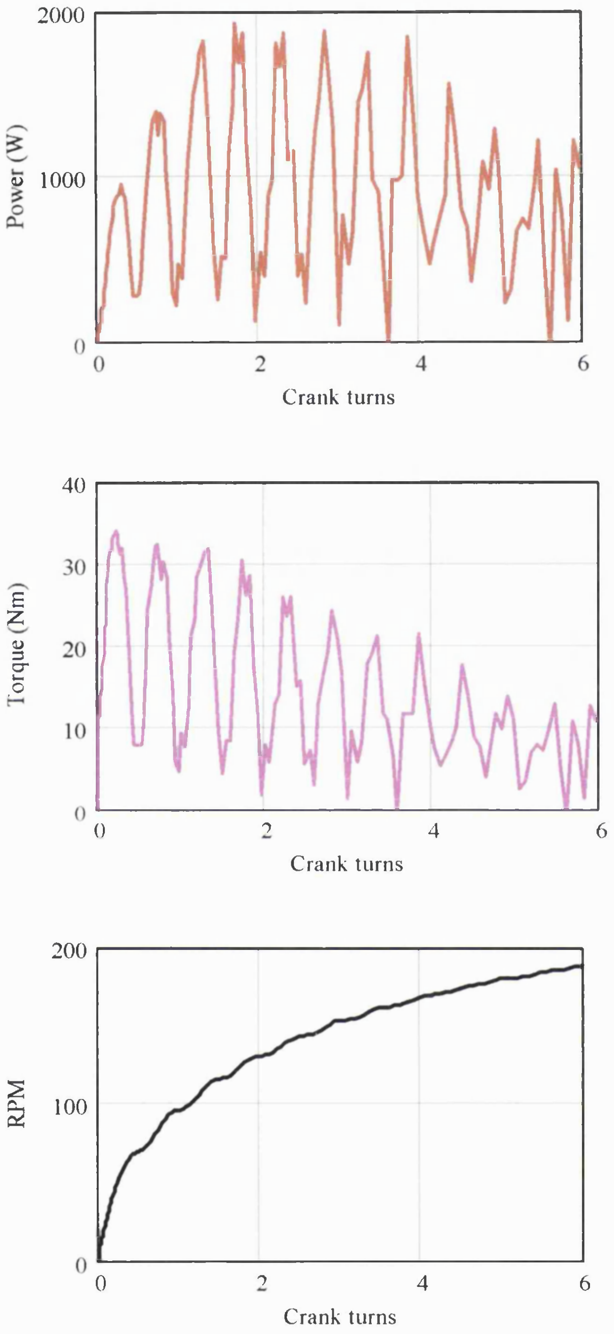


Figure 3.4 Original records of power output, torque and rpm during a single bout of sprint cycling.

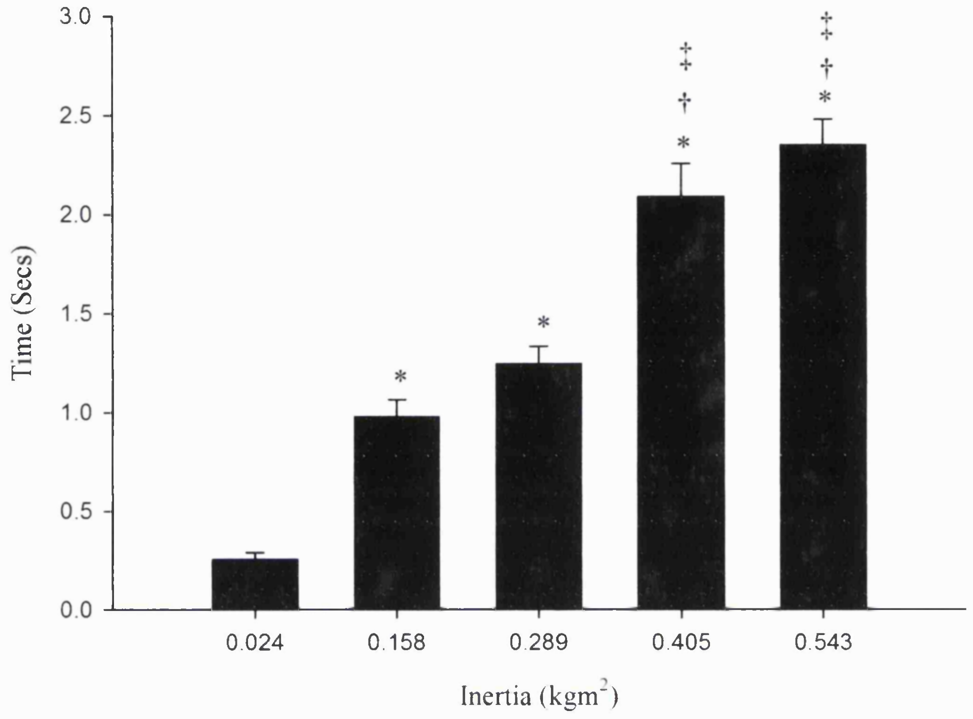


Figure 3.5 The time taken to reach peak power with respect to inertial load. * Significantly different to 0.024 kg m^2. †Significantly different to 0.158 kg m^2 . ‡ Significantly different to 0.289 kg m^2

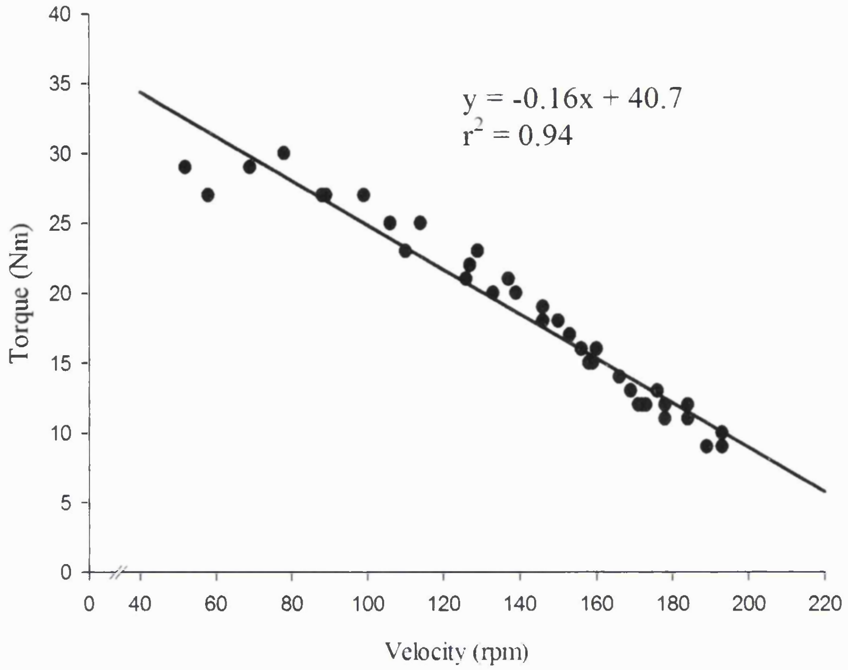


Figure 3.6 Mean group values of torque and velocity across the range of inertial loads for cycling. Crank torque ~ 6x y values.

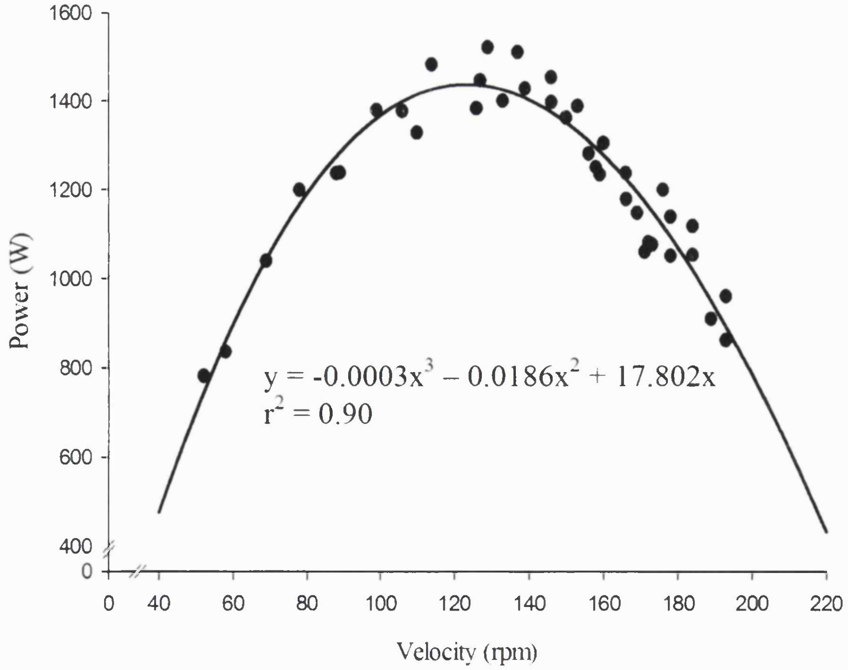


Figure 3.7 Mean group values of power and velocity across the range of inertial loads for cycling.

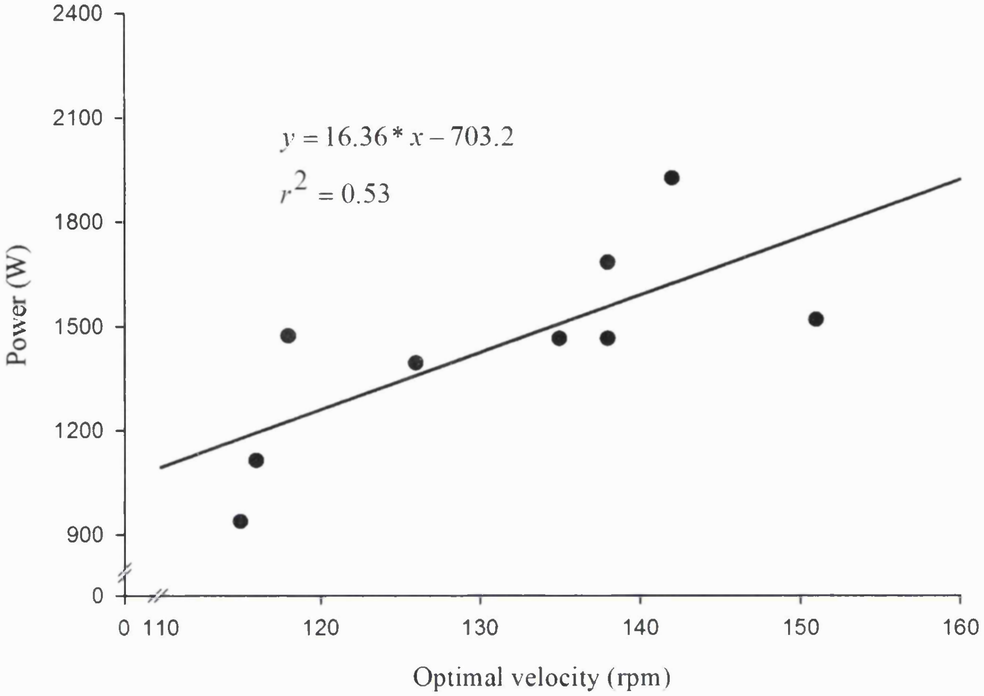


Figure 3.8 Subject values of peak power and optimal velocity during cycling.

3.4 DISCUSSION

Observations on isokinetic vs. inertial cycling protocols

Baron et al. (1999) compared power output during non-isokinetic cycling to that obtained during isokinetic cycling over a 10 second sprint period. They reported values of peak power output during isokinetic cycling of 15.3+/-1.7 W/kg and 14.4+/-1.9 W/kg during non-isokinetic cycling. The non-isokinetic power output was significantly lower than that obtained during isokinetic cycling. The peak power output was determined over a full pedal revolution and therefore would tend to be less than an instantaneous value obtained during a crank turn. This point was illustrated by Lakomy (1986) where he showed that for similar bouts of cycling the sampling rate could have a profound effect on the instantaneous power output (Figure 3.9). Here the power output is averaged over either 0.5 or 0.1 seconds, it can be seen that the power output is greater when the result is averaged over a shorter time period. The fluctuations in power output increased at lower crank velocities where higher torques are produced. Lakomy (1986) reasoned this would be in line with the force – velocity properties of muscle, in that the ability to accelerate the flywheel, hence affect the velocity, is diminished at the higher velocities due to the limitations on torque production.

For instance, the peak power output obtained in this study would equate to approximately 16.9 W/kg. This is higher than the values reported by Baron et al. (1999) in spite of the subjects testing in this study having a greater mass. This is most probably a reflection of the sampling rates used in order to average the

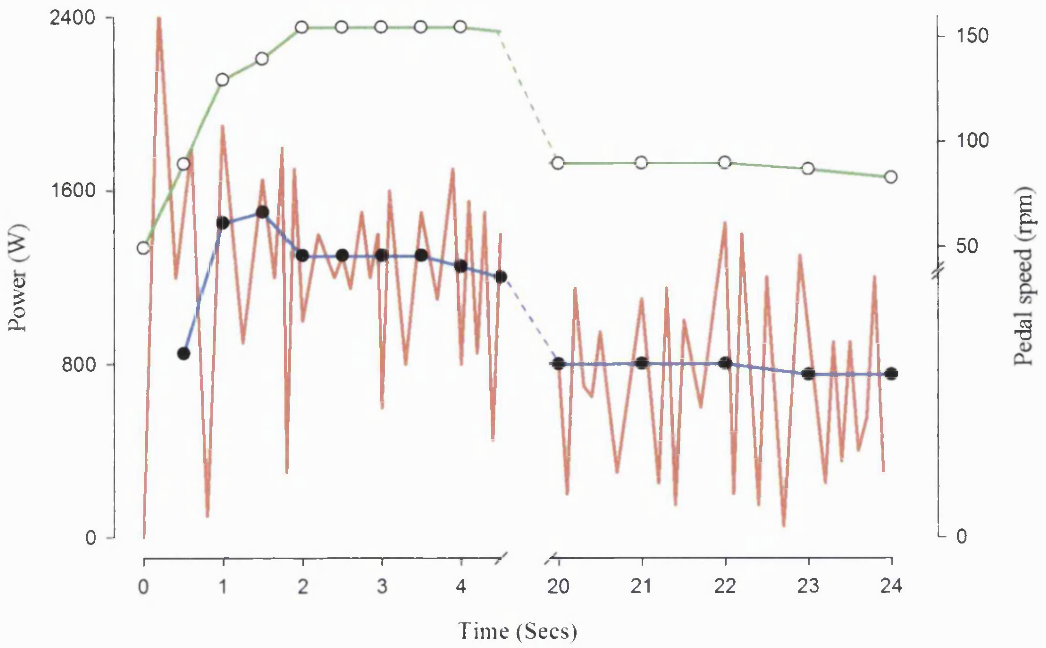


Figure 3.9 From Lakomy (1986) Power output during friction braked cycling. The power output when sampled at 0.1 seconds (—) can be seen to have a greater variation and higher values than power output sampled at 0.5 seconds (—). The variation in power output at the higher velocities of pedal speed (—) can also be seen to be decreased. (See text for explanation).

power output as explained above. Others however have reported results within a range of 13.1 – 16.4 W/kg (Buttelli et al.1996, Dotan et al.1983, Linossier et al.1996, Martin et al.1997, Seck et al.1995).

Of interest is the finding by Baron that the peak power output during isokinetic cycling was higher than that obtained during non-isokinetic cycling. In order to compare the two methodologies they reported maximal values of power obtained from averages of each crank turn over a 10 second isokinetic bout, over a range of isokinetic velocities, to the highest value obtained during a 10 second bout of non isokinetic sprint cycling. The data were fitted with 2nd order polynomials in order to determine the peak power and optimal velocity for each method. The higher power reported during isokinetic cycling was in spite of the fact that the V_{opt} during inertial cycling was higher than that for isokinetic cycling. The V_{opt} during isokinetic cycling was in general agreement with that of Sargeant et al. (1981), who reported a V_{opt} of 110 rpm under isokinetic conditions.

Martin et al. (2000) determined power output and optimal velocity using an inertial cycle, they reported values of 17. 4 W/kg and 124 rpm for subjects in the 20-30 age range, however similarly to Hautier et al.(1996) the subjects were lighter than those in the present study, and trained on the cycle. Martin et al.(1997) utilising a inertial protocol report values for V_{opt} of 122 rpm and a value of power for a full crank revolution of 1317 W. This cannot easily be compared to the values reported here as insufficient data are given in the paper. Other possible differences between cycling protocols used which may affect the values are the sampling rates at which power is sampled. Lakomy (1986) has previously shown that this can have a large effect on the measured power; a shorter averaging period will result in higher values of power.

A possible reason for the finding that isokinetic cycling results in a higher maximal power output is the difference in the dynamics between the two testing methods. During isokinetic cycling, the speed of movement is held constant. This allows the muscle to develop its maximal torque at any given joint angle; maximum power will therefore be generated, where torque is maximal. Whereas under inertial loading conditions the muscle velocity is allowed to increase, according to the force velocity characteristics of muscle, the muscle is therefore less able to generate torque. In this case maximal power will be generated where the product of torque and velocity is maximised. If one now also considers the length tension relationship of muscle, this will not always be at a joint position at which maximal torque can be generated.

The optimal velocity at which peak power was developed in the present experiment, determined using the polynomial fitting method was 125.9 rpm. This is in good agreement with that of Baron et al. (1999) who reported a figure of 127 ± 14 rpm using a friction braked cycle protocol. This optimal velocity was higher than that achieved during isokinetic cycling (115 ± 8.6 rpm). The isokinetic velocity steps were in 10 rpm increments, if they had used a lesser step they perhaps would have found a good agreement between the two methods. Hautier et al. (1996) reported a figure of 120 rpm using a non-isokinetic cycling protocol.

The method used to determine optimal velocity can give different results. If a polynomial fitting method is used on the power velocity data, a smoothed value of velocity is given. For this experiment, the value of optimal velocity using this method is 125.9 rpm; this value is consistent when comparing the optimal velocity at peak power at each of the inertial loads. The polynomial fitting method might underestimate the optimal velocity at which peak power occurs due to the smoothing effect. A similar methodological issue exists concerning the peak power. However in order to compare these results with those previously described results cited in the literature, a similar analysis was used.

Inertial load – order of testing and power output

It is known that temperature affects the power generating capability of muscle (Crowley e al. 1991, Davies et al. 1985, 1983; De Ruiter et al. 2000; Faulkner et al. 1990; Ferretti, 1992; Holewijn and Heus 1992; Oksa et al. 1997; Ranatunga 1998; Rome 1990; Sargeant, 1987). It was postulated that the effect of repeated sprint cycling exertions may increase the temperature of the muscle and therefore increase the muscle potential to generate power. If this was then case then the order of testing may bias the results. However in the case of the protocol used for sprint cycling, no significant differences in power output were found at any inertial load due to loading order and therefore it would appear that there are no significant increases in the muscle temperature during the testing procedure. This is not too surprising in that the sprint generally lasted only a few seconds and a 30 second rest was given between cycling bouts. Additionally the muscles were warmed up prior to testing with a standardised 5 minute cycling bout.

Variable inertial loading, exertion time and peak power output

Martin et al. (1997) reported that the power output during inertial loaded cycling was independent of inertial load. The experiments described in this chapter show that above a certain level of inertial load (critical load) the power output is indeed independent of inertial load and that a plateau is seen for subsequent higher loads. This plateau occurs due to the ability to repeatedly accelerate the inertial load with the cycling action, this allows the muscle to travel along its force – velocity curve until a position at which peak power occurs is met. In theory, this position will always be reached provided sufficient time is allowed for the individual to accelerate the load and the subject does not become fatigued. However, if the inertial load is very low, power output can be seen to be significantly lower than that for all subsequent inertias (Figure 3.3). This is in part due to the inability of the muscle to generate sufficient acceleration at this load to develop optimal levels of torque in order to generate peak power (see Chapter 2 Discussion for the explanation of this point).

Another important factor, in the ability to generate maximal power output, is the absolute time allowed in order to accelerate the load. As inertial protocols are concerned with the accelerative component, if an inertial load is chosen which represents a relatively high load, the time taken to reach peak power will be longer than if a relatively lower inertial load is chosen (Figure 3.4 and 3.5). Very simply, this is due to the muscle at high inertial loads starting at a less optimal position on its force velocity curve and therefore having further to travel along it before reaching an optimal position at which peak power will occur (Figure 3.10).

Therefore, if it is required to determine maximal power output over a given, fixed time period, one must be aware of the possible differences in the time required to reach peak power, dependant on inertial load and the relative ability of the individual to accelerate the load. Thus for a group of subjects who may differ in their muscle torque/power generating characteristics, a range of inertial loads may be required in order to elicit maximal power output.

Comparison of power output - NPR vs. sprint cycling

Peak power output during cycling was shown to be significantly higher than NMPR at all inertial loads apart from the lowest. The muscle is unable to generate sufficient levels of acceleration to generate optimal levels of power output at the lowest inertial load under either protocol. As the inertial load increases however, the peak power is greater during cycling. There are a number of possible reasons

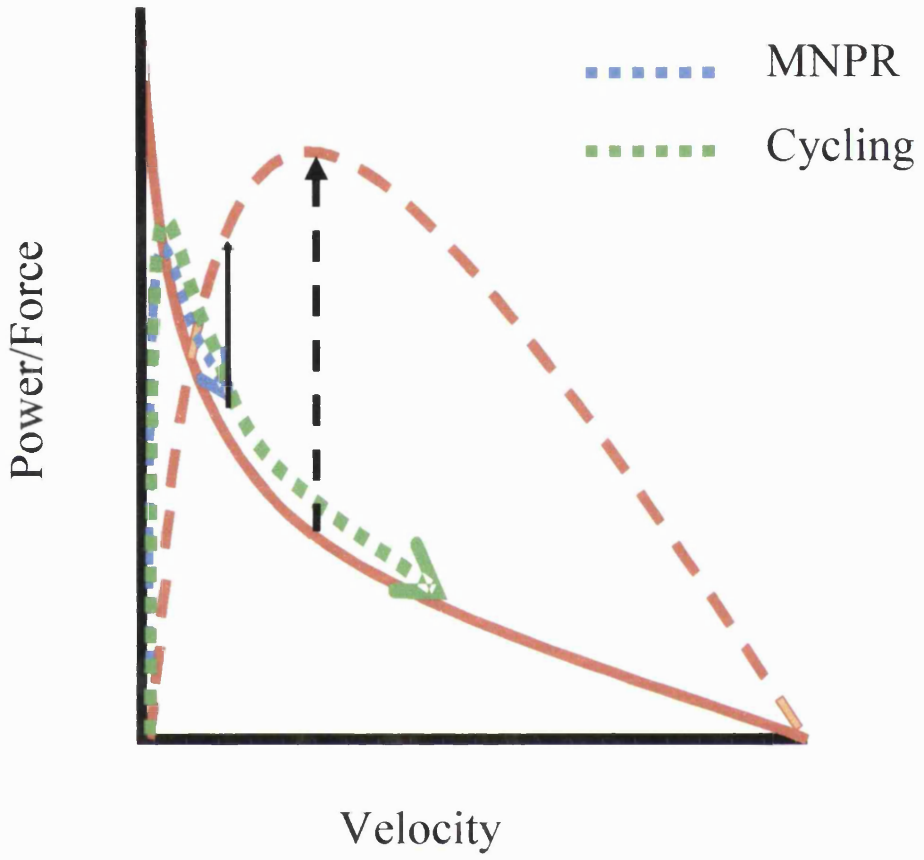


Figure 3.10. Hypothetical characteristics of power generation for cycling and MNPR under identical inertial loading conditions. Here for MNPR (blue dotted line) it can be seen that the acceleration is insufficient to allow sufficient time to arrive at the velocity at which peak power occurs (solid black line). Whereas for cycling (green dotted line) acceleration is continuous hence the 'optimal velocity' at which peak power occurs is passed (dashed black line).

why this is so. The acceleration of the inertia during MNPR is limited to that which can be obtained during a single lower limb thrust. During cycling, at any given inertial load, a greater amount of time is allowed in order for the muscle to fully accelerate the load. This allows for an optimal level of torque and velocity to be reached, and therefore peak power. Figure 3.4 illustrates this point, where it can be seen that as the inertial load increases, the time to peak power increases. Power output for a given bout of cycling shows a parabolic relationship with time (Figure 3.4). In each trace, as velocity continues to increase with each crank turn the torque continues to decrease, hence power output declines.

The anatomical position of the subject during testing also has an effect on power output. Only the lower limb was able to participate in power generation with the MNPR. During sprint cycling, the legs are actively accelerating the inertial load, but also there may be additional input from ancillary muscles. In addition the foot position with respect to the pedal may be different for each protocol, which could affect the ability to generate torque tangential to the crank (See Diagram A p.166).

It has also been reported that the knee extensor muscles are active on the recovery stroke (approximately 40 -50 ^{0} prior to top dead centre) (Faria and Cavanagh 1978). This effect could provide additional ability to generate power by utilising the stretch shortening cycle. Various mechanisms have been suggested to explain the increased output due to the stretch shortening cycle; these include increased time available to generate force (Bobbert et al.1996), reutilisation of stored elastic energy (Hawkins and Hull 1990, Svantesson et al.1991) and reflex contributions (Dietz et al.1978). The arms act as anchors during cycling and may help transfer forces from other muscle groups during cycling action. It has been reported that there is an increased ability to generate power output with the hands holding the handlebars as opposed to not holding the handlebars (Baker et al.2001). The use of toe clips has been shown to increase the power output. Capmal and Vandewalle (1997) reported that both the torque and power output were increased by the use of toe clips, they reasoned this was due to the pulling action during the return of the foot to top dead centre after reaching the bottom of the stroke. In the present study toe clips were used and the levels of absolute peak power generated during cycling are in general agreement with those of Martin et al.(1997), Baron et al. (1999), Martin et al. (2000), Sargeant et al. (1987).

In conclusion this study has shown that for the sprint cycling protocol used, the power output during sprint cycling is greater than that during MNPR exertions. In spite of some similarities between the protocols used in the present study, in that both test the lower limbs, differences are apparent. This is possibly due to a combination of the ability to repeatedly accelerate the load and the assistance of synergistic muscles during cycling. The power output is only independent of inertial load above a 'critical' load during sprint cycling. Time is an important factor to consider if the inertial loading is relatively high, as not all individuals will have a similar ability to accelerate the load. The optimal velocity at which peak power occurs for this study was in general agreement with that of others cited in the literature. The reported optimal velocity during isokinetic cycling is less than that during inertial cycling; this is due in part to the differences in the 'apparent load' seen by the muscle and the interaction of the joint lever arms and the muscle length tension relationship.

PART III

CHAPTER 4

MHC ISOFORMS AND MUSCLE POWER OUTPUT IN YOUNG AND OLD MEN

4.1 INTRODUCTION

MHC composition, power output and optimal velocity

The composition of human muscle in terms of the relative proportions of myosin heavy chain isoforms (MHC) has a direct bearing on the ability of the muscle to generate power output. It has been shown from single fibre studies performed *in vitro* that the power, force output and shortening velocity of fibres containing MHC-II isoforms is higher than that of fibres containing MHC-I isoforms (Larsson and Moss 1993, Bottinelli et al.1996, Harridge et al.1996, Widrick et al.1996, Aagaard and Andersen 1998). It has been reported that fibres containing only MHC-IIX isoforms have approximately nine times the power generating ability and approximately a nine fold increase in maximum shortening velocity of fibres containing MHC-I, (Bottinelli et al.1999) with fibres expressing the MHC-IIA isoform generating approximately four to five times the power of MHC-I fibres and five to six times the maximal shortening velocity. This has implications for not only the power generating capacity of muscle, but also the optimum velocity at which peak power is generated. In a mixed muscle containing different proportions of myosin isoforms the percentage of each should in theory determine the ability of the muscle to shorten against a given load.

In order to examine the underlying physiology with regard to muscle power generation, researchers have examined the composition of the muscles involved in a particular action. Traditionally muscle fibre composition has been determined using ATPase histochemistry. Using this method the results have been somewhat equivocal in that not all the studies showed a correlation of power output to fibre composition. Positive correlations of fibre composition with muscle force/power have been shown by some researchers (Thorstensson et al.1976, Larsson et al.1979, Ivy et al.1981, Häkkinen et al.1984, Macintosh et al.1993). Whilst others have not seen this positive relationship (Clarkson et al.1981, Froese and Houston 1985).

It is well established that muscle mass declines with age. Along with this decline is a reported reduction in the size of fast muscle fibres which would contain MHC-II isoforms (Tomlinson et al.1969, Jennekens et al.1971, Tomonaga et al.1977, Larsson et al.1978, Scelsi et al.1980, Grimby et al. 1984, Grimby et al.1982, Lindboe & Torvik 1982, Nygaard & Sanchez 1982, Aniansson et al.1986, Poggi et al.1987, Lexell 1988, Lexell & Taylor 1991). Reduced muscle mass *per se* would only partly explain the observed differences in muscle output due to ageing, as the velocity at which peak power occurs would be independent of the muscle mass. However the reported reduction in MHC-II isoforms would in part explain a reduction in velocity at which peak power occurs.

The ability to generate peak power and the velocity at which peak power occurs has been shown to decline with age (Davies et al.1983, Martin et al.2000). In addition to an overall loss of MHC-II isoforms, it has been reported that there is an increased co expression of MHC isoforms in the muscle fibres of older individuals (Klitgaard et al.1990, Andersen et al.1999). Ageing also has intrinsic effects on the individual fibres containing different MHC isoforms. Recent studies have shown that that older muscle fibres expressing the same MHC are intrinsically ‘weaker’ than similar fibres from younger men. Larsson et al.(1997) reported that the specific tension in chemically skinned fibres from the vastus lateralis containing MHC-I and IIA was significantly lower in old vs. young men (0.18 vs. 0.19 N/mm^2 MHC-I and 0.18 vs. 0.25 N/mm^2 MHC-IIA). Frontera et al. (2000) reported similarly that in young subjects, vastus lateralis fibres containing MHC-I and MHC-IIA were able to generate significantly more force than similar fibres from old subjects (725 μN vs. 505 μN MHC-I and 792 μN vs. 577 μN MHC-II). The effect of ageing on the kinetics of cross bridges has been recently documented using the *in vitro* motility assay (Hook et al. 2001). They reported in human fibres that actin sliding speed was significantly reduced in fibres expressing MHC-I isoform in the older group (0.56 $\mu m/s$ vs. 0.69 $\mu m/s$). They also showed a trend for the fibres expressing MHC-IIA to have higher values of shortening speed in the young. This is in agreement with the results of Larsson et al. (1997) who showed a significant reduction in maximum shortening velocity (V_{max}) in MHC-II fibres from elderly muscle, as determined using the slack test technique.

These observations might explain the reduced *in vivo* performance of the muscle. Indeed, Harridge et al.(1995) reported a significant correlation between the ability to generate plantar flexor torque at high velocities and the isomyosin content of the gastrocnemius and soleus muscles.

The aims of the present study were thus:

1. To examine and compare the power output, optimal velocity and torque during inertial sprint cycling and MNPR in young and elderly subjects.

2. To examine the relationship between the mechanical output variables and the MHC isoform composition of the quadriceps in young and elderly subjects.

4.2 METHODS

Subjects

All subjects were males, the groups consisted of a 'young' group (n = 7) and an 'elderly' group (n = 7). The physical characteristics are presented in Table 4.1. All subjects were classified as healthy using the health criteria as described by Greig et al (1994). All participating subjects were untrained. Prior to testing, all subjects were informed of the testing procedures and signed a consent form agreeing to take part in the study. The study had the approval of the Royal Free Hospital Ethics Committee.

Group	Age (yrs)	Weight (kg)	Height (cm)
Young	29.4 ± 1.8	83.3 ± 1.1	180.1 ± 1.8
Elderly	73.8 ± 1.6	74.6 ± 2.2	176.3 ± 2.7

Table 4.1 Physical characteristics of subjects. All table values shown as mean ± SE.

Anthropometry

Lower limb volume was estimated from anthropometric measurements comprising segmental circumferences and lengths as described by Jones and Pearson (1969). Skinfold measurements were made at four sites; anterior, posterior mid thigh and lateral, medial mid calf using skin callipers (John Bull Ltd). Skinfold corrections were made using the following regression equations (Personal communication Professor P. R. M. Pearson), anterior thigh ($y = 1.0142 + 0.557 \times$ skinfold value), posterior thigh ($y = 1.368+0.532$ x skinfold value), medial calf ($y = 0.985 + 0.499 \times$ skinfold value) and lateral calf ($0.8701 + 0.3926 \times$ skinfold value), where y represents the corrected skinfold value used in the calculations.

Equipment

See methods section Chapter 3.

Sprint cycle protocol

See methods section Chapter 3. In contrast to the protocol in Chapter 3, 3 exertions were carried out at each of the five different inertial loads, ranging from 0.024 – 0.54 kg m^2.

Cycling mechanical variables - calculation of optimal velocity and torque

See methods section for details of calculations (Chapter 3). A value of maximal power can then be calculated from the values of optimal velocity. For the MNPR data a similar procedure was used, however a second order polynomial was used as this best fitted the data.

The torque at maximal power for both the cycling and MNPR was calculated using a linear fit of the torque – velocity data as this method gave better r^2 values than the polynomial method.

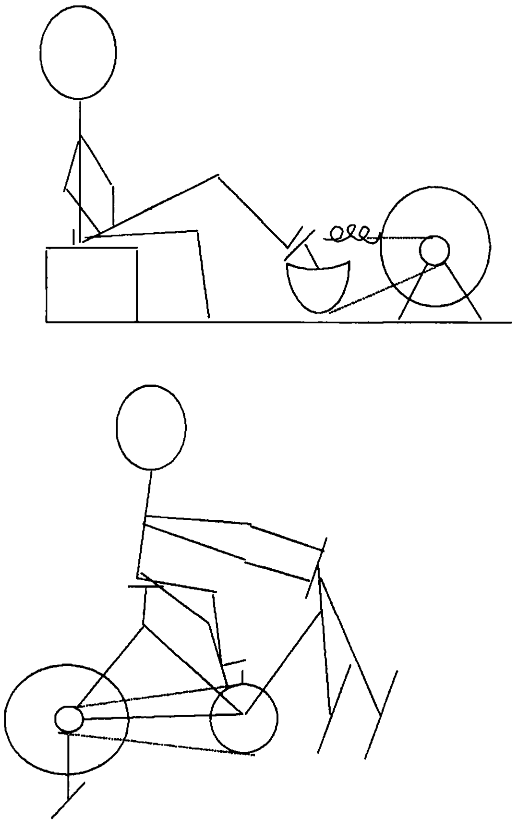


Figure A. Diagram showing positioning of subject for each protocol.

Note in particular the foot position relative to the crank.

Habituation

Due to the 'skilled' nature of cycling the groups were habituated (HAB) on the cycle. This consisted of two exertions at three inertial loads (0.024, 0.29 and 0.54 kg m^2). The habituation was carried out on the same day as the MNPR testing.

MNPR protocol

All exertions of the preferred lower limb were carried out with the subjects seated as previously described (See Chapter 1 section 2). A total of 15 exertions were recorded, 3 trials at each of 5 inertial loads ranging in order from 0.024 – 0.54 kg m^2. The exertion at which the highest value of power occurred within the 3 trials at each inertial load was subsequently used for further analysis. Testing was carried out at identical inertial loads to those for the sprint cycling. Values of maximal power, optimal velocity and torque were calculated using the polynomial method as described.

Muscle tissue sampling

Each subject had a biopsy of muscle tissue taken from the middle of the belly of the vastus lateralis. Prior to the biopsy procedure the site was cleaned with chlorhexidine and a local anaesthetic was administered (2 % lignocaine). An incision was made through the skin and muscle fascia to allow the biopsy needle to pass into the muscle belly. The biopsy was taken using a 5 mm diameter Bergstrom needle with additional suction. The sample weight ranged from 30 –60 mg. The muscle sample taken was immediately frozen in liquid nitrogen and then stored in Eppendorf tubes at –70 ^{0}C. The subject was instructed to rest for approximately 10 minutes and pressure applied to the site to prevent further bleeding. The biopsy site was then closed with steri strips and an adhesive dressing.

Electrophoretic separation of myosin isoforms

The relative proportions of the three MHC isoforms (MHC-I, IIA and IIX), contained in the vastus lateralis biopsy samples from each subject was determined through sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS – PAGE) (Andersen and Aagaard 2000).

i) Preparation of solutions and samples for SDS – PAGE

a) Sample buffer stock solution

A 50ml stock solution of sample buffer consisted of 6.25ml of 0.5 M Tris (pH 6.8), 10 ml of 10% SDS, 10 ml glycerol and 2.5 ml beta-mercaptoethanol. The solution was then made up to 50 ml with distilled water. Bromophenol (25mg), was added in order to stain the solution (Fry, Allmeir and Staron 1994).

b) Muscle tissue preparation

The muscle was homogenised manually with a pestle and mortar prior to adding the stock solution. Each muscle sample had 500 μl of sample buffer added to the tube. The mixture was then heated at 97 ^{0} C for 3 minutes.

c) SDS Gel preparation

The SDS PAGE gel electrophoresis system is a discontinuous system consisting of a stacking and resolving gel. For the stacking gel a 4 % solution was used in order to align the contractile proteins prior to separation.

Resolving gel preparation

In order to produce a 7% resolving gel the following reagents were mixed in specific volumes.

Reagent	Volume (ml)
Acrylamide – Bis (100:1)	3.13
Lower Tris (pH 6.8)	1.34
Distilled H_2O	1.05
Glycine	1.0
10% SDS	0.4
Glycerol	3.0
APS	100 μl
TEMED	8 μl

In order to set the resolving gel a set of glass plates was positioned in a cradle designed to hold the resolving gel plates. Approximately 4ml of the solution was poured into the space between the glass plates. The solution was allowed to set for approximately 2 hours prior to loading the stacking gel.

Stacking gel preparation

In order to produce a stacking gel the following reagents were mixed in specific volumes

Reagent	Volume (ml)
Acrylamide – Bis (37.5:1)	0.5
Upper Tris (pH 8.8)	1.25
Distilled H_2O	3.25
APS	50 μl
TEMED	10 μl

The solution was added on top of the resolving gel and allowed to set for a further 1 hour.

A running buffer consisting of 50 ml buffer concentrate (Biorad Tris, Glycine) added to 450 ml distilled H_2O pre cooled to approximately 5 ^{0} C is placed into the running tank and the samples are loaded into the stacking gel wells (Figure 4.1). A further 200 μl of beta mercaptoethanol was added to the centre of the tank prior to running. The gels were set to run for approximately 26 hours at 5 ^{0} C at 80v.

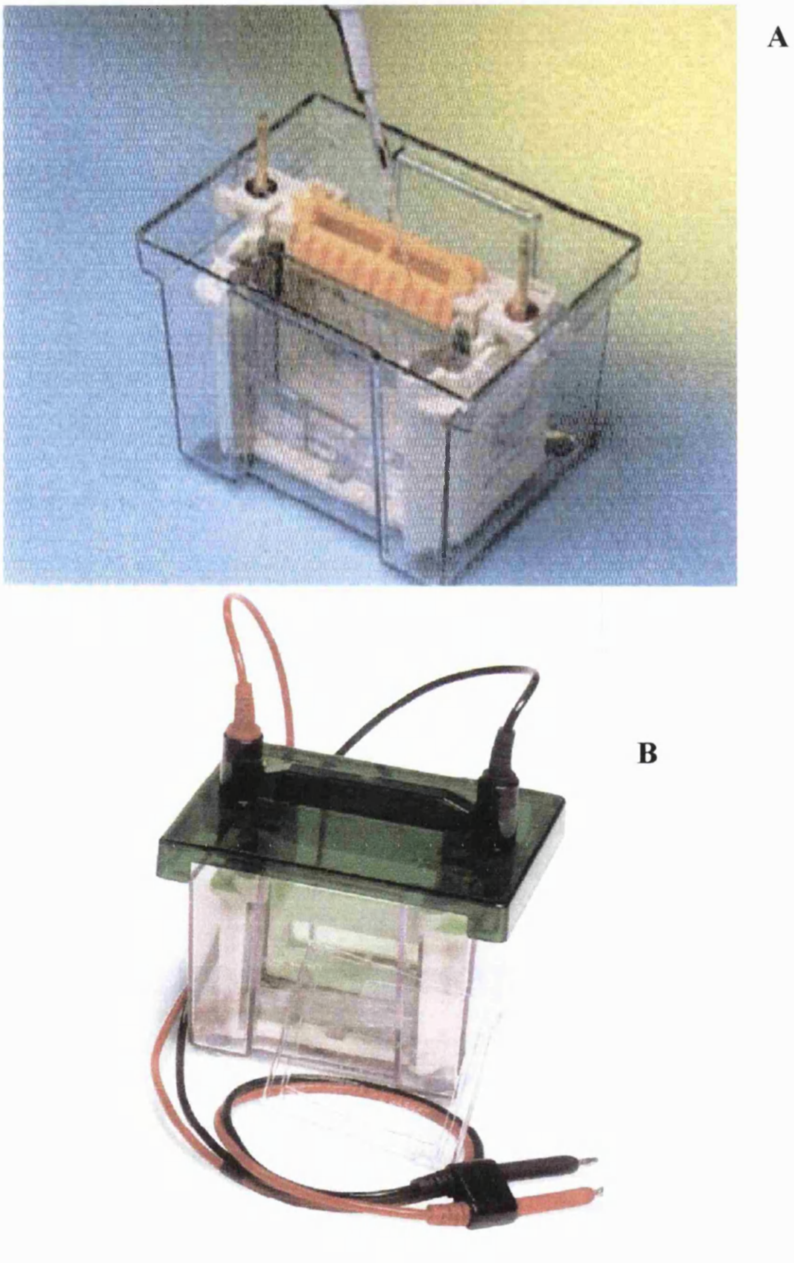


Figure 4.1 Gel electrophoresis setup. A) Loading gel plates with samples B) Loaded tank with supply leads ready to run

Sample staining

The gels were removed from the glass plates and placed into a small container. The gels were rinsed for approximately 10 minutes in distilled water this was then repeated. Next, the gels were covered with 20ml of Invitrogen, 'simply blue safe stain' (LC 6060) and were left staining for approximately 1 hour, depending on the required intensity and the amount of protein present. The stained gels were then rinsed again in distilled water for approximately 2 hours, which decreased the background staining. This improved protein band resolution during light densitometry.

Light densitometry

Once stained the resolving gels were placed into a biorad densitometer (Biorad Geldoc 2000). The gels were scanned and a density map produced along with a calculated proportional percentage with respect to the separate myosin isoform bands using the associated software (Biorad quantity 1 ver 4.2). The gel background was subtracted from the protein band density in order to align the baseline to zero.

Data analysis

Student t tests were used to compare mechanical variables between the two groups. The significance level set was 0.05. In order to determine the association between mechanical variables and the muscle MHC isoform composition, linear regression analyses were used.

4.3 RESULTS

Inertial sprint cycling

Lower limb volumes

Anthropometric measures of the lower limbs for all subjects are shown in Table 4.2

Group	Total limb volume	Lean limb volume
Elderly	7942 ± 210*	5352 ± 160*
Young	10205 ± 224	7670 ± 178

Table 4.2 Anthropometric measures of group mean lower limb volumes (cm^3) for the young and elderly subjects. All values are ± SE. * Significantly different from young group ($P < 0.001$).

Power output and optimal velocity

Figure 4.2 shows the absolute values of average crank power plotted against the associated crank velocity for both the elderly and young groups. It can be seen that the older group have significantly lower levels of both power output and velocity ($P = <0.001$). The mean group optimal velocity (V_{opt}) (120 ± 3.6 rpm, vs. 89 ± 5.8 rpm) ($P = 0.001$), and maximal power being (847 ± 46.8 W vs. 406 ± 52.9 W ($P = <0.001$), for the young and older subjects respectively.

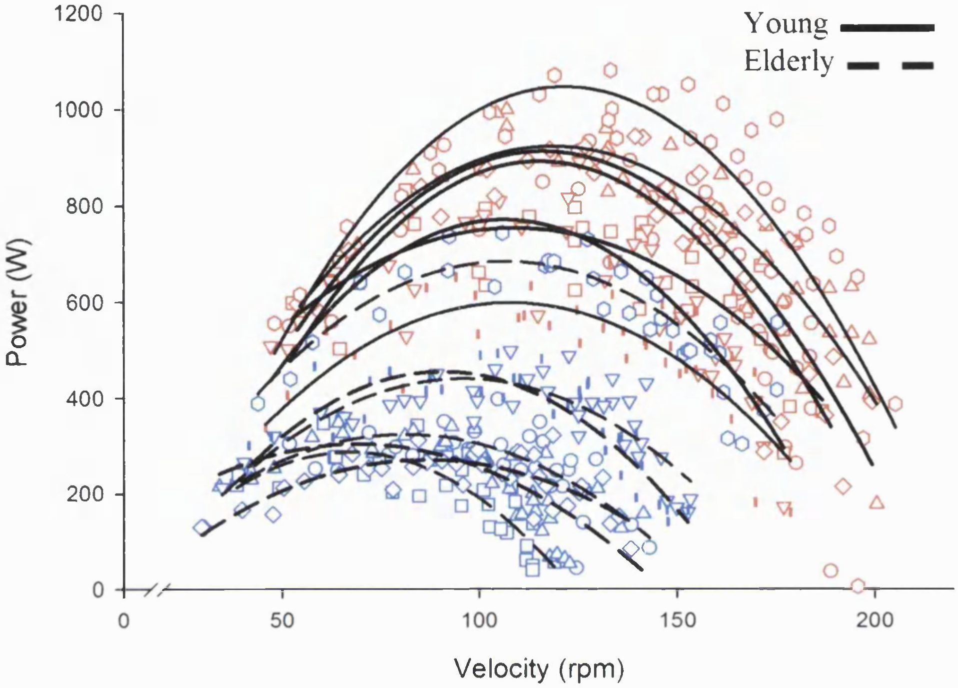


Figure 4.2 Individual subject power output vs. crank velocity during inertial sprint cycling. Each curve represents the polynomial fit to the average power and velocity values for each crank downstroke at all inertial loads tested. Young (●) Elderly (●).

Normalised power output and optimal velocity

Power output was normalised to the lean limb volume (muscle plus bone) in order to allow for the greater volumes observed in the young group. Using the normalised values for the third order polynomial fit, the values for maximal power for the young and elderly group were 0.11 W/cm^3 and 0.08 W/cm^3 respectively. Figure 4.3 shows the normalised group mean power output vs. the associated crank velocity. In spite of the power output being normalised for lean limb volume the younger group still produced significantly greater power output ($P = 0.01$).

Optimal torque

Mean torque was plotted against mean velocity during each crank down stroke (Figure 4.4). The torque generated at maximal power (T_{opt}) was determined for each individual from the line fit generated from the plots in Figure 4.4. The group mean value for T_{opt} was 14.7 ± 0.6 and 8.9 ± 0.7 Nm for the young and old group respectively. The young group generated significantly greater T_{opt} than the elderly group ($P < 0.001$). The torque and velocity obtained by the elderly group as a percentage of the young group is summarised in Figure 4.5.

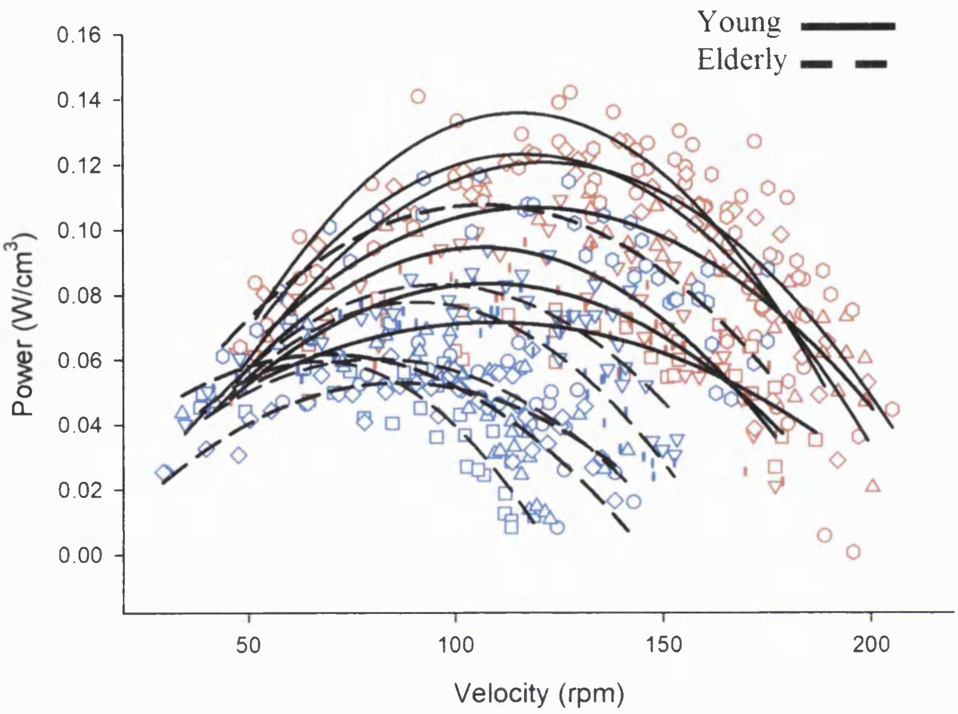


Figure 4.3 Individual normalised power output vs. mean crank velocity during inertial sprint cycling. All plotted data represents individual values at each crank downstroke during sprint cycling over the inertial range tested. Young (●) Elderly (●).

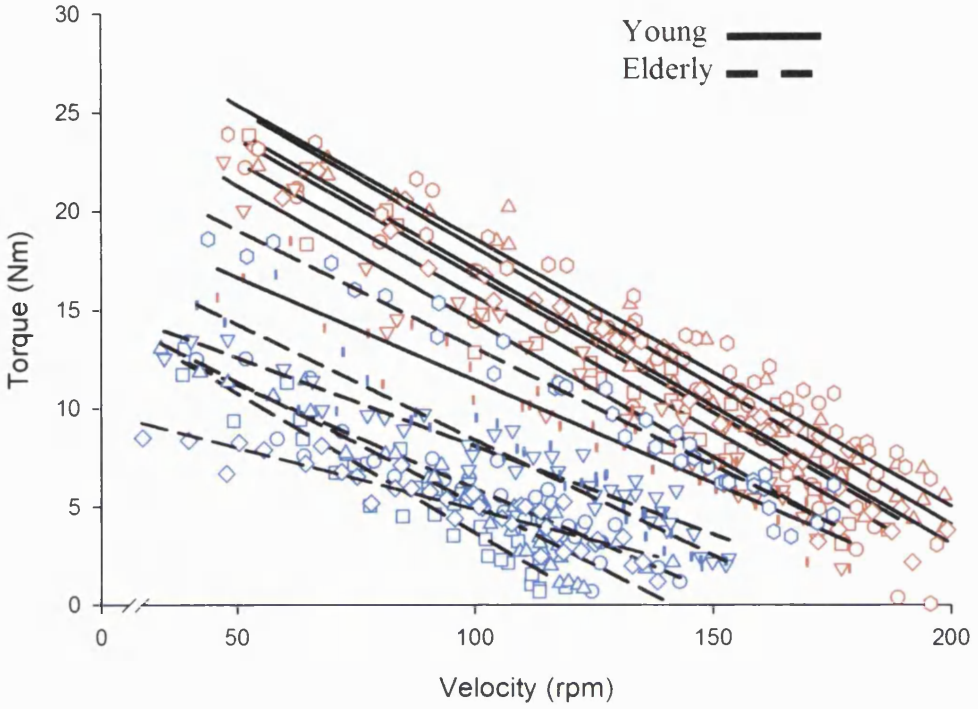


Figure 4.4 Individual plots of torque vs. velocity during inertial sprint cycling. All data represent mean values of torque and velocity during each crank downstroke over the inertial range tested. Young (●) Elderly (●)

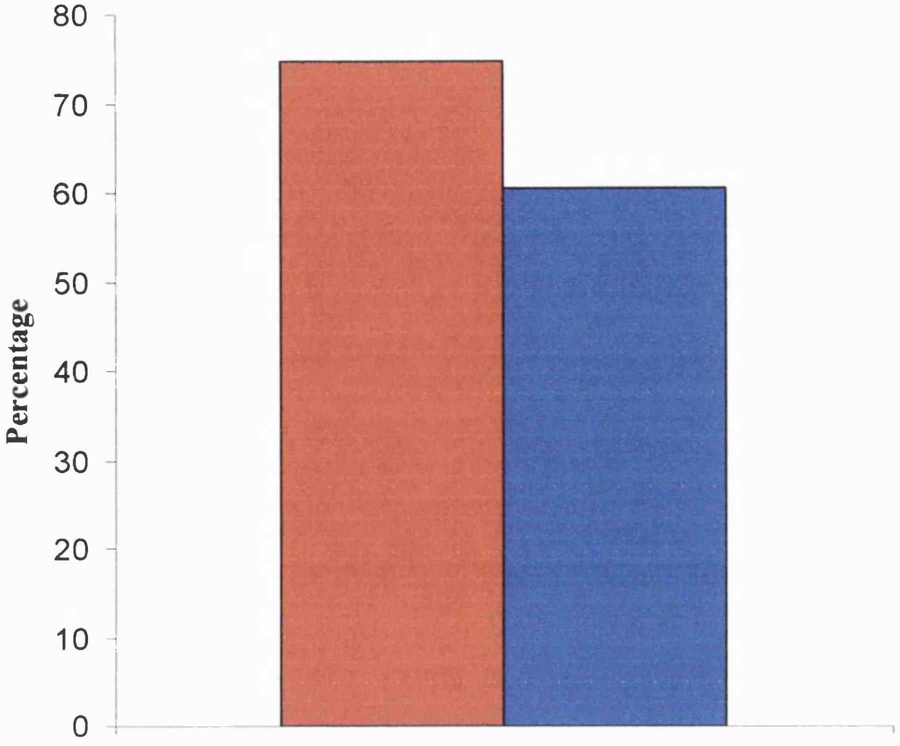


Figure 4.5 Elderly group mean values of velocity and torque at maximal power as a percentage of the young group mean values. Velocity (●) Torque (●).

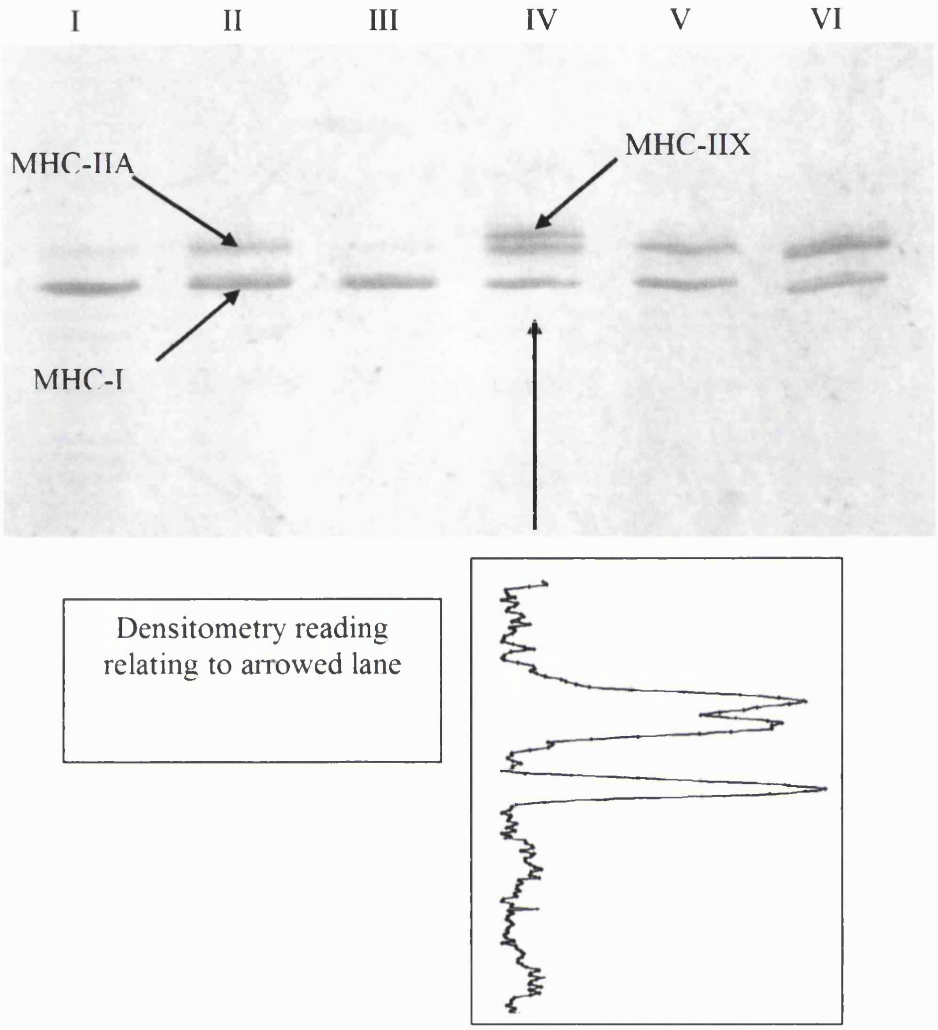
Percentage MHC II composition

The MHC-I and II composition of each subject was determined by SDS PAGE electrophoresis and expressed as a percentage value of the total MHC content. Plate 1 shows an example gel which contains both young and elderly myosin samples. For the purposes of analysis the MHC-II content was pooled (IIA + IIX), this was in part due to the fact that only single young subject had a sample containing both MHC-IIA and IIX .

Figure 4.6 shows the mean values of the relative percentage of MHC-II for the vastus lateralis. The MHC-II content was significantly greater in the younger group ($P = 0.01$). The relative percentage values for the young and elderly group being 52.1 and 25.6 % respectively.

Muscle percentage MHC II composition and optimal velocity

In order to allow the pooling of data, individual regression fit lines were examined to ensure no significant difference with respect to the slope. V_{opt} during sprint cycling is plotted against the percentage of MHC-II determined from the muscle biopsy for each individual (Fig 4.7). The MHC-II composition is seen to be positively related to the V_{opt}. The relationship between the percentage MHC-II composition and V_{opt} is significant ($r = 0.82$; $P < 0.001$).



Densitometry reading relating to arrowed lane

Plate 1. Lane II shows an elderly subject whose MHC composition was 92 % and 8 % MHC-I and II respectively. Lane IV shows a young subject with a MHC composition of 30 % and 70 % MHC-I and II respectively. The range of MHC-II in the young group was from 30 – 70 % whilst in the elderly group it was 8 – 64 %. Inset light densitometry reading showing three distinct peaks, one for each myosin isoform band.

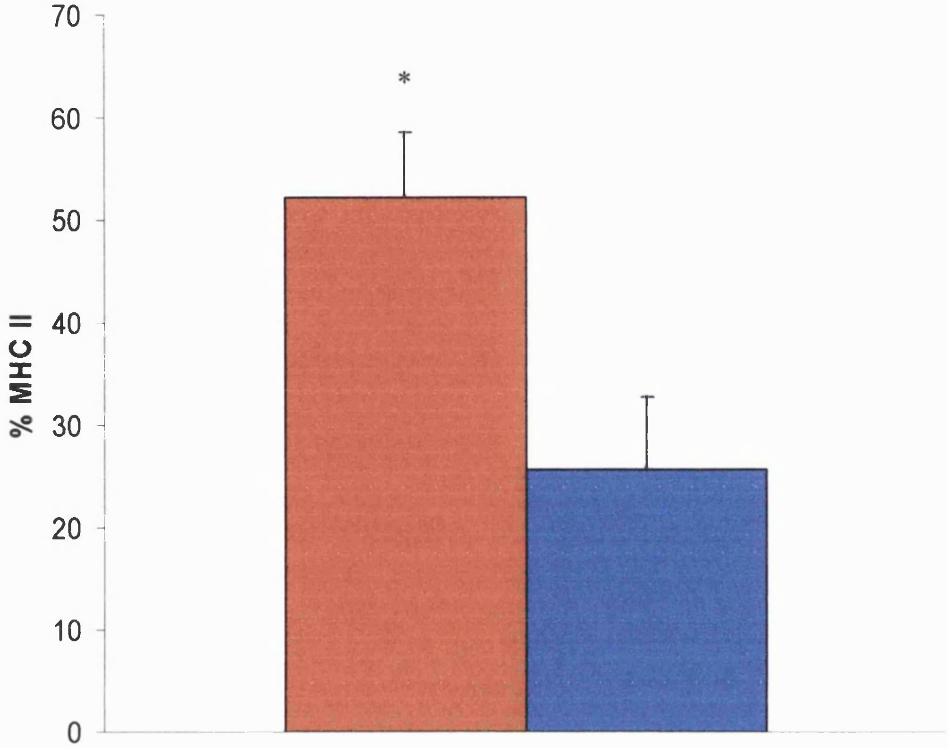


Figure 4.6 MHC-II (IIA + IIX) isoform composition of the vastus lateralis as a percentage of the total. Young (●) Elderly (●). Error bars are + SE. * Significant difference $P < 0.05$.

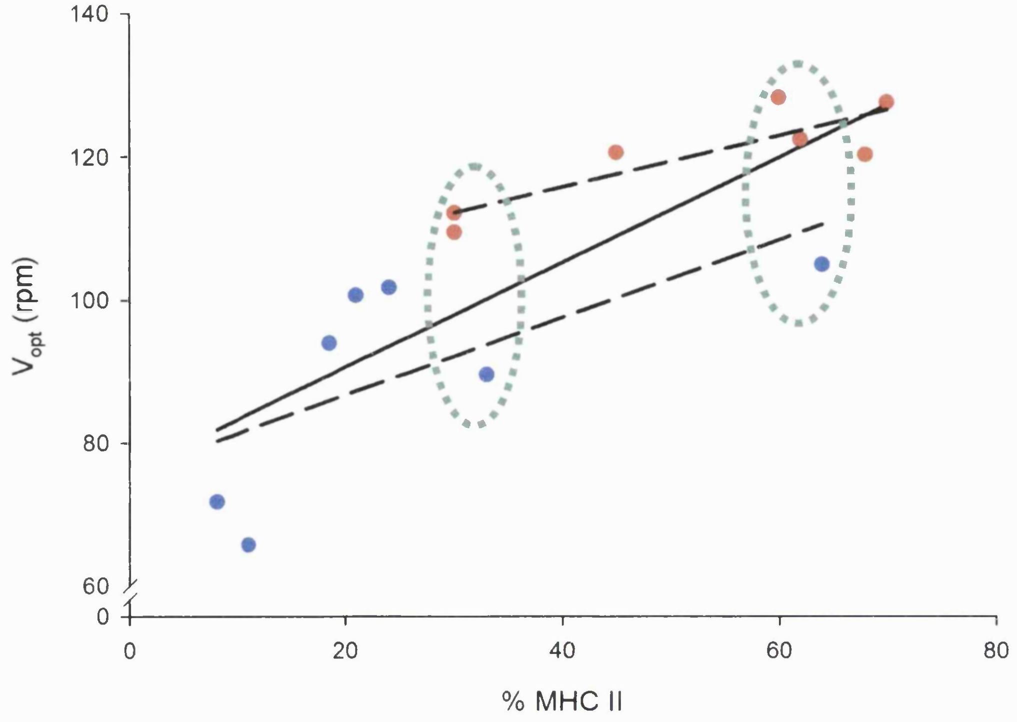


Figure 4.7 Relationship between the percentage MHC-II (IIA + IIX) composition of the vastus lateralis and the V_{opt} during inertial sprint cycling, solid line – pooled results ($y = 76.9 + 0.72x$ $P < 0.001$). Dashed lines show individual regression lines for each group. V_{opt} values for each subject determined from the polynomial curve of power output vs. velocity. Young (●) Elderly (●). Circles indicate differences in V_{opt} between young and old subjects for a similar percentage of MHC-II.

Percentage MHC-II composition and power output - cycle

The calculated power values were plotted against the percentage MHC-II composition of the vastus lateralis muscle. Figure 4.8 shows the relationship between the percentage MHC-II composition and the maximal normalised power output. The relationship is significant between these two variables ($r = 0.61$; $P = 0.02$).

Modified Nottingham Power Rig

Power output and optimal velocity

The mean values of power and velocity are shown in Figure 4.9. Consistent with the results from the sprint cycling is the finding that the elderly group produce significantly lower values of maximal power 165 ± 22 W and 411 ± 33 W for the elderly and young group respectively ($P < 0.001$). The mean V_{opt} was also significantly different between the two groups, being 19 ± 1.8 rads s^{-1} and 25 ± 1.6 rads s^{-1} for the elderly and young group respectively ($P = 0.04$).

Normalised power output and optimal velocity

As for the cycling power output the power output obtained on the modified NPR was normalised to the lean limb volume. Using the normalised values for the third order polynomial fit (Figure 4.10), the values for maximal power for the young and elderly group were 0.057 W/cm^3 ± 0.005 and 0.031 W/cm^3 ± 0.004. The normalised power values were still significantly greater for the young group ($P = 0.002$).

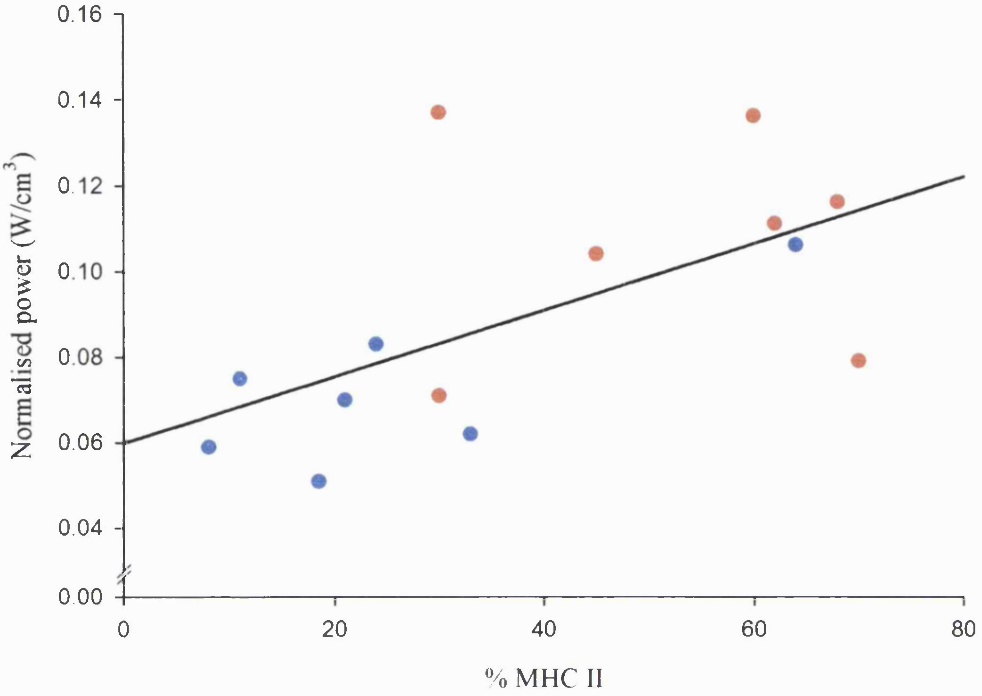


Figure 4.8 Relationship between percentage MHC-II (IIA + IIX) composition of the vastus lateralis and the maximal power output during inertial sprint cycling ($y = 0.06 + 0.0008x$ $P = 0.02$). Young (●) Elderly (●)

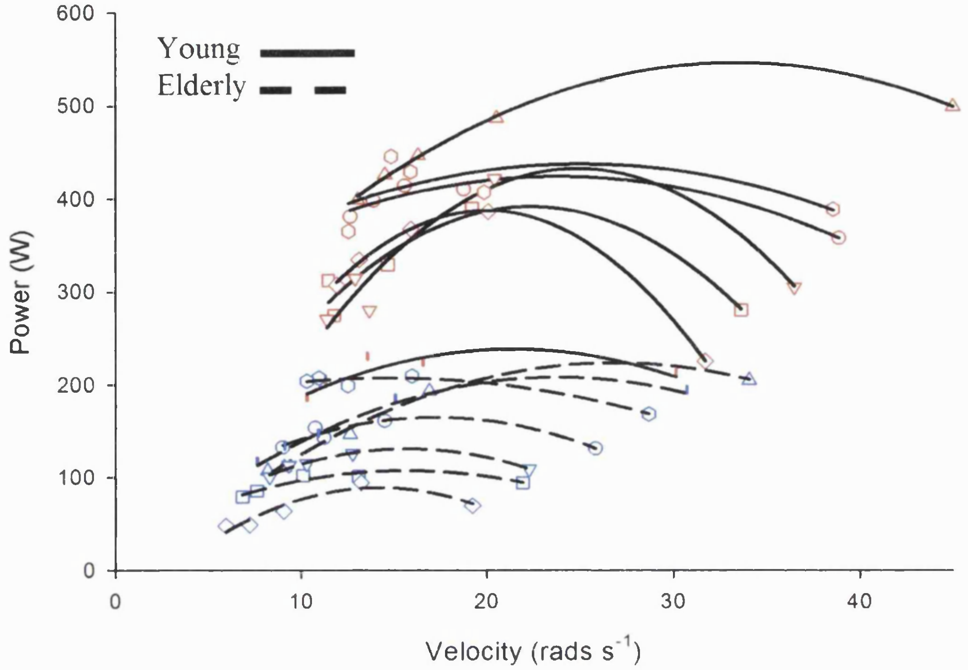


Figure 4.9 Mean power output vs. velocity during NPR exertions over five inertial loads. Young (●) Elderly (●)

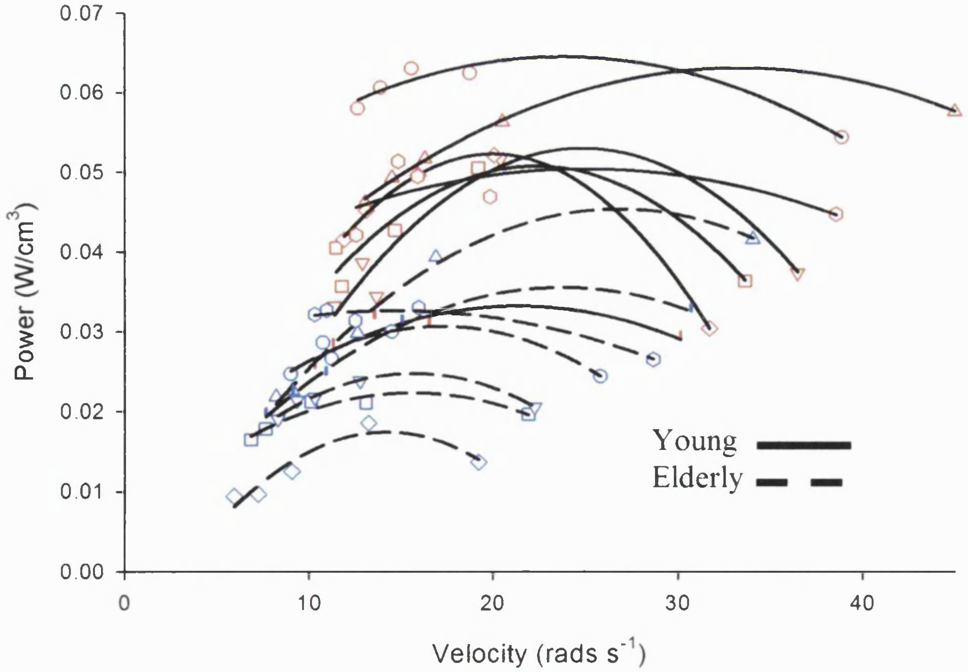


Figure 4.10 Normalised mean power output vs. velocity during modified NPR exertions over five inertial loads. Young (●) Elderly (●)

Optimal torque

In order to examine the relative importance of each component of power, torque - velocity plots were produced. Figure 4.11 shows the results of the modified NPR exertions for the two groups. A significant difference existed between the two groups in terms of optimal torque produced, 15.6 ± 0.9 Nm and 7.6 Nm ± 0.8 for the young and elderly group respectively ($P = <0.001$).

Comparison between the young and elderly group – components of power

The mean values for the young and elderly group for both torque and velocity obtained on the modified NPR have been summarised in Figure 4.12. It can be seen that the major determinant of differences in power output between the two groups is the torque produced. The elderly group produced 78 % and 49 % of the young group with respect to velocity and torque at maximal power.

Muscle percentage MHC-II composition and optimal velocity - MNPR

Figure 4.13 shows the relationship of V_{opt} during exertions on the modified NPR against the % MHC-II composition for each individual. The relationship between the percentage MHC-II composition and the V_{opt} was not significant ($r = 0.31$, $P = 0.27$).

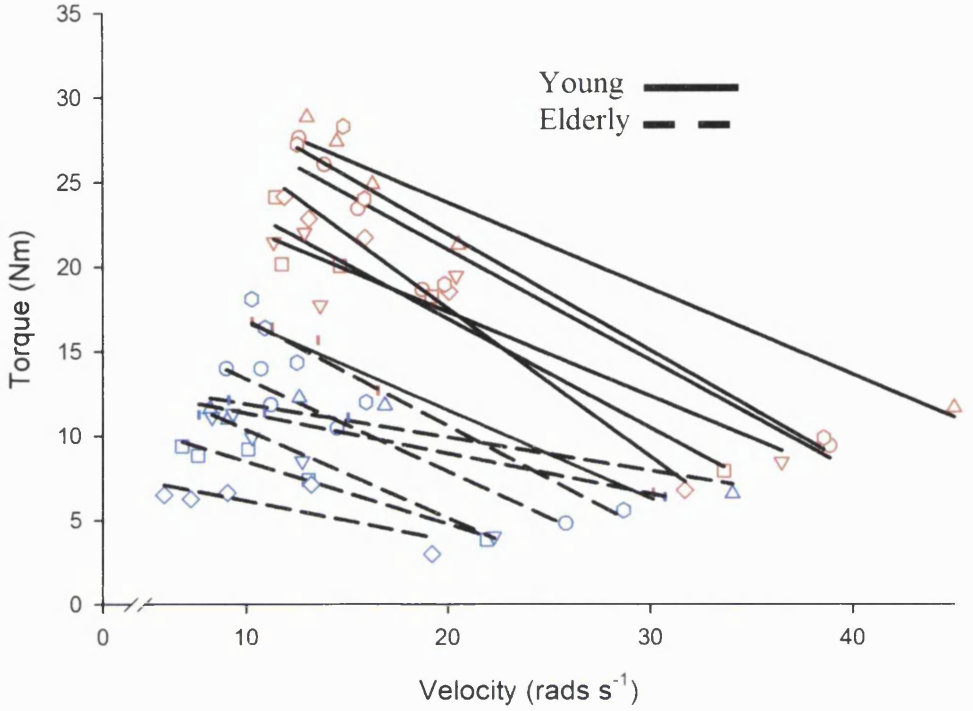


Figure 4.11 Torque vs. velocity during modified NPR exertions over five inertial loads. Young (●) Elderly (●)

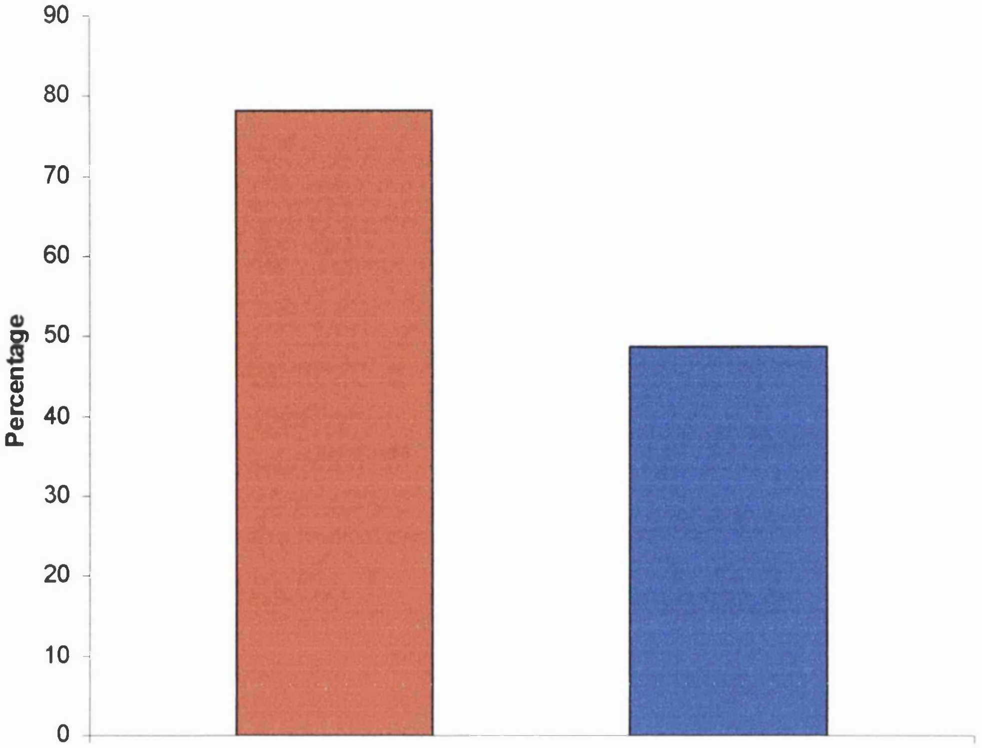


Figure 4.12 Velocity and torque of the elderly group at maximal power as a percentage of the young group. Velocity (●) Torque (●).

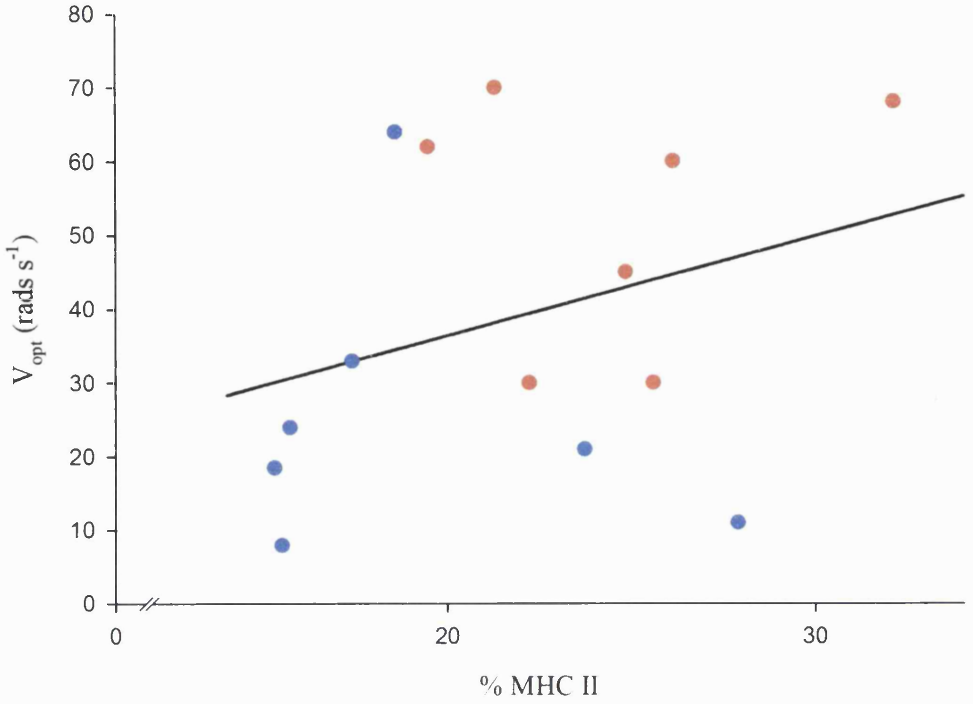


Figure 4.13. Relationship between the % MHC-II composition and the V_{opt} during modified NPR exertions over the inertial range tested ($y = 9.56 + 1.34x$ $P = 0.27$). Young (●) Elderly (●)

Percentage MHC II composition and power output - MNPR

The subject calculated power values for the exertions on the modified NPR at five different inertial loads were plotted against the percentage MHC II composition of the vastus lateralis muscle. Figure 4.14 shows the relationship between the percentage MHC II composition and the maximal power output. The percentage MHC II composition was not significantly related to the power output (r = 0.43, P = 0.12).

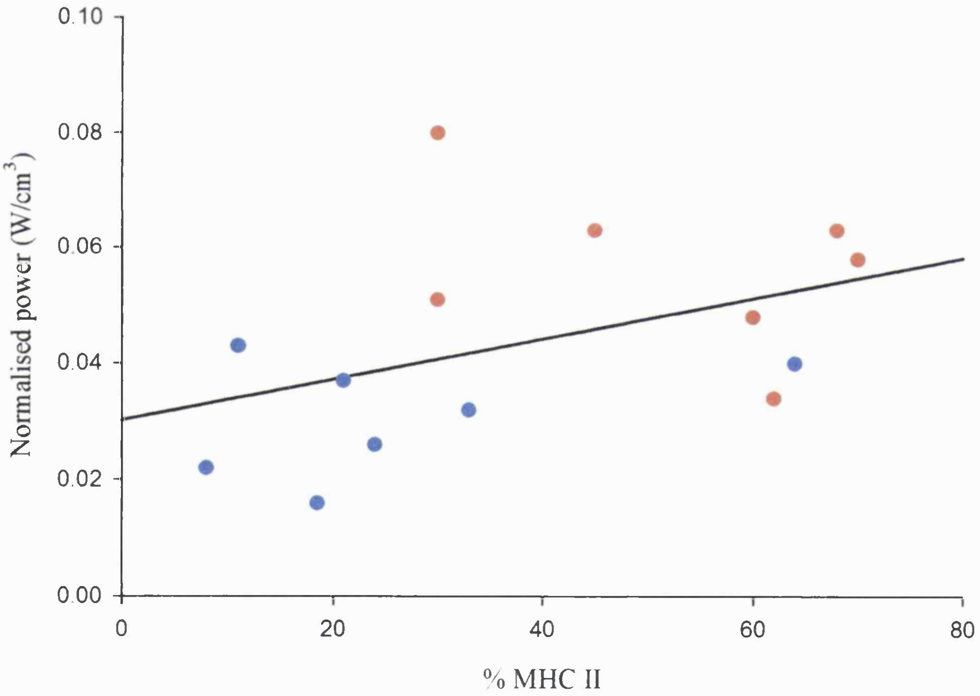


Figure 4.14. The normalised power output for all subjects plotted against the % MHC II ($y = 0.03 + 0.0003x$ $P = 0.12$). Young (●) Elderly (●)

Power output -comparison between modified NPR and sprint cycling protocols

Comparisons were made between the power outputs under the two experimental protocols. Figure 4.15 shows the association between the calculated power outputs for both protocols. It can be seen from the graph shown below that a very strong significant relationship exists between the two different methods of power measurement.

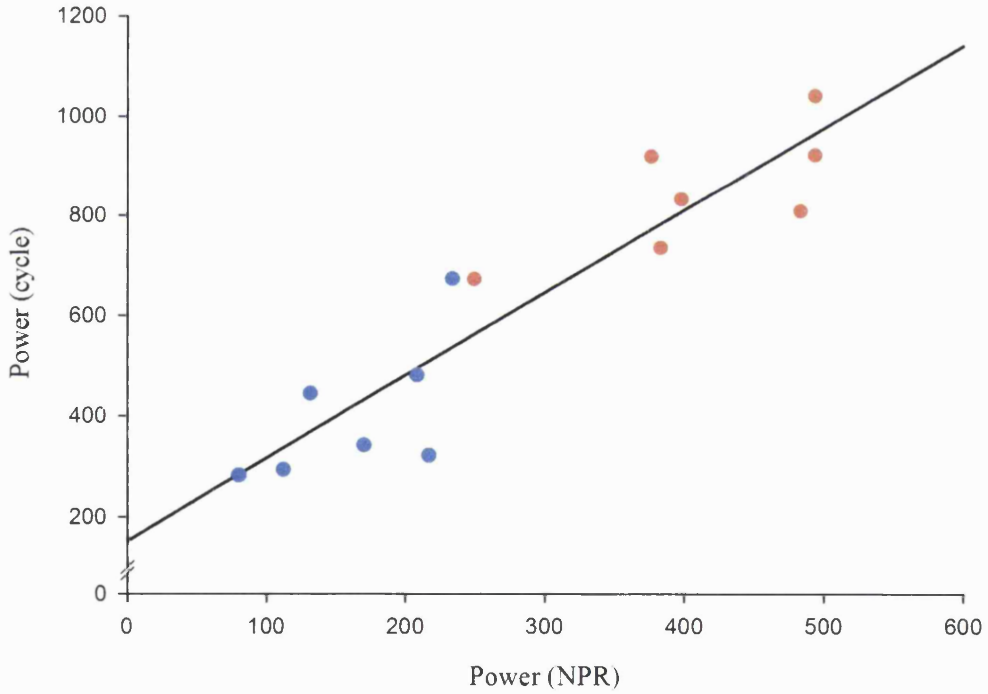


Figure 4.15 Power output during inertial sprint cycling vs. power output during exertions on the modified NPR ($y = 151.4 + 1.65x$; $r = 0.92$; $P = <0.0001$). Young (●) Elderly (●)

4.4 DISCUSSION

The main finding of this study was that the optimal velocity at which peak power occurs during sprint cycling was significantly related to the MHC-II composition of the vastus lateralis muscle in both young and old subjects. Power output was lower in the elderly group on both the modified NPR and the sprint cycling protocols, with the elderly subjects obtaining their peak power at a significantly lower velocity than that of the young group.

Muscle output characteristics and muscle composition

Of the studies carried out to compare the power output and V_{opt} values to the muscle composition during short term maximal cycling, Sargeant et al. (1994) reported that the muscle fibre composition, as determined histochemically, was related to V_{opt}. They observed that in two groups with a significant difference in their percentage of MHC-II fibres, a significant difference existed in their values of V_{opt}. Those with < 50 % of MHC-II fibres in cross section had a V_{opt} of 104 rpm, whereas those with > 50 % MHC-II fibres had a V_{opt} of 119 rpm. From this observation they suggested that the test could be indicative of the relative composition of the muscle fibres. This relates to the fact that the V_{opt} of MHC-II fibres is much higher than that for MHC-I fibres, the rationale therefore being that the V_{opt} of a mixed muscle being reflective of the relative composition of the whole muscle.

Hautier et al. (1996) examined the fibre type composition of the vastus lateralis muscle and related it to the power output and V_{opt} during friction loaded sprint cycling in young subjects (mean age 20. 3 years). They reported that the percentage area of type II fibres was positively and significantly correlated to the V_{opt} ($r = 0.88$).

MHC composition of the vastus lateralis – young vs. old

It was observed that a continuum existed across the age groups in terms of the relative content of the percentage MHC- II. The muscle samples from the young group contained significantly greater percentages of MHC-II and consequently significantly lower percentages of MHC-I. The values of MHC-I are in good general agreement with the data of Klitgaard (1990) who reported that elderly sedentary men had a 27 % higher content of MHC-I than their younger counterparts.

On examination of the evidence with regard to fibre type composition and ageing, early work by Grimby et al. (1982, 1984), reported similar fibre distributions in the vastus lateralis muscle of elderly men to that reported by Glenmark et al. (1994) in young students. They therefore concluded that there was no shift in fibre type distribution with age. However, others suggested that a shift in fibre type is seen towards a slower muscle fibre composition (Larsson et al. 1978, Karlsson and Larsson 1978, Örlander et al. 1978, Larsson et al. 1979, Scelsi et al. 1980). Similarly Jakobsson et al. (1988) who examined the anterior tibialis found differences between young and old muscle composition. Florini (1987) pointed out that caution should be exercised when counting fibres of 'representative' cross sections as not all fibres traverse the full length of the muscle and thus fibre numbers may not be representative of changes with age.

More recently, utilising whole vastus sections, Lexell et al. (1988), Lexell (1995), have suggested that the relative composition of the fibres is constant with ageing but that there is a preferential atrophy of type II fibres. The finding in the present study can be explained, in part, by the report that as individuals age the fast fibres show a preferential loss in size, therefore there will be relatively less fibre area expressing MHC-II. It has also been reported that aged muscle has an increased number of fibres consisting of more than one MHC isoform (Klitgaard et al. 1990, Andersen et al.1999); this is suggestive of an ongoing transformation process in the muscle and could again account for the lower proportions of MHC-II observed in the elderly group. The technique used here to examine the MHC composition of the muscle would identify this ongoing transformation of the muscle proteins with ageing, whereas the more traditional histochemical techniques are less sensitive to hybrid fibres and so may not give as accurate a representation of the muscle composition. A mechanism which could account for the observed lower relative percentages of MHC-II in the elderly group is that the synthesis rates of muscle myofibrillar proteins are lower in the older muscle (Welle et al. 1993, Yarasheski et al. 1993, Proctor et al. 1998, Short and Nair 2000). Balagopal et al. (2001) reported that the effect of age was to lower the transcript levels and synthesis rate of contractile proteins in elderly subjects. They reported a reduction of mRNA levels of MHC-IIA of 38 % from young (20 -27 years), to middle age (47 – 60 years) and a further reduction of 50 % from middle to old age (46 – 79 years).

Whereas mRNA for MHC-IIX decreased 84 % from young to middle age and a further 48 % from middle to old age, there were no reported reductions in the mRNA levels of MHC-I. This reduction in transcription levels of MHC-II is suggested to be due to either defects at the transcriptional level or alternatively DNA damage.

Relationship between muscle output characteristics and MHC isoform composition during sprint cycling

A number of studies carried out have reported that the MHC isoform composition of the muscle is directly related to the mechanical output properties of the muscle. Widrick et al. (1996) showed that there was a continuum of power output from chemically skinned single fibres containing MHC-IIX →IIA →I with the fibres containing MHC-IIX producing up to ten times the power output of the fibres containing MHC-I. Others have shown similar results, in particular the power output and the V_{opt} of fibres containing MHC-II producing up to nine times the power output of fibres containing MHC-I and up to four times the V_{opt} (Bottinelli et al. 1996, Bottinelli et al. 1999).

Larsson and Moss (1993) showed that within fibres expressing the same MHC isoform there was a range of values for the maximum velocity of shortening, such that some of MHC-II fibres were slower than MHC-I fibres. Bottinelli et al. (1994) showed in rat fibres that the variability could be explained by the myosin light chain isoform (MLC) composition. However, Larsson and Moss (1993) in human fibres concluded that no significant relationship existed between the MLC composition and V_{max}, although V_{max} was found to be lower in fibres expressing a combination of the slow and fast MLC isoforms of MLC_2 than in fibres which did not express the slow isoform (MLC_{2s}).

Harridge et al. (1995) examined the *in vivo* torque – velocity relationship of the triceps surae in both young and elderly subjects using electrically evoked contractions of the plantar flexors. They reported that the fast MHC was associated with torque generation at the higher angular velocities, but only in the older subjects. This was suggested to be in part due to the isokinetic speed of movement more closely representing the V_{max} of the elderly group, and therefore presenting a better representation of the myosin limitations to shortening in the elderly group. In general agreement with this study, they also reported higher percentages of fast myosin isoforms in the young group.

When the power output from the two groups was compared, the younger group generated significantly more power in absolute terms. In order to standardise for the larger volume of lean tissue observed in the young group all power data was normalised to lean limb volume (LLV) using skinfold data and anthropometric equations. This technique has limitations however, in that unlike magnetic resonance imaging (MRI) or CT scanning it lacks the ability to determine the volume of muscle alone. Other advantages of MRI and CT scans are that they are able to determine the intramuscular fat content which may be substantial in the elderly group. Thus using the anthropometric method the LLV may be overestimated in the elderly group due to this discrepancy. This may partly explain the young group still generating significantly greater levels of normalised power. However, with these limitations in mind there are suggestions of a qualitative mechanism and this can be explained in part by the differences in the MHC-II composition between the two groups. In support of this was the fact that the V_{opt} was also significantly higher in the young group. This, all things being equal should be independent of muscle mass. In addition, when both groups were pooled, the power output generated during sprint cycling was also positively correlated to the percentage MHC-II of the vastus lateralis. The T_{opt} for the old group can also be seen to be significantly lower than that for the young group, with the mean value for the old group being 61 % of that generated by the young group. The higher percentage of MHC II in the young group might lend itself to greater levels of absolute tension. The specific tension of fibres containing MHC-II has been shown to be greater then those fibres containing MHC-I (Bottinelli et al. 1996, Widrick et al. 1996a). In fibres containing either MHC-I or II isoform from young and old subjects the levels of specific force are lower in the old subjects (Larsson et al. 1997).

Of particular importance is the ability of the muscle to develop force rapidly due to the short time of contraction during the cycling down stoke. The rate of rise of tension being related to both the rate of cross bridge cycling and the diffusion rate of Ca^{++} within the myofibrils (Bottinelli et al. 2000). The rate of force redevelopment in human fibres using a slack test technique has shown that fibres containing MHC-I redevelop force up to ten times slower than fibres containing MHC-IIX (Harridge et al. 1996). The increased sensitivity of fibres containing MHC-II to Ca^{++} (Bottinelli et al. 1999), would allow the faster fibres to generate force at lower concentration levels of Ca^{++} and therefore possibly develop tension quicker. With respect to the Ca^{++} kinetics, it has been reported that with age reductions in the sarcoplasmic Ca – ATPase occur (Klitgaard et al. 1989) and also that there is an uncoupling of the dihydropyridine receptor – ryanodine receptors with age (Renganathan et al. 1997). These factors may be contributory in the reduced rate of force development seen in ageing muscle fibres containing a specific MHC isoform.

When the time to peak torque during cycling is examined at an intermediate level of loading (0.158 kgm^2), it was shown that the elderly group take longer to develop their peak torque than the younger group. The typical times to peak torque during a crank down stroke was approximately 140 ms and 110 ms for the old and young subjects respectively. This ability to rapidly generate torque is a contributing factor for the higher power outputs seen in the younger group especially at the lower inertial loads where contraction times are typically very short.

The correlation between MHC composition and V_{opt} is strong; but 33% of the variance of the V_{opt} cannot be explained on the basis of the MHC-II composition (based on r value of 0.82 – Figure 4.7). One explanation could be that differences in MLC isoform composition have an effect on muscle output. A number of animal studies have shown that differences in the MLC may explain differences in V_{max} in fibres containing the same MHC isoform (Bottinelli et al. 1994, Greaser et al. 1988). Bottinelli et al. (2000) state that the regulatory MLC1f and MLC3f, which are associated with MHC-II, affect the maximum shortening velocity of the muscle fibre, the suggested mechanism include the modulation of the S1 portion thereby altering its flexibility or alternatively strengthening of the actomyosin interaction. However the modulation effects of the MLC isoform on shortening velocity in humans is not as conclusive as the results seen in animal studies. Larsson and Moss (1993) reported no significant correlation between V_{max} and the proportion of fast alkali MLC isoforms from human skeletal muscle.

It has also been reported that qualitative differences exist between fibres possessing the same MHC isoform between young and old muscle (Larsson et al. 1997, Frontera et al. 2000). In general agreement with this it can be seen from Figure 4.7 that young and elderly subjects with a similar MHC-II composition the V_{opt} is greater for the young subjects. It has been suggested that this may be due to as yet unidentified MHC isoforms or a result of the complex interplay of MLC within the fibre; additionally it could be the result of additional, unknown thick and/or thin proteins which are modulating the mechanical properties of the muscle (Frontera et al. 2000). Other confounding variables which could affect the MHC – mechanical output relationship include post translocational modifications to the MHC and the density of the MHC within a given fibre, *in vivo* muscle fibre geometry and differences in the muscle – tendon compliance. In general agreement with the fibre studies, at the molecular level, Hook et al. (2001) has shown using an *in vitro* motility technique that the actin sliding speed from aged human, rat and mouse muscle was 18 - 25% slower than that from the respective young muscle.

Modified NPR and muscle output characteristics

In agreement with the cycling results, the power and V_{opt} values for the young group are significantly higher than those generated by the elderly group. However, the absolute values of power and torque are lower during the modified NPR exertions than during the sprint cycling. For explanation see Discussion (Chapter 3). When the components of power were analysed it could be seen that the torque component was the most affected by age (Figure 4.12). This finding can be explained in part by the fact that the exertion time was typically 300 – 500 ms. When it is known that the position for maximal torque occurs towards the middle of the exertion it is unlikely that sufficient time is allowed for full activation, particularly in the elderly group. Therefore as with the sprint cycling, the ability to develop tension rapidly is an advantage. This along with the contribution of the joint angle which would affect the ability to generate torque due to the muscle length tension / joint- lever relationship, would ultimately determine the ability of the subject to develop optimal tension during a given exertion.

In contrast to the close relationship between MHC-II isoform composition and V_{opt} during sprint cycling, the relationships between the percentage MHC-II, V_{opt} and power was less good for that during exertions on the modified NPR. However there was seen a trend toward an increase in power and V_{opt} with an increase in the percentage MHC-II. The reasons for the association between the MHC-II composition and muscle output characteristics being non significant may be in part due to the fact that there were only five data points (one for each inertial load), on which to fit the polynomial curve. This would affect the accuracy of the fit and the predicted values of power and V_{opt}. Whereas during cycling there were many points as each inertial load produced an overlap of torque and velocity data and each sprint produced approximately 6 – 12 data points. An improvement in the association between percentage MHC-II and power, V_{opt} might be produced if many more intermediate inertial loads had been used

There is also the possibility that the muscle recruitment may not be identical between the two protocols, during the modified NPR exertions, due to the seat position it is possible that a large amount of the thrust comes from the gluteal and hamstring muscle groups (see Figure A). This might affect the correlation between the MHC and the V_{opt} as the biopsy site was the vastus lateralis.

When comparisons of the power output were made between the two methods, a very good relationship was observed (Fig 4.15). This indicated that both methods were measuring a common function and therefore were equally as applicable to determine the muscle output characteristics during short term maximal exertions.

In conclusion the observations from the present experiments have shown that the MHC composition of the vastus lateralis in young and elderly subjects can be related to the muscle output characteristics during maximal exertions under inertial loading conditions, and that during inertial cycling, the ability to generate power at a given velocity appears to be determined by the MHC composition of the quadriceps muscle in both young and old subjects. This is in good general agreement with other human studies comparing muscle output characteristics with muscle composition. Here a simple test can be used which can give a reasonable indication of the fibre MHC-II composition in healthy males across a broad age range.

PART III

CHAPTER 5

A CROSS SECTIONAL STUDY OF MUSCLE POWER AND STRENGTH IN ELITE MASTER WEIGHTLIFTERS OF DIFFERENT AGES

5.1 INTRODUCTION

It is now known that in previously non-trained elderly people, muscle mass can be increased and muscle function improved (Charette et al. 1991, Roman et al. 1993). However, studying master weightlifters who have not just trained for a period of 12 weeks or so, but who have maintained high levels of physical training for many years, may aid in our understanding of the potential limits to which older muscles may function. The two groups studied here (untrained controls and elite master weightlifters aged 40 -87 years), who were age matched, allowed an examination into the differences between subjects who were long-term high resistance trained and those who were not resistance trained, in terms of the absolute level of muscle power, the ability of the muscle to maintain muscle power with age and its relationship to isometric strength.

In contrast to standard resistance training, the sport of Olympic weightlifting, which comprises two disciplines – the clean and jerk and the snatch, involves strength, power and large motor skill elements. The weightlifter is required to accelerate rapidly and brake large inertial loads and to coordinate the movement in order to complete the lifts correctly. For obvious technical and safety reasons Olympic weightlifting does not lend itself as a tool for comparing objectively muscle function of master weightlifters with that of untrained older subjects. However, the inertial testing system developed as part of this thesis provides an objective method for measuring power output in both trained and untrained individuals. Therefore, in the present study muscle function has been compared between these world class elite master weightlifters and non-trained older individuals in terms of lower limb power (using the modified NPR) and in terms of maximum isometric knee extensor strength.

The apparatus also had the advantage of being unfamiliar to both groups. It was hypothesised that, as the nature of Olympic weightlifting involves the rapid acceleration of large inertial loads, and the generation of high power outputs, these weightlifters would exhibit superior dynamic muscle function characteristics even when tested in an unfamiliar piece of apparatus when compared with age matched controls. It was also hypothesised that in light of this ability to generate high power, the age related differences in muscle power output would be less than those of untrained subjects and also that by being stronger the weightlifters would require higher inertial loads to express their maximal power output than their untrained counterparts. In addition, knee extensor strength was measured using isometric dynamometry to establish if the age related differences differed from those of power and also to evaluate the degree of co contraction during isometric knee extension.

Long term high intensity resistance training may also lead to possible differences in older subjects to that of older untrained subjects, with respect to the muscle fibre composition (Klitgaard et al. 1990). DeLuca (1997) suggested that the EMG median density frequency may reflect the muscle composition. Gerdle et al. (1988) reported an association between type I fibre proportion of the vastus lateralis during dynamic isokinetic contractions and the power spectrum of the EMG. The median density frequency of the power spectrum of the EMG signal was analysed here to attempt to identify any differences which may indicate underlying muscle composition differences. The iEMG of the hamstrings and the quadriceps was also used in order to identify the amount of co contraction during isometric knee extension.

The main aims of this study were:

1. Using an inertial system optimised for each individual, to determine whether elite master Olympic weightlifters show similar age related changes in muscle power as healthy non-trained individuals of a similar age
2. To compare and contrast the age related differences in power output and in isometric strength in elite level weightlifters and untrained age matched subjects using the optimised inertial system.
3. To determine if weightlifters require a higher inertial load in order to develop their maximal power output in contrast to the untrained individuals.
4. To determine whether the level of co contraction during an isometric test of strength was affected by the master weightlifters skill in lifting weights.
5. To utilise surface EMG in order to examine the power spectral properties which may be indicative of fibre type.

5.2 METHODS

Subjects

A field laboratory was established close to the competition warm- up area at the World Masters Weightlifting Championships held in Glasgow UK (1999). Fifty-four male competitors volunteered to take part in the study. Data obtained at this meeting were compared with data subsequently obtained on a similar number of untrained men defined as 'healthy' using the criteria of Greig et al. (1994). The physical characteristics of the subjects are shown in Table 5.1. All subjects were informed of the testing procedures prior to testing and signed a consent form agreeing to take part in the study. The study had the approval of Strathclyde University and the Royal Free Hospital Ethics committees.

Table 5.1 – Physical characteristics of the subjects. All table values shown as mean ± SE. Significant difference from control is shown (*)

		Age Group (Yrs)				
		40 – 49 (n=10)	**50 – 59 (n=11)**	**60 – 69 (n=16)**	**70 – 79 (n=13)**	**80 – 89 (n=4)**
Age (Yrs)	**Control**	45.4 (± 0.9)	53.1 (± 0.7)	64.2 (± 0.8)	74.5 (± 0.7)	84.3 (± 1.4)
	Weightlifter	42.7 (± 0.6)	53.9 (± 0.8)	64.1 (± 0.7)	74.3 (± 0.8)	84.2 (± 1.5)
Body Mass (kg)	**Control**	75.7 (± 2.4)	76.6 (± 2.3)	77.0 (± 2.3)	72.7 (± 2.2)	68.6 (± 7.5)
	Weightlifter	87.0 (± 7.8)	83.0 (± 4.6)	78.0 (± 2.8)	77.6 (± 3.5)	72.0 (± 6.5)
Height (cm)	**Control**	177.1 (± 2.3)	177.8 (± 2.4)	174.8 (± 1.5)	171.9 (± 1.5)	167.7 (± 3.5)
	Weightlifter	173.6 (± 2.7)	167.8 * (± 1.6)	168.3 (± 1.9)	167.2 (± 1.7)	164.3 (± 2.5)
Total lower limb volume (cm^3)	**Control**	8734.8 (± 276.3)	8903 (± 366.9)	8676.2 (± 241.5)	7706.3 (± 297.7)	7911.2 (± 507.9)
	Weightlifter	8680.2 (± 831)	7846.5 (± 312.8)	7626.3 (± 365.3)	7318.6 (± 345.8)	6816.1 (± 524.1)
Lean lower limb volume (cm^3)	**Control**	6159.8 (± 287.5)	6341.1 (± 298.0)	6103.5 (± 200.5)	5370.8 (± 251.2)	5359.0 (± 322.6)
	Weightlifter	7276.6 (± 830.7)	6490.0 (± 350.7)	6077.9 (± 300.7)	5477.1 (± 305.1)	4854.6 (± 508.1)

Anthropometry

See chapter 4 methods section.

Power testing

See Chapter 4 methods section. In contrast here 6 inertial loads were utilised and only the MNPR was used. The highest recorded value of peak power from the 18 exertions was taken as the maximal peak power (MPP). Subsequently the torque (TQ_{mpp}) and velocity (V_{mpp}) generated at MPP as well as the time taken to reach MPP were determined. The average power (AP_{mpp}) generated at this inertia was also calculated.

Isometric knee extensor strength

The isometric strength tests were performed on the same limb as that tested on the power dynamometer. The subject was seated upright with the knee flexed at 90^0 to the horizontal. The ankle was fixed in a restraining collar, which was attached to a non-extensible rod attached to a strain gauge assembly as shown in Figure 5.1. The ankle restraining assembly was adjusted to be at 90^0 to the lower limb to ensure accurate measurement of the turning moment. The force transducer signal was amplified and sampled by an A/D system and associated software (CED 1401 Cambridge, UK, Spike 2) at 2 kHz. Each subject carried out a total of three isometric knee extension efforts and three isometric knee flexion efforts. In all tests, the subjects were actively encouraged to give their maximal effort and a 1-minute rest period was given between contractions. In all cases, the test order was first power testing and second strength testing.

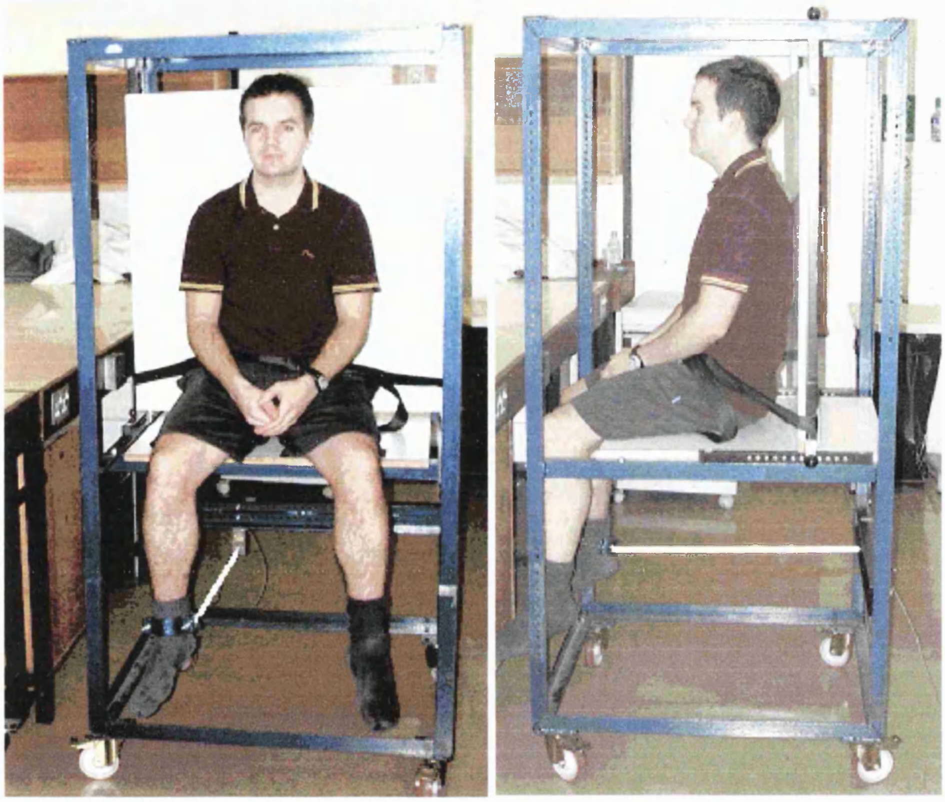


Figure 5.1 Subject positioning during isometric strength testing

sEMG

Simultaneous recording of the sEMG activity from the vastus lateralis and the long head of the biceps femoris muscles was made during the measure of isometric strength. The assumption was made that these two muscles were representative of their constituent groups (Carolan and Cafarelli 1992). Prior to mounting the recording electrodes, the skin surface was prepared by light abrasion and cleaning with alcohol swabs. Two silver/silver chloride bipolar electrodes (Medicotest, type N-10A, UK), with a 20mm inter-electrode distance (centre to centre) were placed midline on the muscle belly halfway between the centre of the belly and the distal myotendinous junction of the prepared site of the muscles. A ground electrode (Dantec Electronics mod. 13S97, UK) was placed around the ankle of the contra-lateral limb. The sEMG was band pass filtered between 3 and 1000Hz (Neurolog Filter NL 125, Digitimer, UK), preamplified (x1000) (Neurolog remote AC preamplifier NL 824, Digitimer, UK), amplified (x2) (Neurolog Isolation amplifier NL 820, Digitimer, UK), and A-D converted (type 1401, CED, Cambridge, UK) at a sampling rate of 2048Hz. To quantify the sEMG amplitude (RMS) and frequency (MDF) components, computer aided analysis was performed over a 1 second window of the plateau phase during the isometric effort. The spectral parameters were evaluated by standard Fast Fourier Transforms over 2048 samples (2^{11}). This procedure has been described in detail elsewhere (Macaluso et al 2000). MDF was adopted as a single measure to represent the power spectrum, because of its lower sensitivity to noise with respect to the mean frequency (MNF) (Stulen and DeLuca 1978). Percentage co-contraction during isometric knee extension was defined as the RMS sEMG of the biceps femoris during maximal voluntary isometric extension, divided by the RMS sEMG activity of the biceps femoris during maximal voluntary isometric flexion.

Data analysis

All data analysis was carried out using SPSS version 10. Data are presented as means ± SE. For clarity the subjects were assigned into decades (see Table 1 and 2). Multiple Student t - tests were applied to the data presented in Tables 1-2 and corrected for multiple comparisons using the Bonferroni method. Statistical comparisons between the weightlifters and untrained subjects controlled with respect to age for the variables measured were made using a general linear model and univariate analysis (ANOVA). For descriptive purposes, linear regression analysis was also performed on each group to determine the rate of change with age. Interpolated values at 45 years of age and 85 years of age were calculated using these regression equations. For between group comparisons, for the inertial load at which peak power occurred, a Student t - test was performed. Levels of significance for all tests were set at $P<0.05$.

5.3 RESULTS

The physical characteristics of the subjects are shown in Table 5.1. No significant differences were observed in body mass, height (apart from the 50-59 age group), age, total and lean lower limb volume between the two groups. However there was a tendency for the untrained subjects to have higher total lower limb volumes at all age groups and conversely for the weightlifters to have higher lean lower limb volumes (muscle and bone), at most age groups.

Competition performance

Figure 5.2 shows the lift performance data from the 1999 World Championships for the competitors tested. The lifts comprise the clean and jerk which is a two stage lift, first to the chest and then above the head and the snatch, which consists of a single lift to above the head. Of the 54 competitors tested, 15 were winners of their respective age and weight categories. A further 11 were silver medallists and 16 others were bronze medal winners (see Appendix I for weightlifter performance table). It can be seen from Figure 5.2 that lift performance declines with age, this is still the case when the results are normalised to body mass.

Power output

The values of MPP and AP_{mpp} generated on the MNPR by each subject can be seen in Figure 5.3 and are summarized by age group in Table 5.2. Both MPP and AP_{mpp} declined as a function of age in both groups in spite of load optimisation. No significant difference in the rate of decline of MPP ($r^2 = 0.54$, $P<0.05$) or AP_{mpp} ($r^2 = 0.48$, $P<0.05$) (see Table 5.4) was observed between the two groups.

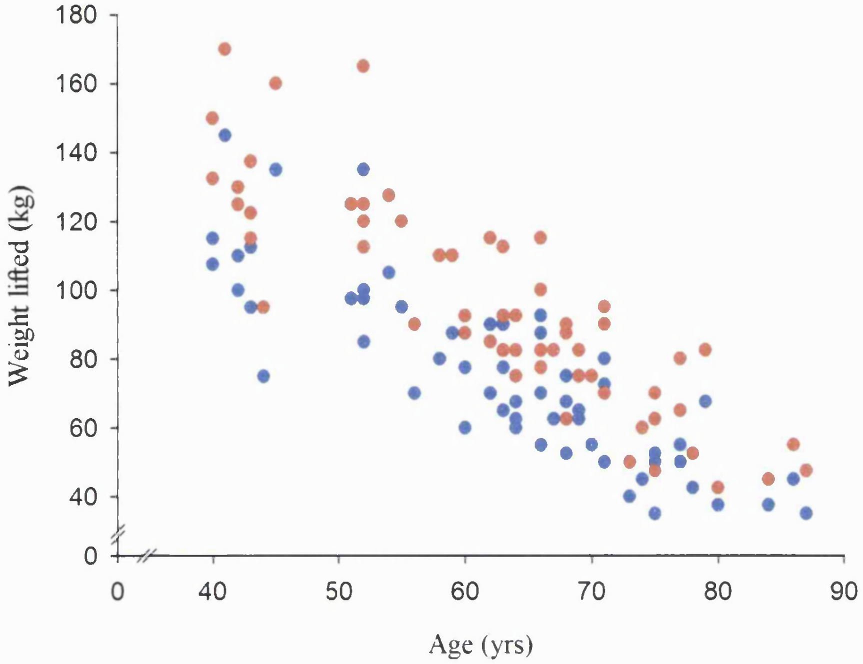
On average, the weightlifters generated 35% more AP_{mpp} ($P<0.05$) and 32% more MPP ($P<0.05$) than the control subjects. Normalisation of power values with respect to body mass or lean lower limb volume made no significant difference to age related differences or group related differences. The difference in decline of MPP between the two groups was 4.5 W/yr, and the 95% CI for the difference was –0.67 – 10.04W/yr.

Figure 5.4 illustrates the number of subjects generating MPP at each of the inertial loads tested. Overall the weightlifters required a higher inertia to express their MPP than the control subjects ($P<0.05$). The torque (TQ_{mpp}) and velocity (V_{mpp}) generated at MPP are shown in Figures 5.5A and B. Figure 5.5C shows the time taken to reach MPP (T_{mpp}). TQ_{mpp} declined with increasing age in both groups ($R^2 = 0.49$, $P<0.05$) but the rates of decline were not significantly different between the two groups. On average, the levels of TQ_{mpp} generated by the weightlifters were 34% greater ($P<0.05$) than those of their untrained counterparts. V_{mpp} declined significantly with age for both groups ($R^2 = 0.35$, $P<0.05$) and on average there was no significant difference between the two groups in the rate of decline with age. T_{mpp} increased with age in both the weightlifters and controls ($P<0.05$) and on average, the values of T_{mpp} were significantly lower for the weightlifters ($P<0.05$).

Isometric strength

An original record of an isometric contraction with the associated sEMG is shown in Figure 5.6. The isometric knee extensor strength data are presented in Figure

5.7 and Table 5.3. Isometric strength declined with age in both the weightlifters and untrained controls ($r^2 = 0.29$, $P<0.05$), but again there was no significant difference in the rate of decline of strength between the two groups (Table 5.4). On average, the weightlifters had significantly ($P<0.05$), higher average values (32%) for isometric extensor strength (See table 5.4).



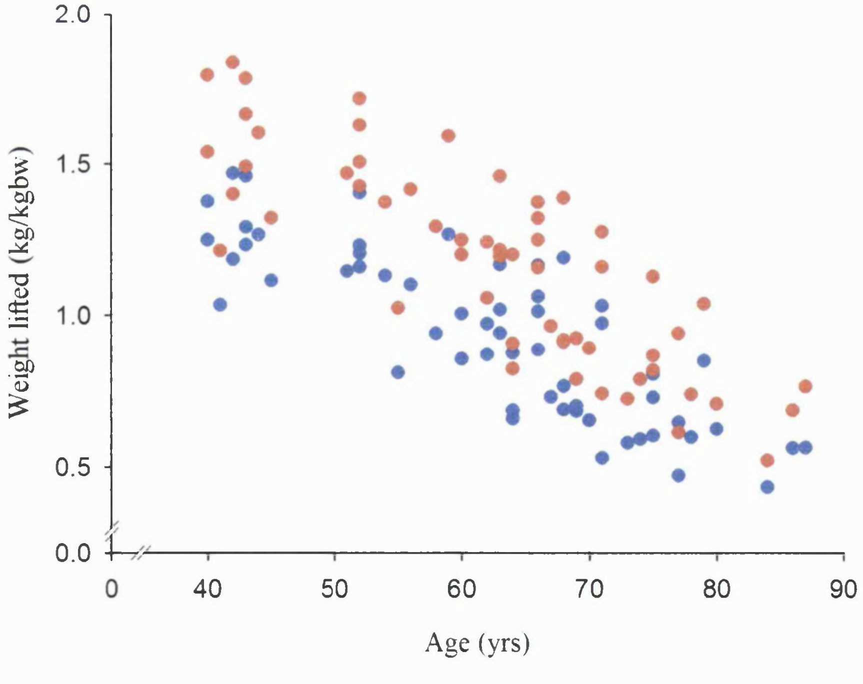


Figure 5.2 Weightlifters performance data. Clean and jerk (●), Snatch (●)

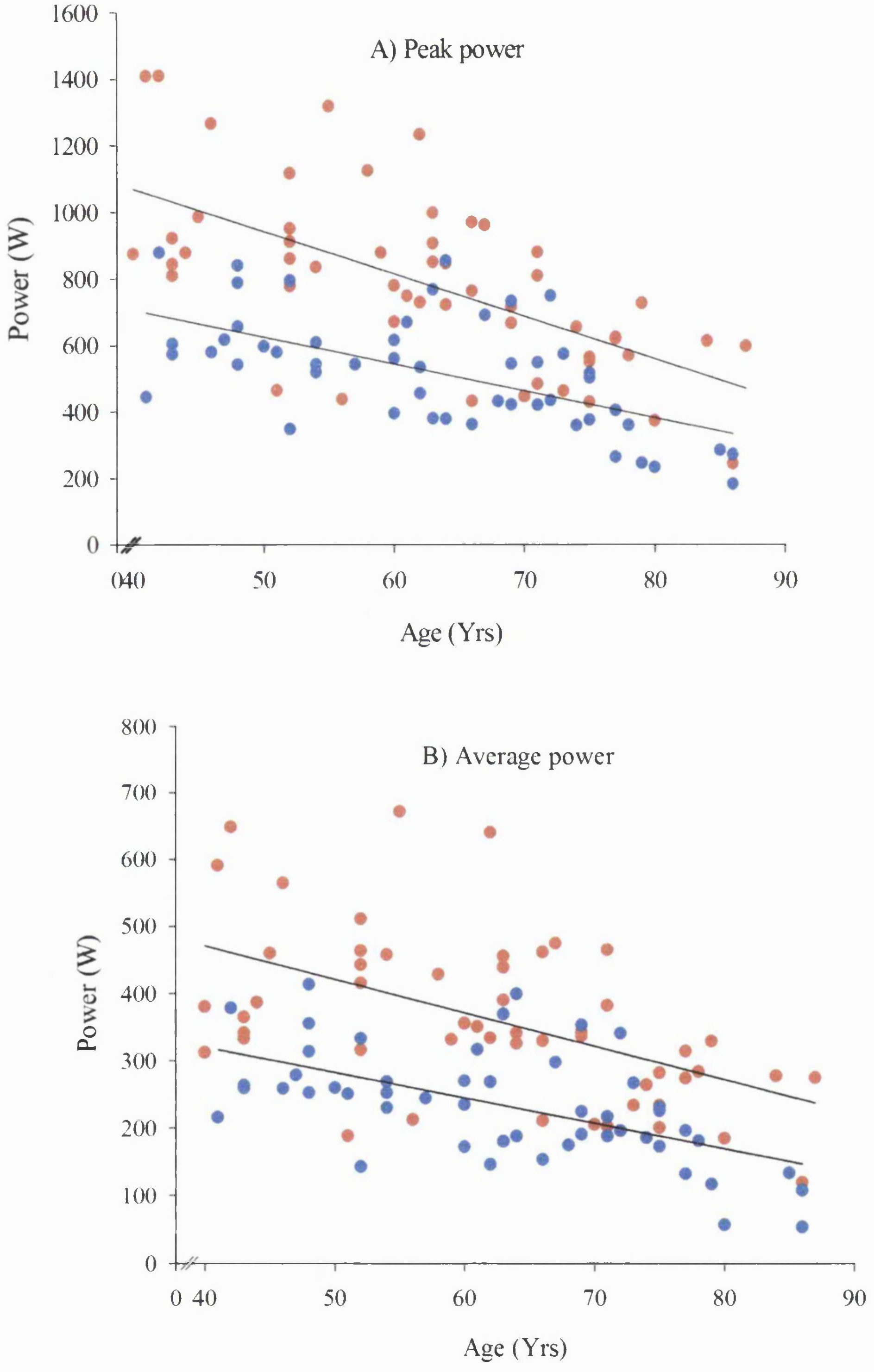


Figure 5.3 Absolute values for A) MPP. (y = 1029 – 8.1x controls, y = 1581 – 12.8x weightlifters) B) AP_{mpp}. (y = 471 – 3.8x controls, y = 669 – 4.9x weightlifters). Control (●), Weightlifters (●)

Table 5.2 Mean values (± SE) for MPP and AP$_{mpp}$. Significant differences from controls are shown (*)

		Age Group (Yrs)				
		40 – 49 (n=10)	**50 – 59 (n=9,11)**	**60 – 69 (n=16)**	**70 – 79 (n=13)**	**80 – 89 (n=4)**
MPP (W)	**Control**	653.1 (± 44.1)	561.4 (± 38.7)	549.4 (± 39.2)	442.1 (± 37.7)	242.6 (± 22.9)
	Weightlifter	1028.4 (± 75.4)*	879.9 (± 79.4)*	811.7 (± 45.1)*	600.9 (± 38.5)*	456.0 (± 89.5)
AP$_{mpp}$ (W)	**Control**	299.5 (± 20.2)	248.7 (± 16.3)	246.7 (± 20.3)	204.2 (± 15.8)	88.1 (± 19.7)
	Weightlifter	438.6 (± 38.2)*	404.0 (± 41.3)*	384.4 (± 23.7)*	282.7 (± 21.3)*	214.5 (± 38.3)

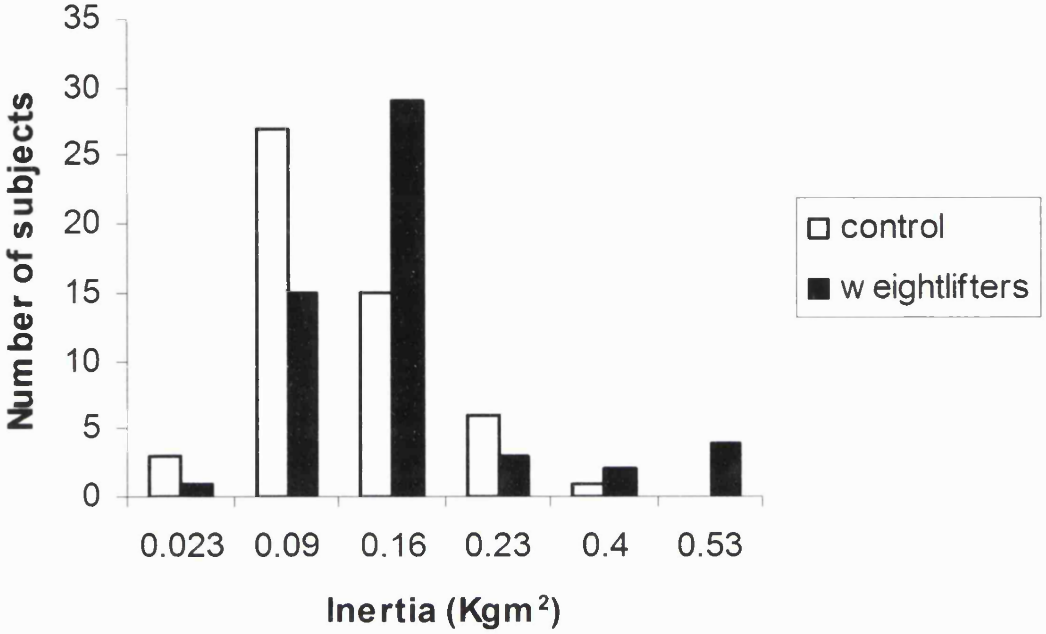
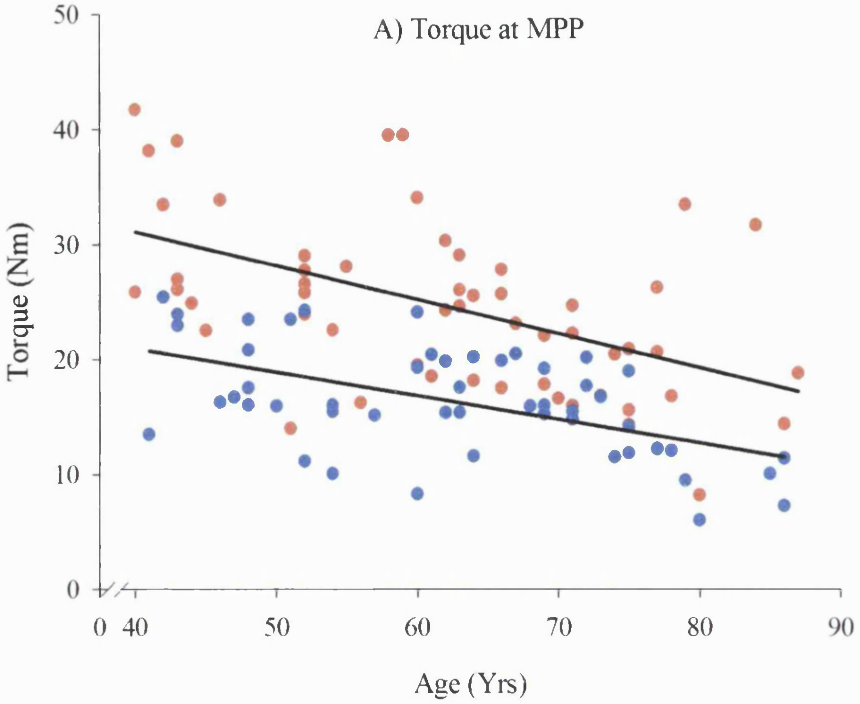
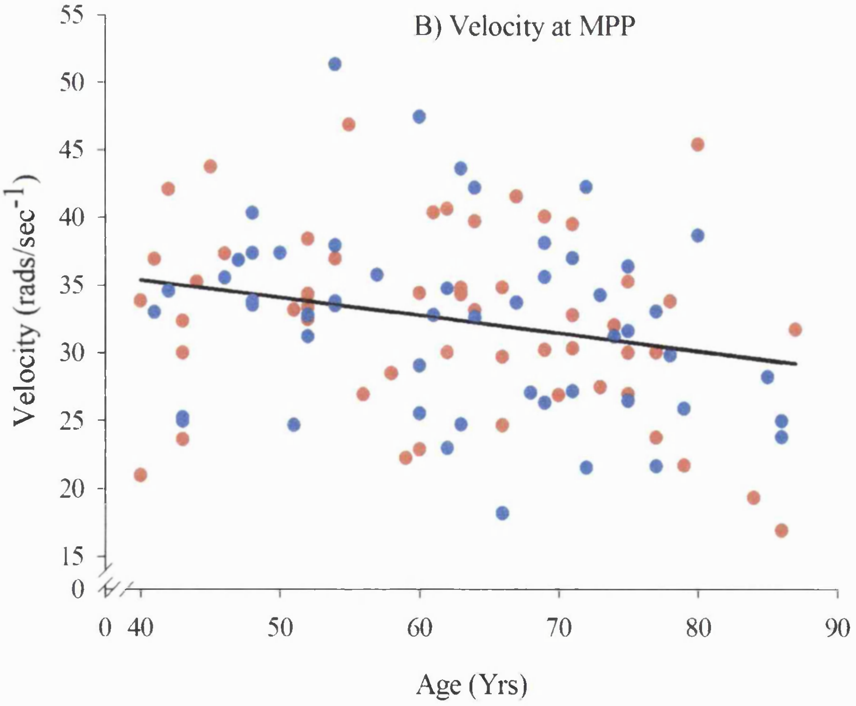


Figure 5.4 Histogram showing number of subjects obtaining MPP at a specific inertia. Weightlifters showing significantly higher values of inertia than controls at maximal peak power ($P<0.05$).


A) Torque at MPP
Torque (Nm)
50
40
30
20
10
0
0
40
50
60
70
80
90
Age (Yrs)


B) Velocity at MPP
Velocity (rads/sec^{-1})
55
50
45
40
35
30
25
20
15
0
0
40
50
60
70
80
90
Age (Yrs)

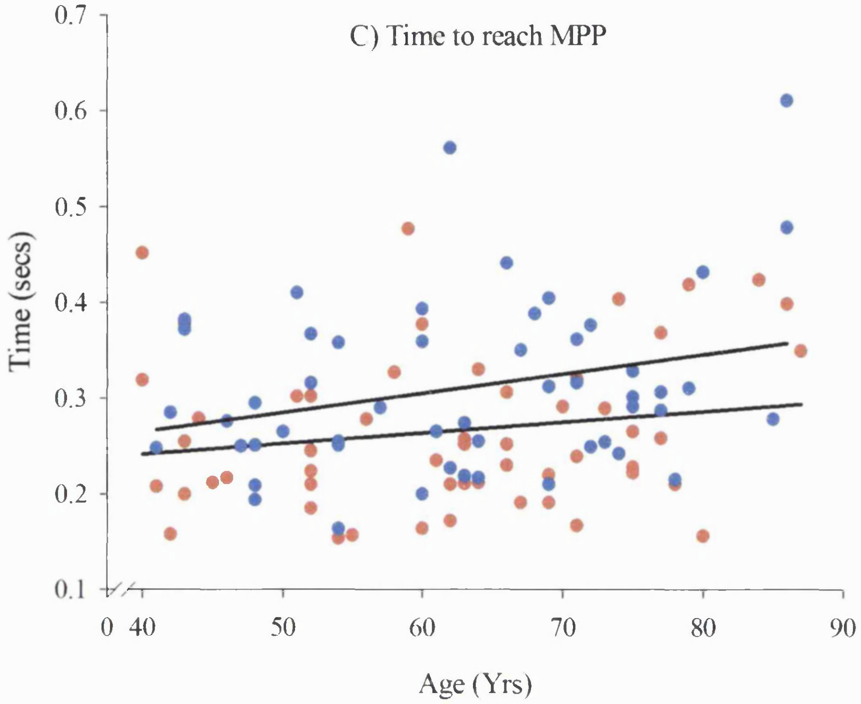


Figure 5.5 A) Torque (TQ_{mpp}) ($y = 29 - 0.21x$ controls, $y = 40 - 0.13x$ weightlifters) B) Velocity (V_{mpp}) ($y = 41 - 0.13x$ controls, $y = 41 - 0.14x$ weightlifters) and C) Time (T_{mpp}) values obtained at MPP ($y = 0.18 + 0.002x$ controls, $y = 0.19 + 0.001x$ weightlifters). Controls (●) and Weightlifters (●)

EMG

The MDF of the sEMG obtained from the vastus lateralis during maximal isometric knee extension is shown in Figure 5.8A. There was no effect of age on MDF in either group. However, weightlifters exhibited higher average values, 66.4Hz ±1.52 as against 58.2Hz ± 1.55 for the control group ($P<0.05$). Figure 5.8B shows the RMS values for the sEMG where there was a significant decline as a function of age in the weightlifters but not in the controls ($r^2 = 0.40$, $P<0.05$). On average, the weightlifters produced significantly higher values of RMS ($P<0.05$). The activation of the biceps femoris muscles during isometric leg extension, (co contraction), expressed as a percentage of the maximum biceps femoris activity is shown in Figure 5.8C. No significant effects of age, rates of decline or differences between the two groups were observed.

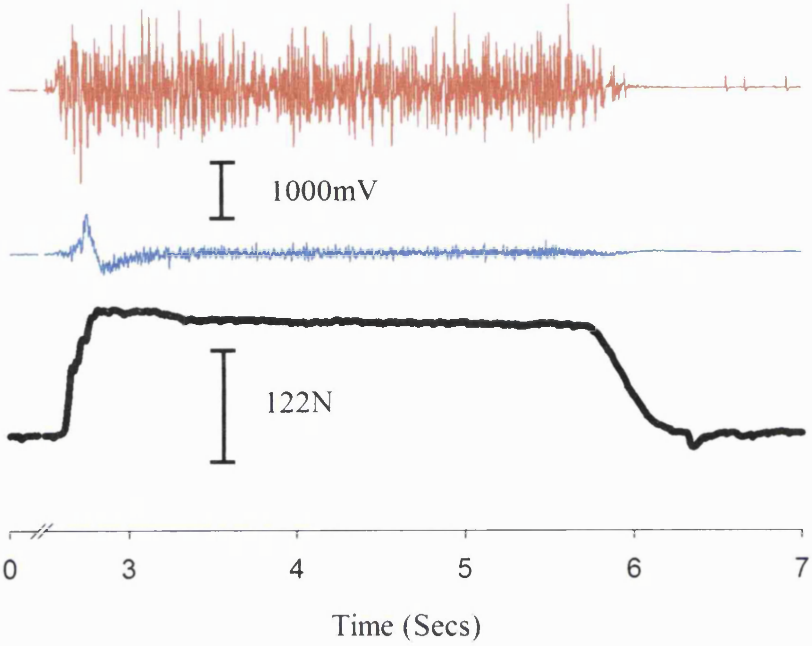


Figure 5.6 An original record showing the strain gauge signal during isometric knee extension (—), along with the associated sEMG from the vastus lateralis (—) and biceps femoris (—) muscle groups. For clarity the sEMG signals have been offset.

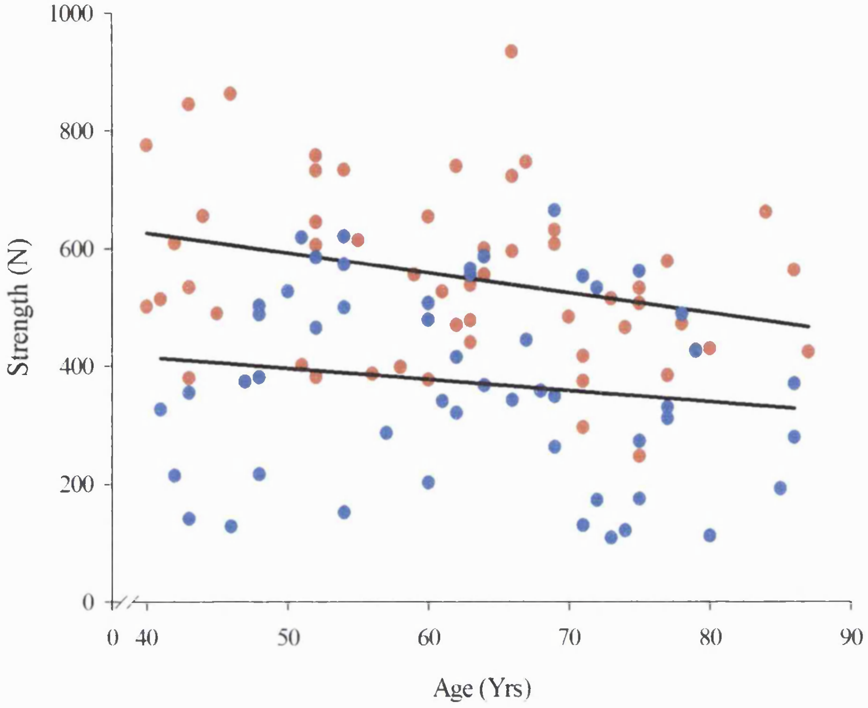


Figure 5.7 Isometric knee extensor strength (y = 491 – 1.9x controls, y = 763 – 3.4x weightlifters). Controls (●), Weightlifters (●)

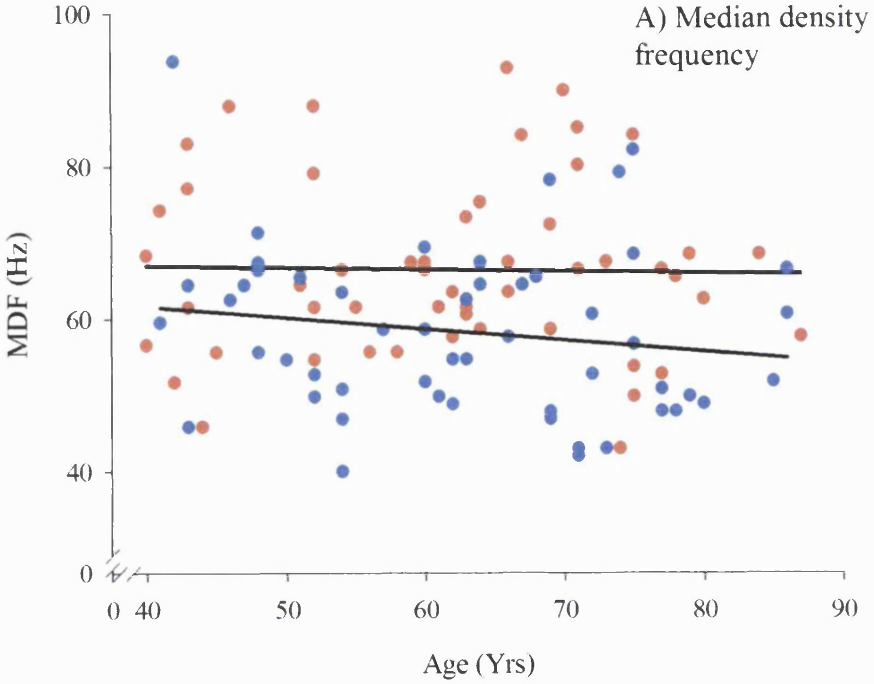
Table 5.3 Mean values for isometric knee extensor strength. Significant differences from controls are shown (*)

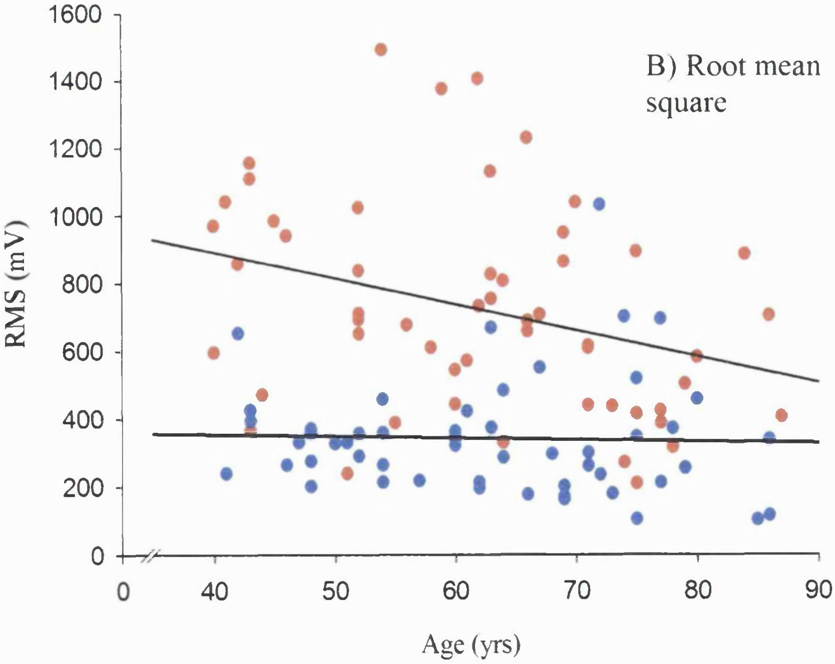
Isometric strength (N)	**Control**	313.1 (± 42.1)	481.3 (± 53.5)	422.7 (± 31.9)	321.8 (± 48.2)	238.4 (± 55.6)
	Weightlifter	617.3 (± 51.9)*	565.4 (± 45.2)	601.3 (± 34.8)*	438.4 (± 26.2)	519.6 (± 57.2)

Table 5.4 –The percentage difference between the mean values of the weightlifters with respect to the control subjects is shown in Column I. The age related decline for both groups, in absolute units per year is shown in columns II-III. The age related decline for both groups in relative units per year (expressed as a percentage of a 45-year-olds value is shown in columns IV-V. The percentage value of an 85-year-old relative to that of a 45-year-old is shown in columns VI-VII.

* Weightlifters significantly different from controls ($P<0.05$). † Significant decline with age, ($P<0.05$).

	I) Average % difference between groups	**II) Weightlifter absolute rate of change**	**III) Control absolute rate of change**	**IV) Weightlifter relative rate of change**	**V) Control relative rate of change**	**VI) Weightlifter 85 yr old as a % of a 45 yr old**	**VII) Control 85 yr old as a % of a 45 yr old**
Normalised clean & jerk ($kg \cdot kgbw^{-1}$)	N/A	$2.2 \bullet 10^{-2}$	N/A	1.4	N/A	44	N/A
Normalised snatch ($kg \cdot kgbw^{-1}$)	N/A	$1.8 \bullet 10^{-2}$	N/A	1.4	N/A	44	N/A
MPP (W)	32*	12.6†	8.1†	1.3	1.2	49	51
AP_{mpp} (W)	35*	4.9†	3.8†	1.1	1.5	55	50
TQ_{mpp} (Nm)	34*	0.29†	0.2†	0.9	1.0	60	59
V_{mpp} (rads sec^{-1})		0.13†	0.14†	0.4	0.4	85	87
T_{mpp} (secs)	-13*	$1.3 \bullet 10^{-3}$ †	$2.3 \bullet 10^{-3}$ †	0.5	0.8	80	70
Isometric strength (N)	32*	3.4 †	1.9†	0.6	0.5	78	81
RMS (mV)	53*	7.7†	0.51†	0.9	0.1	64	94
MDF (Hz)	12*	$2.6 \bullet 10^{-2}$	0.15	$3.9 \bullet 10^{-5}$	0.2	98.3	90





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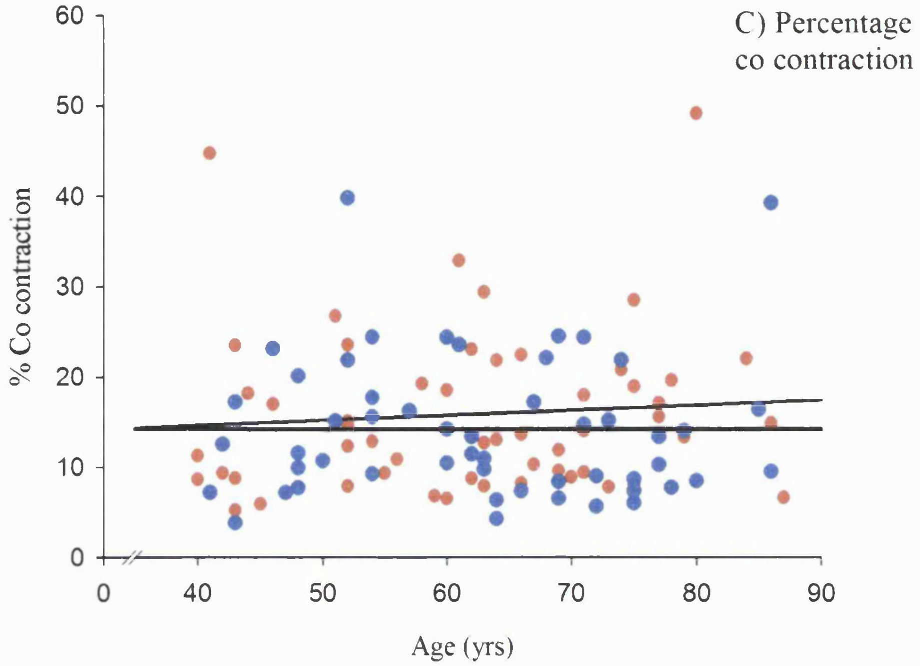

Figure 5.8 A) the median density frequency (MDF) of the power spectrum of the vastus lateralis (y = 67.6 – 0.15x controls, y = 68 – 0.03x weightlifters). B) The root mean squared (RMS) of the vastus lateralis during isometric knee extension (y = 375 – 0.5x controls, y = 1199 – 7.7x weightlifters) C) the percentage biceps femoris involvement during isometric knee extension (y = 14 – 0.001x controls, y = 12 – 0.055x weightlifters). Controls (●), Weightlifters (●).

5.4 DISCUSSION

It is well documented that muscle strength and power decline with increasing age (Izquierdo et al. 1999, Hakkinen et al. 1995, Skelton et al. 1994, Grassi et al. 1991, Vandervoort and McComas 1986). It is also known that resistance training can increase muscle strength in previously untrained people. Klitgaard et al. (1990) showed that in subjects who had been resistance training for approximately 12 – 17 years prior to testing the torque, specific torque and muscle composition was similar to that of young subjects. In order to determine the relative rates of decline in terms of power and strength this study has compared explosive muscle power and isometric strength in the lower limb muscles of elite master weightlifters who were of world class in the sport of Olympic weightlifting and had trained for between 25 – 65 years, with men of a similar age who were healthy but did not undertake any regular physical exercise. The main findings were that, a) both strength and power declined with increasing age in both the weightlifters and the untrained subjects, b) the relative rate of decline was similar for the two groups, with power declining at twice the rate of isometric strength, despite inertial load optimisation and, c) the weightlifters were significantly stronger and more powerful, requiring a higher level of inertia in order to generate their MPP. This was despite the fact that there was no significant difference in lean lower limb volume between the groups as estimated anthropometrically (Table 5.1).

Ageing, power output and performance

The decline in the amount of weight that can be lifted in competitive performance in master's weightlifting has been previously documented by Meltzer (1994) who reported an approximate loss of 1.5% per year in lifting performance. In general agreement with this the present study revealed a decline of 1.4% per year in lifting performance. Two of the competitors tested in the present study were winners in the 85 kg category and represented the extreme of age categories. The winner in the 80+ category, who was also the oldest competitor (87 years old), lifted 55kg in the clean and jerk (see appendix F). This amounted to 36% of the winning lift in the 40-44 year old age group (150kg). From a functional perspective it is worthy of note given that many older people of a similar age i.e. over 80 years of age struggle to rise from a chair let alone be able lift 55 kg above their heads!

In agreement with the decline in competition performance with increasing age, is the observed decline in the performance of the tests of muscle function. In terms of peak muscle power generation, the data from this study suggests a loss equivalent to 1.3% per annum for the weightlifters and 1.2% per annum for the controls. Skelton et al. (1994) has previously reported a progressive decline of approximately 3.5 % per annum in average power in healthy older people ranging in age from 65 to 89 years of age, however, it must be appreciated that these figures are not directly comparable as the denominators used to calculate the decline are different in each case and also Skelton et al. (1994) used the original Nottingham Power Rig but with a fixed single inertial load. In another study of master athletes, Grassi et al. (1991) measured power output using double leg jumps from a force platform. They reported in power athletes (sprinters and jumpers), ranging from a mean age of 17.7 years to 74.3 years, that peak power output of the lower limbs in the older subjects was approximately 50% of that seen in the younger subjects. They also reported that the maximum power output of the power athletes over the age of 70 was 58% of that generated by the subjects in the 40-49 age group. In general agreement, the results of the present study showed that the 70-year-old weightlifters generated 62% of the power generated by the youngest weightlifter group. The methods used by Grassi et al. (1991) are not similar to this study due to the fact that they did not optimise the loading for each individual, but simply used their body weight as the load. This may in fact reflect the reported reductions in power output being slightly higher than the results of this study.

The declines reported in performance for master athletes have been shown to be closely related to strength. Hartley and Hartley (1986) reported that the decline in sprint swimming times with age was associated with declines in strength. Samson et al. (2000) in a study of non athletes also showed that functional decrements in performance were related to decrements in muscle strength. The decrements in strength and power with age have previously been correlated with concomitant reductions in body mass; however in weightlifters this relationship has not been found (Meltzer 1996). This finding could in part be due to the quality of the muscle tissue being different in the weightlifters and also the ratio of body fat to lean tissue.

Functional testing and inertial load optimisation

A further finding of the present study was that the inertial load at which MPP occurred was higher in the weightlifters than the untrained group. This is in agreement with the preliminary suggestions of the earlier chapter (Chapter 1). It is likely that the weightlifters, because they are stronger, are better able to accelerate these higher inertial loads and therefore reach an optimal position on their power velocity relationship during the exertion. Weaker individuals however, must use a greater proportion of their maximal strength to achieve a similar acceleration at a given inertial load. Due to the force-velocity relation, such a contraction must be performed more slowly and thus possibly on a less favourable portion of the power-velocity relationship. Therefore, a lower inertia is more favourable for the weaker subjects, for when MPP was broken down into its torque and velocity components, the velocity component was found to be not significantly different between the two groups (Figure 5.5B). However, the torque generated by the weightlifters at their optimal inertia was significantly greater (Figure 5.5A) than the torque generated by the control subjects at their lower optimal inertia. In addition, the weightlifters required significantly less time than the untrained group to reach MPP (Figure 5.5C). Thus, not only are the master weightlifters achieving higher levels of MPP through higher torque generation, they are also generating torque more quickly.

The results also raise the question, in spite of no significant differences between the two groups in terms of LLV why then are the weightlifters still able to generate significantly greater levels of power output? It is possible that the result of high intensity resistance training results in a greater packing density of the muscle fibres (Aagaard et al. 2001); this would alter the pennation angle of the fibres and also the effective pull length. The reduction in effective pull length could result in a decrease in shortening velocity, therefore the resulting increases in force and decrease in velocity would have opposing effects on the power output. It would be expected that this would result in increased power output at low velocities or where the force is high, this is what is seen with the weightlifters as they are able to generate their MPP at a higher inertial load where forces are high and velocities low. The above factors are probably not as important overall as the MHC composition of the muscle, and it is very possible that the weightlifters have a higher relative MHC-II content in their muscle fibres than that of the untrained subjects.

Ageing and training adaptability

An important finding in the present study was that the decline with age in both average and peak power was similar for the weightlifters and the control subjects (Table 5.4) suggesting an obligatory decline in muscle function with age. However, the weightlifters generated significantly greater absolute levels of both MPP and AP_{mpp} at all age groups, such that the power generated by an 85-year-old competitor was equivalent to that of a 65-year-old non-trained subject, representing an apparent gain of some 20 years. This was also the case when the data were normalised to body mass or lower limb volume. A number of researchers have carried out studies to examine the potential 'trainability' of muscle from elderly subjects in contrast to that of younger subjects. This has been carried out at a number of different levels from *in vitro* measurements to *in vivo* measurements. Some of which examined changes in voluntary strength/power and changes in muscle size (Hakkinen et al. 2000, Ivey et al. 2000, Harridge et al. 1999, Rantanen et al. 1997, McCartney et al. 1995, Skelton et al. 1995, Fiatarone et al. 1990, Moritani and deVries 1980). Recently Ivey et al. (2000) in a study consisting of resistance training 3x per week for 9 weeks, reported that the older subjects (mean age 69 years) had similar responses to increases in thigh muscle volume (approximately 10%) as that by the younger subjects (mean age 25 years), suggesting that ageing *per se* does not affect the trainability of older muscle. Harridge et al. (1999) in a study of elderly subjects (age range 85 – 97 years), strength trained the subjects for 12 weeks 3x per week, and showed that the increases in lean quadriceps CSA was approximately 9.8 %, with increases in 1RM of 134 %. In the present study no detailed anatomical measures were possible in the field setting described, nor was it possible to obtain muscle biopsy samples. The anthropometric data gathered here suggested that the weightlifters showed a trend towards a higher proportion of lean tissue, this however was not significant. However, it has to be understood that the anthropometric method used has limitations in terms of the ability to detect lean tissue (see Chapter 4 Discussion for explanation). But the fact that the weightlifters still showed a significant advantage in terms of functional power output when the data was normalised to lean leg volume, suggests that there were qualitative differences between the two groups and also within the groups as a function of age.

The strength training study carried out by Hakkinnen et al. (2000) reported that the elderly group (age range 62 – 77) showed similar hypertrophic increases and increases in voluntary strength to that of the younger group. It was suggested that the increases in strength were predominantly due to neural aspects (reduced co contraction, increased activation), also that the elderly group showed a blunted training response after 41 weeks whereas the younger group responded up until week 48. In the present study, in terms of the 'trainability', an approximate comparison between the youngest and oldest groups show a larger difference between the oldest groups in terms of MPP, the oldest weightlifter group having a relative 88% greater value and the youngest weightlifter group having a 57 % greater value than their equivalent untrained age group. However, numbers in the oldest group were small (n = 4).

Resistance training and intrinsic adaptations

At the cellular level, in order to adapt to resistance training the muscle must repair the damage caused by training and in doing so, enable the muscle to undergo further stimuli. The ability of elderly subjects to resynthesise muscle proteins has been reported to be similar to that for younger subjects (Hasten et al. 2000). Intrinsic differences in fibres containing the same MHC isoforms have been shown between young and elderly groups of subjects. Larsson et al. (1997) reported that in young subjects, the specific tension and V_0 values of fibres consisting of MHC-I or IIA isoforms were significantly higher than values for the elderly subjects. This would in part, help explain the decline shown with age in the untrained group studied here. The weightlifters showed a similar decline in

MPP with age when compared to the untrained group although in relative terms they showed a 20-year advantage. Klitgaard et al. (1990), reported that elderly resistance trained subjects had similar MHC isoform composition, specific strength and speed of movement to the young control group. However, in his study, the elderly group was of mean age 68 +/- 0.8 years and the young control group was 28 +/- 0.1 years. This would suggest an approximate 40-year advantage for the weight-trained group. The differences in advantage shown due to weight training with the results of this study could in part be due to the difference in age of the subjects tested, but also in the different testing protocol used. Klitgaard et al. used an isokinetic method of testing, whereby the torque was measured at a given limb angle. It is known that the angle, at which peak torque occurs, changes with the velocity for an isokinetic movement (Kannus and Beynnon 1993). This could result in a less than optimal torque at a given joint angle, which could result in a reduction in peak power. This effect may not be equivalent in the weight trained and control groups, resulting in a biased comparison of power output.

Increases in the number of fibres expressing more than one MHC isoform with ageing could be part of the explanation of why muscle functional output decreases with age. Aged muscle shows a large number of fibres containing more than one MHC isoform (Williamson et al. 2000, Andersen et al. 1999, Klitgaard et al. 1990, Klitgaard et al. 1990). This has been suggested to be in part due to motor unit remodelling (see Literature Review for details). The results reported in Chapter 4 are in general agreement with this idea in that the relative MHC-II proportion of older men is less than in the younger group. The results of Chapter 4 also showed that the velocity at which peak power was generated and the peak power were significantly related to the percentage MHC-II isoform present in the muscle. Qualitative differences were also seen, and that for a similar MHC-II composition the elderly muscle was slower (see Figure 4.7). The incidence of hybridised muscle fibres in the elderly has been shown to be reduced with resistance training (Williamson et al. 2000, Williamson et al. 2001). Williamson et al. (2000) showed that training increased the number of MHC-I fibres and decreased the number of mixed fibres (I/IIA, I/IIA/IIX, IIA/IIX) in untrained men (mean age 74 years).

Strength trained individuals have been shown to have a larger type II fibre area than non-trained individuals (Tesch et al. 1984, Tesch and Larsson 1982). In the present study it was not possible to obtain muscle biopsies from the competitors. However, electromyographic studies have revealed significant correlations between muscle fibre type and the MDF of the sEMG power spectrum (Stulen et al. 1981), while Bilodeau et al. (1994) suggested that the power spectrum of the sEMG might be more related to the fibre diameter than the fibre type. A relationship between the fibre area and the power spectrum at 70% MVC was also shown by Gerdle et al. (1997). The higher values of MDF for the weightlifters from the power spectrum analysis (Fig 5.7A), might suggest either a higher proportion of type II fibres or larger diameter type II fibres in the weightlifters. Klitgaard et al. (1990) reported that in male bodybuilders the fibre type distribution was different from untrained men. The bodybuilders showed a greater proportion of muscle fibres containing MHC-II isoforms than did their untrained counterparts. Similarly in a resistance training study of sprinters it was found that after the training period there was an increase in the expression of fibres containing MHC-IIA isoform, this was suggested to be due to bi directional transformation from MHC-I and IIX towards MHC-IIA isoforms (Andersen et al. 1994). In a longitudinal study of elderly people, Roman et al. (1993) reported that older men showed a preferential hypertrophy (37.2%) of the fast fibres in the biceps brachii following 12 weeks of resistance training of the elbow flexors.

Recent mechanical studies on human chemically skinned single fibres (Larsson et al. 1997) and using the *in vitro* motility assay, (Hook et al. 2001) suggest that there may be intrinsic changes with age that may slow cross bridge cycling which is directly linked to the velocity of shortening. In a recent study of chemically skinned fibres, Trappe et al. (2000) reported that in elderly men (74 +/- 2 years), who strength trained 3X/week for 12 weeks, that the shortening velocity increased by 75% in MHC-I and by 45% in MHC-IIA fibres, P_0 increased by 36% and 20% in MHC-I and MHC-IIA fibres respectively, contributing to an increase of 48% and 66% in power output for the MHC-I and IIA fibres respectively. Thus the increased power output in the weightlifters shown in the present study might be reflective of intrinsically faster contracting fibres as a result of strength training.

Isometric strength EMG and co contraction

The results of the sEMG support the notion that the weightlifters can activate their muscles during an isometric contraction with a more synchronous firing pattern as indicated by the RMS data. In fact, the RMS, which can be used as a measure of general muscle activation, was significantly higher in the weightlifters in all age groups. A factor in the measurement of isometric strength that may affect the result is the amount of co contraction present during the effort. Many researchers do not take this into account and therefore erroneous results may occur. In these two groups, it is possible that there is a difference in the ability to focus the contraction effort into the muscles that are important in the isometric force measurement. This postulate is based on the fact that the weightlifters require skill in order to carry out their discipline and therefore may be able to reduce the amount of co contraction present during an isometric test of strength. Previously Aagaard et al. (2000) has shown that the level of co contraction is reduced in resistance trained subjects. Therefore the untrained group may be less able to generate isometric force due to an inability to control the co contraction of the antagonistic muscles in comparison to the weightlifters.

However, similar levels of co contraction are seen between the two groups (Figure 5.7C). This may, in part, be due to the fact that a number of weightlifters described having some knee discomfort during the test and it was possible that the weightlifters utilised the antagonist muscles to help protect the knee during these isometric contractions.

Comparison of isometric strength and power

In terms of isometric knee extensor strength, the weightlifters were on average 32% stronger than the controls with the decline in strength occurring at 0.6% per annum, this was approximately half the rate observed for muscle power. Skelton et al. (1994) reported that average power declined at 3.5% of a 77 year olds value per annum between 65 and 85 years old, but that isometric strength declined at a lower rate (~1.5% per annum). The results of the present study in terms of relative decline of power and strength are in general agreement with those of Skelton et al. (1994) in that power is lost at approximately twice the rate of isometric strength. In the study by Skelton et al. (1994), power was determined using a single, relatively high inertial load. As already alluded to, in a protocol which uses a single lower limb thrust, it is possible that the weaker older people are at a disadvantage as they are unable to sufficiently accelerate the heavy flywheel in order to obtain a velocity to allow them to generate their optimal power. In the present study we have optimised the inertial load for power generation, yet the rate of decline in power was still greater than that of isometric strength, suggesting that in addition to declines in strength, alterations in shortening speed may play also a role. In this regard, although the decline in TQ_{mpp} was more dramatic (Figure 5.5A) there was also an age related slowing of the V_{mpp} (Figure 5.5B).

Summary

In conclusion, the results of the present cross-sectional study have shown that the apparent decline in lower limb muscle function occurs even in highly trained weightlifting athletes and that the relative rate of decline is similar to that which occurs as a result of normal ageing. The optimisation of inertial load for each individual for power testing was shown to be important as the weightlifters developed their maximal power at a higher inertial load than the untrained control subjects. When looked at in absolute terms, the data in the present study show that weight lifters are considerably stronger and more powerful, with the oldest weightlifters having muscle power characteristics of untrained individuals some 20 years younger than themselves. In functional terms however, it is probably the ability to generate levels of force and power relative to body weight, which determine the ability of older people to perform physical tasks. As there were no significant differences between the weightlifters and controls with respect to body weight, the higher levels of absolute power generated by the weightlifters showed that they were advantaged in terms of their power to weight ratio which has implications for carrying out functional tasks. These results therefore suggest that high resistance strength training for older people might not slow the rate of decline in muscle function with increasing age, but may result in a shift upwards to a new slope which starts at a higher absolute level, but which has a similar relative rate of decline. The data presented here suggest that activities that require repetitive high levels of muscle power to be generated as in weightlifting may help improve muscle function in later life.

PART III

CHAPTER 6

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

This thesis examined the validity of an inertial device (NPR) used for measuring the average power output of the lower limbs. It was revealed that the underlying assumptions of constant acceleration and hence linear velocity (Bassey and Short 1990), used in order to calculate power output were not consistent with the experimental findings (see Appendix A). A system was therefore designed and developed which did not rely on the above assumptions and had the advantage of allowing for the measurement of power, torque and velocity throughout the exertion period.

An analysis of the interaction between inertial loading and power output revealed that peak power output followed a parabolic relationship. It was hypothesised that at the lowest inertial loads, insufficient time to develop optimal levels of torque may be in part responsible for the lower levels of power output. An isometric release device was designed and built in order to examine this possibility. A pilot study indicated that substantial increases in power output could be achieved if an isometric pre release period was allowed. Subsequently it was shown that power output could be significantly increased (17 %) at the lowest inertial loads by allowing a pre set level of isometric torque to be developed prior to a dynamic exertion.

The reduction of power output at the higher inertial loads was hypothesised to be due to an inability to fully accelerate the load to a point at which maximal power would be reached. The inertial loading device was mounted in the rear wheel assembly of a static cycle to examine this possibility. It was seen that during sprint cycling a plateau was obtained in the inertial load power output relationship above a critical load. In contrast to the NPR, cycling exercise allows for the repeated acceleration of the load. It was thus concluded that maximal power should always be attained against an inertial load provided sufficient time is allowed for the load to be accelerated to a point on the torque – velocity relationship where the optimal velocity for peak power occurs. However, when carrying out brief exertions over a fixed range of movement using an inertial protocol (modified NPR), a range of inertial loads must be used in order to elicit maximal power.

In order to identify any differences in the muscle myosin isoform composition between young and old subjects, gel electrophoresis (SDS – PAGE) was utilised to identify and quantify the relative proportions of the three MHC isoforms from biopsy samples taken from the vastus lateralis muscle. Sprint cycling was used to identify any differences in mechanical output between a young and old group. The older subjects possessed a significantly lower percentage of type II MHC when compared with the younger subjects. The normalised power output was also significantly lower in the older group, as was the velocity at which peak power occurred. A significant relationship ($r = 0.82$) between the percentage MHC-II and the optimal velocity at which peak power occurred was observed. However the relationship between MHC composition and the NPR was not as good, possibly due to the limitations to accelerate the load, hence express true maximal power.

Resistance training is known to maintain or increase muscle strength and power even in very elderly people. As part of this thesis there was a unique opportunity to study men of ages up to 87 years old who were competitors at the World Masters Weightlifting Championships. These men had trained for many years and represented the potential to which ageing muscles may function in terms of power output. The results showed that the master weightlifters declined in power output at similar rates to untrained subjects, however the absolute levels of power were shown to be significantly greater in the weightlifters representing an apparent 'gain' of some twenty years. Furthermore, the observation that these athletes require a significantly higher level of inertia to express their peak power confirms the utility of using a testing system in which the inertial load could be varied, thus allowing optimisation for each individual.

In conclusion the development and use of a variable inertial loading device has allowed an objective assessment of dynamic human muscle function. The attributes of different applications of the device have been explored by using the device in a cycling protocol, and whilst it has been used to study the lower limb muscles in this thesis, it could easily be housed in test systems to allow other muscles to be studied. The mechanical data obtained has been coupled with biochemical data and has shown strong a relationship between the contractile proteins expressed and the velocity at which peak power is produced (at least during cycling) under inertial loading. This indicates that with increasing age not only is power reduced as a function of a weaker muscle, but also because the aged muscle is slower. The further study of athletes who represent the elite of their age, such as those used in the present study, will help in our understanding of the interaction between age and exercise in helping to maintain muscle function in later life.

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APPENDIX A - INVESTIGATION OF THE VELOCITY PROFILE DURING A TYPICAL EXERTION ON THE NPR

The NPR calculates the average power applied to a spinning flywheel. In order to calculate average power the exertion time must be determined. The method utilised by Bassey and Short (1990) to determine the time of exertion uses a system of 4 pins mounted into the hub of the flywheel at 90^0 intervals in order to determine the time of exertion. As the flywheel turns, the pins pass through an optoswitch, which is interfaced, to a PC, this logs the time that each pin passes through the optoswitch. This method was reliant on the underlying assumptions that acceleration was constant, and hence increase in velocity of the flywheel linear during the exertion phase. The method used employed the time taken for one full revolution of the flywheel after the exertion period. From this time the exertion period was estimated by use of the equations of linear motion as shown.

$$s = {}^{v}\!/_{2} \times t \quad \text{①}$$

Where s = the flywheel distance travelled during the exertion, v = the flywheel final velocity and t = the time for one full revolution after the exertion period. Let x = the number of revs during the exertion and let T = the time of exertion

Then

$$2 \times \pi \times x = {}^{2 \times \pi}\!/_{2 \times t} \times T \quad \text{②}$$

Therefore $T = 2 \times x \times t$

The CODA *mpx* 32 motion analysis system which used an infra red detection method was used to determine the acceleration profile during a typical exertion on the NPR. This was performed by a young subject. Markers were attached to the flywheel and a fixed horizontal reference point on the NPR to allow relative rotary displacement to be calculated. From the angular displacement data it was determined that the velocity was not linear (see Figure 1) and thus acceleration not constant. Therefore the underlying assumptions used to calculate the time of exertion were not valid (see Figure 1). This fact was further confirmed using the rotary encoder system (described in Chapter 1) and a typical plot of the flywheel acceleration during an exertion can be seen in Figure 2.

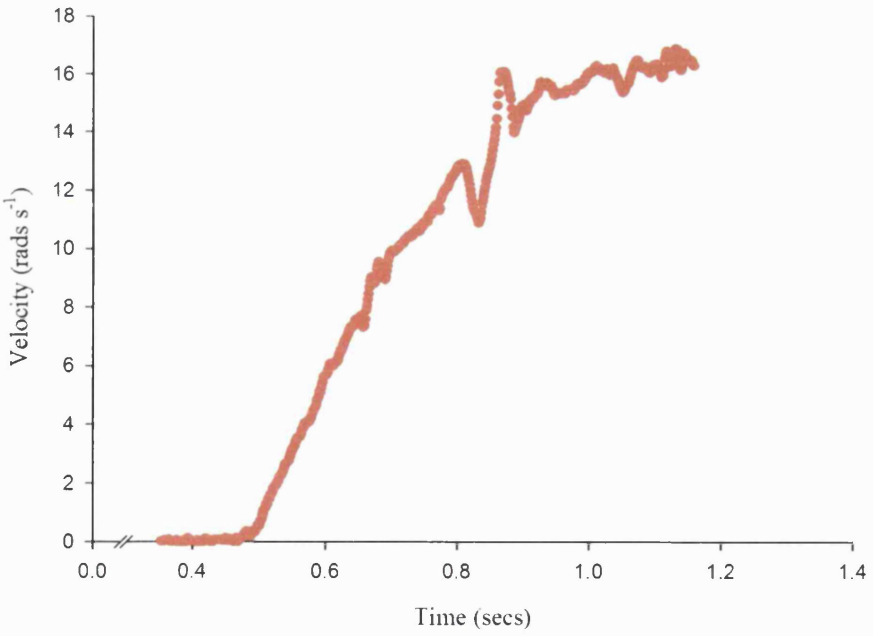


Figure 1 Plot of angular flywheel velocity during exertion phase. Data was captured using the CODA 32 mpx motion analysis system, sampled at 800 Hz

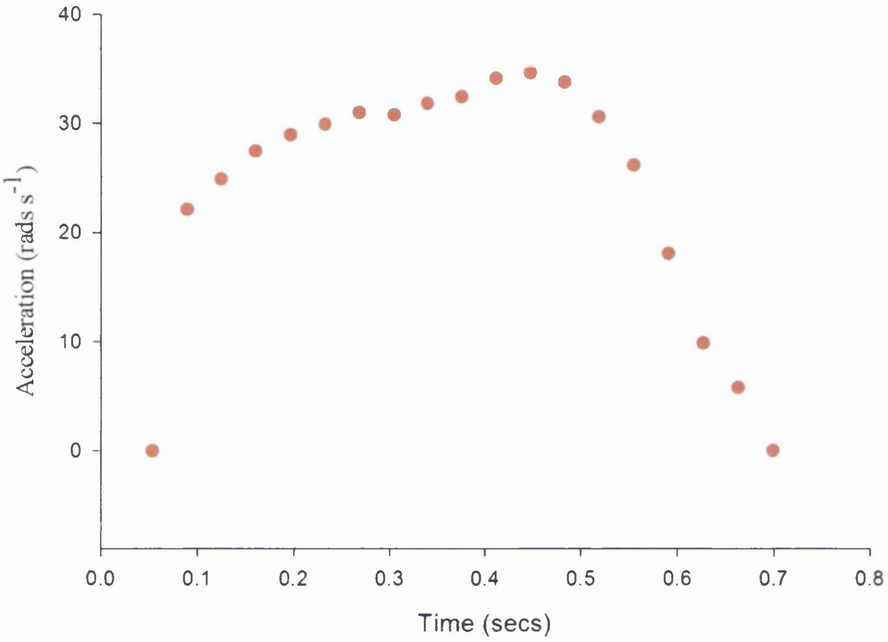
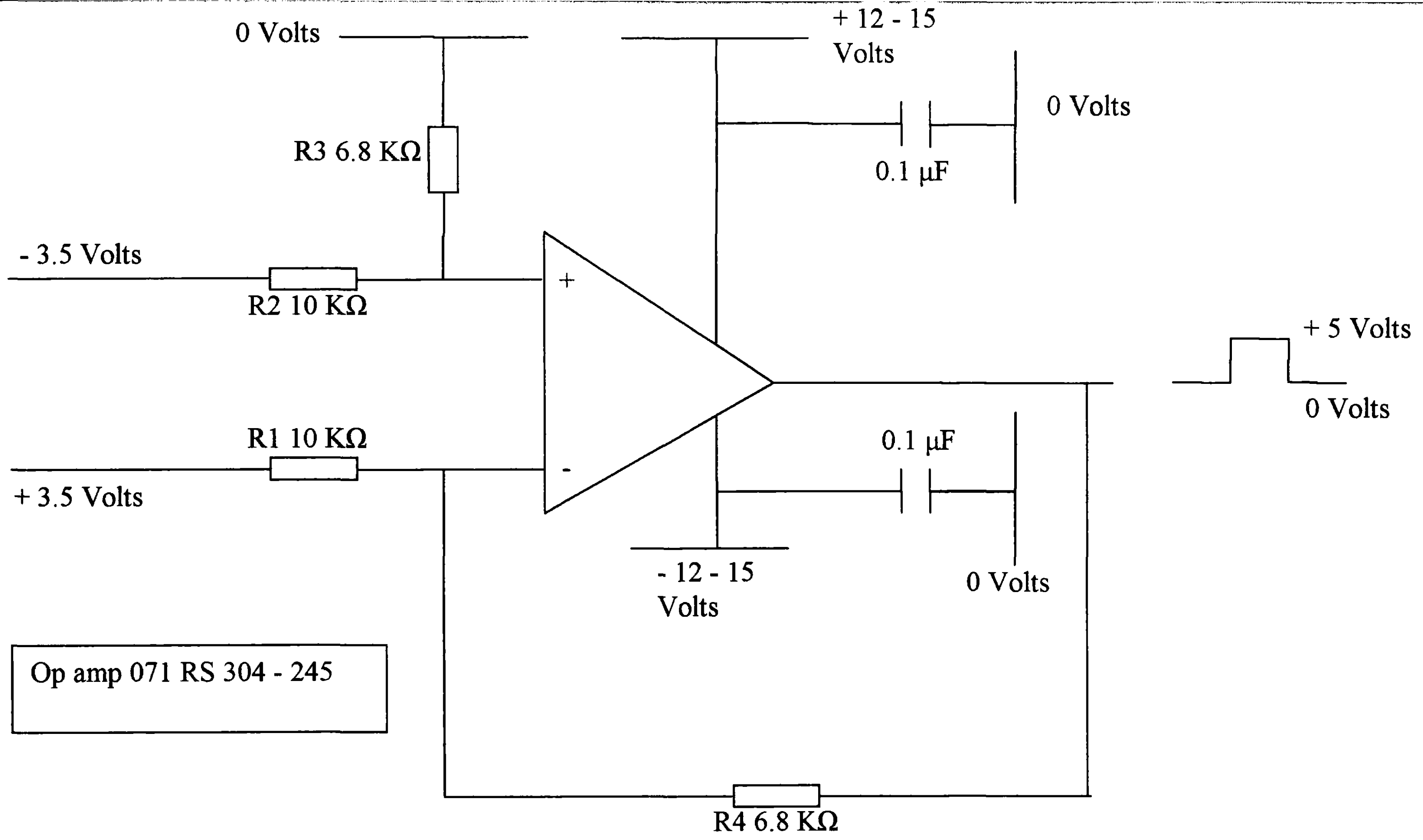


Figure 2 A typical acceleration trace during an exertion on the NPR



APPENDIX B ROTARY ENCODER DIGITAL PULSE CONVERTER CIRCUIT

APPENDIX C SPIKE 2 SCRIPTS AND MATHCAD PROGRAMME

SPIKE 2 SCRIPT TO CONVERT RAW ANALOGUE AND DIGITAL DATA INTO A TEXT FILE SUITABLE FOR READING INTO MATHCAD

```
'SonDump.s2s
'Declare variables:
var ok%,chan%,dh%,th%,close%:=0, fmt%:=0, fmt$[1], delim$ := ",", fin;

fmt$[0] := "Real";

if ViewKind()<>0 then
        dh% := FileOpen("", 0, 0, "SON file to dump as text");
        if dh%<=0 then Message("Error|No file selected to dump"); halt endif;
        close% := 1;                                        ' say we should close
file
else
        dh% := View();                                              ' get the
starting view handle
        Message("File '%s' selected for dump",FileName$());
endif;

View(dh%).WindowVisible(3);
CursorSet(1);
Interact("Please select end position for file dump",0);
fin := Cursor(1);

Query("SON file dump|This script is not able to dump files which contain gaps on
one\n"
          "data channel only. It can, however, process files with gaps on\n"
          "all channels simultaneously correctly.","Continue","Abort");

DlgCreate("SON file dump");                         ' Start new dialog
DlgChan(1,"Choose the channels to dump",262271);
DlgString(2,"Item delimiter (\\t=tab)",2);
DlgList(3,"Format for the output",fmt$[]);
ok% := DlgShow(chan%, delim$, fmt%);     ' ok is 0 if user cancels

if delim$ = "\\t" then delim$ := "\t" endif;

if ok% then                                                  ' dump if user
selects channels
        if delim$<>"," then
                if not Query("Are you sure?|Using a symbol other than a comma
for the delimiter\n"
                                "could slow the dump down considerably. Do you
really\nwant to use your symbol?") then
                        Message("A comma will be used instead");
```

```
                        delim$ := ",";
                endif;
        endif;

        th% := FileOpen("", 8, 1, "File to dump channels to");
        if th% > 0 then
                Dump(dh%, th%, chan%, fmt%);
                View(th%);FileClose();                          ' close the text file
        endif;
endif;

if close% then                                          ' if we opened the file..
        View(dh%);                                              ' ...then move to it...
        FileClose();                                    ' ...and close it again
endif;
halt;

Proc Dump(dh%, th%, ch%, fmt%)
        if ch% = -1 then ch% := 127; endif;

        view(dh%);
        var temp%[1];
        var chans%[ChanList(temp%[],ch%)+1];

        ChanList(chans%[],ch%);

        const BSZ% := 100;              ' buffer size
        ch% := chans%[0];                                       ' store number of channels
        var buff[ch%][BSZ%];                                    ' data buffer
        var t[ch%], n%;                                                 ' start time, items read
this loop
        var gapbin%[ch%];                                       ' the bin in which we hit the
gap
        var hitgap%[ch%];                                       ' has each channel hit the
gap?
        var numhitgap% := 0;                            ' how many channels have hit the gap

        for x%:=0 to ch%-1 do
                hitgap%[x%]:=0;
                t[x%]:=-1;
        next;

        View(dh%);                                                      ' data file view
        var fini%;                                                      ' have we finished
yet?
        var x%, maxn%;

        repeat
```

```
        fini% := 1;
    ' assume finished
        maxn% := 0;
        for x% := 0 to ch%-1 do                                      ' loop
around each channel
            gapbin%[x%] := -1;
            var chan%;
            chan% := chans%[x%+1];
            if ChanKind(chan%)=1 and not hitgap%[x%] then    ' if we
haven't hit the gap on this waveform channel
                if t[x%] >= fin then
                    n% := 0
                else
                    n% := ChanData(chan%, buff[x%][], t[x%],
fin, t[x%]);
                endif;

                if n% > 0 then                                ' if we
got data, then
                    t[x%] := t[x%] + n% * BinSize(chan%);

    ' store time of next point
                    fini% := 0;
    ' and flag not finished yet
                    gapbin%[x%] := n%;                    ' we
store here the number of bins we filled
                    maxn% := max(n%,maxn%);              ' and
here the max. number of bins filled on any channel
                endif;
            endif;

            if ChanKind(chan%)>1 and not hitgap%[x%] then    ' if we
haven't hit the gap on this marker/event channel
                buff[x%][0] := 0;
                n% := 0;
                var fini1%;
                fini1% := 0;
                while not fini1% do
                    buff[x%][n%] := NextTime(chan%, t[x%]);

                    if buff[x%][n%] > -1 then      ' if we got
data, then
                        t[x%] := buff[x%][n%];        ' store
time of last point
                        fini% := 0;
    ' and set not finished yet flag
                        n% += 1;
    ' go onto next buffer item
```

```
                    else
                        fini1% := 1;
                    endif;

                    if n% = BSZ% then
                        fini1% := 1;
                    endif;
                wend;

                gapbin%[x%] := n%;                          ' we store here
the number of bins we filled
                maxn% := max(n%,maxn%);                     ' and here the
max. number of bins filled on any channel
            endif;
        next;

        var pos%;
        pos% := 0;
        while pos% < maxn% do
            var printTo%;
            printTo% := maxn%;

            var minidx%;
            minidx% := -1;
            for x%:=0 to ch%-1 do
                if not hitgap%[x%] and gapbin%[x%] > -1 and
gapbin%[x%] <= printTo% then
                    printTo% := gapbin%[x%];
                    minidx% := x%;
                endif;
            next;

            if delim$ = "," and numhitgap% = 0 and printTo% > pos%
then
                View(th%).Print(buff[][pos%:printTo%-pos%]);
            else
                for x% := pos% to printTo%-1 do
                    var y%;
                    for y% := 0 to ch%-1 do
                        if not hitgap%[y%] then

    View(th%).Print("%g",buff[y%][x%]);
                        endif;

                        if y% < ch%-1 then
                            View(th%).Print(delim$);
                        else
                            View(th%).Print("\n");
```

```
                        endif;
                    next;
                next;
            endif;

            pos% := printTo%;

            if pos% < BSZ% and minidx% > -1 then
                hitgap%[minidx%]:=1;
                numhitgap% += 1;

                if pos% = maxn% and numhitgap% = ch% then
                    for x%:=0 to ch%-1 do
                        hitgap%[x%]:=0;
                        if x% < ch% - 1 then
                            View(th%).Print(delim$);
                        endif;
                    next;
                    View(th%).Print("\n");

                    numhitgap% := 0;
                endif;
            endif;
        wend;
    until fini%;
end;
```

DATA ANALYSIS PROGRAMME WRITTEN IN MATHCAD

Mathcad programme to calculate power output from modified NPR

Import file

$In :=$ C:\..\anrtwp7a.txt

$En := In^{\langle 0 \rangle}$

$zero := 1$

Zero first encoder pulse

$Enc := \text{submatrix}(En, zero, \text{rows}(En) - 1, 0, 0) - En_{zero}$

Get the strain gauge signal
using the first 100 points as BL

$BL := \text{mean}(\text{submatrix}(In, 0, 50, 2, 2))$

$strain := In^{\langle 2 \rangle} - BL$

Enter sample frequency for strain signal

$k := 0 .. \text{rows}(strain) - 1$

$TimeS_k := k \cdot 0.0004 - En_{zero}$

Set the time for which the push record should last

$PushTime := Enc_{390}$

Correct SG input using calibration formula

$Strain := \frac{strain}{.0029} \cdot 0.03$

$\Delta t := TimeS_1 - TimeS_0$

Generate range variable for deconvolution

$kk := 1 .. \text{rows}(strain) - 2$

Set time window for calculations of mechanical variables

$inc := \frac{PushTime}{points}$

$DelT := inc$

$points := 20$

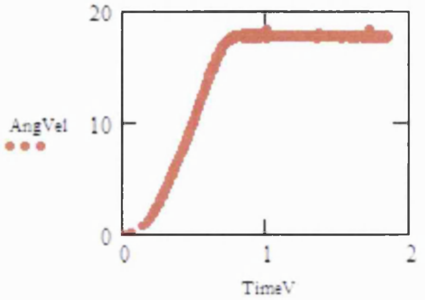
Examine the raw velocity trace calculated from the rotary encoder signal

$SEsens := 360$

$j := 1 .. \text{rows}(Enc) - 5000$

$$AngVel_j := \frac{2 \cdot \frac{\pi}{SEsens}}{Enc_j - Enc_{j-1}}$$

$$TimeV_j := \frac{Enc_j + Enc_{j-1}}{2}$$



Set calculation block to calculate position, velocity and acceleration from encoder data

```
$Calcs :=$ for $r \in 0 .. points - 1$
    $ts \leftarrow DelT \cdot r + Enc_0$
    $te \leftarrow DelT \cdot (r + 1) + Enc_0$
    $tc \leftarrow (r + 0.5) \cdot DelT$
    $k \leftarrow 0$
    for $rr \in 0 .. 390$
      if $(Enc_{rr} \geq ts) \cdot (Enc_{rr} \leq te)$
          $tim_k \leftarrow Enc_{rr}$
          $ang_k \leftarrow rr \cdot deg$
          $k \leftarrow k + 1$
    if $k > 2$
        $reg \leftarrow regress(tim, ang, 2)$
        $acc_r \leftarrow 2 \cdot reg_5$
        $vel_r \leftarrow reg_4 + 2 \cdot reg_5 \cdot (tc + Enc_0)$
        $pos_r \leftarrow reg_3 + reg_4 \cdot (tc + Enc_0) + reg_5 \cdot (tc + Enc_0)^2$
        $time_r \leftarrow tc$
        $lastone \leftarrow r$
    if $k = 2$
        $acc_r \leftarrow 0$
        $reg \leftarrow regress(tim, ang, 1)$
        $vel_r \leftarrow reg_4$
        $pos_r \leftarrow reg_3 + reg_4 \cdot (tc + Enc_0)$
    if $k = 1$
        $acc_r \leftarrow 0$
        $vel_r \leftarrow 0$
        $pos_r \leftarrow if(r = 0, 1 \cdot deg, pos_{r-1} + 1 \cdot deg)$
    if $k = 0$
        $acc_r \leftarrow 0$
        $vel_r \leftarrow 0$
        $pos_r \leftarrow 0$
    $time_r \leftarrow tc$
$\begin{pmatrix} time \\ pos \\ vel \\ acc \\ lastone \end{pmatrix}$
```

Query programme block to allow output of time, velocity and acceleration

$Calcs_4 = 19$ $\quad Time := \mathrm{submatrix}(Calcs_0, 0, Calcs_4, 0, 0)$ $\quad Pos := \mathrm{submatrix}(Calcs_1, 0, Calcs_4, 0, 0)$

$Acc := \mathrm{submatrix}(Calcs_3, 0, Calcs_4, 0, 0)$ $\quad Vel := \mathrm{submatrix}(Calcs_2, 0, Calcs_4, 0, 0)$

$r := 0 .. \mathrm{rows}(Time) - 1$

Deconvolute the strain gauge signal using the tor value entered here

$tor \equiv 0.0045$

$$StrainD_{kk} := Strain_{kk} + tor \cdot \frac{\left(Strain_{kk+1} - Strain_{kk-1}\right)}{2 \cdot \Delta t}$$

Get strain values for intervals using the same time centres as for the above calculations

```
Torque :=  for r ∈ 0 .. rows(Time) - 1
             ts ← DelT·r
             te ← DelT·(r + 1)
             tc ← (r + 0.5)·DelT
             k ← 0
             for rr ∈ 0 .. rows(StrainD) - 1
               if (TimeS_rr ≥ ts)·(TimeS_rr ≤ te)
                 tim_k ← TimeS_rr
                 str_k ← StrainD_rr
                 k ← k + 1
             reg ← regress(tim, str, 2)
             out_r ← reg_3 + reg_4·tc + reg_5·tc^2
           out
```

Assume a value for the three mechanical constants of the system

Spring $stiff := 185$ in N m $\quad cograd := 0.03$ $\quad RotStiff := stiff \cdot cograd^2$

$Inertia \equiv ipl3$ $\quad Spring := RotStiff$ $\quad Friction := 0.092$ $\quad Stiction := swheel$

Calculate the expected strain from acceleration*inertia + velocity* friction + displacement * spring

$EStrain_r := Acc_r \cdot Inertia + (Acc_r \cdot Inertia) \cdot 0.0012 + Spring \cdot Pos_r + Stiction$

Plot the expected strain against the observed strain
Are they linearly related with a slope of 1 and no intercept. If not then something is wrong:

If the above test is past then calculate the power etc

find time point one beyond the last zero in Acc

```
TLZ := | k ← rows(Acc) - 1
       | while Acc_k = 0
       |     | k ← k - 1
       |     | break  if k = 0
       | k + 1
                                TLZ = 4
```

Trim the Estrain record to remove the zeros

Trim Strain to match

Get slope and intercept of EStrain vs Strain

Calc the fitted line

```
EStrainTm := submatrix(EStrain, TLZ, rows(EStrain) - 3, 0, 0)
TorqueTm := submatrix(Torque, TLZ, rows(EStrain) - 3, 0, 0)

ESSlope := slope(EStrainTm, TorqueTm)

ESInter := intercept(EStrainTm, TorqueTm)

PStrain := ESInter + ESSlope·EStrainTm
TimeTm := submatrix(Time, TLZ, rows(EStrain) - 3, 0, 0)

AccTm := submatrix(Acc, TLZ, rows(EStrain) - 3, 0, 0)

VelTm := submatrix(Vel, TLZ, rows(EStrain) - 3, 0, 0)

ro := 1 .. rows(TimeTm) - 1      zero := rows(TimeTm)      AccTm_(zero-1) := 0
```

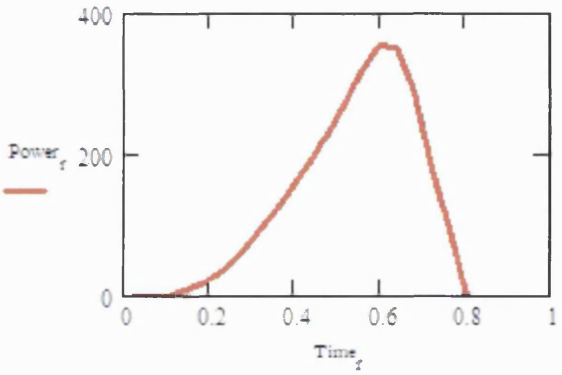
Checks that the two Strain signals are agreeing when both are present

```
ESSlope = 2.474        ESInter = -1.242        corr(EStrainTm, TorqueTm) = 0.997
```

Calculate power

```
EndPush := rows(Time) - 1        Power_r := Torque_r·Vel_r

Power_EndPush := 0
```



All records of displacement, velocity, and acceleration were calculated from the rotary encoder. The rotary encoder had a resolution of 1^0 (0.01745 rads). The rotary encoder returned a time for every degree of wheel rotation. This allowed a record of wheel angle (W) against time (t) to be collected. For each test the time to complete the push was divided up into 20 equal periods. Each period was fitted by a second order polynomial $W = P + Q \bullet t + R \bullet t^2$. The velocity and acceleration were then calculated for the mid point of the time period (tc), from the polynomial coefficients as follows-:

$\text{Velocity(V)} = Q + 2 \bullet R \bullet tc$

$Acceleration(A) = 2 \bullet R$

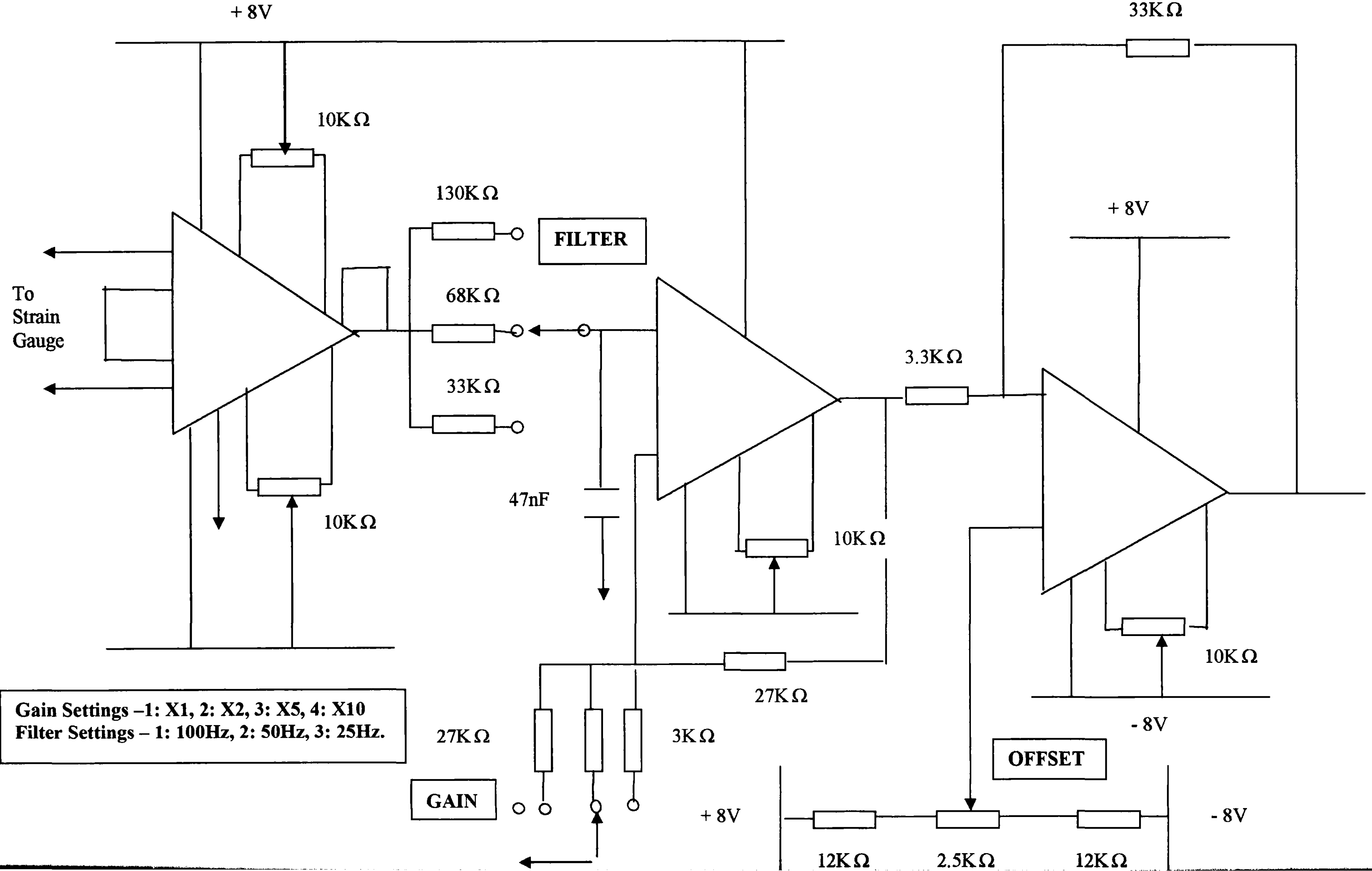
The strain gauge system lag is accounted for by using a deconvolution method, which adds to the measured strain a product of its first time derivative and the time constant.

Power is calculated as a product of the velocity and torque throughout the push.

Work done is calculated as the time integral of power from the start of the push.

Average power for the test is calculated as total work done divided by the time of the push.

APPENDIX D STRAIN GAUGE AMPLIFIER CCT



Gain Settings –1: X1, 2: X2, 3: X5, 4: X10
Filter Settings – 1: 100Hz, 2: 50Hz, 3: 25Hz.

APPENDIX E METHOD TO CALCULATE INERTIA AND FRICTIONAL CONSTANTS OF FLYWHEEL SYSTEM

From the equations of linear motion under constant acceleration: -

$$S=\left(\frac{u+v}{2}\right)\bullet t \quad \text{②}$$

Where t is the time taken for the mass to fall, v is the velocity of the falling mass, and u is the initial velocity of the mass = 0.

Rearranging equation ② for v

$$v=\frac{2\bullet S}{t} \quad \text{③}$$

Also knowing that $v = r \bullet \omega$ ....④

Rearranging equation ④ for ω

$$\omega = \frac{v}{r} = \frac{2 \bullet S}{r \bullet t} \quad \text{⑤}$$

Where r is the radius of the hub around which the mass is attached

Let n_1 = the number of revs of the flywheel during the fall of the mass.

Let n_2 = the number of revs of the flywheel between the mass hitting the floor and the flywheel coming to rest.

$$n_1 = \frac{S}{2 \bullet \pi \bullet r} \quad \text{.......} ⑥$$

After the mass has hit the floor the kinetic energy in the flywheel is dissipated as frictional energy, hence: -

$$\tfrac{1}{2} \bullet I \bullet \omega^2 = W' \bullet f \quad \text{.......} ⑦$$

Where $W' \bullet f$ represents the work done against friction in n_2 revs.

The work done against friction by the flywheel in n_1 revs is: -

$$W_f = \frac{n_1}{n_2} \bullet W' \bullet f = \frac{n_1}{n_2} \bullet \tfrac{1}{2} \bullet I \bullet \omega^2 \quad \text{.......} ⑧$$

Substituting for ⑧ in equation ①: -

$$M \bullet g \bullet S = \frac{1}{2} \bullet I \bullet \omega^2 + \frac{1}{2} \bullet \frac{n_1}{n_2} \bullet I \bullet \omega^2 + \frac{1}{2} \bullet M \bullet v^2 \ldots\ldots\ldots\ldots \text{⑨}$$

Combining terms and rearranging in ⑨: -

$$\frac{1}{2} \bullet I \bullet \omega^2 \bullet (1 + \frac{n_1}{n_2}) = M \bullet g \bullet S - \frac{1}{2} \bullet M \bullet v^2$$

$$I \bullet \omega^2 (\frac{n_1 + n_2}{n_2}) = 2 \bullet M \bullet g \bullet S - M \bullet v^2 \ldots\ldots\ldots\ldots \text{⑩}$$

Substituting for ⑤ in ⑩: -

$$I \bullet (\frac{2 \bullet S}{r \bullet t})^2 \bullet (\frac{n_1 + n_2}{n_2}) = 2 \bullet M \bullet g \bullet S - M \bullet v^2$$

Finally rearranging for I: -

$$I = \frac{r^2 \bullet t^2 \bullet n_2}{2 \bullet S^2 \bullet (n_1 + n_2)} \bullet (M \bullet g \bullet S - \frac{1}{2} \bullet M \bullet v^2 \ldots\ldots\ldots\ldots \text{⑪}$$

APPENDIX F

A pilot study was carried out to examine the effect of preloading on power output. Isometric preloading was examined at a high (0.543 kgm^2) and low (0.024 kgm^2). The effect of preloading on power output was tested on the same subject used for the experiments in chapter 1. An arbitrary level of preloading was chosen (21Nm) Figure 1 shows an example of the effects of an isometric preload of $(21\text{N} \bullet \text{m})$ prior to dynamic movement at both a high and low inertial load in comparison to exertions at the same loads without an isometric preload. Peak power values of 883W and 574W for the low and high inertia respectively were only different for the low inertia when compared to the no preload contraction for this subject.

The use of this preloading exertion at the low inertia resulted in an increase in peak power of 95% when compared with the exertion without preloading. At the high inertia however there was little difference between the two types of exertion.

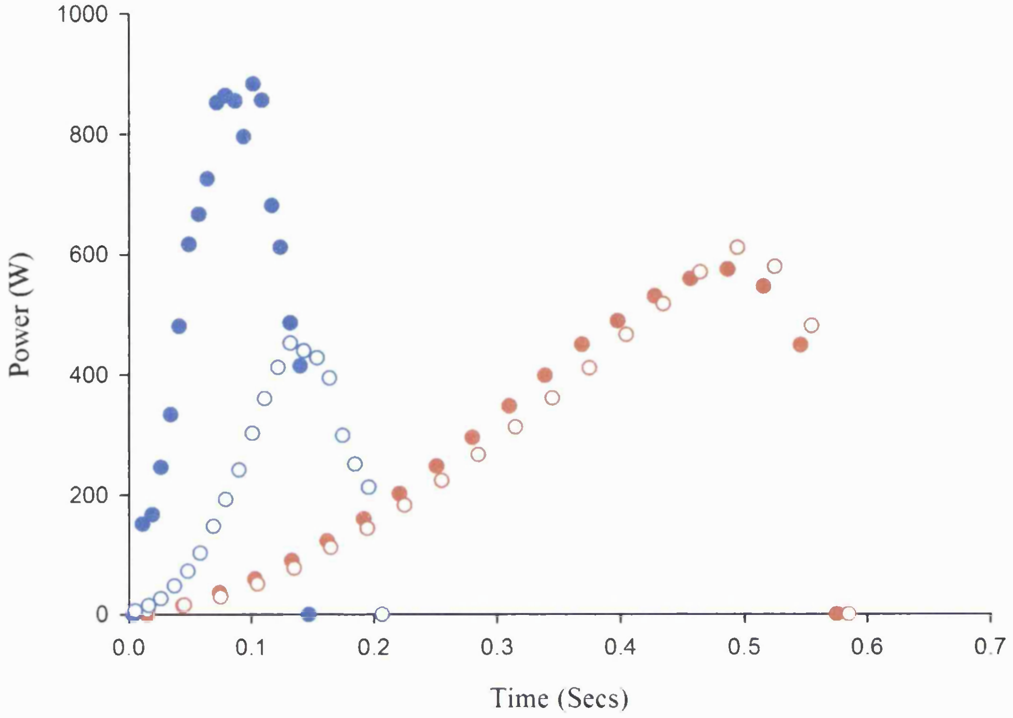
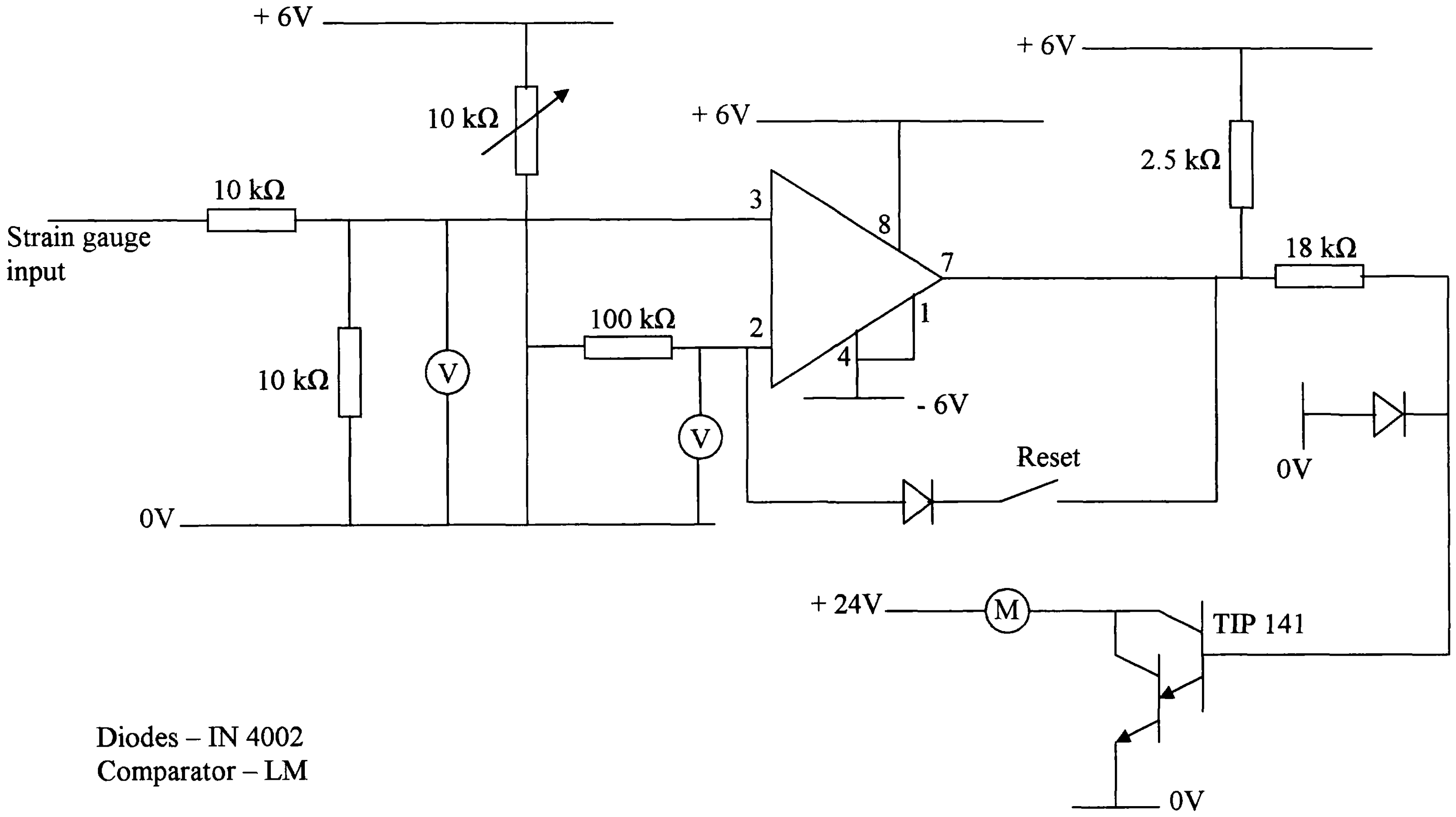


Figure 1 Power output of an individual subject attained against a high (●) and a low (●) inertia following an isometric pre-loading, and release after an isometric torque of 21Nm had been attained. Open circle plots represent exertions at the same inertial loads without the isometric pre release.



Diodes – IN 4002
Comparator – LM

APPENDIX G ELECTROMAGNET CONTROLLER CIRCUIT DIAGRAM

APPENDIX H MATHCAD PROGRAMME FOR CYCLING DATA ANALYSIS

Get the encoder signal $\mathrm{In} :=$ C:\..\cmwp1b.txt $\mathrm{En} := \mathrm{In}^{\langle 0 \rangle}$

set file start and finish parameter $\mathrm{start} := 2$ $\mathrm{end} := \mathrm{newcogratio} \cdot 6$

Assume a value for the mechanical constant of the system $\mathrm{Inertia} := \mathrm{ipl7}$

correct encoder start time $\mathrm{Enc} := [\,\mathrm{submatrix}[\mathrm{En}, \mathrm{start}, (\mathrm{end}), 0, 0]\,] - \mathrm{En}_{\mathrm{start}}$

Set the time for which the push record should last and define fitting period

$\mathrm{PushTime} := \mathrm{Enc}_{\mathrm{end-start}}\mathrm{inc} := \dfrac{\mathrm{PushTime}}{\mathrm{points}}$ $\mathrm{DelT} := \mathrm{inc}$

$\mathrm{SEsens} := 360$ $j := 1 .. \mathrm{rows}(\mathrm{Enc}) - 1$

$$\mathrm{AngVel}_j := \frac{2 \cdot \dfrac{\pi}{\mathrm{SEsens}}}{\mathrm{Enc}_j - \mathrm{Enc}_{j-1}} \quad \mathrm{TimeV}_j := \frac{\mathrm{Enc}_j + \mathrm{Enc}_{j-1}}{2} \quad \mathrm{AngAcc}_j := \frac{\mathrm{AngVel}_j - \mathrm{AngVel}_{j-1}}{\mathrm{Enc}_j - \mathrm{Enc}_{j-1}}$$

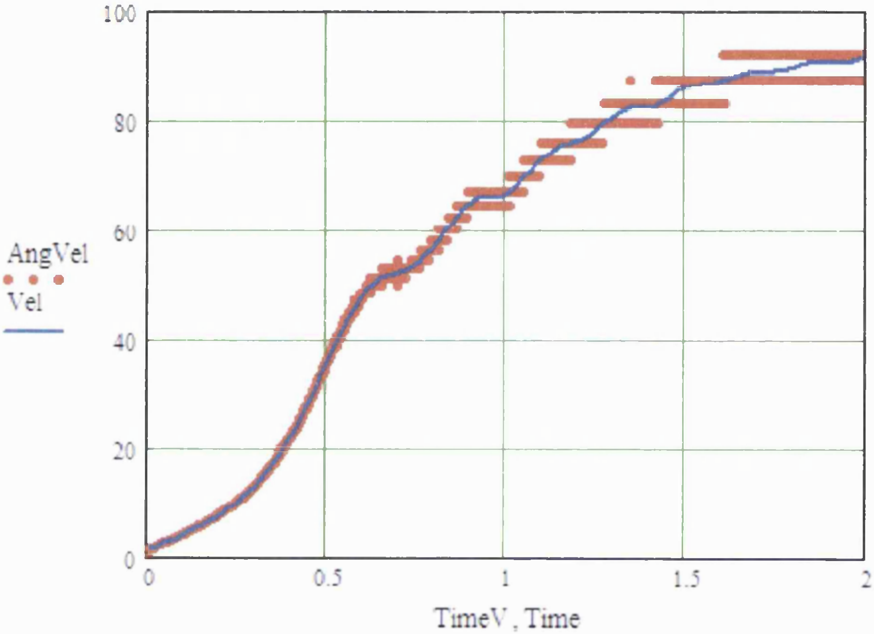
Make a time scale inc intervals on which the first encoder pulse is at time zero and Calculate the acceleration position and velocity from the encoder data at time intervals of inc sec

$\mathrm{Calcs}_4 = 149$

$\mathrm{Time} := \mathrm{submatrix}(\mathrm{Calcs}_0, 0, \mathrm{Calcs}_4, 0, 0)\mathrm{s} := \mathrm{submatrix}(\mathrm{Calcs}_1, 0, \mathrm{Calcs}_4, 0, 0)$

$\mathrm{Acc} := \mathrm{submatrix}(\mathrm{Calcs}_3, 0, \mathrm{Calcs}_4, 0, 0)\mathrm{el} := \mathrm{submatrix}(\mathrm{Calcs}_2, 0, \mathrm{Calcs}_4, 0, 0)$

Look at match between raw data and fitted data in order to decide on fitting parameter



fitting parameter and interval

points = 150 DelT = 0.018

Define mechanical formulae to calculate required variables

$$\text{Torque} := \overrightarrow{[\,\text{Acc}\cdot\text{Inertia} + [(\text{Acc}\cdot\text{Inertia})\cdot 0.0012]\cdot 2\,]}\text{io} \equiv 1020 \quad \text{newcogra}$$

$$\text{crankrat1} := \frac{\text{Ratio}}{360} \quad \text{crankrat2} := \frac{\text{newcogratio}}{360}$$

$$\text{rpm} := \overrightarrow{\frac{60\cdot\text{Vel}}{2\cdot\pi\cdot\text{crankrat2}}}\ \text{crank} := \frac{\left(\frac{\text{Pos}}{\text{deg}}\right)}{\text{newcogratio}} \quad \text{Power} := \overrightarrow{(\text{Torque}\cdot\text{Vel})}$$

$$\text{Wo}_0 := \frac{1}{2}\cdot\text{Pos}_0\cdot\text{Torque}_0 := 1 .. \text{rows}(\text{Time}) - 1$$

$$\text{Wo}_p := \text{Wo}_{p-1} + (\text{Pos}_p - \text{Pos}_{p-1})\cdot\left(\frac{\text{Torque}_p + \text{Torque}_{p-1}}{2}\right)$$

$$\text{crWo} := \text{linterp}(\text{crank}, \text{Wo}, 1)\text{n} := \text{linterp}(\text{crank}, \text{Time}, 1)\text{MeancrPower}$$

$$\text{pedtq} := \left(\frac{\text{Torque}}{0.03}\right)\cdot 0.18$$

Index to determine row at max power

```
index1 := | k ← 0
          | for  r ∈ 0 .. rows(Power) − 1
          |   k ← r  if  Power_r = max(Power)
          | k
```

```
breeturns :=  i ← 0
              ii ← 0
              iii ← 0
              iv ← 0
              v ← 0
              vi ← 0
              P1 ← 0
              P2 ← 0
              P3 ← 0
              P4 ← 0
              P5 ← 0
              P6 ← 0
              for r ∈ 0.. rows(crank) − 1
                if rev1(crank_r) = 1
                    P1_i ← Power_r
                    V1_i ← rpm_r
                    T1_i ← Torque_r
                    crk1_i ← crank_r
                    C1 ← lookup(max(P1), P1, crk1)
                    Vel1 ← lookup(max(P1), P1, V1)
                    Tor1 ← lookup(max(P1), P1, T1)
                    i ← i + 1
              for r ∈ 0.. rows(crank) − 1
                if rev2(crank_r) = 1
                    P2_ii ← Power_r
                    V2_ii ← rpm_r
                    T2_ii ← Torque_r
                    crk2_ii ← crank_r
                    C2 ← lookup(max(P2), P2, crk2)
                    Vel2 ← lookup(max(P2), P2, V2)
                    Tor2 ← lookup(max(P2), P2, T2)
                    ii ← ii + 1
              for r ∈ 0.. rows(crank) − 1
                if rev3(crank_r) = 1
                    P3_iii ← Power_r
                    V3_iii ← rpm_r
                    T3_iii ← Torque_r
                    crk3_iii ← crank_r
                    C3 ← lookup(max(P3), P3, crk3)
                    Vel3 ← lookup(max(P3), P3, V3)
                    Tor3 ← lookup(max(P3), P3, T3)
                    iii ← iii + 1
              for r ∈ 0.. rows(crank) − 1
                if rev4(crank_r) = 1
                    P4_iv ← Power_r
                    V4_iv ← rpm_r
                    T4_iv ← Torque_r
                    crk4_iv ← crank_r
                    C4 ← lookup(max(P4), P4, crk4)
                    Vel4 ← lookup(max(P4), P4, V4)
                    Tor4 ← lookup(max(P4), P4, T4)
                    iv ← iv + 1
              for r ∈ 0.. rows(crank) − 1
                if rev5(crank_r) = 1
                    P5_v ← Power_r
                    V5_v ← rpm_r
                    T5_v ← Torque_r
                    crk5_v ← crank_r
                    C5 ← lookup(max(P5), P5, crk5)
                    Vel5 ← lookup(max(P5), P5, V5)
                    Tor5 ← lookup(max(P5), P5, T5)
                    v ← v + 1
              for r ∈ 0.. rows(crank) − 1
                if rev6(crank_r) = 1
                    P6_vi ← Power_r
                    V6_vi ← rpm_r
                    T6_vi ← Torque_r
                    crk6_vi ← crank_r
                    C6 ← lookup(max(P6), P6, crk6)
                    Vel6 ← lookup(max(P6), P6, V6)
                    Tor6 ← lookup(max(P6), P6, T6)
                    vi ← vi + 1
                     ⎛ "Power"  "AvgPower"  "AvgTq"   "AvgVel"   "crankpos"  "Torque"  "Velocity" ⎞
                     ⎜ max(P1)  mean(P1)    mean(T1)  mean(V1)   C1_0        Tor1_0    Vel1_0     ⎟
                     ⎜ max(P2)  mean(P2)    mean(T2)  mean(V2)   C2_0        Tor2_0    Vel2_0     ⎟
              Out ←  ⎜ max(P3)  mean(P3)    mean(T3)  mean(V3)   C3_0        Tor3_0    Vel3_0     ⎟
                     ⎜ max(P4)  mean(P4)    mean(T4)  mean(V4)   C4_0        Tor4_0    Vel4_0     ⎟
                     ⎜ max(P5)  mean(P5)    mean(T5)  mean(V5)   C5_0        Tor5_0    Vel5_0     ⎟
                     ⎝ max(P6)  mean(P6)    mean(T6)  mean(V6)   C6_0        Tor6_0    Vel6_0     ⎠
```

Set parameters for each crank turn

$\mathrm{rev1}(x) \equiv (0.1 \le x \le 0.5)$ $\mathrm{rev2}(x) \equiv (0.51 \le x \le 1.0)$ $\mathrm{rev3}(x) \equiv (1.01 \le x \le 1.5)$

$\mathrm{rev4}(x) \equiv (1.51 \le x \le 2.0)$ $\mathrm{rev5}(x) \equiv (2.01 \le x \le 2.5)$ $\mathrm{rev6}(x) \equiv (2.51 \le x \le 3.0)$
$\mathrm{rev7}(x) \equiv (3.01 \le x \le 3.5)$ $\mathrm{rev8}(x) \equiv (3.51 \le x \le 4.0)$ $\mathrm{rev9}(x) \equiv (4.01 \le x \le 4.5)$

$\mathrm{rev10}(x) \equiv (4.51 \le x \le 5.0)$ $\mathrm{rev11}(x) \equiv (5.01 \le x \le 5.5)$ $\mathrm{rev12}(x) \equiv (5.51 \le x \le 6.0)$

Get data from matrix

$\mathrm{sixcrankturns} := \mathrm{augment}(\mathrm{submatrix}(\mathrm{sixturns}, 1, 1, 0, 6), \mathrm{submatrix}(\mathrm{sixturns}, 2, 2, 0, 6))$

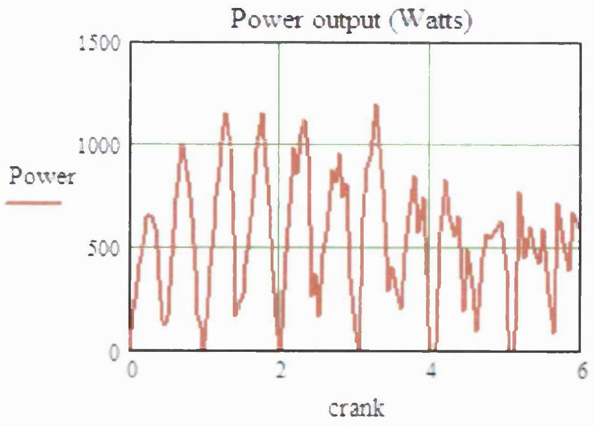
sixcrankturns =

	0	1	2	3	4	5	6
0	74.387	79.494	4.799	52.685	5.187	9.877	151.85

array of results

$\mathrm{results} := \left(\max(\mathrm{Power})\ \ \mathrm{rpm}_{\mathrm{index1}}\ \ \mathrm{Vel}_{\mathrm{index1}}\ \ \mathrm{Torque}_{\mathrm{index1}}\ \ \mathrm{crank}_{\mathrm{index1}}\ \ \mathrm{Time}_{\mathrm{index1}}\right)$

All parameters expressed in terms of crank turns



APPENDIX I 1999 WORLD MASTERS CHAMPIONSHIP

WEIGHTLIFTING MEETING PERFORMANCE TABLE

Subject	Age (years)	Age –group (years)	Weight category (kg)	Clean & jerk (kg)	Snatch (kg)	Total (kg)	Placing
W. Oyafuso	75	75 – 79	56	47.5	35	82.5	1
S. Ference	80	80+	62	42.5	37.5	80	2
V. Younger	87	80+	62	47.5	35	82.5	1
K. Saitl	75	75 – 79	62	70	50	120	2
L. D'Arcy	78	75 – 79	77	52.5	42.5	95	3
J. Garbutt	75	75 - 79	77	62.5	52.5	115	2
W. Mrosack	86	80+	85	55	45	100	1
C. Meole	84	80+	85	45	37.5	82.5	2
J. Santana	79	75 – 79	85	82.5	67.5	150	1
R. Howes	77	75 - 79	94	80	55	135	1
C. Lano	77	75 - 79	105+	65	50	115	1
S. Grant	74	70 – 74	69	50	40	90	3
A. Pietrowsky	74	70 - 74	77	60	45	105	3
B. Deni	72	70 - 74	77	90	80	170	1
D. Takeuchi	71	70 - 74	77	95	72.5	167.5	1
N. Munly	70	70 - 74	85	75	55	130	2
W. Underhill	70	70 - 74	85	82.5	62.5	145	1
K. Rosenburger	71	70 - 74	94	70	50	120	3
Y. Haatanan	66	65 - 69	62	82.5	70	152.5	1
D. Stewart	68	65 - 69	69	62.5	52.5	115	4
P. Serrano	68	65 - 69	69	87.5	75	162.5	1
M. Nivault	66	65 - 69	62	77.5	55	132.5	3
G. Gonzalez	67	65 - 69	94	82.5	62.5	145	3
M. Huszka	66	65 - 69	94	115	92.5	207.5	1

Subject	Age (years)	Age –group (years)	Weight category (kg)	Clean & jerk (kg)	Snatch (kg)	Total (kg)	Placing
D. Ramos	66	65 - 69	85	100	87.5	187.5	1
R. Crisp	65	65 - 69	94	75	62.5	137.5	5
N. Lewis	65	65 - 69	94	82.5	60	142.5	3
A. Huber	69	65 - 69	105	75	65	140	4
G. Macinko	68	65 - 69	105	90	67.5	157.5	3
M. Barton	60	60 - 64	69	87.5	60	147.5	6
F. Binkowski	63	60 - 64	69	82.5	65	147.5	5
W. Deppner	64	60 - 64	77	92.5	67.5	160	7
M. Gilz	63	60 - 64	77	92.5	77.5	170	6
H. Heid	60	60 - 64	77	92.5	77.5	170	5
E. Chevier	63	60 - 64	77	112.5	90	202.5	2
G. Byng	63	60 - 64	85	85	70	155	6
R. Strange	63	60 - 64	94	115	90	205	3
G. Hilley	56	55 - 59	62	90	70	160	2
S. Braithwaite	59	55 - 59	69	110	87.5	197.5	1
B. Ulfving	58	55 - 59	85	110	80	190	3
L. Graber	55	55- 59	105+	120	95	215	3
B. Halgand	52	50 - 54	69	112.5	85	197.5	3
V. Fontanal	51	50 - 54	85	125	97.5	222.5	3
S. Kozlowski	52	50 - 54	85	120	97.5	217.5	6
J. Sokolowski	52	50 - 54	85	125	100	225	2
L. McConnell	54	50 - 54	94	127.5	105	232.5	2
S. Jakobsson	52	50 - 54	105	165	135	300	1
J. Swiecici	45	45 - 49	105	160	135	295	2

Subject	Age (years)	Age –group (years)	Weight category (kg)	Clean & jerk (kg)	Snatch (kg)	Total (kg)	Placing
M. Cartwright	40	40 - 44	85	150	115	265	1
K. Hogg	40	40 - 44	94	132.5	107.5	240	2
G. Skinner	42	40 - 44	94	130	110	240	3
I. Borozan	44	40 - 44	62	95	75	170	DNF
L. Modis	42	40 - 44	69	125	100	225	DNF
K. Forster	44	40 - 44	77	115	95	210	DNF
G. Lenk	43	40 - 44	77	122.5	95	217.5	DNF
L. Kurti	43	40 - 44	77	137.5	112.5	250	DNF
L. Grayson	41	40 - 44	105+	170	145	315	1

RE-INVENTING THE WHEEL - MUSCLE POWER IN AGEING ITS MEASUREMENT & IMPLICATIONS

S.J.Pearson, S.D.R. Harridge, A.Young*, R.C.Woledge† & D.Grieve†

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Ageing is synonymous with the decline in the effectiveness of the various physiological systems. One such system is the human neuromuscular system. This system is of particular interest due to its importance in the ability to carry out everyday functional tasks. Of significance in the ability to carry out daily functional tasks is muscle power. It is thought that the decline in muscle power with ageing consists of many complex interactions. These interactions are further complicated by the fact that differences may exist between muscles used for different functions.

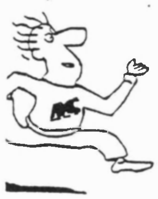
Of particular interest to this study are the muscles of the lower limbs. So chosen because without these muscles functioning correctly many tasks we take for granted such as chair rising walking and stair climbing would be impossible Along with this comes an increase in the risk of falls and the associated morbidity/mortality. Muscle power of the lower limbs as measured using the Nottingham Power Rig (NPR)[1] has been reported to decline at a greater rate than strength[2].

By measuring muscle power and determining the changes due to ageing some insight may be gained as to development of effective methods to reduce the effect of ageing on muscle power loss. In vivo muscle power measurement was made utilising an inertial wheel system known as the Nottingham Power Rig. This system was chosen due to the inertial load system closely mimicking everyday actions. The NPR utilises fixed inertial loads, this may in part explain the observed greater loss of power than strength. As the loads presented to the elderly may appear as a higher percentage of their maximum, forcing them to work at a lower velocity and possibly on a less favourable portion of their power curve[3].

We initially determined that certain assumptions relied on for the calculation of power by the NPR were erroneous. Also in order to investigate more closely muscle power loss design changes to the NPR were derived. Subsequently, three major areas of modification were initiated. These included

1. Mathematical equations to correct the NPR power calculation.
2. Mechano-electrical modifications.
3. A new data acquisition programme

From the changes that we have made to the original NPR system, we will be able to capture a greater variety of muscle contraction parameters including a) Instantaneous muscle power, b) Average power, c) Peak power, d) Instantaneous strength, e) Work done, f) Knee angle in relation to power/strength, and g) Optimal inertial loading for peak power.



National Institute for Medical Research
Mill Hill, London 21st-23rd April 1999

M&D MUSCULAR DYSTROPHY GROUP

The Physiological Society

British Heart Foundation

AMC ALTERNATIVE MUSCLE CLUB

1. Bassey E.J, Short.A.H. (1990). A new method for measuring power output in a single leg extension: feasibility, reliability and validity.

2. Skelton D.A et al. (1994). Strength, power and related functional ability of healthy people aged 65-89 years.

3. Harridge S.D.R, Young A. (1997). Skeletal muscle. In Principles and Practise of Geriatric Medicine.Pathy M.S.J. Wiley and Sons Ltd.

Telemetric recording of heart rate, motor activity and body temperature in small mammals during chronic cold exposure

D. Deveci, N. Ambrose * and S. Egginton †

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A variable inertial system for measuring the contractile properties of human muscle

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For many years human skeletal muscle power output has been studied using the inertial properties of flywheels (e.g. Hill, 1922). Inertial loading of a muscle has particular relevance for function, as when performing everyday activities muscles face gravitational and inertial challenges and are not, for instance, required to rotate joints at constant angular velocities (i.e. isokinetically).

The apparatus described here comprises a flywheel system, whose inertia can be varied, and where the mechanical properties of a muscle or muscle group are inferred from data collected by sensitive detection of the flywheel's rotation and simultaneous measurement of the torque applied to the flywheel.

With appropriate housing the system could be used to test different muscle groups. It is described here having been mounted in the apparatus described by Bassey & Short (1990) which measures lower limb extensor power during a single maximal leg thrust against a pedal and chain assembly. The present system consists of a lightweight flywheel to which can be attached additional steel plates, allowing inertia to be increased from 0·024 to 0·609 kg m^2. Rotation of the flywheel is detected by a rotary encoder (1 deg resolution) mounted via a flexible coupling on to the flywheel shaft. Torque applied to the wheel is recorded via a strain gauge mounted in a chain connecting the pedal assembly with the flywheel. A modified cam system ensures constant leverage to the flywheel. A facility for isometric pre-loading prior to movement is provided by an electromagnetic catch assembly controlled by a comparator receiving torque input from the strain gauge.

Square wave digital signals from the rotary encoder and analogue data from the strain gauge are sampled by an A/D convertor (Micro 1401 and Spike 2, Cambridge Electronic Design, UK). The analogue torque data are sampled at 2·5 kHz and the encoder signals with a time resolution of 2 μs. Limb movement data may also be sampled simultaneously (e.g. using an electrogoniometer). The data are then processed (Mathcad 7, Adept Scientific) for determination of mechanical properties (Fig. 1).

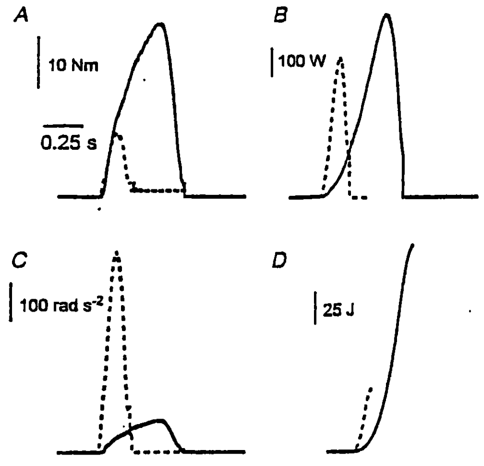

Figure 1. Torque (*A*), power (*B*) angular acceleration (*C*) and work done (*D*) during a single maximum lower limb extensor thrust against both a light inertia (0·024 kg m^2, shown as dashed line) and heavy inertia (0·410 kg m^2, continuous line) from a young male subject.

On six male subjects (aged 25–36 years) the highest average power recorded during a single contraction on this system ranged from 231 to 469 W and these values occurred at different levels of inertia (ranging from 0·092 to 0·232 kg m^2).

This study had approval from the local ethics committee.

S.H. is a Wellcome Trust Fellow.

Bassey, E.J. & Short, A.H. (1990). *Eur. J. Appl. Physiol.* 60 385–390.

Hill, A.V. (1922). *J. Physiol* 56, 19–21.

editing routine based on absolute values, plateau slope, and timing eliminated spurious readings with an accuracy of 98% when compared with readings by eye. Values were averaged over successive 1 min bins, and we demonstrated, by comparison with arterialized venous blood gas samples, that a minimum of six valid readings for each bin was required to give a meaningful mean $P_{\mathrm{ET,CO_2}}$ value during speech. Ambulatory heart rate was measured to indicate stressful episodes and severe exertion.

With ethical permission, we studied 25 normal subjects (8 males, 17 females) over 4 h during activities of daily living including gentle activity, talking, eating, reading etc., either within the hospital ($n = 18$) or at home ($n = 7$). We excluded asthma and other organic conditions. A mean of 172 min (S.D. 44, range 53–229) of data remained after editing. Mean $P_{\mathrm{ET,CO_2}}$ was 38·2 (S.D. 0·8) for males, and 36·9 mmHg (S.D. 3·2) for females. The lowest limit of the range (mean − 2S.D.) was between 30 and 35 mmHg in fifteen subjects and below 30 mmHg in four (1 male, 3 females). There were no significant differences between data collected by telemetry and logger, or between work and home. $P_{\mathrm{ET,CO_2}}$ did not correlate with respiratory frequency (mean 18·3, S.D. 2·4), Speilberger State or Trait anxiety scores, Beck Depression inventory scores, or heart rate (mean 76·6 beats min^{-1}, S.D. 6·3). These results show that some normal subjects can have spontaneous $P_{\mathrm{ET,CO_2}}$ values well below the accepted lower limit and within the range of 14–29 mmHg known to induce hypocapnic symptoms in humans. These subjects may be particularly susceptible to developing hyperventilation syndrome.

Gardner, W.N. (1996). *Chest* 106, 516–534.

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Muscle power in elite master weightlifters aged 43–87 years of age

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Muscle power declines with increasing age. The mechanisms responsible for this phenomenon are still not completely understood. Studying elite master athletes may aid in our understanding of the potential limits to which older muscles may function. In the present study, approved by the ethics committee of Strathclyde University, we measured the explosive power of the lower limb muscles in 42 men (aged 43–87 years) who were competitors in the World Masters Weightlifting championships (Glasgow, UK, 1999). It was hypothesized that, as the nature of Olympic weightlifting involves the rapid acceleration of large inertial loads, these athletes would also be capable of generating more power per unit leg volume when compared with age-matched controls. Power measurements were made using a testing system (Bassey & Short, 1990) with a modified flywheel assembly (Pearson *et al.* 2000). Subjects were required to perform three maximal lower limb extensor efforts against inertial loads, ranging from 0·024 to 0·550 kg m^2. These data were compared with measurements made on age-matched controls (age 42–86 years). Measurements of power were normalised to lower limb volume (Jones & Pearson, 1969). The values for peak (P_k) and average (P_a) power generated were compared

Weightlifters generated significantly more absolute P_a (45%, $P < 0·05$) and P_k (39%, $P < 0·05$) than the controls. When normalised to lower limb volume P_a was on average (40%, $P < 0·05$) greater and P_k (34%, $P < 0·05$) greater when compared with controls. Figure 1 shows that significantly more normalised P_a and P_k was generated by most age groups of weightlifters when compared with the control subjects.

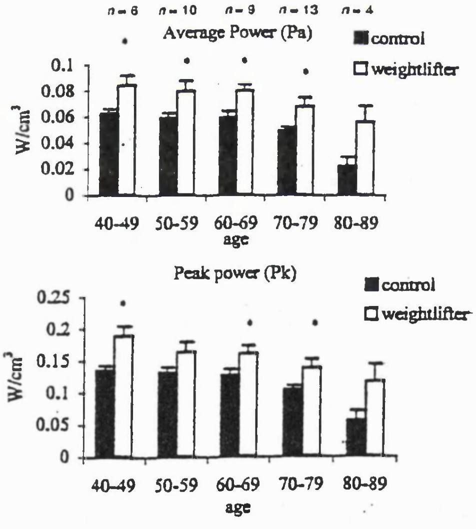

Figure 1. Data (mean ± S.E.M.) for (a) P_a and (b) P_k for the weightlifters and controls, normalised to lean lower leg volume. *$P < 0·05$ as determined by two-way ANOVA and *post hoc* unpaired *t* tests.

The data indicate that lower limb explosive muscle power in elite master weightlifters is significantly greater when compared with non-trained age-matched individuals. This is the case in both absolute terms and when expressed relative to lower limb volume. The difference was such that the mean maximum P_a for the 80+-year-old weightlifters equated to that of the 60- to 69-year-old control group.

Bassey, E.J. & Short, A.H. (1990). *Eur. J. Appl. Physiol.* 60, 385–390.

Jones, P.R.M. & Pearson, J. (1969). *J. Physiol.* 204, 63–66P.

Pearson S.J., Harridge, S.D.R, Grieve, D., Young, A. & Woledge, R.C. (2000). *J. Physiol.* 523.P, 135P.

S.D.R.H. is a Wellcome Trust Research Fellow.

Cardiovascular changes during tilt table testing before and during tilt training in patients with recurrent neurocardiogenic syncope

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In patients with neurocardiogenic syncope dramatic changes in heart rate and arterial blood pressure have been observed during tilt table testing, leading to syncope. After repeated tilt tests we observed a rapid and sustained improvement of tilt tolerance with disappearance of syncope, already after a few days of tilt training. Therefore tilt training is considered as a therapy in our departement (Reybrouck *et al.* 2000).

The purpose of the present study was to analyse cardiovascular changes before and during tilt table training (TT) in a group of heavily symptomatic patients. This study was approved by the ethical committee. 109 patients (59 males and 50 females, mean age 32·1 ± 2·0 (S.E.M.) years) with recurrent neurocardiogenic syncope were admitted to the hospital and underwent a diagnostic tilt table test with 60 deg inclination (Westminster protocol), without pharmacological provocation. Forty-four patients were diagnosed as vasodepressor type of syncope (VD), 34 with cardio-inhibitory (CI) type (asystole for at least 3 s) and 31 with mixed type (MX). During the tilt test heart rate was recorded by ECG (telemetry) and arterial blood pressure was continuously monitored by an oscillometric method (Dinamap). The tilt test was continued until syncope or severe orthostatic intolerance. The patients were tilted daily until syncope or until a normal value of at least 45 min was reached.

Table 1.

Type of syncope	Tilt duration (min)	ΔHR (b.p.m.)	ΔSBP (mmHg)
VD (n = 44)			
Before TT	21·5 ± 1·8	−3 ± 4·1	−54·4 ± 6·2
End hospit	48·4 ± 1·4†	5 ± 1·8*	−1·9 ± 2·2†
CI (n = 34)			
Before TT	14·5 ± 1·7‡	−74·8 ± 5·2	−112·2 ± 2·8
End hospit	47·8 ± 1·2†	7·0 ± 1·8†	−2·2 ± 1·3†
MX (n = 31)			
Before TT	22·6 ± 2·2	−33·4 ± 4·4	−77·2 ± 7·4
End hospit	45·8 ± 0·9†	4·0 ± 1·7†	−3·2 ± 1·9*

Values are reported as means ± S.E.M. * $P < 0{\cdot}05$, † $P < 0{\cdot}001$: paired comparison before TT *vs.* end hospitalisation (hospit) (Student's paired *t* test). ‡ $P < 0{\cdot}05$: comparison between three groups of syncope, before tilt training (ANOVA). ΔHR and ΔSBP, changes in heart rate and systolic blood pressure between start and termination of tilt test. b.p.m., beats per minute.

The results show at the diagnostic tilt test a dramatic decrease in systolic blood pressure in the three patient groups, which was associated with bradycardia in patients with mixed type of syncope or with asystole (mean: 13·0 ± 2·0 s) in the patients with the cardioinhibitory type of syncope. At discharge from the hospital (after 3·4 ± 0·1 days of TT on average) all patients could sustain the tilt test for at least 45 min (normal value) without dramatic changes in heart rate and blood pressure.

In conclusion, this study shows that a programme of regular tilt training can condition the cardiovascular orthostatic reflexes with disappearance of syncope already after a few sessions of tilt training. However, tilt training has to be continued daily, otherwise syncope recurs again (Reybrouck *et al.* 2000).

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Effects of prior concentric training on exercise-induced muscle damage of the elbow flexors

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Exercise-induced muscle damage (EIMD), particularly that which follows strenuous eccentric exercise, is well documented. So too is the observation that a prior bout of eccentric exercise reduces the severity of symptoms of EIMD after a subsequent bout. This has been attributed to an increase in sarcomeres in series. Lynn & Morgan (1994) and Lynn *et al.* (1998) observed increases in the number of sarcomeres in the vastus intermedius muscle of rats after

32.6 l min^{-1}), and a-v O_2 difference (~155 to 171 ml l^{-1}). There was only a small difference in arterial saturation between leg and combined arm and leg exercise.

Table 1. Mean values ± S.D. in diagonal skiing (DIA), running (RUN), and double-poling (DP) for heart rate (f_H; beats min^{-1}), blood lactate concentration (HLa; mmol l^{-1}) and oxygen uptake ($\dot{V}_{O_2}$; l min^{-1}) ($n = 7$)

Study A	f_H	HLa	$\dot{V}_{O_2}$
DIA_{max}	189 ± 7.6	10.0 ± 1.0	6.23 ± 0.47
RUN_{max}	189 ± 5.6	9.2 ± 1.7	6.00 ± 0.42
DP_{max}	186 ± 5.6	8.2 ± 1.5	5.36 ± 0.28

We interpret these data to indicate that upper body muscle mass even in a well-trained athlete is not sufficient to elicit maximal oxygen uptake. Combining arm and leg exercise does increase peak oxygen uptake, but the difference is small and markedly less than could be anticipated from the $\dot{V}_{O_2}$ reached when performing either lower or upper body exercise, suggesting a central limitation, i.e. systemic oxygen delivery sets an upper limit. Arterial desaturation is only to a minor extent a contributing factor; rather the capacity of the heart to provide a cardiac output sets the upper functional limit.

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Power output characteristics during sprint cycling using a variable inertial system

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During a single lower limb extensor movement over a fixed distance, the power–velocity relationship obtained against an inertial load, provided by an adjustable flywheel is parabolic (Pearson *et al.* 2000). The reason for the decline in the peak power that can be obtained at high inertias may result from the completion of the movement before the load can be accelerated to an optimal velocity for peak power generation. In contrast to a single movement, a repeated action performed against a high inertial load, such as cycling, should allow for the progressive acceleration of an inertial load. It was thus hypothesised that the peak power output measured during sprint cycling would, within limits, be independent of the level of inertia, but would depend upon the time allowed for the acceleration of the load.

In the present study approved by the local ethics committee, we examined the power generated by the lower limb muscles of nine healthy men; mean age (29.4 ± 2.2 years) using two different test protocols. Following a 5-min warm up, the subjects performed a single maximal lower limb thrust (T1), on a variable inertial flywheel system (Pearson *et al.* 2000). Each subject performed two exertions at five inertial loads in a randomised order ranging from 0.024 to 0.54 kg m^2. In T2, performed on a separate occasion, two sprints were performed at each of the same inertias on a modified cycle ergometer attached to the flywheel assembly. The best exertion at each inertia was used for purposes of analysis.

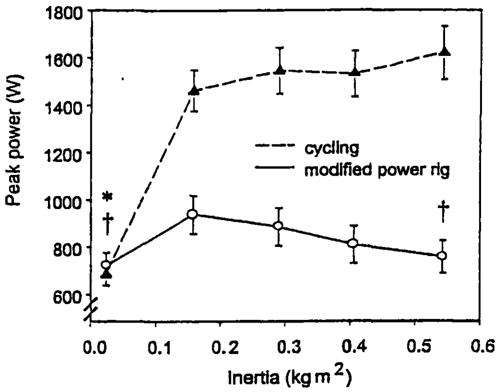

Figure 1. Peak power output T1 (○) and T2 (▲). Values are means ± S.E.M., $n = 9$. *Difference from subsequent inertias (T2); †difference from 0.158 kg m^2 (T1) ($P < 0.05$) using repeated measures ANOVA with Tukey *post-hoc* pairwise comparisons.

In T1 the maximal values for peak power occurred at an inertia of 0.158 kg m^2 and declined thereafter. In T2 there was no significant change in peak power beyond 0.158 kg m^2. The time to reach peak power was 0.99 ± 0.10 s (~1.4 crank revs) and 2.39 ± 0.14 s (~2.8 crank revs) at 0.158 and 0.54 kg m^2, respectively ($P < 0.05$). It is likely that a similar value of peak power will always be achieved during cycling against different inertial loads. This is because as an inertial load accelerates, a muscle will move down its force–velocity relationship and eventually achieve a velocity that corresponds to its optimum for power generation (~30 % V_{max}). This is providing that (i) the muscle is not fatigued at this point and (ii) the inertia is of sufficient magnitude to allow forces to be produced that are initially greater, or equal, to those at which peak power can be generated.

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S.D.R.H. is a Wellcome Trust research fellow.

Changes in physical fitness in elite youth team soccer players, aged 11 and 12 years old over a soccer season

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During a competitive soccer season, young players are subjected to physical exertion during both training sessions and matches. A combination of this physical exertion and increases in strength due to maturation may result in changes in physical fitness (Armstrong & Welsman, 1997). The aim of this study was to investigate changes in physical fitness in elite youth team soccer players over a competitive season.

Fourteen boys, aged 11–12 years were recruited from the soccer academy of a nationwide league side. Fitness, as predicted by sprint times over 10 m, performance during the multistage shuttle run (MSR), agility (measured by a run through cones), and hamstring flexibility (sit and reach test) were monitored at 7 week intervals over the complete soccer season. Somatotype was calculated using the Heath & Carter (1967) method.

Linear regression coefficients (LRCs) for each measured parameter were calculated and one-tailed *t* tests used to detect any significant changes in the LRCs over the season. Pearson's correlation coefficients were calculated to test the relationships between parameters.

Over the season there were significant increases in the LRCs for height, weight endomorphy and mesomorphy, accompanied by significant decrease in ectomorphy ($P < 0.05$) with no overall change in body fat. Fitness improved as measured by time taken to complete the agility run ($P < 0.05$) but decreased as indicated by increased time taken for the sprint. Hamstring flexibility and MSR performance showed no significant change.

Table 1. Correlation coefficients for fitness and anthropometric characteristics ($P < 0.05$ where values shown)

	Sprint time	MSR	Agility
Ectomorphy	n.s.	n.s.	n.s.
Endomorphy	n.s.	n.s.	0.61
Mesomorphy	–0.312*	n.s.	0.323*
Flexibility	0.324	n.s.	n.s.

*NB The sign for the correlations with 10 m sprint and agility have been reversed.

The significant negative correlation between both weight and mesomorphic characteristics with sprint times could explain this decrease in performance.

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The Chester College ethics committee provided ethical approval.

Orthostatic tolerance and resting blood pressure in humans: the effect of isometric exercise training of the arms

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To prevent syncope during orthostasis, the cardiovascular system must maintain blood pressure (BP). A lower baseline systolic BP (SBP) has been reported in subjects with low orthostatic tolerance, compared with more tolerant subjects (Stevens, 1966). Therefore, reduced resting BP may influence tolerance to simulated orthostasis (LBNP). Previously, a reduction in resting BP has been reported following isometric exercise training (Wiley *et al.* 1992). The purpose of this study was to reduce resting BP using isometric exercise training and assess its effect on LBNP tolerance.

LBNP tolerance and resting BP were determined before (first LBNP test) and after (second LBNP test) a 5 week period of training, in a normotensive training group (T) ($n = 8$; mean ± S.D.; age 21.0 ± 1.4 years; height 177.1 ± 9.9 cm; mass 78.3 ± 10.3 kg) and a normotensive control group (C) ($n = 8$; age 24 ± 4.3 years; height 174.0 ± 9.4 cm; mass 73.7 ± 10.7 kg). During the training

In situ experiments were performed on GM muscle–tendon complexes of anaesthetised (urethane, 1.5 g kg^{-1} i.p.) male Wistar rats ($n = 12$, body mass 243–302 g). Proximal and distal muscle parts (each $n = 6$) were activated maximally. The muscles were subjected to eccentric contractions at eight different velocities (5, 10, 15, 20, 40, 60, 100, 150 mm s^{-1}) in random order. Stretches (3 mm) started from a maximal isometric force plateau (F_{before}) at 2.5 mm below optimum length. After reaching peak eccentric force (F_{peak}), stimulation was continued for 100 ms to obtain isometric force after stretch (F_{after}). Normalised eccentric force was calculated as $(F_{peak} - [F_{after} - F_{before}])/F_{before}$ (de Ruiter *et al.* 2000) and expressed as a percentage. After the experiments, the animals were humanely killed.

Maximal isometric forces were 5.1 ± 1.7 and 5.4 ± 1.8 N (mean ± s.d.) for the proximal and distal part ($P > 0.05$, Student's unpaired *t* test), respectively. There was a significant effect of velocity on eccentric force, with no differences between muscle parts ($P < 0.05$, ANOVA repeated measures). Maximum normalised forces were obtained at 60 mm s^{-1} and were 157 ± 3 and 153 ± 6 % for the proximal and distal part, respectively. In conclusion, oxidative and glycolytic parts of rat GM have similar eccentric force–velocity relationships despite differences in metabolic, isometric and concentric contractile properties. Apparently, fibre type composition affects muscle function more when muscles are used as a motor than when used as a brake.

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All procedures accord with current National guidelines.

A calcium-dependent non-cross-bridge stiffness in frog skeletal muscle

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Our previous data showed that static stiffness increase following the stimulation both in twitch or tetanic contractions has a time course distinct from that of tension and roughly similar to that of internal calcium concentration. We hypothesized that static stiffness could be attributed to elastic properties of elements of the sarcomere structure whose stiffness increases in a calcium-dependent way. The experiments reported here were made to test the validity of this hypothesis. To investigate the effects of intracellular calcium we measured the static stiffness in single frog muscle fibres under different conditions in which isometric tension was inhibited either by reducing calcium release or by a direct inhibition on the actomyosin interaction.

Frogs (*Rana esculenta*) were killed by decapitation followed by double pithing. Single fibres, dissected from tibialis anterior muscle, were mounted between the lever arms of a force transducer and a moving coil stretcher by means of aluminium clips. Average sarcomere length in a selected segment (1–2 mm long) of the fibre was measured using a striation follower device. The temperature was maintained constant at 14°C. The experiments were made in Ringer and test solutions containing one of the following agents: 2,3-butanedione monoxime (BDM) at 1–5 mM concentration, dantrolene and methoxyverapamil (D600) at 5–10 μM concentration, deuterium oxide Ringer (98 % of water substituted with D_2O) and hypertonic solution up to 1.6 normal tonicity. To measure the static stiffness the fibres were rapidly stretched (up to 40 nm per half-sarcomere and about 0.5 ms duration) and hold for a period longer than the twitch time course. The fast tension transient was followed by a period during which the tension remained constant at a level that greatly exceeded the isometric force. This level is the static tension and the ratio between the static tension and the accompanying sarcomere length is the static stiffness. The complete time course of static stiffness was determined by applying stretches with different delays with respect to the stimulation.

The results show (Table 1) that static stiffness is almost unaffected by BDM and hypertonic solution, agents that inhibit tension generation mainly by affecting actomyosin interaction reducing cross-bridge formation, even at BDM concentration or Ringer tonicity that reduced twitch tension by more than 90 %. In contrast, a similar degree of tension inhibition was accompanied by strong static stiffness reduction when deuterium oxide, dantrolene or D600, agents which mainly reduce the calcium release, were used to inhibit tension generation.

Table 1. Relative values to normal Ringer solution

Tension inhibitors	Concn	Mean relative tension	± s.e.m.	Mean relative static stiffness	± s.e.m.	*n*
D600	20 μM	0.134	0.049	0.248	0.079	4
D_2O	98 %	0.081	0.022	0.569	0.066	5
Dantrolene	6.25 μM	0.202	0.038	0.641	0.038	4
BDM	2.5 mM	0.116	0.007	0.936	0.020	4
Hypertonicity	1.4 T	0.279	0.041	0.977	0.030	5

Our data show that the static stiffness increase in frog skeletal muscle is calcium dependent, in line with our hypothesis, suggesting that the elastic sarcomere structure responsible for static stiffness may be calcium sensitive.

All procedures accord with current National guidelines.

A lesser proportion of fast myosin heavy chain isoforms in older men is closely related to a lower velocity at which peak power occurs during inertial sprint cycling

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Peak power output and the velocity at which peak power occurs (V_{opt}) during sprint cycling has been shown to be lower in older people (Davies *et al.* 1983). The lower power output can be explained in part by reduced muscle mass; however, this should not explain the lower V_{opt}. It was the aim of the present study to determine if the lower V_{opt} in older people is related to a lower proportion of fast myosin isoforms in their quadriceps muscle.

We examined the power–velocity characteristics of 14 healthy men (7 young, 29 ± 2 years; 7 old, 74 ± 2 years) using a recently developed inertial cycle system (Pearson *et al.* 2002). Prior to testing, a standardised 5 min warm-up was undertaken, two maximal sprints were then performed at five inertial loads, ranging from 0.16 to 0.54 kg m^2. The best performance was used for analysis. V_{opt} was determined by a third-order polynomial fitting technique. Following local anaesthesia (1 % Lignocaine), muscle biopsy samples were obtained from the vastus lateralis muscle using the needle technique and were subsequently analysed for relative MHC isoform composition using gel electrophoresis (SDS-PAGE).

The mean (± S.E.M.) peak power, V_{opt} and % MHC-II (MHC-IIA + MHC-IIX) were 847 ± 47 *vs.* 406 ± 53 Watts, 120 ± 4 *vs.* 89 ± 6 r.p.m. and 52 ± 7 *vs.* 25 ± 7% for the young and older groups, respectively. All measures were shown to be significantly lower in the elderly group when compared with the young ($P < 0.05$, Student's unpaired *t* test). V_{opt} was found to correlate significantly with the % MHC-II (Fig. 1).

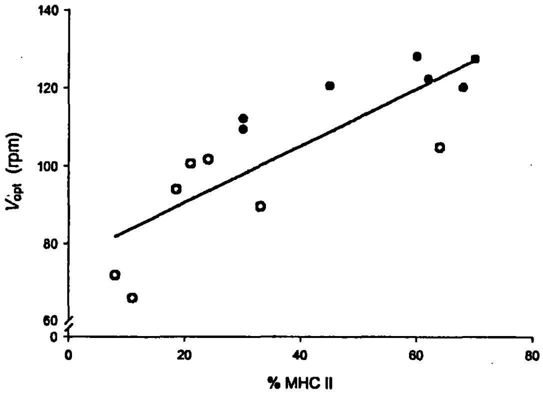

Figure 1. Relationship between %MHC-II and V_{opt}. ●, young; ○, old. $V_{\mathrm{opt}} = 76.9 + 0.72\,\mathrm{MHC\text{-}II}$ ($r = 0.82$, $P < 0.001$, $n = 14$).

The results show that the lower V_{opt} in older men relates closely to a lower proportion of the fast contracting MHC-II isoforms in their quadriceps muscle.

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Pearson, S.J. *et al.* (2002). *J. Physiol.* 539.P, 59–60P.

S.D.R.H. is a Wellcome Trust Research Fellow.

All procedures accord with current local guidelines and the Declaration of Helsinki.

Nandrolone decanoate treatment affects the steady-state inactivation of contraction and the sarcoplasmic reticulum Ca^{2+} uptake in rat soleus muscle

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The widespread use and abuse of anabolic androgenic steroids (AAS) have generated tremendous interest in effects, side effects, and raised ethical questions related to the use of these agents, in athletics populations (Shahidi, 2001). However, few studies have been devoted to the analysis at the cellular level of the change due to AAS treatment on the skeletal muscle excitation– contraction coupling mechanism.

Twenty male Wistar rats were divided into two groups; one group received weekly (for 6 weeks) an intramuscular injection of nandrolone decanoate (15 mg kg^{-1}) and the second group received similar doses of vehicle. One week after the last injection, rats were anaesthetised by an ether vapour flow and killed by cervical dislocation; soleus muscle was quickly excised and placed in oxygenated solution. Isolated small bundles (2–4 cells) were dissected for intact and saponin-skinned experiments and mounted in experimental chambers as already described by Joumaa *et al.* (2002).

In intact fibre bundles, the activation curve of the $[\mathrm{K}^+]_\mathrm{o}$ contracture was obtained by a rapid change from the control solution to one containing an elevated potassium concentration (20–146 mM $[\mathrm{K}^+]_\mathrm{o}$). The inactivation curve of $[\mathrm{K}^+]_\mathrm{o}$ contracture was obtained by measuring test 146 mM $[\mathrm{K}^+]_\mathrm{o}$ contracture amplitude after submaximal depolarisation for 2 min in a conditioning $[\mathrm{K}^+]_\mathrm{o}$. In this solution, the $[\mathrm{K}^+][\mathrm{Cl}^-]$ product was kept constant to allow rapid recovery of resting membrane potential and restoration of the amplitude of tension response. Membrane potentials were measured in the usual way (Joumaa *et al.* 2002). No significant change was observed in 146 mM $[\mathrm{K}^+]_\mathrm{o}$ contracture characteristics after drug treatment (amplitude normalised to saponin-maximal tension: control, 85.7 ± 2.9%; treated, 91.9 ± 3.1%; time-to-peak: control, 13.5 ± 0.5 s; treated, 12.9 ± 0.6 s; time constant of relaxation: control, 5.5 ± 0.3 s, treated, 6.1 ± 0.5 s; $n = 12$, mean ± S.E.M., ANOVA one-way statistical test). In treated muscle, a shift to more negative potential of the steady-state inactivation curve was found (membrane potential for 50% of inactivation (mV): control, –40.5 ± 1.1; treated, –48.7 ± 1.2; $n = 10$; $P < 0.05$), whereas no significant change was detected on the voltage dependence activation curve (membrane potential for 50% of activation (mV): control, –38.7 ± 0.4; treated, –38.1 ± 0.6, $n = 10$).

In saponin-skinned fibres, the amount of Ca^{2+} taken up at different loading times in pCa 7.0 solution was estimated by using the amplitude of the contracture due to caffeine application (10 mM). The semilogarithmic plot of the relative tension against time during the different loading time allows estimation of the rate of the Ca^{2+} uptake by the sarcoplasmic reticulum. The loading rate was significantly decreased after 6 weeks of nandrolone decanoate treatment (control: 0.0079 ± 0.0012 s^{-1}; treated: 0.0039 ± 0.0007 s^{-1}, $n = 8$, $P < 0.05$).

It has been shown previously that, in frog muscle (Même & Léoty, 1999), the decay of tension in skeletal muscle during prolonged steady-state depolarisation depends not only on inactivation of the process regulating Ca^{2+} release from the sarcoplasmic reticulum, but also on the ability of the sarcoplasmic reticulum to pump Ca^{2+}. Then, in the absence of a significant difference in the relaxation phase of $[\mathrm{K}^+]_\mathrm{o}$ contracture between treated and control soleus fibres, it could be proposed that, in treated muscle, the effect of the shift in the steady-state inactivation curve was compensated for by the slowing of Ca^{2+} uptake by the sarcoplasmic reticulum.

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Même, W. & Léoty, C. (1999). *Acta Physiol. Scand.* 166, 209–216.
Shahidi, N.T. (2001). *Clin. Ther.* 23, 1355–1390.

All procedures accord with current National guidelines.

Stimulation of sarcoplasmic reticulum Ca^{2+}-ATPase by disulfiram induces changes in voltage activation of contraction in rat slow-twitch skeletal muscle

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In skeletal muscle fibres, potassium contractures have been widely used as a convenient experimental model for the study of depolarisation–contraction coupling. It is generally recognised

APPLIED SCIENCES

Biodynamics

A variable inertial system for measuring the contractile properties of human muscle

STEPHEN J. PEARSON, STEPHEN D. R. HARRIDGE, DONALD W. GRIEVE, A. YOUNG, and ROGER C. WOLEDGE

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ABSTRACT

PEARSON, S. J., S. D. R. HARRIDGE, D. W. GRIEVE, A. YOUNG, and R. C. WOLEDGE. A variable inertial system for measuring the contractile properties of human muscle. *Med. Sci. Sports Exerc.*, Vol. 33, No. 12, 2001, pp. 2072–2076. **Purpose:** A flywheel system of variable inertia is described for inferring the mechanical properties of human muscle during a single explosive movement. **Methods:** The system consists of a lightweight aluminum disk mounted on a shaft onto which a driving cog is mounted. The inertia of the system can be varied from 0.024 to 0.69 $kg \cdot m^2$ by attaching semicircular steel plates to the disk. A rotary encoder detects displacement of the wheel with a resolution of 1°. Digital signals from the encoder are collected using an A/D converter interfaced to a PC. The data are then processed for the calculation of torque, velocity, power, work done, and acceleration. The mechanical properties of the muscles employed are inferred from calculations of flywheel displacement, time, and force. In addition, a pretension release mechanism can be incorporated into the system to allow isometric force to be developed before movement. This can increase power generation at the low inertias where the time of contraction is typically less than 200 ms. Seven subjects were test-retested using the device. Measures of both average and peak power were made **Results:** When mounted in the apparatus described by Bassey and Short, the maximum values for peak and average power were on average 965 ± 103 and 448 ± 47 W, respectively. Upon retesting, these results were found to be reliable (cv = 3.3% and 3.0%, respectively). **Conclusions** The inertial system described has been shown to have validity in reproducibility and provided a suitable method of determining a number of muscle output properties during short-term single exertions. This tool could prove useful in a research or clinical setting and may also prove useful as a training device as it negates the need for a strain gauge or goniometer attachment.

Key Words: INERTIA, POWER, TORQUE, FLYWHEEL, MUSCLE

Over recent decades, isotonic, isokinetic, hydrodynamic, and other systems have been designed and used to measure the dynamic contractile properties of human muscle. However, the acceleration of inertial loads, most closely resembles the challenges faced by skeletal muscles in everyday exertions. A. V. Hill was one of the first to realize the potential use of inertia in the form of a flywheel to study muscle in the 1920s (6). In a purely rotational inertial system, such as a flywheel, wheel angular acceleration is directly proportional to the torque applied to it. From determination of the inertia and rotational movement of the flywheel alone, it should therefore be possible to infer the mechanical properties of the muscular exertions accelerating the flywheel, assuming that the body and the machine are tightly coupled. Others (1,4) have also described systems for studying human muscular exertions against inertial loads, which involve the use of strain gauges to directly measure applied torque and goniometers to measure joint rotation. Recently, the average muscle power developed during a lower limb extensor thrust was measured by only determining the motion of a flywheel (3). In this study, the terminal velocity of a heavy flywheel of known inertia was measured (thereby obtaining its kinetic energy). Average power output was estimated on the assumption that the wheel's acceleration (and therefore the applied torque) was constant throughout the exertion.

In this paper, we describe a system that has practical advantages over strain-gauge–based systems by using a rotary encoder for the sensitive detection of the rotation of a flywheel, which may be rapidly accelerated to velocities in excess of 480 rpm within 200 ms. From derivation of the rotational properties of the flywheel, several features of limb thrust may be inferred without additional strain gauge or goniometer attachments. The instrument described allows for the inertia of the flywheel to be varied and for static preloading contractions to be performed. It is described here installed in a modification

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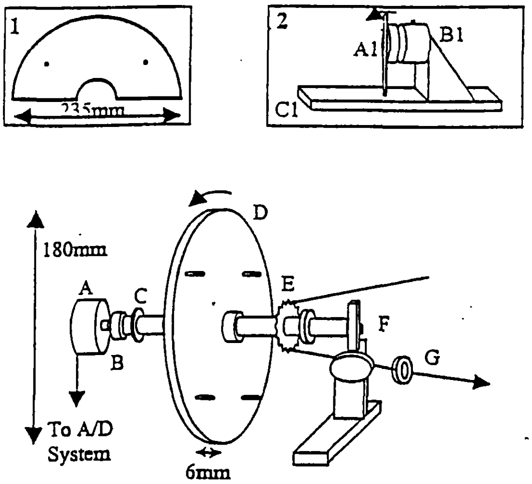

FIGURE 1—Inertial system. Inset 1, inertial loading plates. Inset 2, release mechanism; A1, locking arm and armature; B1, electromagnetic solenoid. C1, fixing plate. Main: A, rotary encoder; B, flexible coupling; C, self-aligning low-friction bearings; D, inertial wheel; E, drive cog and freewheel mechanism; F, locking latch; G, strain gauge assembly.

of the system described by Bassey and Short (3) but may be housed differently in order to study other muscle groups.

MATERIALS AND METHODS

Instrumentation. The inertial flywheel system is shown in Figure 1. A lightweight aluminum disk (6 mm thick, 180 mm diameter), with moment of inertia (MI) 0.024 kg·m^2, was mounted centrally via two low friction self-aligning bearings onto a steel shaft. Semicircular steel plates (2 mm thick, 235 mm diameter), which can be mounted on the disk in pairs, acted as inertial loading plates (Fig. 1, inset). The shaft also held a driving cog (60 mm diameter), which had an inbuilt freewheeling mechanism. Mounted on one end of the shaft via a flexible coupling (Hengstler 1.076.014; Hengstler GmBH, Aldingen, Germany) was a rotary encoder (Hengstler RI58 0/360AS0.41RB). On the other end, a locking latch was mounted for a release mechanism. Displacement of the wheel was measured by the rotary encoder, which produced one pulse for every degree of wheel rotation.

A ring transducer mounted in a chain driving the cog can measure force applied to the flywheel. This had four strain gauges (RS 632-124; RS Components Ltd, Northhants, United Kingdom) connected to form a Wheatstone bridge. The observed time constant of the strain gauge system was 4.5 ms, which was allowed for in the calculations. When the system is mounted in the apparatus described by Bassey and Short (3), the chain is connected to drive pedals via a modified constant lever system. The system can also be mounted in a purpose built knee extension rig, e.g., Aagaard et al. (1).

TABLE 1. Typical loss values: total work was calculated from strain gauge values, i.e., inclusive of losses.

Inertia (kg·m^2)	Losses (J)	% Work Due to Losses	Total Work (J)
Low (0.024)	7.5	15.4	48.6
High (0.54)	16.9	9.8	173.3

A large electromagnet (Emessem G MH × 100; Solenoid Company Ltd, Middlesex, United Kingdom) with a holding force of 3000 N was aligned with the locking latch. This electromagnet was controlled via a comparator device, which allowed the wheel to be released at a preset level of force.

Data aquisition. The analog signal from the strain gauge was amplified via a purpose built strain gauge amplifier. The digital signal from the rotary encoder was converted from quadrature pulses to a TTL compatible signal. Both analog and digital signals were then sampled by an A/D converter (CED micro1401; Cambridge Electronic Design Ltd, Cambridge, United Kingdom). The analog channel was sampled at 2.5 kHz; the digital channel set to detect the falling edge of pulses had an event resolution of 2μs. A PC, utilizing Spike 2 software (CED Ltd.), logged the sampled data. Analysis was carried out by utilizing a mathematical software package (Mathcad Ver. 7; see Appendix for details of calculations). Data were averaged by utilizing a smoothing algorithm based on a second order regression fitting procedure and a moving window based on the push time divided by the number of sample points to produce a set

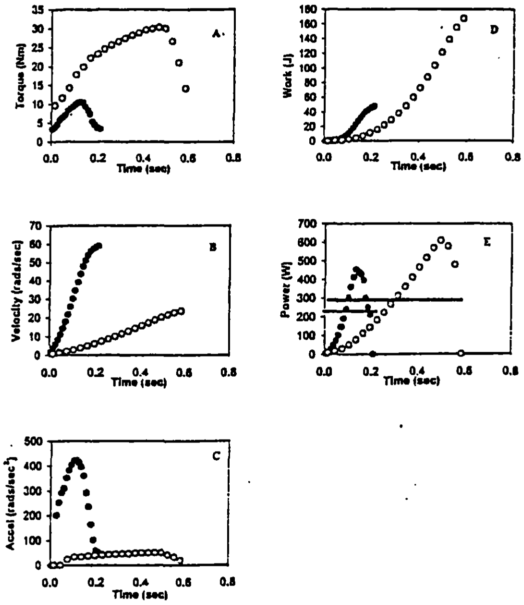

FIGURE 2—An example of A) torque, B) angular velocity, C) angular acceleration, D) work done, and E) power during a single exertion against low (●) and high (○) inertias created by a single subject. Calculated mean power is shown in graph E (___).

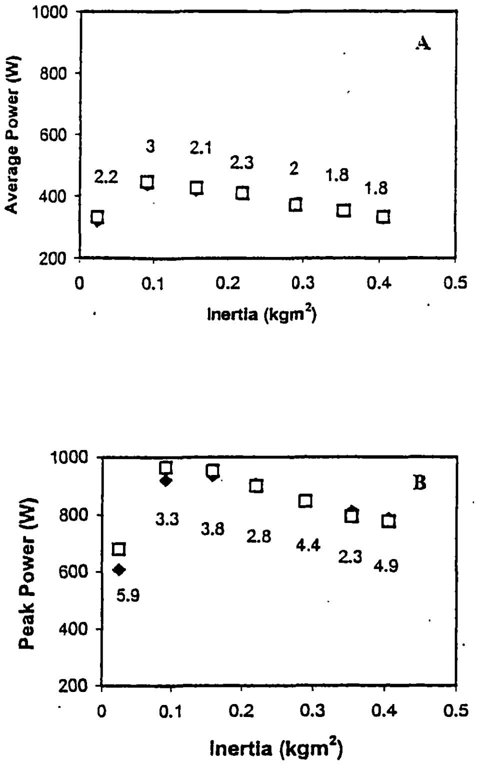

FIGURE 3—Mean group values for A) average and B) peak power, N = 7. Test 1 (☐) Test 2 (◆). The mean coefficient of variation is given below the symbol. Overlap of symbols reflects agreement of test-retest results.

number of data points per sample (usually 20); this reduced the noise present when using second derivative procedures but allowed sufficient resolution to identify physiological events. Data were then fitted using first and second order polynomial techniques to allow power, velocity, acceleration, work done, and torque data to be calculated. Maximal values, as well as averages, can be obtained from the data.

Calculation of system losses. In a rotary system, other factors in addition to inertia are always present, and a constant torque opposing wheel rotation was detected during a run down test. This test involved suspending a known mass from the inertial wheel at a given distance from the floor and allowing the mass to fall. Time of fall was recorded along with rotations of the wheel both while mass was falling and after it had landed. This allowed calculation of the opposing torque. When mounted in the apparatus described by Bassey and Short (3), work is also done against the return spring. Attaching known masses to the spring and recording length change determined the spring stiffness (see Table 1). The calculated system losses included work done against the return spring and work done to overcome the constant torque opposing motion, which includes frictional losses in all adjacent bearing surfaces.

Data collection. The repeatability within subjects using the device was examined on a group of seven male subjects. The physical characteristics, mean (SD), of the subjects were age (yr) 25.8 (5.8), height (m) 1.8 (0.1), and weight (kg) 76.3 (6.5). The subjects each performed four contractions at seven different inertial loads, range 0.024–0.405 kg·m^2, on two separate occasions. The best value for each subject was used for the purposes of analysis of average and peak power. Subject testing was separated by between 2 and 4 d. Experimental data collection was carried out with the approval of the local ethics committee. Written informed consent was obtained from all subjects.

All exertions of the lower limb were carried out with the subjects seated as previously described (3). Briefly, the tests consisted of the subject being seated in an upright position in the dynamometer with the knee flexed. The dynamometer has a very low backrest (10 cm) to minimize the possibility of utilizing the back extensor muscles in the exertion. A seat belt was attached across the hips securing the subjects into the seat, while performing a single maximal leg thrust against the pedal, attached to the wheel via a chain drive. The subjects were actively encouraged to give their maximum effort.

RESULTS

Typical values for torque, velocity, acceleration, work done, and power obtained from a single subject at low (0.024 kg·m^2) and high (0.543 kg·m^2) inertia are shown in Figure 2. With the larger inertia considerably greater torque was applied to the flywheel, which in this instance despite the slower angular velocity resulted in a greater peak and average power generation when compared with the low inertia.

The data obtained on seven young subjects is summarized in Figure 3. Maximum values for both average and peak power occurred over a range of inertias from 0.09 to 0.16 kg·m^2 (ranging from 318 to 448 W for average power and 608 to 965 W for peak power). The coefficient of variation for average power and peak power generated during the leg

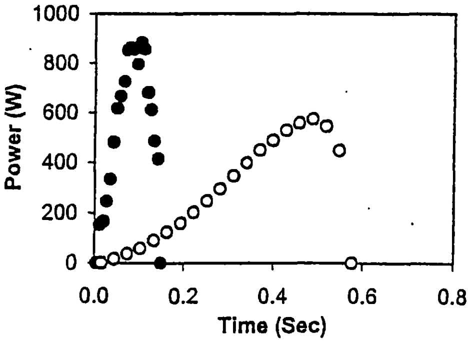

FIGURE 4—Power of an individual subject attained against a high (○) and a low (●) inertia after an isometric preloading and release after an isometric torque of 21 Nm had been attained.

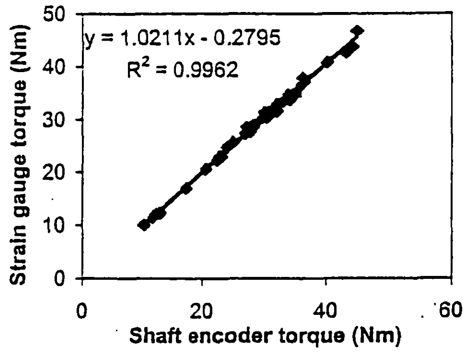

FIGURE 5—Relationship between the measured torque (strain gauge) and calculated torque (shaft encoder) for an arbitrary number of exertions made by a single subject across a range of inertias (0.024–0.69 kg·m²)

thrust ranged from 1.8 to 3.0% and 2.3 to 5.9%, respectively. A student paired two-tailed *t*-test revealed no significant difference ($P = 0.09$ and $P = 0.25$) between test 1 and test 2 for both average and peak power, respectively. Similar coefficients of variation were observed for the mean and peak torques.

Figure 4 shows an example of the effects of an isometric preload of (21 N·m) before dynamic movement at the high and low inertial loads shown in Figure 2. Peak power values of 883W and 574W for the low and high inertia, respectively, were only different for the low inertia when compared with the no preload contraction for this subject (Fig. 2E).

The use of this preloading exertion at the low inertia resulted in an increase in peak power of 95% when compared with the exertion without preloading. At high inertia, however, there was little difference between the two types of exertion.

The relationship between the measured torque (strain gauge) and calculated torque (shaft encoder) for an arbitrary number of exertions across a range of inertias (0.024–0.69 kg·m²) is shown in Figure 5. The r^2 value (0.9962) indicated a close agreement between the measured torque and that obtained through determination of the displacement of the flywheel.

DISCUSSION

One of the earliest recordings of dynamic human muscle function used an inertial loading system (6). Since then, others have utilized this principle to study human muscle power output (2,5,8); one such group (1) utilized a modified Hill flywheel driven through a clutch mechanism that allowed changes in the inertia presented to the muscle. To calculate the muscle output, a strain gauge system was attached in the cord, which wound around the flywheel and attached at the other end to the ankle. This was used in conjunction with an electrogoniometer, which was attached across the knee joint to monitor the movement. Inertial flywheels have also been designed to load muscle in non-gravity environments (4). This system (4) allowed the muscle to be loaded in both concentric and eccentric modes by initially imparting energy into the flywheel using a concentric muscle action and then by using the stored energy in the spinning flywheel to perform a resistive or eccentric muscle action. As with the apparatus designed by Aagaard et al. (1), muscle testing utilized a goniometer and strain gauge system for muscle function evaluation.

The apparatus developed by Bassey and Short (3) was designed to measure the average power that could be generated while accelerating a heavy flywheel. This was achieved by measuring the terminal velocity of the flywheel after a contraction had ended, through the use of four steel pins that passed through an optoswitch mounted in the wheel hub. Assuming that acceleration was constant to the point of terminal velocity, they calculated the time of contraction and used this to calculate average power. Developments in computing and data acquisition have allowed us to develop a system with high resolution, which allows us to measure contraction time directly, without making assumptions regarding the acceleration (see Fig. 2C). The present system allows for the accurate timing of muscle exertion by tracking the rotation of a flywheel, with a resolution of one degree. Another advantage of being able to track the wheel at discrete intervals is that it allows torque and velocity to be determined throughout the exertion period. We utilized a strain gauge system in parallel with the rotary encoder to compare the torque applied directly to the flywheel with that calculated from the flywheels rotational properties. When using the encoder system to calculate the torque applied to the flywheel, close agreement with the strain gauge torque values over the range of inertial loads was obtained (see Fig. 5), indicating that the strain gauge may be dispensed with in future dynamic studies.

Varying the inertia of the flywheel through the addition of steel plates allows the inertia at which a subject may express their maximum power, velocity, and force to be determined. In the present group of young male subjects, the inertias at which maximum peak and average power occurred ranged from 0.09 to 0.16 kg·m².

Isokinetic devices are limited in their ability to capture data at high speeds (>5.5 rad·s^{-1}). James et al. (7) stated that "it was not routinely possible to record forces above 2.1 rad·s^{-1} due to the small forces and large mechanical artifacts," whereas data collection is not impaired at high speeds of movement for the device presented. This fact allows for the detection of peak values of power, which occur at high movement speeds. At the lowest inertia, time of contraction was typically 200 ms or less (see Figs. 2 and 4), which is probably insufficient for the lower limb muscles to be fully activated. For this reason, a preload mechanism was incorporated (Fig. 4). The isometric preload mechanism was shown to improve muscle performance at high speeds, in part, as a result of the additional time for force to be generated.

The average power data presented were in general agreement with that presented by Bassey and Short (3). This system is most similar to the device presented here. Others (8.9.10) report values for peak power that are higher than those reported here. This could be explained in part by the fact that the devices were different by design. i.e.. cycling versus single leg thrusts. Also. the loading protocol was different. isokinetic versus inertial. Finally, the time of test was different: the push times for the device presented here could be under 200 ms. whereas the test time for the cycling was in the order of seconds. This could have a profound effect on the power output as shown in the isometric release example (Fig. 4).

In summary, the system described in this paper allowed a number of mechanical properties of human muscle to be inferred from the discrete measurement of the rotating properties of a flywheel through the use of a rotary encoder and high-resolution digital data acquisition. The approach has been tested against a strain gauge system and has been shown to allow reproducible measurements of short-term power to be made.

The technical assistance of Alan Snook, Bill Potter, and Chris Brad is gratefully acknowledged. S.D.R.H. is a Wellcome Trust Research Fellow.

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APPENDIX

All records of displacement. velocity, and acceleration were calculated from the rotary encoder. The rotary encoder had a resolution of 1° (0.01745 rads). The rotary encoder returned a time for every degree of wheel rotation. This allowed a record of wheel angle (W) against time (t) to be collected. For each test the time to complete the push was divided up into 20 equal periods. Each period was fitted by a second order polynomial $W = P + Q \cdot t + R \cdot t^2$. The velocity and acceleration were then calculated for the midpoint of the time period (tc), from the polynomial coefficients as follows:

$$\text{Velocity (V)} = Q + 2 \cdot R \cdot tc$$

$$\text{Acceleration(A)} = 2 \cdot R$$

The strain gauge system lag is accounted for by using a deconvolution method, which adds to the measured strain a product of its first time derivative and the time constant.

Power is calculated as a product of the velocity and torque throughout the push.

Work done is calculated as the time integral of power from the start of the push.

Average power for the test is calculated as total work done divided by the time of the push.

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Muscle function in elite master weightlifters

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ABSTRACT

PEARSON, S. J., A. YOUNG, A. MACALUSO, G. DEVITO, M. A. NIMMO, M. COBBOLD, and S. D. R. HARRIDGE. Muscle function in elite master weightlifters. *Med. Sci. Sports Exerc.*, Vol. 34, No. 7, pp. 1199–1206, 2002. **Purpose:** To determine whether explosive power and isometric strength of the lower-limb muscles in elite master Olympic weightlifters declines at a similar rate to nontrained healthy controls with increasing age. **Methods:** 54 elite level masters weightlifters (aged 40–87), who were competitors at the World Masters Weightlifting Championships (1999), were compared with a similar number of aged-matched, healthy untrained individuals. Isometric knee extensor strength and lower-limb explosive power were tested. Extent of antagonist co-contraction during isometric knee extension was determined by EMG and power loading characteristics by using a variable inertial system. Muscle volume was estimated using anthropometry. **Results:** On average, the weightlifters were able to generate 32% more peak power ($P < 0.05$) in the lower limbs and 32% more isometric knee extensor force ($P < 0.05$) than the control subjects. No significant differences in lower-leg volume were observed between the two groups. Peak power declined at a similar rate with increasing age in the weightlifters and controls (1.2 and 1.3% of a 45-yr-old's value per year), as did strength, but at a lower rate (0.6 and 0.5% per year). The inertial load at which the weightlifters achieved their maximal peak power output was greater ($P < 0.05$) than the controls. The torque generated at this optimal inertia was also greater in the weightlifters ($P < 0.05$), whereas the time taken for the weightlifters to reach their maximal peak power was on average 13% shorter ($P < 0.05$). No differences in antagonist co-contraction during isometric knee extension were observed between the two groups. **Conclusions:** Muscle power and isometric strength decline at a similar rate with increasing age in elite master weightlifters and healthy controls. In spite of inertial load optimization, muscle power declined in both groups at approximately twice the rate of isometric strength. Although similar rates of decline were observed, the absolute differences between the weightlifters and controls were such that an 85-yr-old weightlifter was as powerful as a 65-yr-old control subject. This would therefore represent an apparent age advantage of ~20 yr for the weightlifters. **Key Words:** MUSCLE, EXERCISE, WEIGHTLIFTING, AGEING, POWER, STRENGTH, SURFACE ELECTROMYOGRAPHY

The decline in maximal power generation with age has important implications for independent living in later life (2,13). It has been reported previously, using a fixed inertial loading device, that muscle power declines at twice the rate of isometric strength with increasing age (13). The mechanisms underlying such a phenomenon are unclear. One possible explanation might be that a lack of optimization of inertial loading results in slower and less powerful contractions in weaker, older subjects. Studying master weightlifters who have maintained high levels of physical training for many years may aid in our understanding of the potential limits to which older muscles may function. In this cross-sectional study, the strength and power of the lower-limb muscles was measured in 54 master weightlifters who were competitors at the World Masters Weightlifting Championships (Glasgow, U.K., 1999). Their data were compared with those obtained subsequently from healthy, but nontrained, age-matched subjects. The youngest competitor was 40 and the oldest 87 yr of age. The main aim of the present study was to determine whether elite master Olympic weightlifters show similar age-related declines in muscle strength and power as healthy nontrained individuals of a similar age.

APPROACH TO THE PROBLEM

In contrast to standard resistance training, the sport of Olympic weightlifting, which comprises two disciplines—the clean and jerk and the snatch, involves strength, power, and large motor-skill elements. The weightlifter is required to accelerate rapidly and brake large inertial loads and to coordinate the movement to complete the lifts correctly. For obvious technical and safety reasons, Olympic weightlifting does not lend itself as a tool for comparing objectively muscle function of master weightlifters with that of untrained older subjects. Therefore, in the present study we have compared muscle function between these master weightlifters and nontrained older individuals in terms of maximum isometric knee-extensor strength and in terms of lower-limb power. Lower-limb power was tested using a multi-joint testing system (Fig. 1), which was modified to allow optimization of inertial loading (12). This apparatus is designed for safely measuring explosive power in older people (1). The power values in this apparatus have been

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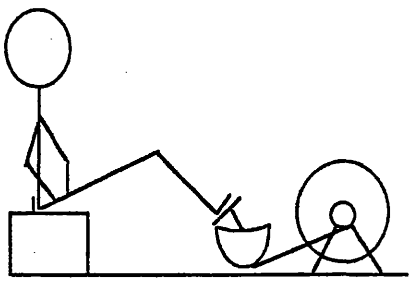


FIGURE 1—Diagram showing subject positioning for the inertial power test (1).

shown to relate closely to the ability to perform functional tasks in older people (2). This apparatus also had the advantage of being unfamiliar to both groups. It was hypothesized that, as the nature of Olympic weightlifting involves the rapid acceleration of large inertial loads and the generation of high power outputs, these weightlifters would exhibit superior dynamic muscle function characteristics even when tested in an unfamiliar piece of apparatus when compared with age matched controls. It was also hypothesized that in light of this ability to generate high power the rates of decline in muscle power output would be less than that of untrained subjects and also that by being stronger the weightlifters would require higher inertial loads to express their maximal power output than their untrained counterparts. In addition, knee extensor strength was measured using isometric dynamometry to establish whether the rate of decline differed to that of power and also to evaluate the degree of co-contraction during isometric knee extension.

METHODS

Subjects. A field laboratory was established close to the competition warm- up area at the World Masters Weightlifting Championships held in Glasgow, United Kingdom (1999). Fifty-four male competitors volunteered to take part in the study. Data obtained at this meeting were compared with data subsequently obtained on a similar number of untrained healthy men defined using the criteria of Greig et al. (5). The physical characteristics of the subjects are shown in Table 1. All subjects were informed of the testing procedures before testing and signed a consent form agreeing to take part in the study. The study had the approval of Strathclyde University and the Royal Free Hospital Ethics committees and was in agreement with the policy statement regarding human subjects of the American College of Sports Medicine.

Power testing. All exertions of the preferred lower limb were carried out with the subjects seated as previously described (12) shown in Figure 1. Test-retest data of peak muscle power in young subjects have shown a CV of 3.3% (12). Briefly, the tests consisted of the subject being seated in an upright position in the dynamometer with the knee flexed, a seat belt was attached across the hips securing them into the seat. The dynamometer, which has a very low backrest (10 cm) to minimize the possibility of utilizing the back extensor muscles in the exertion, comprised an inertial flywheel linked to a pedal assembly via a chain and freewheel mechanism. The exertion consisted of performing a single maximal leg thrust against the pedal. The subjects were actively encouraged to give their maximal effort. A total of 18 exertions were recorded, three trials at each of six inertial loads ranging in order from 0.024–0.543 $\mathrm{kg{\cdot}m^{-2}}$. The highest recorded value of peak power from the 18 contractions was taken as maximal peak power (MPP). Subsequently, the torque (TQ_{mpp}) and velocity (V_{mpp}) generated at MPP as well as the time taken to reach MPP were determined. The average power (AP_{mpp}) generated at this inertia was also calculated. Power measurements were made by measuring the torque and displacement of the inertial wheel. The torque applied to the wheel was determined via a strain gauge located in the chain and was amplified via a purpose-built strain-gauge amplifier. A rotary encoder, which was mounted end on to the shaft, detected displacement of the inertial wheel. Both analog and digital signals were then sampled by an A/D converter (CED micro1401, Cambridge, U.K.). Analysis of the power data was carried out with a mathematical software package (Mathcad Ver. 7 Mathsoft, Cambridge, MA); this procedure has been described in detail elsewhere (12). Data were then fitted using second-order polynomial techniques to allow velocity data to be calculated. Power was then calculated as the product of torque and velocity.

Isometric knee extensor strength. The isometric strength tests were performed on the same limb as that tested on the power dynamometer. The subject was seated upright with the knee flexed at 90° to the horizontal. The ankle was fixed in a restraining collar, which was attached to a nonextensible rod attached to a strain-gauge assembly. The force transducer signal was amplified as above and sampled by an A/D system (CED 1401 Cambridge, U.K.) at 2 kHz. Each subject carried out a total of three isometric knee extension efforts and three isometric knee flexion efforts. In all tests, the subjects were actively encouraged to give their maximal effort, and a 1-min rest period was given between contractions. In all cases, the test order was first power testing and second strength testing.

sEMG. Simultaneous recording of the sEMG activity from the vastus lateralis and the long head of the biceps femoris muscles was made during the measure of isometric strength. The assumption was made that these two muscles were representative of their constituent groups (3). Before mounting the recording electrodes, the skin surface was prepared by light abrasion and cleaning with alcohol swabs. Two silver/silver chloride bipolar electrodes (Medicotest, type N-10A, St. Ives, U.K.), with a 20-mm interelectrode distance (center to center) were placed midline on the muscle belly halfway between the center of the belly and the distal myotendinous junction of the prepared site of the muscles. A ground electrode (Dantec Electronics, mod. 13S97, Bristol, U.K.) was placed around the ankle of the contralateral limb. The sEMG was band pass filtered between 3 and 1000 Hz (Neurolog Filter NL 125, Digitimer, Welwyn Garden City, U.K.), preamplified (×1000) (Neu-

TABLE 1. Physical characteristics of the subjects; all table values shown as mean ± SE; significant difference from control are shown (*).

	Age Group (yr)				
	40–49 ($N = 10$)	50–59 ($N = 11$)	60–69 ($N = 16$)	70–79 ($N = 13$)	80–89 ($N = 4$)
Age (yr)					
Control	45.4 (±0.9)	53.1 (±0.7)	64.2 (±0.8)	74.5 (±0.7)	84.3 (±1.4)
Weightlifter	42.7 (±0.6)	53.9 (±0.8)	64.1 (±0.7)	74.3 (±0.8)	84.2 (±1.5)
Body mass (kg)					
Control	75.7 (±2.4)	76.6 (±2.3)	77.0 (±2.3)	72.7 (±2.2)	68.6 (±7.5)
Weightlifter	87.0 (±7.8)	83.0 (±4.6)	78.0 (±2.8)	77.6 (±3.5)	72.0 (±6.5)
Height (cm)					
Control	177.1 (±2.3)	177.8 (±2.4)	174.8 (±1.5)	171.9 (±1.5)	167.7 (±3.5)
Weightlifter	173.6 (±2.7)	167.8* (±1.6)	168.3 (±1.9)	167.2 (±1.7)	164.3 (±2.5)
Total lower-limb volume (cm^3)					
Control	8734.8 (±276.3)	8903 (±366.9)	8676.2 (±241.5)	7706.3 (±297.7)	7911.2 (±507.9)
Weightlifter	8680.2 (±831)	7846.5 (±312.8)	7626.3 (±365.3)	7318.6 (±345.8)	6816.1 (±524.1)
Lean lower-limb volume (cm^3)					
Control	6159.8 (±287.5)	6341.1 (±298.0)	6103.5 (±200.5)	5370.8 (±251.2)	5359.0 (±322.6)
Weightlifter	7276.6 (±830.7)	6490.0 (±350.7)	6077.9 (±300.7)	5477.1 (±305.1)	4854.6 (±508.1)

rolog remote AC preamplifier NL 824) amplified (×2) (Neurolog Isolation amplifier NL 820), and A-D converted (type 1401, CED, Cambridge, U.K.) at a sampling rate of 2048 Hz. To quantify the sEMG amplitude (RMS) and frequency (MDF) components, computer-aided analysis was performed over a 1-s window of the plateau phase during the isometric effort. The spectral parameters were evaluated by standard fast Fourier transforms over 2048 samples. This procedure has been described in detail elsewhere (10). MDF was adopted as a single measure to represent the power spectrum, because of its lower sensitivity to noise with respect to the mean frequency (MNF) (14). Percentage co-contraction during isometric knee extension was defined as the RMS sEMG of the biceps femoris during maximal voluntary isometric extension, divided by the RMS sEMG activity of the biceps femoris during maximal voluntary isometric flexion.

Anthropometry. Lower-limb volume was estimated from anthropometric measurements comprising segmental circumferences and lengths as described by Jones and Pearson (8). Skin-fold measurements were made at four sites: anterior, posterior mid-thigh and lateral, medial mid-calf by using skin callipers (John Bull Ltd. West Sussex, U.K.). Skin-fold corrections were made using the following regression equations (personal communication, Professor P. R. M. Pearson), anterior thigh ($y = 1.0142 + 0.557 \times$ skin-fold value), posterior thigh ($y = 1.368 + 0.532 \times$ skin-fold value), medial calf ($y = 0.985 + 0.499 \times$ skin-fold value) and lateral calf ($0.8701 + 0.3926 \times$ skin-fold value). Where y represents the corrected skin-fold value used in the calculations.

Data analysis. All data analysis was carried out using SPSS version 10. Data are presented as means ± SE. For clarity, the subjects were assigned to decades (see Tables 1 and 2). Multiple Student's t-tests were applied to the data presented in Tables 1 and 2 and corrected for multiple comparisons by using the Bonferroni method. Statistical comparisons between the weightlifters and untrained subjects with respect to age for the variables measured were made using a general linear model and univariate analysis (ANOVA). For descriptive purposes linear regression analysis was also performed on each group to determine the rate of change with age. Interpolated values at 45 yr of age and 85 yr of age were calculated using these regression equations. For between-group comparisons, for the inertial load at which peak power occurred, a Student's t-test was performed. Levels of significance for all tests were set at $P < 0.05$.

RESULTS

The physical characteristics of the subjects are shown in Table 1. No significant differences were observed in body mass, height (apart from the 50–59 age group), age, and total and lean lower-limb volume between the two groups. However, there is a tendency for the untrained subjects to have higher total lower-limb volumes at all age groups and conversely for the weightlifters to have higher lean lower-limb volumes (muscle and bone) at most age groups. Figure 2 shows the lift performance data from the 1999 World Championships for the competitors tested. Of the 54 competitors tested, 15 were winners of their respective age and

TABLE 2. Mean values for isometric knee extensor strength, MPP and AP_{mpp}; significant differences from controls are shown (*).

	Age Group (yr)				
	40–49 ($N = 10$)	50–59 ($N = 9,11$)	60–69 ($N = 16$)	70–79 ($N = 13$)	80–89 ($N = 4$)
MPP (W)					
Control	653.1 (±44.1)	561.4 (±38.7)	549.4 (±39.2)	442.1 (±37.7)	242.6 (±22.9)
Weightlifter	1028.4 (±75.4)*	879.9 (±79.4)*	811.7 (±45.1)*	600.9 (±38.5)*	456.0 (±89.5)
AP_{mpp} (W)					
Control	299.5 (±20.2)	248.7 (±16.3)	246.7 (±20.3)	204.2 (±15.8)	88.1 (±19.7)
Weightlifter	438.6 (±38.2)*	404.0 (±41.3)*	384.4 (±23.7)*	282.7 (±21.3)*	214.5 (±38.3)
Isometric strength (N)					
Control	313.1 (±42.1)	481.3 (±53.5)	422.7 (±31.9)	321.8 (±48.2)	238.4 (±55.6)
Weightlifter	617.3 (±51.9)*	565.4 (±45.2)	601.3 (±34.8)*	438.4 (±26.2)	519.6 (±57.2)

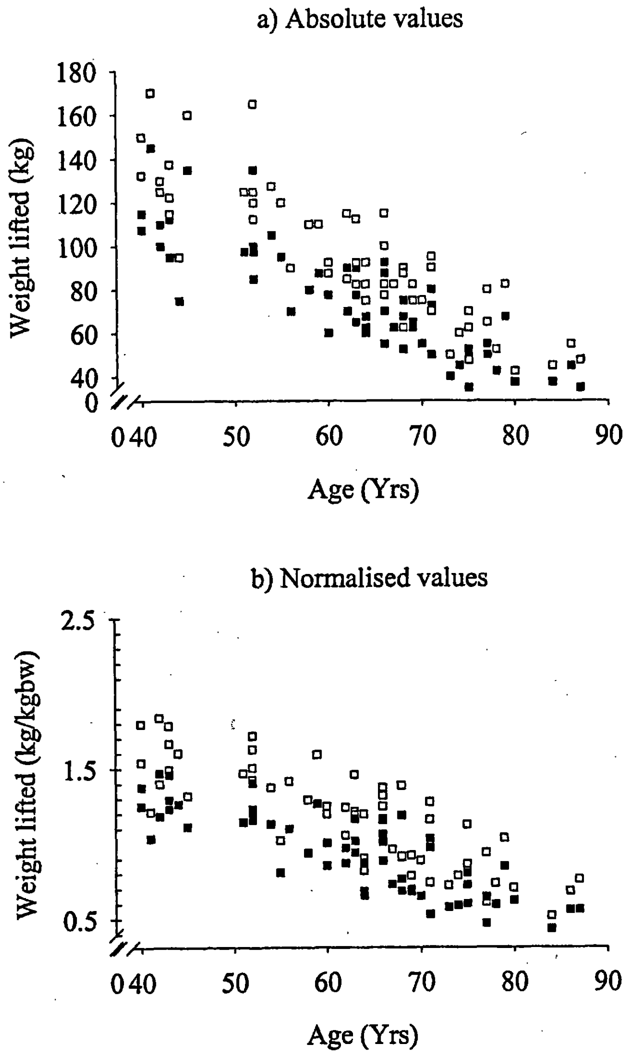


FIGURE 2—Performance data for all weightlifters tested (Glasgow World Masters Weightlifting Championships 1999). Clean and jerk (□), snatch (■). Regression of clean and jerk with age, $y = -2.1\cdot(\text{age}) + 223.86$ ($R^2 = 0.45$), and for the snatch, $y = -1.7\cdot(\text{age}) + 181.34$ ($R^2 = 0.47$). Regression of clean and jerk normalized to body weight with age, $y = -2.2\cdot10^{-2}(\text{age}) + 2.6$ ($R^2 = 0.64$) and for the normalized snatch, $y = -1.8\cdot10^{-2}(\text{age}) + 2.1$ ($R^2 = 0.67$).

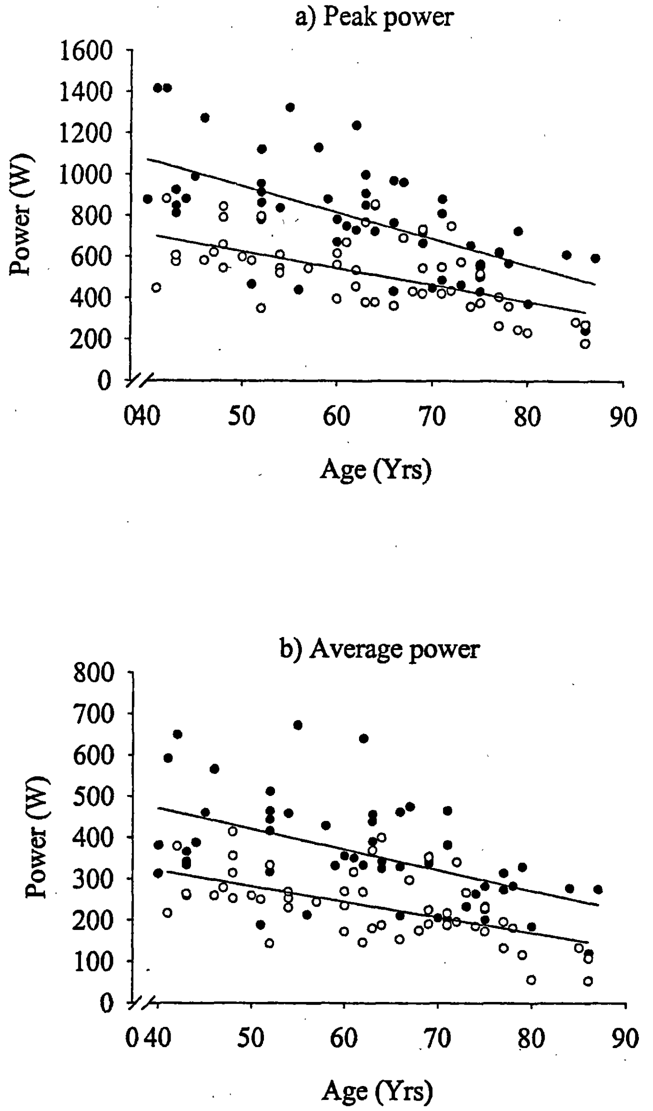


FIGURE 3—Absolute values for a) MPP and b) AP_{mpp}. Control (○), weightlifters (●). Values for MPP regression lines $y = -8.1\cdot\text{age} + 1029.2$ and $y = -12.8\cdot\text{age} + 1580.7$ for the controls and weightlifters, respectively. Values for the regression lines for AP_{mpp} $y = -3.8\cdot\text{age} + 471$ and $y = -4.9\cdot\text{age} + 669.1$ for the controls and weightlifters, respectively.

weight categories. A further 11 were silver medallists, and 16 others were bronze medal winners.

The values of MPP and AP_{mpp} can be seen in Figure 3 and are summarized by age group in Table 2. Both MPP and AP_{mpp} declined as a function of age in both groups, and there was no significant difference between the groups in rate of decline of MPP ($R^2 = 0.54$, $P < 0.05$) or AP_{mpp} ($R^2 = 0.48$, $P < 0.05$) (see Table 3). On average, the weightlifters generated 35% more AP_{mpp} ($P < 0.05$) and 32% more MPP ($P < 0.05$) than the control subjects. Normalization of power values with respect to body mass or lean lower-limb volume made no significant difference to age-related or group-related differences. The difference in decline of MPP between the two groups was 4.5 $W\cdot yr^{-1}$, and the 95% CI for the difference was −0.67–10.04 $W\cdot yr^{-1}$. Therefore, it is unlikely that the weightlifters would have declined more than 10 $W\cdot yr^{-1}$ greater than the controls. A statistically calculated power of 0.8 was shown to be sufficient to show a difference in rate of decline of 8.11 $W\cdot yr^{-1}$.

Figure 4 illustrates the number of subjects generating MPP at each of the inertial loads tested. Overall, the weightlifters required a higher inertia to express their MPP than the control subjects ($P < 0.05$). The torque (TQ_{mpp}) and velocity (V_{mpp}) generated at MPP are shown in Figures 5a and b. Figure 4c shows the time taken to reach MPP (T_{mpp}). TQ_{mpp} declined with increasing age in both groups ($R^2 = 0.49$, $P < 0.05$), but the rates of decline were not significantly different between the two groups. On average, the levels of TQ_{mpp} generated by the weightlifters were 34% greater ($P < 0.05$) than those of their untrained counterparts. V_{mpp} declined significantly with age for both groups, and on average there was no significant difference between the two groups. T_{mpp} increased with age in both the weightlifters

TABLE 3. The percentage difference between the mean values of the weightlifters with respect to the control subjects is shown in column I; the age-related decline for both groups, in absolute units per year is shown in columns II and III; the age-related decline for both groups in relative units per year (expressed as a percentage of a 45-yr-olds value is shown in columns IV–V; the percentage value of an 85-yr-old relative to that of a 45-yr-old is shown in columns VI–VII.

	I) Average % Difference between Groups	II) Weightlifter Absolute Rate Change	III) Control Absolute Rate Change	IV) Weightlifter Relative Rate Change	V) Control Relative Rate Change	VI) Weightlifter 85 yr-old as a % of a 45-yr-old	VII) Control 85 yr-old as a % of a 45-yr-old
Normalised clean & jerk ($kg \cdot kgbw^{-1}$)	N/A	$2.2 \cdot 10^{-2}$	N/A	1.4	N/A	44	N/A
Normalised snatch ($kg \cdot kgbw^{-1}$)	N/A	$1.8 \cdot 10^{-2}$	N/A	1.4	N/A	44	N/A
MPP (W)	32*	12.6†	8.1†	1.3	1.2	49	51
AP_{mpp} (W)	35*	4.9†	3.8†	1.1	1.5	55	50
TQ_{mpp} (Nm)	34*	0.29†	0.2†	0.9	1.0	60	59
V_{mpp} ($rads \cdot s^{-1}$)		0.13†	0.14†	0.4	0.4	85	87
T_{mpp} (s)	−13*	$1.3 \cdot 10^{-3}$†	$2.3 \cdot 10^{-3}$†	0.5	0.8	80	70
Isometric strength (N)	32*	3.4†	1.9†	0.6	0.5	78	81
RMS (mV)	53*	7.7†	0.51†	0.9	0.1	64	94
MDF (Hz)	12*	$2.6 \cdot 10^{-2}$	0.15	$3.9 \cdot 10^{-5}$	0.2	98.3	90

* Weightlifters significantly different from controls ($P < 0.05$).
† Significant decline with age ($P < 0.05$).

and controls ($P < 0.05$), and on average the values of T_{mpp} were significantly lower for the weightlifters ($P < 0.05$).

The isometric extensor strength data are presented in Figure 6 and Table 2. Isometric strength declined with age in both the weightlifters and untrained controls ($R^2 = 0.29$, $P < 0.05$), but again there were no significant differences in the rates of decline of strength between the two groups (Table 3). On average, the weightlifters had significantly ($P < 0.05$) higher average values (32%) for isometric extensor strength.

The MDF of the sEMG is shown in Figure 7a. There was no effect of age on MDF in either group. However, weightlifters exhibited higher average values, 66.4 Hz ± 1.52 as against 58.2 Hz ± 1.55 for the control group ($P < 0.05$). Figure 7b shows the RMS values for the sEMG where there was a significant decline as a function of age ($R^2 = 0.40$, $P < 0.05$). On average, the weightlifters produced significantly higher values of RMS ($P < 0.05$). The activation of the biceps femoris muscles during isometric leg extension (i.e., co-contraction) expressed as a percentage of the maximum biceps femoris activity is shown in Figure 7c. No significant effects of age, rates of decline, or differences between the two groups were observed.

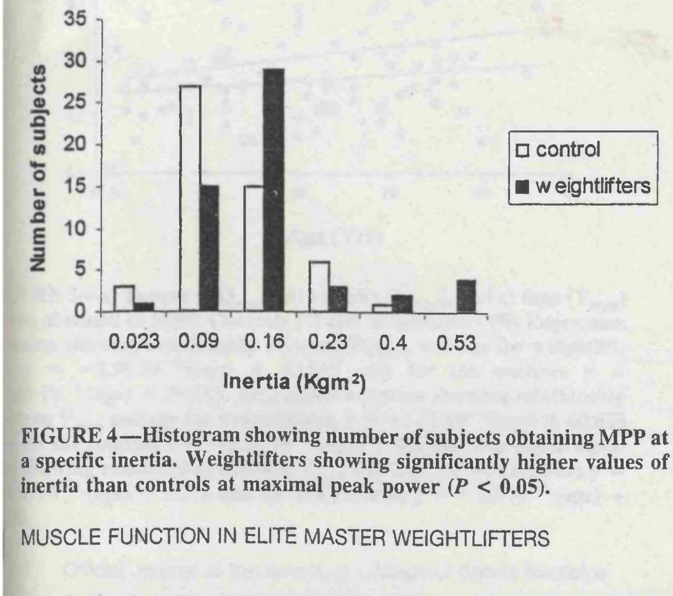


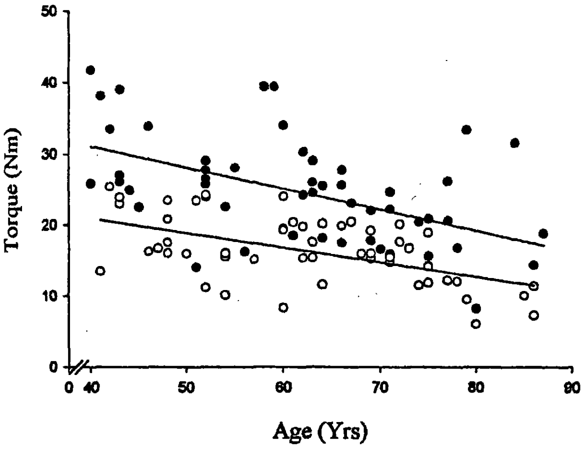
FIGURE 4—Histogram showing number of subjects obtaining MPP at a specific inertia. Weightlifters showing significantly higher values of inertia than controls at maximal peak power ($P < 0.05$).

DISCUSSION

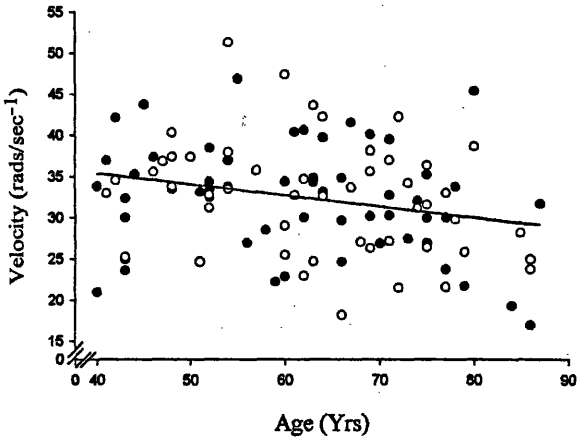
This study has compared explosive muscle power and isometric strength in the lower-limb muscles of elite master weightlifters with men of a similar age who were healthy but did not undertake any regular physical exercise. The main findings were that i) both strength and power declined with increasing age in both the weightlifters and the untrained subjects; ii) the relative rate of decline was similar for the two groups, with power declining at twice the rate of isometric strength, despite inertial load optimization; and iii) the weightlifters were significantly stronger and more powerful, requiring a higher level of inertia to generate their MPP. This was despite the fact that there was no detectable difference in lean lower-limb volume between the groups as estimated anthropometrically (Table 1).

The decline in the amount of weight that can be lifted in competitive performance in masters weightlifting has been previously documented (11). This decline is confirmed in the present study. Two of the competitors tested in the present study were winners in the 85-kg category and represented the extreme of age categories. The winner in the 80+ category, who was also the oldest competitor, lifted 55 kg in the clean and jerk. This amounted to 36% of the winning lift in the 40- to 44-yr-old age group (150 kg). From a functional perspective, it is worthy of note given that many older people of a similar age, i.e., over 80 yr of age struggle to rise from a chair let alone be able lift 55 kg above their heads.

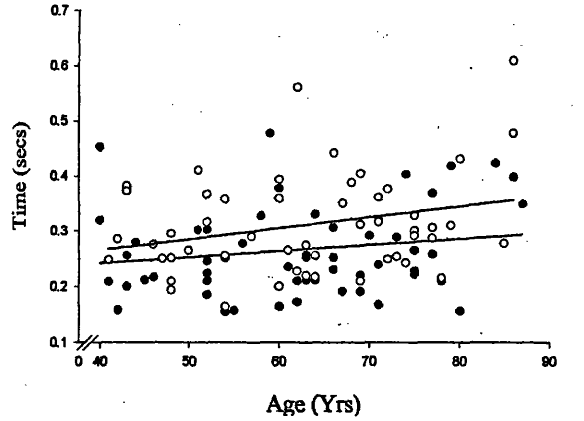
In agreement with the decline in competition performance with increasing age is the observed decline in the performance of the tests of muscle function. In terms of peak muscle power generation, the data suggest a loss equivalent to 1.3% per annum for the weightlifters and 1.2% per annum for the controls. Skelton et al. (13) have previously reported a progressive decline in average power in healthy older people ranging in age from 65 to 85 yr of age. This was tested using a single inertial load. In another study of master athletes, Grassi et al. (4) measured power output by using double leg jumps from a force platform. They reported in



b) Velocity at MPP



c) Time to reach MPP



IGURE 5—a) Torque (TQ_{mpp}), b) velocity (V_{mpp}), and c) time (T_{mpp}) alues obtained at MPP. Controls (○) and weightlifters (●) Regression quation showing relationship between TQ_{mpp} and age for weightlift-rs $y = -2.96 \cdot 10^{-1} \cdot (\text{age}) + 42.955$ and for the controls $y = \cdot 2.05 \cdot 10^{-1} \cdot (\text{age}) + 29.153$. Regression equation showing relationship tween V_{mpp} and age for weightlifters, $y = -1.31 \cdot 10^{-1} \cdot (\text{age}) + 40.623$ d for the controls $y = -1.35 \cdot 10^{-1} \cdot (\text{age}) + 40.358$. Regression equa-on showing relationship between T_{mpp} and age for weightlifters $y = \cdot 1.33 \cdot 10^{-3} \cdot (\text{age}) + 0.221$ and for the controls $y = -2.3 \cdot 10^{-3} \cdot (\text{age}) +$ 198.

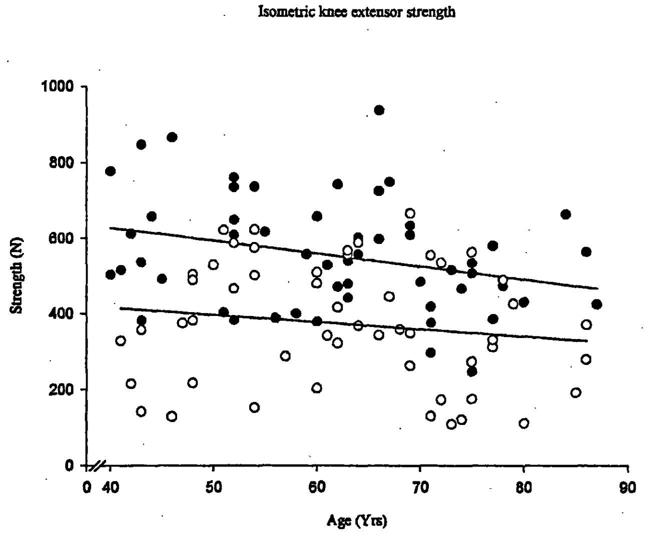
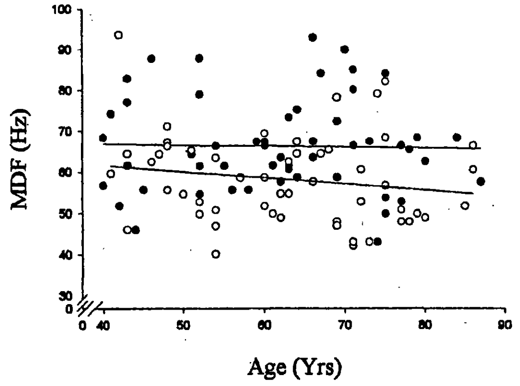


FIGURE 6—Isometric knee extensor strength. Controls (○), weightlifters (●). Regression equation for relationship between weightlifter absolute isometric strength and age, $y = -3.417 \cdot (\text{age}) + 763.04$ and for controls, $y = -1.89 \cdot (\text{age}) + 491.33$.

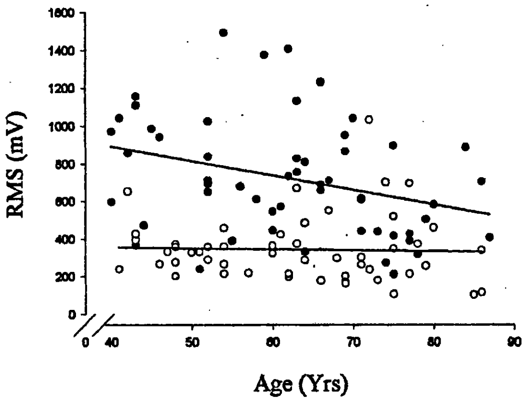
power athletes (sprinters and jumpers), ranging from a mean age of 17.7 yr to 74.3 yr, that peak power output of the lower limbs in the older subjects was approximately 50% of that seen in the younger subjects. They also reported that the maximum power output of the power athletes over the age of 70 was 58% of that generated by the subjects in the 40–49 age group. In general agreement, the results of the present study showed that the 70-yr-old weightlifters generated 62% of the power generated by the youngest weightlifter group. An important finding in the present study was that the decline with age in both average and peak power was similar for the weightlifters and the control subjects (Table 3). However, the weightlifters generated significantly greater absolute levels of both MPP and AP_{mpp} such that the power generated by an 85-yr-old competitor was equivalent to that of a 65-yr-old nontrained subject, representing an apparent gain of some 20 yr. This was also the case when the data were normalized to body mass or lower-limb volume.

A further finding of the present study was that the inertial load at which MPP occurred was higher in the weightlifters than the untrained group. It is likely that the weightlifters, because they are stronger, are better able to accelerate these higher inertial loads and therefore reach an optimal position on their power velocity relationship during the exertion. Weaker individuals, however, must use a greater proportion of their maximal strength to achieve a similar acceleration at a given inertial load. Due to the force-velocity relation, such a contraction must be performed more slowly and thus possibly on a less favorable portion of the power-velocity relationship. Therefore, a lower inertia is more favorable for the weaker subjects, for when MPP was broken down into its torque and velocity components, the velocity component was found to be not significantly different between the two groups (Fig. 5b). However, the torque generated by the weightlifters at their optimal inertia was significantly

a) MDF of the vastus lateralis during isometric knee extension



b) RMS of the vastus lateralis during isometric knee extension



c) Percentage cocontraction during isometric knee extension

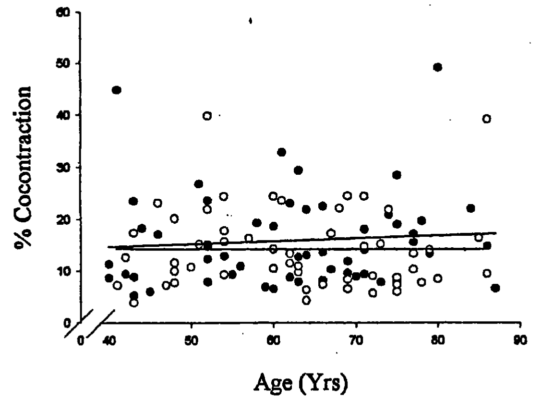


FIGURE 7—Plots of a) the mean density frequency (MDF) of the power spectrum of the vastus lateralis $y = -2.3 \cdot 10^{-2} \cdot (\text{age}) + 67.98$ and $y = -1.5 \cdot 10^{-1} \cdot (\text{age}) + 67.6$ represent the regression relationships with respect to age for the weightlifters and controls, respectively; b) the root mean squared (RMS) of the vastus lateralis during isometric knee extension; c) the percentage of biceps femoris involvement during isometric knee extension. Controls (○), weightlifters (●).

greater (Fig. 5a) than the torque generated by the control subjects at their lower optimal inertia. In addition, the weightlifters required significantly less time than the untrained group to reach MPP (Fig. 5c). Thus, not only are the master weightlifters achieving higher levels of MPP through higher torque generation, they are also generating torque more quickly.

No detailed anatomical measures were possible in the field setting described, nor was it possible to obtain muscle biopsy samples. However, earlier data on strength-trained older individuals might suggest a number of mechanisms explaining their superior muscle function. These include a greater lean-leg volume (6), a relatively greater size of their Type II fibers (9), and that strength-trained individuals have been shown to have a larger Type II fiber area (15), although a training adaptation toward faster contracting fibers expressing the same MHC isoform cannot be excluded (16).

In terms of isometric knee extensor strength, the weightlifters were on average 32% stronger than the controls with the decline in strength occurring at 0.6% per annum; this was approximately half the rate observed for muscle power. Skelton et al. (13) reported that average power declined at 3.5% per annum between 65 and 85 yr (of a 77-yr-old's value) but that isometric strength declined at a lower rate (~1.5% per annum). In that study, power was determined using a single inertial load. As already alluded to, it is possible that this puts the weaker older people at a disadvantage as they are unable to sufficiently accelerate a heavy flywheel to obtain a velocity to allow them to generate their optimal power. In the present study, we have optimized the inertial load for power generation, yet the rate of decline in power was still greater than that of isometric strength, suggesting that in addition to declines in strength, alterations in shortening speed may play also a role. In this regard, although the decline in TQ_{mpp} was more dramatic (Fig. 5), there was also an age related slowing of the V_{mpp}. Recent mechanical studies on human chemically skinned single fibers (9) and using the *in vitro* motility assay (7) suggest that their may be intrinsic changes with age that may slow cross-bridge cycling.

The results of the sEMG support the notion that the weightlifters can activate their muscles during an isometric contraction with a more synchronous firing pattern as indicated by the RMS data. In fact, the RMS, which can be used as a measure of general muscle activation, was significantly higher in the weightlifters in all age groups. It has been suggested by Stulen and Deluca (14) that the MDF of the power spectrum for the sEMG is related to muscle fiber composition. The higher values of MDF for the weightlifters from the power spectrum analysis (Fig. 7a), might suggest a higher proportion of Type II fibers in the weightlifters.

It was hypothesized that weightlifters may produce less antagonist co-contraction than the controls during isometric knee extension. However, similar levels of co-contraction are seen between the two groups (Figure 7c). This may, in part, be due to the fact that a number of weightlifters described having some knee discomfort during the test and it was possible that the weightlifters utilized the antagonist muscles to help protect the knee during these isometric contractions.

In conclusion, the results of the present cross-sectional study have shown that the apparent decline in lower-limb muscle function occurs even in highly trained weightlifting athletes and that the relative rate of decline is similar to that which occurs as a result of normal aging. For each individual, the optimization of inertial load for power testing was shown to be important as the weightlifters developed their maximal power at a higher inertial load than the untrained control subjects. In functional terms, however, it is the ability to generate absolute levels of force and power that determine the ability of older people to perform physical tasks. When looked at in absolute terms, the data in the present study show that weightlifters are considerably stronger and more powerful, with the oldest weightlifters having muscle power characteristics of individuals some 20 yr younger than themselves. These results therefore suggest that high-resistance strength training for older people might not slow the rate of decline in muscle function with increasing age but may result in a shift upward to a new slope that starts at a higher absolute level but that has a similar relative rate of decline. The data presented here suggest that activities which require repetitive high levels of muscle power to be generated as in weightlifting may help improve or maintain muscle function in later life

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MUSCLE POWER LOSS IN OLD AGE: FUNCTIONAL RELEVANCE AND EFFECTS OF TRAINING

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ABSTRACT. Old age is characterised by a number of physiological changes and probably none is more obvious than the decreased ability to perform physical tasks. This relates in a large part to the loss of skeletal muscle power. Power is the product of force of contraction and speed of movement. Thus a reduction in either of these will cause power to be reduced. In reality it is the reduction in muscle force production, which in turn is most closely associated with a loss of muscle mass (sarcopenia), which is fundamental to power loss. Power is reduced by the same amount as force, but when overcoming loads which require a high percentage of the maximum force generating ability an even greater decline may occur due to the slower contraction needed to perform the task. A reduction in the amount of fast myosin heavy chain isoforms and an intrinsic slowing in the unloaded speed of shortening may further contribute to power loss. Strength or progressive resistance training, is effective in improving muscle strength and by implication power, even in very frail very elderly people.

RIASSUNTO. *L'età avanzata è caratterizzata da un certo numero di cambiamenti fisiologici, il più ovvio dei quali è, probabilmente, la diminuita abilità di eseguire compiti fisici. Questo è in larga misura riconducibile alla perdita di potenza del muscolo scheletrico. La potenza è il prodotto della forza di contrazione e della velocità di movimento. Perciò, una riduzione in una di queste due variabili causerà una riduzione della potenza. In realtà, è la riduzione della produzione di forza muscolare, strettamente associata alla perdita di massa muscolare (sarcopenia), che è cruciale per la perdita di potenza. La potenza risulta ridotta nella stessa misura della forza, ma questa riduzione risulta ancora maggio-*

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re quando si affrontano carichi che richiedono una percentuale più alta della massima capacità di generare forza, in ragione del fatto che è richiesta una contrazione più lenta per eseguire il compito. Anche una riduzione nella quantità di isoforme di catene pesanti di miosina ed un intrinseco rallentamento nella velocità di accorciamento senza carico contribuiscono alla perdita di potenza. Un allenamento progressivo alla forza o alla resistenza è efficace nel migliorare la forza muscolare e la potenza anche in soggetti molto anziani.

The world is undergoing considerable demographic changes; birth rates are falling and people are living longer. For instance, in the UK, it is predicted that the number of men and women living over the age of 85 will have increased from 0.8 million in 1987 (1.4% of the population) to 1.4 million in 2025 (2.3% of the predicted population)(1). Ageing is accompanied by numerous physiological changes (2) which impair the ability of the older population to live independently. This has negative consequences both for elderly people, in terms of their overall quality of life, and for the financing of health and social services charged with the care of those no longer able to look after themselves.

This chapter will focus on the age related changes in one of these physiological systems, skeletal muscle. Specifically, on how ageing results in its decreased ability to generate power, the practical consequences of this power loss, in terms of the performance of every day physical tasks, and how these age related changes in muscle function might be improved through exercise training.

WHAT IS POWER?

Muscle *strength* can be defined as the amount of force that a muscle can generate, whilst *power* is the rate of performing mechanical work, or calculated as the product of force of muscular contraction and the speed at which the contraction occurs:

$$\text{Power (Watts)} = \text{Force (N)} \times \text{Velocity (ms}^{-1}\text{)}$$

Power is therefore generated in any body action that involves movement. In this chapter we will consider power to mean 'explosive power', or that generated in a single or just a few maximal contractions where muscle metabolism does not limit performance. This is different from 'sustained power' (3) which occurs during activities of a longer duration such as endurance running or swimming and which describes the ability to maintain a submaximal level of power output over a period of time.

Power can be derived from the force-velocity relationship of muscle and it is clear from Figure 1 that power is determined by three things i) the maximum isometric force (P_0) ii) the maximum velocity of shortening (V_{max}) and iii) the curvature of the force velocity relationship (a/P_0 - as described by Hill 1938(4)). So anything that affects the maximum force or the shortening properties of the muscle will by definition affect power output.

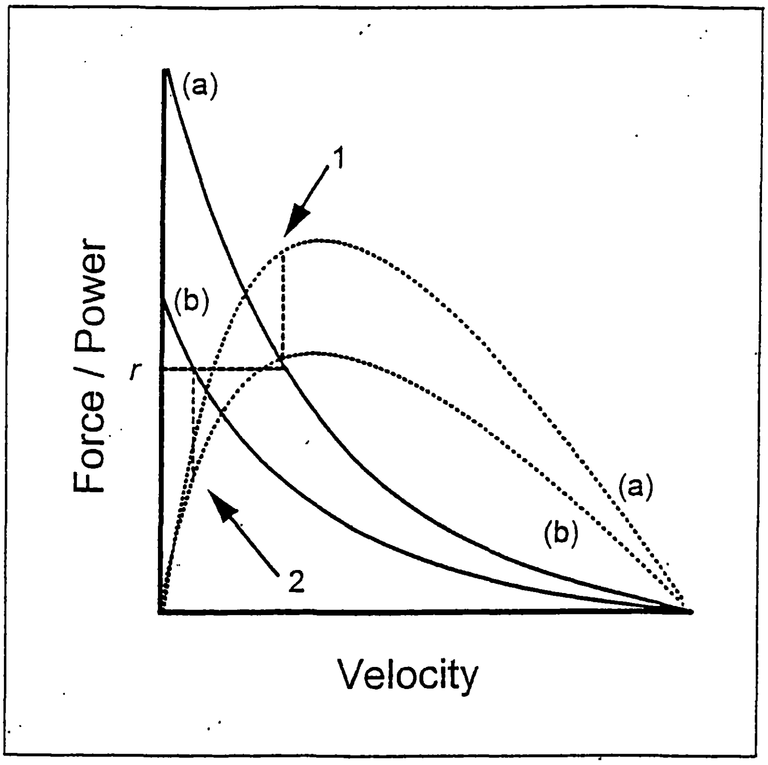

FIGURE 1. Schematic representation of the force-velocity relationship of skeletal muscle and its derivative, power (dotted lines). This hyperbolic relationship is described by the Hill (1938) equation:

$$(P + a)\ (V + b) = (P_o + a)b$$

where P = force obtained at velocity V, P_o = isometric force and a and b are constants obtained from thermal or mechanical measurements.

HOW IS POWER MEASURED?

In exercise physiology laboratories explosive power in young people has been traditionally measured using a number of simple whole body movements (see 5) such as vertical jumping (e.g. Sargeant jump, or on a force plate), sprinting up a short flight of stairs (e.g. Margaria stair test) or sprinting on a cycle ergometer (e.g. Wingate test). In general these tests are not readily applicable to older people.

Isokinetic (constant rotational velocity) dynamometers allow power to be determined in single muscle groups under safe and standardised conditions, from the torque measured whilst the limb rotates at a constant angular velocity:

Power (Watts) = Angular Velocity (rad s^{-1}) x Torque (Nm)

Although useful testing devices, isokinetic contractions do not reflect how muscles work in real life where muscles have to contract, not at a constant angular velocity, but at velocities which vary depending on the fixed resistances that they must overcome. Isokinetic dynamometers are also often prohibitively expensive. With these factors in mind Bassey and Short (6) developed a piece of apparatus which measures the average power generated by the lower limb muscles during a single leg extensor thrust against a pedal which accelerates a heavy flywheel of a known inertia. From the terminal speed of flywheel rotation measured directly after the push (representing kinetic energy) and the time of the push (calculated assuming a constant level of acceleration during the push) they derived average power for the leg extensor movement:

$$\text{Power (Watts)} = I\omega^2 / 2T$$

Where I = inertia, ω = angular velocity of flywheel rotation (i.e. kinetic energy) and T = time.

Cross-sectional data (7) collected on elderly men and women using this apparatus, suggest that between the ages of 65 and 95 years, lower limb power declines at a rate of 3-4% pa (of a 77-year-olds value).

WHAT CAUSES THE LOSS OF MUSCLE POWER WITH INCREASING AGE?

Anything that effects force production or the shortening characteristics of a muscle will affect its power output.

1. Loss of force

Muscle power production will be reduced by at least an amount that is equal to any reduction in force. The force that a muscle can generate, whether a single fibre or an intact whole muscle, is ultimately dependent on its cross-sectional area (all other things being equal). At the molecular level this reflects the number of cross-bridges working in parallel. It is clear that as we get older muscle size is reduced and this loss of muscle, or 'sarcopenia', has the expected impact on the generation of force.

It has been apparent for many years (e.g. since Quetelet 1815 (8)) that old age results in a series of structural and functional changes in muscle. In the last 30 years however, a wealth of information has been generated concerning the loss of muscle strength in older people. It is not the purpose of this chapter to provide an extensive review of this topic as there are several recent reviews (9, 10, 11, 12). However, as an indication, cross-sectional data indicate that the postural muscles of elderly people aged around 70 years have approximately 60% of the force generating ability of young adults aged 20-30 years (e.g. 13, 14). Between the ages of 65 and 90 muscle strength appears to be 'lost' at a rate of 1-2 %per annum a (of a 77 -year olds value, (7)). The same being apparently true for both men and women. Note that this figure is less than the 3-4% p.a. reduction in explosive power recorded in the same subjects (see later discussion).

It is clear that force and power will be decreased by an amount, at least equivalent to the loss of muscle mass. But like force, power per unit muscle volume will be further underestimated if the amount of actual contractile tissue is overestimated and further still if, even after accurate measurement of muscle cross-sectional area, specific force is still reduced (see 12 for review). One of the likely reasons for this phenomenon is the overestimation of CSA in elderly people as the amount of 'muscle' occupied by non-contractile components i.e. occupied by fat and connective tissue is increased in elderly people (15).

A second explanation for a reduction in force per unit area could be a decline in the ability to activate fully the total muscle mass during a voluntary effort. In the calf muscle a comparison of plantar flexor forces obtained with voluntary and maximally electrically evoked contractions, reveals similar deficits in force between old and young people (13), suggesting that the weakness in this muscle at least is not due to poor neural drive. But are elderly people able to activate fully their muscles during fast dynamic contractions i.e. when power is being generated? As the threshold for recruitment of the high force motor units is lowered with increasing speed of contraction (16), it would seem that there is less of a likelihood of poor activation during fast dynamic contractions than during isometric ones. Indeed, the results of a study comparing stimulated and voluntary plantar flexor torques performed at 5 rad. s^{-1} (17) supports the contention that elderly people are no different to young in this regard, at least during very simple movements.

At the cellular level, muscle activation could be impaired in old age by alterations known to occur in the Ca^{2+} release and sequestration systems of the sarcoplasmic reticulum (18,19). However, even when the contractile machinery is fully activated (pCa 4.5) in chemically skinned single fibre preparations (where the sarcolema has been removed), fibres which express the MHC-IIA isoform have a lower specific tension (0.26 versus $0.36 N/mm^2$) if they originate from the muscles of an elderly person as opposed to a young person (20). Furthermore, recent studies on elderly women suggest that oestrogen may play a rôle in the loss of specific force in the adductor pollicis muscle, as those post menopausal women who are on hormone replacement therapy do not show such a dramatic decline (21). This hormonal influence on the contractile properties of the adductor pollicis has been confirmed in a subsequent longitudinal study by the same group, studying the same muscle (22), but this effect is not a universal finding (23, 24).

The data of Skelton et al. (7) for lower limb extension and more recently Metter et al. (25) from arm cranking, confirm the observations of Davies et al. (26) that muscle power is reduced by an amount which is greater than that expected by the reduction in strength alone. Davies and co-workers measured explosive power of young and elderly men (aged 69 years) sprint cycling on an isokinetic dynamometer (where power was determined from the forces generated at the cycle cranks and the speed of crank rotation) and jumping from a force plate. They observed that peak power occurred at a slower rotational velocity (100RPM as opposed to 128RPM). During the vertical jump, the peak force generated was 84% of that produced by the young, but peak power only 49%. The nature of the force-velocity relationship explains in part, these observations. To overcome a fixed load or resistance such as bodyweight during a vertical jump, a weaker elderly muscle has to use a relatively greater proportion of its maximum force generating ability. The force-velocity relationship dictates that this more forceful contraction be performed at a slower speed. This slower speed may result in a movement away from the optimal velocity for power generation (Figure 2) and hence less power is generated. This may also explain the greater decline in power compared to isometric strength reported by Skelton et al. (7) using the Nottingham power rig where only a fixed flywheel inertia was used for testing, irrespective of the degree of weakness.

2. Slower speed of shortening

The shortening characteristics of a muscle are determined ultimately by the expression of the different myofibrillar proteins (myosin heavy chains I, IIA, & IIX). The slower contracting (type I) fibres exhibit a V_{max} approximately one third of that of the IIA fibres. Fibres expressing exclusively MHC-IIX are even faster, but are rare as MHC-IIX isoforms usually occur in hybrid fibres co-existing with MHC-IIA (27). In animal muscle, modulation of shortening velocity among fibres expressing the same MHC may be explained by the type of alkali light chain expressed (28), although this has yet to be clearly shown in man due to the co-expression of fast and slow regulatory light chains in the same fibre (29). MHC-I, IIA and IIX fibres generate on average 0.5, 1.9 and 3.4 Watts of peak power per litre of muscle respectively, when studied at 12°C (30).

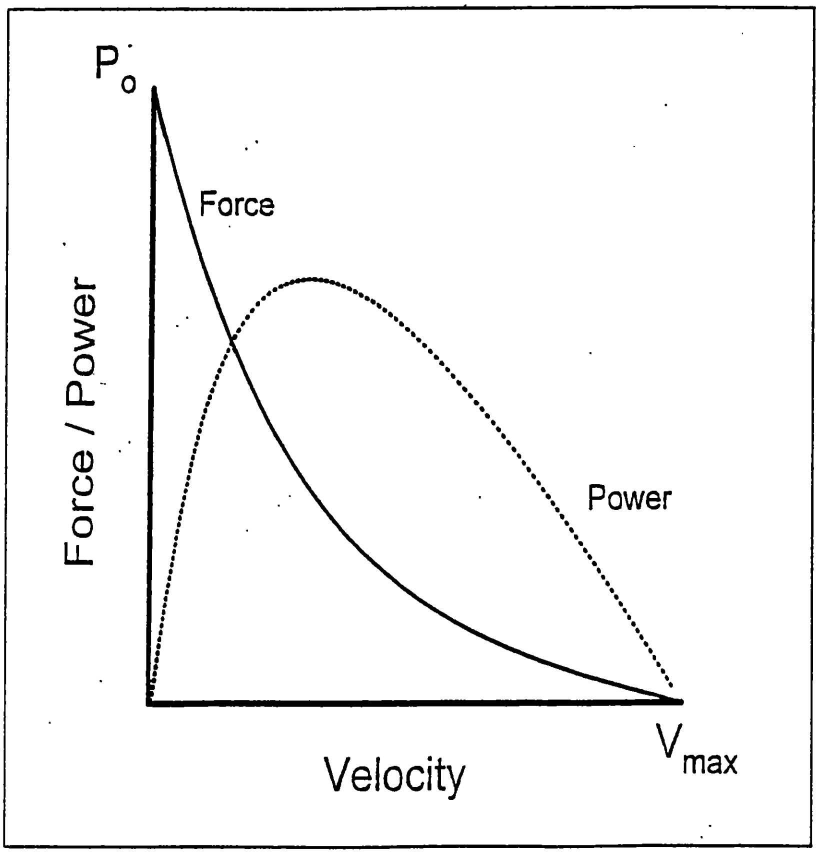

FIGURE 2. Schematic representation of how a weaker muscle (b) can have a greater decrement in power (dotted lines) when it is forced to contract more slowly against a given resistance (*r*) resulting in a less optimum velocity for power generation as indicated by arrow 2. Arrow 1 indicates the power obtainable when resistance *r* is generated by a stronger muscle (a).

Using the slack test to determine maximum shortening velocity (V_o) Larsson et al. (20) reported that single fibres which originated from older people, exhibited a significantly slower speed of shortening when compared with fibres expressing the same MHC's but originating from young muscles. The mean V_o values for MHC-I fibres were 0.18 (old) v 0.25 fibre lengths s^{-1} (young) and for MHC- IIA fibres 0.83 (old) v 0.98 fls^{-1} (young). A similar phenomenon was reported by the same group in rat muscle fibres (31). In whole muscle experiments (mouse) however, Brooks and Faulkner (32) reported that V_{max} (extrapolated from force-velocity data) was unchanged in the aged mice when compared to young mice in both the soleus and extensor digitorum longus muscles. The reasons for the discrepancy are not clear.

In whole human muscle, Harridge et al. (33) reported that the normalised torque generated by the plantar flexors of elderly men aged 70 years by stimulated release contractions at 5 rad s^{-1} (the highest angular velocity that could be tested) was related to the relative amount of MHC-II expressed in their gastrocnemius and soleus muscles. Only a few subjects were used in this study and the lower mean values of %MHC-II observed in both muscles did differ statistically from those of the young subjects.

In has been suggested that the muscles of elderly people have more type I fibres and this contributes to the loss of muscle power. However, the balance of evidence suggests that that fibre type proportions do not change as a function of age (34). The most extensive data on this topic comes from Lexell et al. (35) supports this lack of change. They measured fibre composition (using ATPase histochemistry) of whole sections of vastus lateralis muscle taken from cadavers of different ages. However, there is evidence that the size of the type II fibres may become reduced in advanced old age. This alone would result in less overall type II MHC expression, which in turn would be likely to have an impact on muscle power production (36). Furthermore, there is evidence that in old age muscle fibres themselves become more heterogeneous in nature, with an increase in the number of muscle fibres expressing more than one MHC isoform (27).

In addition to MHC expression, speed of shortening and muscle power output may also be affected by other factors such as nitric oxide (37) and importantly for elderly people, muscle temperature (30, 38). Elderly people are less able than young adults to maintain their core and muscle temperatures when exposed to a low ambient temperature (39). Thin elderly people are affected most in this regard, not merely because of their poorer insulation but because they are less able to mount a protective increase in their metabolic rate (40). This may be due to their smaller muscle mass but it is possible that there are qualitative changes which results in a decreased ability to stimulate substrate cycles both in muscle and in other tissues (41). Even greater reductions in power are thus likely in thin and cold elderly people.

FUNCTIONAL CONSEQUENCES OF A LOSS OF MUSCLE POWER IN OLD AGE

Effective muscle contractility is essential for the physical tasks of ordinary everyday life. In youth, muscles like most organs, have generous spare capacity; their strength, power and endurance being more than ample for the walking, stepping, climbing, holding and lifting requirements of normal activities of daily living. Although muscle performance starts to decline in middle age, it is not until advanced old age that a healthy individual's muscle function will have deteriorated to such a degree that their ability to perform such basic tasks is lost, or even threatened. When this happens, however, the consequences are potentially severe for continuing independence and for safe personal mobility.

Three different, but closely related approaches, have been useful in the exploration of the laboratory correlates of everyday functional abilities. Simple linear regression has been less revealing.

The 'threshold' concept (42) was prompted by clinical observation of the functional abilities of individual patients as they grew stronger or weaker (e.g. 43). It was explored by Skelton et al. (7) in healthy men and women aged from 65 to 89 years, who determined the greatest height of step that could be mounted without using the hands. Although step height correlated significantly with lower limb extensor power, it was not possible to identify a universally applicable threshold value, even after adjusting for limb length. Arguing that each individual might have their own, unique threshold value for each height of step, a slightly different approach was taken (44). From this analysis, it was apparent that a unilateral power/weight ratio of 2.5 $W \cdot kg^{-1}$ formed what might be considered a population threshold; above this value virtually no one failed to mount a 50 cm step without using their hands but below this value an increasing proportion of people would be unable to mount the step. An equivalent observation applied to a power/weight ratio of 1.5 $W \cdot kg^{-1}$ and a 30 cm step.

A somewhat different, but compatible model (based on an inverse transformation of strength), was adopted by Buchner et al. (45) to examine the relationship between lower limb strength (a composite of the strength of 4 muscle groups) and self-chosen walking speed. Their data supported the hypothesised model. Nevertheless, there was wide inter-individual variation, with the result that they too concluded that a "universal threshold" may not exist, suggesting that this might be because it may be possible to compensate for deficiencies in strength by using reserve capacity in other determinants of walking speed.

With the help of these three representations of the threshold phenomenon, it is easy to appreciate that muscle performance will reach critically low levels at a younger age in the presence of disease, through an enforced reduction in habitual physical activity, poor nutrition, or disease related cachexia (e.g. in cancer, heart failure, or chronic airflow limitation). It is also clear that women, even in health, are at a disadvantage; their relatively smaller muscle mass and the resultantly lower power/weight ratio means that they reach functional 'threshold' values of muscle performance some 20 years younger than men (46).

Consider, for example, the modest environmental challenge posed by a step 30 cm high (46). In the English National Fitness Survey, nearly half of all women aged only 70-74, but only 15% of men of the same age, had a unilateral lower-limb-power/weight ratio less than 1.5 W.kg^{-1} (the value below which increasing numbers of people will not be able to mount the step without using their hands (44). It seems likely that the gender difference in body composition which underlies this difference in functional muscle performance may contribute to the greater prevalence of disability and falls amongst elderly women than amongst elderly men.

As Buchner points out, the same approaches also make it easy to recognise that small changes in physiological capability can have large effects on the functional ability of a frail person, whereas quite large changes in physiological capability may produce little or no change in the everyday function of a more robust person (e.g. someone who is younger, healthier or male!).

CAN MUSCLE POWER BE REGAINED THROUGH EXERCISE TRAINING?

I. CROSS-SECTIONAL PERSPECTIVE

In most studies muscle power has not been directly measured but mechanical function reported in terms of isometric strength, or the amount of weight that can be lifted without reference to the speed of contraction. However, as is clear from the previous sections any adaptation to exercise that will increase either force or speed of contraction, all other things being equal, will increase muscle power.

Muscle usage can take two extreme forms; high resistance short duration work (e.g. weightlifting) or low intensity, long duration work (e.g. endurance running). In young individuals strength training is known to increase muscle mass and strength, but has relatively little effect on aerobic power. In contrast endurance training enhances aerobic capacity on the systemic (cardiovascular) as well as muscle levels, but has little impact on muscle strength. This raises the important issue of specificity of training and the benefits of different types of training for maintaining explosive power in old age.

Cross-sectional data suggest that elderly men who undertake endurance based physical activity are likely to be no stronger (47) or only slightly stronger (48,36) both in absolute terms and when strength is normalised to body mass (47), lean body mass (48) or muscle cross-sectional area (36) when compared with men who do not undertake any exercise. This is despite these trained athletes having a high level of aerobic power (e.g. mean VO_2 max of 42ml. $kg^{-1}min^{-1}$ in those aged 70-76 years, (47)). In contrast, those elderly men who undertake strength and weight training activities are considerably stronger in both absolute and normalised terms, compared with those who do not undertake any exercise (36, 48) and can be as strong as non-active young males (36). Explosive power measured during a single standing vertical jump in male endurance trained cyclists aged 60-69 years was no greater than that measured in men of a similar age who did not undertake any exercise (49). This was the case in terms of absolute power and also when power was normalised to body weight or active muscle volume. However, power athletes (sprinters and jumpers) of a similar age were able to generate 66% more absolute power and 49% more power when expressed per unit volume of muscle, when compared with the inactive subjects.

The power per unit volume generated by chemically skinned single fibres expressing MHC-I or MHC-II isoforms obtained from the gastrocnemius muscle of master endurance runners aged 44 years was similar to that obtained from sedentary age matched controls (50).

These cross-sectional data support the contention that muscle function relating to explosive power is best maintained with training activities which involve high forces and power outputs and not those in generating submaximal power outputs.

II. LONGITUDINAL PERSPECTIVE

In the last decade a number of studies have been performed in which elderly people have undergone rigorous progressive resistance training programmes. These have similar structures to those undertaken by young people. i.e. 12 week programmes where subjects train 3 times per week performing 3-4 sets of 8 repetitions of an exercise at an intensity that corresponds to 70-80% of the amount of weight that can be lifted once (1-RM). It is apparent that in terms of the changes in 1) 1-RM, 2) maximum voluntary isometric force, 3) muscle cross-sectional area that the adaptations to this type of exercise are similar to those reported for young people performing this type of exercise. This is in terms of the magnitude of these changes and also in terms of the nature of the training response, namely dramatic increases in the amount of weight that can be lifted, but considerably less improvements in isometric strength and still smaller changes in muscle size.

TABLE 1. Summary of studies which have examined the effects of progressive resistance training on muscle strength and size of the knee extensor muscles in elderly men and women. *Controlled and ** randomized and controlled trials.
Training example: (12 3 3 x 8 x 80%) = 12 weeks of training, 3 sessions per week, each session comprising 3 sets of 8 repetitions at a weight equal to 80% of the 1-repetition maximum. CSA = Anatomical cross-sectional area measured by CT (computerised tomography), or MRI (magnetic resonance imaging). [1] Open brackets unaffected and closed brackets affected leg following (7 months previous) hip fracture.

	SUBJECTS			**TRAINING**			**% CHANGE**		
Author	**Age**	**Gender**	***n***			**(see Legend)**	**1-RM**	**Isometric**	**CSA**
Harridge et al. (53)	85-97	M+F	11	2	3	3 x 8 x 80%	138	17	10 MRI
Sherrington & Lord (56)**	64-94	M+F	35	4	5	General weight bearing ex.	-	22 (53)[1]	-
Welle et al. (57)	62-72	M+F	8	12	3	3x8x80%(3-RM)	71(3-RM)	-	6 MRI
Skelton & McLaughlin (55)	74-79	F	10	8	3	3x4-8 Body wt./elastic tubing	-	20	-
Sipilä & Suominen (58)**	76-78	F	16	16	3	4x8-10x60-75%	14	14	5 CT
Skelton et al. (52)**	76-93	F	20	12	3	3x4-8 Body wt./elastic tubing	-	27	-
Lexell et al. (59)	70-77	M+F	28	11	3	3x6x85%	163	-	-
McMurdo & Rennie (60)*	83	M+F	55	26	3	Seated group exercise	-	18 median	-
Fiatarone et al. (51)**	72-95	M+F	25	10	3	3x8x50-80%	113	-	3 CT vs. control
Grimby et al. (61)	78-84	M	9	8-12	3	3x8	Isokinetic	10 at 30°s^{-1}	- 3 CT
Fiatarone et al. (62)	86-96	M+F	10	8	3	3x8x80%	174	-	11 CT
Brown et al. (63)	60-70	M	14	12	3	2-4x15x50-90%	63	-	10 CT
Frontera et al. (64)	60-72	12M	12	12	3	3x8x80%	107	17	9 CT

Table 1 summarises the studies which have undertaken strength training of the knee extensor muscles in elderly people ranging from those in their 60's to those in their 90's. It seems that even very elderly people are able to benefit from progressive resistance training (51, 52, 53). However, in only one study has muscle power been directly measured. Skelton et al. (52) reported an 18% increase in lower limb power in healthy elderly women in the (measured in the Nottingham Power Rig) following 12 weeks of strength training utilising elastic tubing. Importantly out of 14 functional tasks tested only one (rising from the kneeling position) showed a significant improvement with training. The specificity of training is a well documented phenomenon. Rutherford et al. (54) for example observed an a 19% increase in isometric strength in young men following 12 weeks of knee extensor strength training (where 1-RM increased by 160%), but detected no change in lower limb power as measured by sprinting on an isokinetic cycle ergometer. In older people the question of specificity was addressed in a subsequent study by Skelton and McLaughlin (53) who combined strength training with sessions in which a number of functional tasks were also practised. They again reported significant improvements in isometric strength (20%), but now also significant improvements (10-20%) in 5 (stair walking time, times floor rise, step rate, timed up and go, timed chair rise) of the 9 functional tasks measured.

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