MUSCLE POWER AFTER STROKE

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning.

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ABSTRACT

Stroke is the leading cause of disability worldwide. It often leads to mobility limitations resulting from deficits in muscle performance. While reduced muscle strength and rate of force production have been reported, little is known about the power generating capability of people after stroke and its relationship to mobility. Research in other populations has found that measures of muscle power may have a greater association with activity performance than do measures of muscle force alone. Consequently, in an attempt to optimise power, investigators have focused on identifying ideal parameters within which to train for power. One such parameter is the identification of the loading level at which maximal power is generated. Literature reporting optimal loads from both young athletic and healthy older populations has yielded mixed results, making the applicability to a hemiparetic population difficult.

The purpose of this study was to investigate muscle power performance at differing loads and to determine at what load muscle power is best elicited in hemiparetic and age and gender matched control groups. A secondary aim was to ascertain whether there is a relationship between the muscle power values obtained and activities such as gait, stair climbing and standing from a chair.

Twenty nine hemiparetic volunteers and twenty nine age and gender matched controls were evaluated. Involved and uninvolved legs of the stroke group and a comparison leg of the control group underwent testing. Leg press muscle power was measured using a modified supine leg press machine at 30%, 50% and 70% of a one-repetition maximum (1-RM) load. Participants were positioned on the leg press machine and asked to push, with a single leg, as hard and as fast as they could. Data was collected via a mounted force platform and a linear transducer connected to a platform on which the participants lay. From these, power was able to be calculated. The activities were timed while being performed as fast as possible.

The results showed that peak muscle power values differed significantly between the involved, uninvolved and control legs. Peak leg power in all three leg groups was greatest when pushing against a load of 30% of 1-RM. Involved leg peak power tested at 30% of 1-RM (Mean:240; SD:145 W) was significantly lower ($p<0.05$) than the uninvolved leg (Mean:506; SD:243 W). Both the involved and uninvolved legs

generated significantly lower peak power ($p<0.05$) than the control leg (Mean:757; SD:292 W). Correlations were found between the involved leg peak power and gait speed and involved leg peak power and stair climbing (r=0.6-0.7, $p<0.05$). No correlation was found between paretic leg peak power and chair stands. The control group leg peak power demonstrated significant associations with the performance of all three activities.

In summary, there were significant differences between the involved and the uninvolved leg in power production after stroke. As well, there are significant differences between the uninvolved leg and the leg of those not affected by stroke. Power was related to a number of activities.

CHAPTER 1-INTRODUCTION

1.1 The problem

Stroke is the third biggest cause of death and the leading cause of disability in New Zealand and in developed countries worldwide (Feigin, Lawes, Bennett, & Anderson, 2003). With improved acute management, the survival rate is high and there are an estimated nine million stroke survivors worldwide per year with predictions that this will rise by 30% between the years 1983 and 2023 (Wolfe, 2000). Of those that do survive after stroke, nearly half are left dependent (Warlow, Sudlow, Dennis, Wardlaw, & Sandercock, 2003) and will often present with some sort of residual physical deficit, resulting from muscle weakness, that impacts on their independence and participation (Feigin, Lawes, Bennett, & Anderson, 2003; Foulkes, Work, Price, Mohr, & Hier, 1988; New Zealand Guidelines Group, 2004; Stroke Foundation, 2004; Strokecenter, 1998-1999).

Weakness after stroke has been well established (Andrews & Bohannon, 2000; Bourbonnais & Vanden Noven, 1989; Davies, Mayston, & Newham, 1996; Hachisuka, Umezu, & Ogata, 1997; Harris, Polkey, Bath, & Moxham, 2001; Newham & Hsiao, 2001) People who have sustained a stroke lose muscle strength in that they (or their muscles) lose the ability to exert muscular force to overcome a load, under a given set of conditions. As a consequence, they not only lose muscle strength, they also suffer from mobility issues (Ada, Dorsch, & Canning, 2006; LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Morris, Dodd, & Morris, 2004; Patten, Lexell, & Brown, 2004; van de Port, Wood-Dauphinee, Lindeman, & Kwakkel, 2007).

It is also worthy of note that the incidence of stroke increases with increasing age (Feigin, Lawes, Bennett, & Anderson, 2003; New Zealand Guidelines Group, 2004; Stroke Foundation, 2004; Strokecenter, 1998-1999; Warlow, Sudlow, Dennis, Wardlaw, & Sandercock, 2003). Therefore, many people who have a stroke are likely to be of older ages. Age in itself brings about its own declines in strength and mobility (Fiatarone Singh, 2002; Latham, 2004; Latham, Bennett, Stretton, & Anderson, 2004). As such, presentation after a stroke may very well have contributions from both aetiologies and a better understanding of these both will allow for a better appreciation of the neurologically related deficits seen.

Both cross-sectional and trial-designed studies have demonstrated limitations in muscle strength that have been linked to decreased mobility in both the aged (Chandler, Duncan, Kochersberger, & Studenski, 1998; Fiatarone et al., 1994; Lamoureux, Sparrow, Murphy, & Newton, 2002; Seynnes et al., 2004) and stroke populations (Bohannon, 1986; Canning, Ada, Adams, & O'Dwyer, 2004; Nadeau, Arsenault, Gravel, & Bourbonnais, 1999; Ouellette et al., 2004; Weiss, Suzuki, Bean, & Fielding, 2000). Consequently, people affected by stroke, or hemiparetics, will most likely present with some form of muscle weakness that can impact on their ability to carry out many activities such as walking, standing up from a chair and using stairs.

When compared with strength, muscle power, defined as the ability to generate muscle force quickly, may have a larger influence on activity performance. Schulz (1995) and Cuoco et al. (2004) have proposed that, in the older adult, performance of many activities of daily living (ADL)'s does not require large amounts of joint torque and may rely more heavily on the ability to quickly develop the required torque. Indeed, many authors investigating function in the older adult are now reporting a significant relationship between muscle power and function. Leg power was the strongest predictor of ambulatory and functional status as compared to other variables, including strength in a number of investigations (Bassey et al., 1992; Bean, Kiely et al., 2002; Foldvari et al., 2000; Rantanen & Avela, 1997; Suzuki, Bean, & Fielding, 2001). Bean et al. (2003) further concluded that not only does muscle power consistently explain more of the variance (22%-38%) than strength in many activity-based measures, but also that low power would result in a two to threefold greater risk of mobility limitations than low strength. Perhaps another indication of the role muscle power plays in function can be taken from trials that compared strength training to power training to no training. Those who power trained showed greater functional gains and even similar strength gains as compared to strength training alone or to no training (Bean et al., 2004; Miszko et al., 2003).

In the stroke population, there is also increasing appreciation that muscle force is not the only factor to consider. For instance, Olney, Griffen, Monga & McBride (1991) demonstrated significant correlations between hip and ankle power and gait velocity. As well, Davies, Mayston & Newham (1996) demonstrated a significant relationship between maximum knee extension velocity and 10 metre gait time but a non-significant one with isometric maximum voluntary contraction (MVC) and gait time. The

significance of the velocity component of power was also highlighted by Pohl et al. (2002) who investigated peak force, rate of force development (RFD) and walking speed in participants with stroke. Twelve percent of the variance in gait speed was explained by combining peak isometric force with the RFD of the knee extensor muscles. When peak force was removed, the model predictions did not change significantly. However, when the RFD was removed, the predictive power was reduced by almost half. In addition, in a cross-sectional design study, LeBrasseur, Sayers, Ouellette & Fielding (2006) used stepwise regression models to demonstrate the significant effect deficits in muscle power have with gait and stair climbing in community dwelling participants with stroke. They concluded that muscle power explained nearly twice the variably as muscle strength for stair climbing in this population. Therefore, the role of muscle power in activity performance has been linked to an increasing number of populations, including stroke.

A number of trials have investigated the effect of muscle training on activity performance in the stroke population (Kim, Eng, MacIntyre, & Dawson, 2001; Ouellette et al., 2004; Teixeira-Salmela, Olney, Nadeau, & Brouwer, 1999; Weiss, Suzuki, Bean, & Fielding, 2000). While effective at improving muscle force, strength training programmes without a power component have not led to significant gains in activity performance tasks such as gait and stair climbing speed (Meek, Pollock, & Langhorne, 2003; Morris, Dodd, & Morris, 2004; van de Port, Wood-Dauphinee, Lindeman, & Kwakkel, 2007). Although in a neurologically impaired person there are many potentially confounding factors affecting motor control and performance, the outcome from these studies may be partially explained by the fact that muscle strength performance comprises of several components (force and velocity) and that progressive resistance training in its traditional application does not address both of these components.

If power training is to be implemented in stroke rehabilitation, the identification of the best load or resistance at which maximal muscle power can be generated is a necessary step. Several authors have investigated optimum load profiles that best elicit maximum power in other populations. Kaneko, Fuchimoto, Toju & Suei (1983) noted maximal power in the upper limb in young healthy males occurred after testing and training at 30% of maximum isometric strength for twelve weeks. A comparison of differing loads in the same population by Siegel, Gilders, Staron & Hagerman (2002) demonstrated that

maximal muscle power was produced during a bilateral squat exercise at loads of 50-70% of maximal force. Fielding et al. (2002) found that in the older adult, loads of 70% of maximum isoinertial strength, or one-repetition maximum (1-RM) allowed for maximum lower limb power production. In their intervention trial, de Vos, Singh, Ross, Stavrinos, Orr & Fiatarone Singh (2005) found that older adult power increased similarly when training across low (20% of 1-RM), medium (50% of 1-RM) and high (80% of 1-RM) loads and noted that strength and endurance increased when training at high loads and therefore recommended the higher load for training. While this may be sound reasoning if aiming for optimal muscle performance in isolation, it is important to also consider its functional application. For instance, Cuoco et al. (2004) concluded that power output at 40% of 1-RM explained more of the variability in habitual gait velocity than did power at 70% of 1-RM and stressed the importance of further investigation of lower loads because of their relevance to function. As such, appropriate optimal load that maximises activity performance may be a crucial component to improving function. However, to date, there does not seem to be consensus on one load that optimises power performance and explains activity performance in the neurologically intact population.

With increasing attention and support for not only strength but power training in stroke rehabilitation, clear training parameters need to be established. To date, there is nothing in the literature that addresses optimum load for power training in the stroke population. Therefore, more stroke specific data need to be available in order to optimise and promote muscular performance that may impact on physical function.

1.2 Purpose of the study

The primary aim of this project was to compare muscle power generated at different loads (30%, 50%, 70% of 1-RM) during a leg press task using the involved and uninvolved limbs of individuals with stroke and to compare those values with those generated by age and gender matched participants without stroke. A secondary aim was to examine the relationship between power and performance of three activities (gait, stair climbing, and standing from a chair).

1.3 Significance of the problem

In many activities of daily living, the rate at which muscles can produce work, or power production, is the critical performance variable (Enoka, 2002). Some authors would argue it is the most important variable (Bassey et al., 1992; Bean et al., 2003). The

ability to produce muscle power, once the sole domain of athletes participating in sporting endeavours, is now being acknowledged in other populations for its contribution to purposeful movement, mobility and even safety. With improved acute management, the survival and subsequent disability rates after stroke are rising. Many of these people, therefore, have goals of attaining their pre-morbid functional and participation level. As such, there is growing demand for effective rehabilitation aimed at achieving these goals. The investigation of power production after stroke has received little attention in rehabilitation research to date. Therefore, the current study will contribute to the growing focus and available literature on optimal loads and ultimately to intervention methods aimed at improving muscle performance after stroke.

CHAPTER 2 – REVIEW OF THE LITERATURE

2.1 Introduction

This chapter presents current literature that provides information and background on a number of issues related to the production of muscle power after stroke. First is a review of the structural and physiological factors involved in force and power generation in skeletal muscle. In order to appreciate the deficits seen after a stroke, this section will be further subdivided to include a review of these factors in the young healthy adult, the aged adult as well as after stroke. Trials that have investigated parameters and training considerations that optimise power output in each population are then discussed. Finally, studies addressing muscle power's association with mobility and performance is presented.

2.2 Factors influencing force and power generation in skeletal muscle

2.2.1 Introduction

This section will review the structural and physiological properties of the normally functioning neuromuscular system and its ability to generate muscle force, velocity and power. Firstly, a description of the mechanics of muscle contraction will be followed by a discussion on the relationship of the contributing factors in power production: force and velocity. Next, the influences of muscle architecture on the behaviour of muscle will be presented including reference to both the non-contractile as well as the contractile elements. Finally, the neural involvement and factors associated with muscle activation will be examined.

2.2.2 Basic muscle fibre and muscle contraction mechanics

A muscle can contain a few hundred to as many as a million muscle fibres (Feinstein, Lindegard, Nyman, & Wohlfart, 1955; McComas, 1996). These fibres, which are composed of myofibrils, house the contractile element of the muscle: the sarcomere. The striated appearance of these sarcomeres corresponds to the two sets of highly organised filaments of thick myosin and thinner actin as demonstrated by H.E Huxley (1953). Huxley (1953) examined thin sections of frog sartorius and rabbit psoas muscles via electron microscope and noted that each contained a thick and thin filament. As is now understood, the thick filaments contain the main protein of myosin whose heads are the force generating sites in the muscle and utilise the energy derived from adenosine triphosphate (ATP). The two strings of actin molecules make up the thin

filament and contain the contraction regulatory unit of tropomyosin with its adjoining troponin complex (A. Gordon, Homsher, & Reginer, 2000).

The observed consistency in length of these two filaments during contraction and a proposition that they slid alongside each other, supplied by HE Huxley and Hansen (1954) as well as AF Huxley and Niedergerke (1954), led AF Huxley (1957) to propose the cross-bridge theory (earlier hypotheses suggested a folding or coiling of some sort). In this theory, cross-bridges from the myosin filament attach in a cyclical manner to specific binding sites on the actin filament. Contraction was thought to be produced by a power stroke of these cross-bridges (A. Huxley, 1957; H. Huxley, 1969). These cross-bridges could also remain in some form of attachment, a theory that was later refined and included reference to the existence of stable states of cross-bridge attachment (A. Huxley & Simmons, 1971). Despite more recent investigations in single cross-bridge interactions with findings that are in disagreement with what would be expected based on this model (Finer, Simmons, & Spudish, 1994), and discussion around other models (Herzog & Ait-Haddou, 2003), the cross-bridge attachment theory is still the most widely used model explaining the mechanisms of contraction.

2.2.3 Force, velocity and power relationships

Muscle's behaviour under differing shortening speeds was observed by Fenn & Marsh (1935) and then Hill (1938). This force-velocity relationship was based on observations of thermal activity and energy liberation with whole frog muscle contraction. The increase in rate of heat production was noted to be proportional to the speed of shortening. Also observed was that the total extra heat produced during shortening was proportional to the distance shortened. As well, the rate of extra energy liberation during shortening was inversely proportional to the load applied to the muscle in afterloaded shortening experiments. From these observations, Hill (1938) proposed a hyperbolic curve to reflect the loss of force with increasing shortening velocity for maximally stimulated muscle at optimal length, along with a widely used general equation to describe it. AF Huxley's (1957) sliding filament theory is, to some extent, also able to be integrated with this work. This relationship has maximally generated muscle force dependent on its contraction velocity. Briefly, the force a muscle is able to produce is dependent on the number of cross-bridges and the degree of overlap. When the speed of contraction is zero and the muscle is at its optimum length, maximum cross bridge formation is attained. As velocity of contraction increases, the rate at which

cross bridges must attach and detach also increases as does motor unit activation time, thus limiting the amount of force generated at fast limb-movement velocities (Edman, 1988; Perrine & Edgerton, 1978). As a result, fewer cross bridges form solid connections resulting in less actual force production.

This relationship has generally been found to be consistent in isolated animal whole muscles (Hill, 1938) as well as single muscle fibres (Edman, Reggiani, Schiaffino, & te Kronnie, 1988) but with differences of lower then predicted forces at low velocities or with lower contraction speeds at higher forces (Edman, Reggiani, Schiaffino, & te Kronnie, 1988). The in-vivo force-velocity relationship of human knee extensors was assessed by Perrine & Edgerton (1978) and later by Wickiewicz, Roy, Powell, Perrine & Edgerton (1984) using isokinetic testing. They too noted a biphasic relationship with the curve plateauing at relatively low velocities, suggesting that the Hill model overestimates force at low shortening velocities. While the original Hill model did not include force-velocity findings with muscle lengthening, Katz (1939) noted even higher forces when muscle is eccentrically lengthened possibly due to the mechanisms in cross-bridge detachment. However, differences in this force magnitude exist between electrically stimulated and voluntarily contracted muscle (Westing, Seger, & Thorstensson, 1990). The fact that in vivo voluntary contractions do not reach similar force levels to those of electrically stimulated muscle have led authors to suggest that a self-regulatory mechanism of inhibition and protection may be a factor (Harridge & White, 1993; Perrine & Edgerton, 1978; Westing, Seger, & Thorstensson, 1990; Wickiewicz, Roy, Powell, Perrine, & Edgerton, 1984). The additional finding that, under supra-maximal stimulation, human muscle does not present the same force inhibition further supports this hypothesis (Thomas, White, Sagar, & Davies, 1987). As well, in those trials involving voluntarily activated movement, antagonistic activity may have also played a factor in decreasing agonist force production (Kellis & Batzopoulos, 1997). A final word of caution should also be noted when applying this force-velocity curve in multi-jointed or more complex tasks that involve joint loading and eccentric work. The effects of inertia or gravity as well as the contribution of both the series elastic components and the nervous system must also be factored in, likely altering the resultant relationship between force and velocity (Macaluso & De Vito, 2004; Siff, 2000).

The power developed by a muscle in a contraction, therefore, can be derived and plotted from the force-velocity relationship and is equal to the product of the load and velocity with which it moves. The force-velocity curve indicates that power will be zero when the load acting on the muscle is zero as well as when the load is so heavy, it cannot be moved resulting in zero velocity. At some intermediate point, power production will be optimised. There is thus an optimal force and an optimal velocity at which maximum power is developed. By using certain assumptions to determine the constants, this can be mathematically calculated using Hill's equation (Hill, 1938).

2.2.4. Muscle architecture, size and shape

Despite very little difference between sarcomere structure and function, muscle behaviour and use varies widely. Differences in force and velocity characteristics can, in large part, be explained by sarcomere number and architecture, defined as the arrangement of muscle fibres relative to the axis of force generation. In fact, some would argue that it can have a greater influence than can the intrinsic biochemical properties (Wickiewicz, Roy, Powell, & Edgerton, 1983). There are three key architectural components that can affect muscle force and velocity generation: cross sectional area of a muscle; the length of the fibres within a muscle; and the angle of pennation, or angle at which the fibres are positioned, relative to the pull of the tendon. Each of these factors will now be discussed in more detail.

Firstly, the maximum force a muscle can produce is directly related to the physiological cross sectional area (PSCA), that is, the cross-section that cuts the fibres at a right angle, and is proportional to the number of sarcomeres arranged in parallel (Edgerton, Roy, Gregor, & Rugg, 1986; Lieber & Bodine-Fowler, 1993; Narici, 1999; Roy & Edgerton, 1992; Wickiewicz, Roy, Powell, Perrine, & Edgerton, 1984). The intrinsic force generating capacity of the muscle fibres can then be further calculated by normalising the force to the PCSA to determine the specific tension. Animal muscle specific tension appears to be relatively constant at 22.5 Ncm^{-2} with ranges from 15 to 30Ncm^{-2} (Close, 1972; Powell, Roy, Kanim, Bello, & Edgerton, 1984; Spector, Garniner, Zernicke, Roy, & Edgerton, 1980). However, suggestions of human skeletal muscle specific tensions have not been as decisive with them ranging from 23 to 42 and even as high as 100 N cm^{-2}. This large range may be explained, at least in part, to the inaccurate calculation of PCSA. When Narici (1999) normalised tensions for the PCSA's of human quadriceps muscles, the resultant average specific tension value was 25 Ncm^{-2} which is similar to

that based on animal data. This variation may also reflect differences in proportions of muscle fibre types in the muscle examined since Larsson, Li & Frontera (1997) demonstrated significant variations in specific tensions between fibre types in isolated young human knee extensor muscle fibres and found that the faster fibres produced the above noted specific tension of 24 Ncm^{-2} while the slower ones reached tensions of only 19 Ncm^{-2}. Other factors, such as variations in myofilament density and non-contractile structures can also have influences on force transmission as will be discussed in the following section. Hence muscles with greater PCSA will be able to produce greater amounts of force and power but at lower contraction velocities.

Secondly, the length of the fibres within a muscle reflects the number of sarcomeres in series. Longer muscle fibres with sarcomeres arranged in series, when stimulated, produce greater contraction excursion over a given time. As such, muscles with longer fibres will have the faster shortening velocity (Edgerton, Roy, Gregor, & Rugg, 1986; Lieber & Bodine-Fowler, 1993; Roy, Monti, Lai, & Edgerton, 2003). Therefore, muscle velocity potential is proportional to the number of active sarcomeres in series and muscle fibre length. As Roy & Edgerton (1992) have noted, however, there have been inconsistent reports on fibre lengths, most likely due to confusion over clear divisions of fibres, the use of fibre bundles instead of individual fibres and the difficulties with differentiating muscle fibres that are anatomically arranged as compared to functionally arranged and belonging to one motor unit (Roy & Edgerton, 1992; Roy, Monti, Lai, & Edgerton, 2003). Nevertheless, muscles with longer myofibrils will be able to produce peak power at higher velocities.

Thirdly, the angle of pennation will affect the amount of pull on the tendon. Larger angles will mean that less force and speed, produced by the muscle fibre, will be transmitted to the external tendon (Wickiewicz, Roy, Powell, & Edgerton, 1983). Of further consideration is that with muscle contraction, pennation angle changes (Herbert & Gandevia, 1995) further affecting the eventual transfer of force. However, since pennate muscles allow for a greater number of sarcomeres to be arranged in parallel, its PCSA will likely be greater per ACSA with a resultant greater specific tension (Challis, 2000; Narici, 1999; Sacks & Roy, 1982). Additionally, because of the angle, muscles with pennated fibres can utilise the length-tension relationship more effectively resulting in more range of optimal muscle length (Roy & Edgerton, 1992). While Wickiewicz and colleagues (1983, 1984) have argued that most of the pennation angles

are small and would not have a significant functional effect, the trade-offs that occur with pennation likely reflect the functional role and anatomical constraints of each muscle.

The consequences of muscle architecture on force and velocity production were highlighted by the work of Spector, Garniner, Zernicke Roy & Edgerton (1980). While investigating the soleus and gastrocnemius of the cat, these authors noted many differences between the two muscles. The soleus was composed of slower twitch fibres, smaller pennation angle and longer fibre length as compared to the gastrocnemius that had a high proportion of fast twitch fibres, larger pennation angle and shorter fibres. When assessing for peak force, the gastrocnemius produced force five times that of soleus, as would be expected from its muscle fibre type proportions. But, when all the other architectural parameters such as muscle volume, fibre length and pennation angle were included, the resultant specific tension was similar between the two muscles. Additionally, gastrocnemius was found to have fibres that exhibited a contraction velocity three times that of soleus. But, when assessing for maximum contraction velocity at the tendon, because of the pennation angle and fibre length, its speed was reduced to one and a half times that of soleus. As such, architecture in itself can substantially influence force and velocity properties.

As muscle power is the product of both force and velocity, different sarcomere arrangements will influence the power potential of different muscles (Edgerton, Roy, Gregor, & Rugg, 1986; Lieber & Bodine-Fowler, 1993). While a muscle with a pennated and parallel arrangement and a muscle with sarcomeres arranged in series will both be able to produce power, due to greater peak velocity, those that arranged in series will produce peak power at higher velocities.

2.2.5 Non-contractile components

Although the contractile elements are often the main focus of force and power production, more consideration has been directed towards the contribution of connective tissue, or the elastic components, to muscle tension. Forces produced by the muscle fibres are in part conveyed through and/or assisted by this connective tissue.

The structures of the elastic components, arranged in parallel (PEC) or series (SEC) appear to have a significant role in storing potential energy. Purslow & Trotter (1994)

described the honeycomb intramuscular collagen based connective tissue of the endomysium. They demonstrated that as muscle length increases, these collagen fibrils align themselves with the long axis of the muscle, providing elastic potential energy. Inactive muscle fibres, serially connected to active ones, also contribute to the elastic energy as does the extracellular matrix via transmembrane structural proteins such as titin and the desmin proteins (Monti, Roy, Hodgson, & Edgerton, 1999; Wang, McCarter, Wright, Beverly, & Ramirez-Mitchell, 1991). The endomysium, together with the perimysium which surrounds the muscle fascicles, form the intramuscular connective tissue (IMCT). These then merge with the epimysium to form the tendons and aponeurosis (Roy, Monti, Lai, & Edgerton, 2003). The collagen component of this tissue allows it to resist tensile loading while the small elastin component plays a role in energy absorption (Culav, Clark, & Merrilees, 1999). The tendon itself contains bundles of collagen fibres that also exhibit a crimped arrangement (Kannus, 2000) as well as cross-links that add to its strength (Culav, Clark, & Merrilees, 1999). This configuration allows the muscle-tendon unit to store significant amounts of potential energy. As increasing force is applied to the tendon, the collagen fibrils un-crimp and align, reflecting the increased stiffness, then stretch and store energy until a point where only very high forces will be able to contribute (Huijing, 1992). Eccentric forces, as seen from the force-velocity curve, are higher than others and are involved with this end of the curve. In situations of jumping, running or walking, this eccentric loading, prior to concentric action, allows for the SEC to maximally store potential energy that, when released quickly, results in a potentiated concentric contraction, contributing to an explosive, or powerful action. This mechanism is involved in the stretch-shortening cycle SSC (Komi, 1984) and enables the tendon to facilitate high velocity and high power movements without imposing these high velocities on muscle fibres (Huijing, 1992).

The role of the IMCT has become increasingly apparent from a structural, developmental as well as mechanical point of view (Purslow, 2002). Besides storing potential energy, the appreciation of lateral transmission of forces from the sarcomeres to the extracellular matrix is gaining more attention (Goldberg, Wilson, & Shall, 1997; Huijing, Baan, & Rebel, 1998; Monti, Roy, Hodgson, & Edgerton, 1999). Goldberg, Wilson & Shall (1997) found that despite removing a significant portion of force generating or transmitting structures from the eye lateral rectus muscle of the cat, the resultant force produced was only minimally affected, suggesting an internal

mechanism for transmitting force through the IMCT to the tendon. Similarly, Huijing et al. (1998) performed progressive tenotomies on the distal ends of dissected extensor digitorum longus (EDL) muscles of the rat and noted that despite progressively losing the attachment of distal ends of the muscle, the muscle was still able to maintain a disproportionate amount of force, likely due to the shear stress exerted on parallel myofibrils from the basal lamina and/or endomysium. Further fasciectomies between the myofibrils in the same EDL muscle resulted in additional declines in force, despite no actual interference with any of the remaining contractile elements, all of which suggests that the fascicle connective tissue sheaths are important force transmitting structures (Huijing, Baan, & Rebel, 1998). The amount of muscle force transmission will, subsequently, be dependent on the amount of lateral or shear strain. Therefore, the effectiveness of ultimate force transmission may differ for muscles of varying amounts of pennation and for the same muscle under different lengths.

In vivo studies of muscle-tendon behaviour during concentric contractions have been done using ultrasonography. This technology provides several advantages with regards to being less invasive as well as yielding more functionally relevant results for humans. However, methodological issues of how tendons are defined and assessed as well as the attempt to discriminate between the IMCT or the tendon and aponeurosis when they are inherently connected raise both reliability as well as validity questions. In vivo studies to date have also applied different loads to the muscle-tendon unit under examination making comparisons difficult. Additionally, different muscles have been investigated. These differing muscles would have different functional roles and as such, may have different force transmission characteristics. Differences in studies, leading to divergent results can be seen from the work Maganaris & Paul (2000) and from Magnusson, Hansen et al. (2003). Maganaris & Paul (2000) studied load-elongation characteristics of the tibialis anterior muscle-tendon unit of young healthy men. Eliciting isometric contraction from progressively larger amounts of electrical stimulation, they found that the tendon was three times stiffer than the aponeurosis. However, in the opposing Achilles tendon complex, Magnusson, Hansen et al. (2003) reported that during active plantarflexion, the tendon of their young male participants was almost six times more compliant than the aponeurosis. Discrepancies in protocol and results reflect the complexities of both the functional role each of these muscles-tendon units have as well as the methods used in investigating them in vivo.

2.2.6 Motor units

Motor units vary in size, having innervation ratios from only a few to thousands of muscle fibres per motorneuron (Loeb & Ghez, 2000; McComas, 1996; Monti, Roy, & Edgerton, 2001) and can be described as the elementary functional unit of the motor system. Understandably, in order to maintain flexibility in its actions (and produce force and power), the motor system demonstrates a variability and specificity in characteristics and classification of both the muscle fibres and their corresponding motorneuron. Although these two are very closely interrelated, the motor unit components of muscle fibres and motorneurons will be reviewed separately.

2.2.6.1Muscle fibres

While sarcomeres present similarly, muscle fibres demonstrate different structural and behavioural characteristics. These have been studied by a number of methods that have typically sought to identify markers that correlate with contractile speed. Both myosin ATPase isoforms studied by Brooke & Kaiser (1970) as well as myosin heavy chains (MHC) investigated by Reiser, Moss, Biulian, & Greaser (1985), have been linked to cross-bridge cycling rate, and hence contraction velocity. These two methods appear to support one another as the ATPase based classification of I, IIA, IIB, IIC generally corresponds to the MHC classification of I, IIa, IIx, IIb (Enoka, 2002; MacIntosh, Gardiner, & McComas, 2006). These markers, in addition to the presence or absence of other factors such as mitochondria, glycogen and capillary density, suggest differences in contractile behaviour with regard to speed and fatigability and also suggest a continuum of capabilities that may be subject to plasticity (Clamann, 1993; Gordon & Pattullo, 1993). Throughout this thesis, the simplified classifications of slow twitch (ST) and fast twitch (FT) will be used to represent the two main divisions of muscle fibres.

Despite some findings to the contrary (Bottinelli, Pellegrino, Canepari, Rossi, & Reggiani, 1999; Close, 1972; Larsson, Li, & Frontera, 1997), there is greater acceptance that there are insignificant differences between the fibre types with regard to specific tension, especially when normalised to the same cross-sectional area (Faulkner, Claflin, & McCully, 1986; Green, 1986; Herzog, 2004; Lucas, Ruff, & Binder, 1987). However, there is more agreement on the differences between fibres with regards to speed of shortening, reflecting the ATPase activity with myosin (Barany, 1967; Edman, 1988). Faulkner et al. (1986), investigating the behaviour of slow and fast human vastus lateralis muscle fibres, noted that while ST fibres had equal maximal isotonic

tetanic force to FT fibres, they contracted at speeds one third slower. This has been confirmed by Bottinelli et al. (1999) who also studied fibres taken from the knee extensors and reported a four fold difference in contraction velocity.

The difference in fibre type properties is particularly important for power production. Authors investigating contractile properties of human skeletal muscle fibres have presented data in the form of force-velocity, power-velocity and power-force curves that clearly demonstrate differences between fibre types (Bottinelli, Pellegrino, Canepari, Rossi, & Reggiani, 1999; Faulkner, Claflin, & McCully, 1986). Biopsied vastus lateralis muscle fibres have been differentiated into types and subsequently tested for contractile performance values such as isometric tetanic tension, maximal shortening velocity and maximum power output, to establish the force-velocity-power characteristics of each fibre type (Bottinelli, Pellegrino, Canepari, Rossi, & Reggiani, 1999; Faulkner, Claflin, & McCully, 1986). When Faulkner and colleagues (1986) normalised ST fibre tension to maximum isometric tetanic tension on the assumption of constant specific tensions between the two types, they noted that ST fibres, for a given force, contracted at a slower velocity relative to the FT fibres and proposed force constants of 0.15 and 0.25 for ST and FT fibre respectively. When included in Hill's equation (1938), this ST fibre constant results in a force-velocity curve with a greater curvature than for FT fibres. These findings were more recently confirmed by Bottinelli et al. (1999) who noted a four fold difference in maximal shortening velocity between the types. Both research groups were also able to calculate power outputs of the different fibres and noted that FT fibres produced greater power at all shortening velocities. Additionally, the load at which maximum power was reached was greater in FT as compared to ST fibres. Consequently, power that is produced by FT fibres has been shown to be four times (Faulkner, Claflin, & McCully, 1986) to nine times (Bottinelli, Pellegrino, Canepari, Rossi, & Reggiani, 1999) that of the ST fibres. This difference is most likely due to a greater shortening velocity for a given afterload.

As such, power performance can be related to percentage of FT fibres and those muscles with higher percentages of FT fibres perform most effectively during activities requiring rapid force development, especially with maximum contractions involving high velocities or when the rate of force development (RFD) is high (Green, 1986; MacIntosh, Herzog, Suter, Wiley, & Sokolsky, 1993). In 'mixed' muscles with ST and FT fibres, peak power is only 55% to that of a muscle with FT fibres exclusively

(Faulkner, Claflin, & McCully, 1986) with the ST fibres contributing only slightly to power performance at lower velocities. This ST fibres' contribution also declines with increasing speeds. This may be attributable or explained by the observation that, at lower levels of force, additional recruitment is through additional motor units, whereas at higher levels, the additional input is by an increased firing rate—targeting FT fibres more and excluding the ST ones (Moritani, 1993). In fact, McCartney, Heigenhauser & Jones (1983) reported that the differences in muscle fibre composition alone could account for 85% of difference in maximum power. Clearly, muscle fibre type plays a considerable role in the production of power.

2.2.6.2 Motorneurons

It has long been known that muscle fibres belonging to the same motor unit manifest nearly identical biochemical, histochemical and contractile characteristics (Burke, Levine, & Zajac, 1971). This evidence points to the pivotal role of the motorneuron in influence and behaviour. This is supported by the finding that after surgically switching motorneurons between FT fibres and ST fibres, Buller, Eccles, & Eccles (1960a; 1960b) and later Gordon, Thomas, Stein & Erdebil (1988) found that both groups of fibres showed evidence of transformation towards that of their new motorneurons' properties.

The morphology of the motorneuron has specific properties. Those that innervate the slower contracting muscle fibres, for example, have smaller cell bodies and fewer ion channels in parallel, resulting in a higher overall resistance. The resultant excitatory post-synaptic potential is thus, according to Ohm's Law, greater and hence these motorneurons have lower firing thresholds and are the first to be recruited (Burke, 1967; Kernell, 1983; Loeb & Ghez, 2000). Together, the type of motor neuron and its corresponding muscle fibres demonstrate specificity with regards to gradation and selection of muscle contraction. This has implications for recruitment as will be discussed in the next section.

2.2.7 Activation

2.2.7.1 Motor unit coordination

The differences seen in motor unit properties play a role in smooth and graded muscle activation. Motor unit pools, when elicited voluntarily, are activated by peripheral or descending inputs to produce muscle force and contraction velocity. Increases in either of these are subsequently produced by increases in the recruitment of additional motor

units as well as the increase of firing frequency in a given motor unit, otherwise called rate coding (Adrian & Bronk, 1929; Moritani, 2003). When increasing force, it appears that these two strategies are employed differently across muscles. The early work of Milner-Brown, Stein & Yemm (1973), using spike-triggered averaging to study the first dorsal interosseous muscle of the hand, showed that half of the motor units had already been recruited when only 10% of the maximal force had been developed. This would suggest that recruitment was preferentially used at lower loads, followed by increased firing rates to bring the force closer to maximum. This finding was later confirmed by Kukulka & Clamann (1981) who also demonstrated little additional motor unit recruitment once the adductor pollicis muscle reached about 50% of maximum force.

However, when Kukulka & Clamann (1981) repeated the testing on the larger and more proximal biceps brachii, they found that additional motor unit recruitment occurred over a greater range of forces concluding that in these muscles, recruitment is an important factor at all force levels. Similar findings from others (DeLuca, LeFever, McCue, & Xenakis, 1982; Desmedt & Godaux, 1978; Moritani, Muro, Kijima, & Berry, 1986) would suggest that small distal muscles rely more on increasing firing rates when needing to produce higher forces whereas larger proximal muscles continue to recruit additional motor units even at greater force levels.

An additional element of versatility in proportional muscle contraction is the use of slow (ST) and fast (FT) motor units. It is generally accepted that they are recruited in order according to their size. This has been termed the size principle (Henneman, Somjen, & Carpenter, 1965). This orderly progression of activation from ST to FT motor units reflects the biochemical and contractile properties of ST and FT motor units and ensures force increases will be proportional to the activation, which results in smooth increases or decreases in force output. There is also general agreement that in voluntary, steady or repetitive contractions, ST motor units are recruited first (Desmedt & Godaux, 1977, 1978; Gollnick, Piehl, & Saltin, 1974). Using histochemical staining for both fibre typing and glycogen content, Gollnick et al. (1974) studied muscle fibre specimens from volunteers who were either cycling or isometrically contracting their quadriceps muscles. They were able to demonstrate that in both of these sustained activities, ST fibres were the first to show signs of glycogen depletion when working submaximally. At supramaximal levels, however, FT fibres also showed depletion. This orderly motorneuron recruitment and rate coding has also been found to exist

without voluntary input but with transcranial magnetic stimulation (TMS) of the upper limb muscles in a small group of volunteers between the ages of 32 and 49 years (Bawa & Lemon, 1993).

However, there is less agreement about recruitment order in other situations. Desmedt & Godaux (1977) investigated motor unit behaviour in voluntary ramped and ballistic actions of the tibialis anterior muscle in young men. While they found that the threshold of motor unit firing was lower during ballistic movements and that the discharge rate was considerably higher, particularly early in the burst, there was no change in the order at which motor units were recruited. The muscle was able to meet the powerful demands of the task by earlier recruitment of all the motor units. These authors had similar findings when testing the upper limb, soleus and finger abduction muscles (Desmedt & Godaux, 1977, 1979). Van Cutsem, Duchateau & Hainaut (1998) also found that even with 12 weeks of explosive training on the dorsiflexors of young volunteers, the motor unit recruitment order was preserved but all the units were activated earlier and had a greater maximal firing frequency during the voluntary explosive contractions.

On the other hand, it has been proposed that this order and discharge pattern may vary depending on a number of other factors. For example, training via biofeedback was shown to alter the recruitment order of thumb motor units in some human volunteers (Basmajian, 1963). As well, stimulation via cutaneous electrical stimulation preferentially activates the larger FT motor units, likely through inhibition to ST and excitation to the FT units at the level of the spinal cord (Delitto & Snyder-Mackler, 1990; Garnett & Stephens, 1981). Altered motor unit thresholds have also been found in the biceps brachii, depending on whether it was flexing, supinating or externally rotating the humerus (Gielen & Denier van der Gon, 1990). Additionally, Nardone, Romano & Schiepatti (1989) found that there were fast conducting motor units that were only active during eccentric contractions and proposed that FT motor units were selectively activated during lengthening contractions. This preferential FT activity was also found in the eccentric stretching phase of a hopping action (Moritani, Oddsson, & Thorstensson, 1991). At the muscle level, investigations into the paw-shake response of rapidly alternating flexion and extension of a cat paw showed that the predominantly ST soleus, which is normally constantly active, is preferentially inactivated while the shaking is produced by activation of the fast-twitch muscles (Smith, Betts, Edgerton, &

Zernicke, 1980). Similarly, in humans, it has been shown that some high-threshold units in the toe extensor muscles did not fire except in rapid corrective movements (Grimby, 1984). Therefore, despite an orderly recruitment for most activities, in certain circumstances, this order may change resulting in selective recruitment of FT fibres.

2.2.7.2 Supraspinal drive

Voluntary movement, generated by the motor cortex, is initiated by a need or idea to move (Prochazka, Clara, Loeb, Rothwell, & Wolpaw, 2000). Selective stimulation of different regions of the primary motor cortex can produce the same movement (Penfield & Boldrey, 1937; Sessle & Wiesendanger, 1982) suggesting the existence of multiple functional efferent zones resulting in movements that are controlled by a network of neurons distributed throughout the motor cortex. This allows tremendous flexibility through convergence of overlapping cortical territories on to single motor units as well as divergence from one cortical site to many lower motor neurons (Huntley & Jones, 1991). Limb joints are also represented in the cortex more than once, but with different configurations to enable a large repertoire of target-muscle activation. As a result, the system can sustain various movement combinations (Rossini, Calautti, Pauri, & Baron, 2003). The cortical activity that occurs before a movement is widespread and involves not only the motor cortex (Tanji & Evarts, 1976), but also the supplementary and premotor areas (Weinrich, Wise, & Mauritz, 1984), cerebellum and basal ganglia (Ghez & Thach, 2000; Hoover & Strick, 1993; Thach, 1978). This is reflected in the findings of earlier work (Galea & Smith, 1994; Jane, Yashon, Demyer, & Bucy, 1967) that suggest that only 30% of the corticospinal tract fibres originate in the primary motor cortex. These cortical areas represent functional movement-specific areas and demonstrate plasticity in response to various stimuli such as after injury, physical training or, skill acquisition (Jenkins, Merzenich, Ochs, Allard, & Guic-Robles, 1990; Karni et al., 1995; Muller et al., 1997; Weiss et al., 1998).

The central command, originating from the motor cortex and transmitting to lower neurons, requires that cortical neurons be released from inhibition and depolarised enough to reach threshold. Kujirai and colleagues (1993) first described a paired pulse paradigm using transcranial magnetic stimulation (TMS) to test the excitability of inhibitory and excitatory intracortical circuits within the human motor cortex and to evaluate their role in the modulation of motor cortical output. There is evidence that short interstimulus intervals (ISIs) reflect the activation of GABAergic interneurons that

exert intracortical inhibition on the corticospinal neurons, whereas the facilitation seen at longer ISIs reflects the activation of glutamatergic interneurons that provide excitatory effects on the corticospinal neurons (Cicinelli et al., 2003). Ridding, Taylor & Rothwell (1995) found that with voluntary contraction, there was a reduction in the activity of inhibitory circuits in cortical areas that projected to the voluntarily contracted muscles in a group of 30-47 year old volunteers. These authors used TMS in paired-pulses to examine the cortical activity during a minimally tonic contraction of the first dorsal interosseous (FDI) muscle and noted that voluntary drive produced different cortical responses than when tested during relaxation. They also found that when another muscle that was not being tested, such as biceps brachii, was voluntarily contracting, there was no effect on the relaxed FDI, reinforcing the notion of a high degree of movement and muscle specificity in the cortex. This finding was further refined by Chen, Yaseen, Cohen & Hallett (1998) who found that in a similar group of volunteers performing thumb abduction, there was increased excitability in the area of the cortex projecting to the agonist muscle approximately 20ms before the movement. Reynolds & Ashby (1999) then used a paired-pulse TMS protocol to assess cortical inhibition in a similar sample group performing wrist extension and flexion actions. They found that selective focal inhibition of only the corticospinal neurons projecting to the agonist muscles occurred approximately 95ms prior voluntary contraction. Combining these results, it has been suggested that this selective reduction in inhibitory cortical circuit activity well before the movement could serve to focus the subsequent excitatory drive onto the corticospinal neurons that produce the intended movement (Floeter & Rothwell, 1999).

Because of their brevity, explosive or ballistic movements, defined as movements in which subjects accelerate an object or their own body mass throughout the propulsive phase (Newton & Kraemer, 1994) are pre-programmed actions, meaning that once the central command is transmitted to the lower motorneurons, modification based on proprioceptive feedback or a new command, is at best, limited (Behm & Sale, 1993). As such, in power production, this central voluntary drive has been suggested as a key component in performance (Young & Bilby, 1993). The neural intent to perform an explosive contraction has been suggested as more important, or at least equal to, actually training at high velocities (Behm & Sale, 1993). These authors had volunteers contract both their dorsiflexors with maximum explosive effort over sixteen weeks. One ankle was immobilised while the other was allowed to contract quickly over a

range. Both limbs improved in voluntary and evoked force-time characteristics prompting them to conclude that as long as the attempts to perform the action explosively, and subsequent contractions with high rates of force development, were made, the actual load and movement velocity were not as important for explosive performance.

Another factor in effective central motor drive is its ability to fully activate all available motor units. One method of assessing voluntary muscle activation is by determining if there is a difference in the muscle force by twitch interpolation (TI), in which one or a brief series of electrical stimuli are delivered to the muscle during a voluntary effort contraction (Macaluso & De Vito, 2004). TI techniques have been widely used to test voluntary activation. Belanger & McComas (1981) used this technique while investigating the tibialis anterior and plantarflexor in a group of healthy adults aged 19 to 45 years. They found that while the tibialis anterior appeared to be able to be fully activated, the plantarflexors could not be. However, Dowling, Konert & Ljucovic (1994), suggested that, because of the large signal to noise ratio at higher MVC's, extra forces could be missed and subsequently also included the use of triggered averaging when testing voluntary contraction in the biceps brachii. They concluded that full activation was not achieved in this muscle. Despite this, other authors have subsequently concluded that younger volunteers appear to be capable of complete biceps brachii muscle activation by voluntary command (Allen, Gandevia, & McKenzie, 1995; De Serres & Enoka, 1998).

However, even in the studies proclaiming full activation, it is important to note that this state is not achieved with every trial, with a fully activated state achieved between 25%-50% of the time. As well, as Herbert & Gandevia (1999) note, at forces greater than 90% of maximal voluntary force, interpolated twitch amplitude is insensitive to slight changes in motorneuron pool excitation. Hence, the definition of full activation may be ambiguous. Despite this, different muscles do seem to perform differently which may be due to their synaptic connections and functional roles. For example, the differences in activation between the dorsiflexors and plantarflexors found by Belanger & McComas (1981) may reflect the more postural and reflexive role of the plantarflexors suggesting that descending voluntary drive is not as powerful an input as it is in the tibialis anterior muscles. Additionally, quadriceps seem to have even lower activation levels than either of the ankle muscles (Roos, Rice, Connelly, & Vandervoort, 1999).

While these studies have investigated voluntary activation levels during isometric actions, activation levels have also been assessed by Gandevia, Herbert & Leeper (1998) in biceps brachii during powerful concentric contractions. These authors noted that in powerful dynamic contractions of the elbow flexors, similar levels of voluntary activation were achieved as compared to isometric contractions. In the quadriceps muscle, however, concentric activation levels were found to be lower than levels obtained during isometric contractions by Babault, Pousson, & Ballay (2001). Finally, as these investigations have used single joint movements, caution should be used in applying these findings to a more complex multi-joint movement (Shield & Zhou, 2004).

The significance of supraspinal drive can also be inferred from studies investigating the effect of motor imagery on muscle performance. Yue & Cole (1992) found that when they compared participants who had trained either with actual maximal isometric contractions or with imaginary contractions of the abductor digiti minimi muscle for four weeks to a control group, isometric strength increased by 30% and 22% respectively in the training groups while the controls' increase was 3.7%. Herbert, Dean & Gandevia (1998), however, found no significant differences in elbow flexor strength between an actual practice group and an imagined and control groups. They proposed that the differences between their results and that of Yue & Cole (1992) could be due to the different muscles examined. Since elbow flexors are used more often functionally, there may be less potential gain by mental practice. Ranganathan, Siemionow & Liu (2004) subsequently assessed gains in strength in the abductor digiti minimi and elbow flexors after 12 weeks of a structured mental imagery protocol as compared to an exercise and control group. They found greater gains of strength in the abductor digiti minimi (40%) than the elbow flexors (13.5%), supporting the hypothesis by Herbert et al. (1998). And while the gains made in the imagery group were slightly less than an actual exercise group, they conclude that the significant increases in strength in the imagery group do provide evidence of the ability of an improvement in the descending command to affect muscle performance.

2.2.7.3 Muscle coordination

While not directly related to a muscle's ability to produce force or power, the ability to synergistically coordinate all the involved muscles creates the optimal environment allowing the prime mover to produce actions that are forceful or powerful. The role

postural muscles play in laying a foundation for meaningful performance was highlighted by Wilson, Murphy & Walshe (1996) who found that after eight weeks of training, the improvements in performance were greatest in the tests involving postures that were used during training.

A clear example highlighting the differing roles of muscles for a given task was presented by Schieber (1995) who used percutaneous EMG to study motor unit activity of several adjacent forearm muscles when trained rheus monkeys performed individual flexion and extension movements of the digits and wrist. Using flexor digitorum profundus as an example, he pointed to its role as a synergist with digits 2 and 3 flexion and wrist flexion, its role as antagonist in digit 2 extension as well as its role during digit 5 extension where it acted as a stabiliser by limiting movement of digits 2 and 3 that would have resulted from the attempt to extend digit 5.

The effect of synergistic muscles has also been investigated by Jamison & Caldwell (1993) in humans. Utilizing EMG techniques as well as MRI, these authors showed that the torque produced in elbow flexion by a group of young men changed by as much as 25% depending on the amount of supination or pronation that was maintained and was reflected in the relative involvement of brachioradialis, triceps brachii, and the different heads of biceps brachii (Jamison & Caldwell, 1993). These findings lend support to the notion of dynamic synergism that is related to the task in all the applicable degrees of freedom. The authors go on to speculate that the concept of motorneuron task groups, as proposed by Loeb (1985), may assist in the motor control of such a diverse and redundant neuromuscular system. Loeb suggested that individual muscles need not be represented explicitly in the CNS but are seen as pools of motorneurons that are functionally compatible, although they may be physically located in different anatomically defined muscles. This idea is supported by the findings of Karst & Hasan (1991) who established that upper limb reaching followed some stereotypical muscle activity, regardless of the trajectory of the arm in reaching for the end target. The earlier presented findings of different motor cortical areas contributing to the same movement by Penfield & Boldrey (1937) and Sessle & Wiesendanger (1982) support this coordinated efferent output.

Muscle coordination in the form of antagonist co-contraction has been shown to be prominent in high-velocity or explosive actions (Carpentier, Duchateau, & Hainaut,

1996; Marsden, Obeso, & Rothwell, 1983; Osternig, Hamill, Lander, & Robertson, 1986). Such co-contraction may be particularly detrimental to high force or power generation, decreasing the net agonist torque through both opposing force and reciprocal inhibition (Baratta et al., 1988; Kellis & Batzopoulos, 1997; Sale, 2003; Tyler & Hutton, 1986). There is some evidence from cross-sectional studies that there is reduced co-contraction in strength and power trained subjects as opposed to endurance trained or control participants (Baratta et al., 1988; Osternig, Hamill, Lander, & Robertson, 1986). Furthermore, less co-contraction was noted as a training effect by Carolan & Cafarelli (1992) very early on in an 8 week knee extension training programme. They found that this change contributed to about one-third of the increase in knee extension strength and suggested that it was a clear indication of the neural effects of training, leading to increases in muscle force. However, co-contraction likely has a protective role in maintaining joint stability (Carpentier, Duchateau, & Hainaut, 1996; Tyler & Hutton, 1986), especially with unfamiliar tasks or when finer joint control may be limited. Additionally, it may provide a coordinating or even synergistic role such as the triceps contracting when the biceps supinates the forearm to prevent undesirable elbow flexion (Sale, 2003). It is still unclear whether training reduces the absolute level of co-contraction, the antagonist/agonist ratio, or even if, with agonist hypertrophy, a concomitant increase in antagonist activity would have to occur in order to maintain joint stability.

2.3 Changes with ageing

2.3.1 Introduction

The incidence of stroke increases with age (Feigin, Lawes, Bennett, & Anderson, 2003; Warlow, Sudlow, Dennis, Wardlaw, & Sandercock, 2003). As such, in order to better understand the primary effects of stroke, it is important to first understand those associated with ageing. This section will present changes that result from ageing that affect muscle properties and performance of strength, velocity and power.

Changes in function and mobility are seen with ageing (Fiatarone Singh, 2002; Foldvari et al., 2000; Latham, Bennett, Stretton, & Anderson, 2004; Skelton, Greig, Davies, & Young, 1994; Suzuki, Bean, & Fielding, 2001). Cross-sectional studies have shown that muscular strength peaks between the second and third decade, remains unchanged until the fourth or fifth decade and then declines at a rate of approximately 12%-15% per decade until the eighth decade (Frontera, Hughes, Lutz, & Evans, 1991; Larsson,

Grimby, & Karlsson, 1979; Lindle et al., 1997; Vandervoort & McComas, 1986). By the age of eighty, strength and other muscular measures decline to almost half that of a young adult (Booth, Weeden, & Tseng, 1994; Campbell, McComas, & Petito, 1973; Grimby & Saltin, 1983; Lexell, Taylor, & Sjostrom, 1988). Longitudinal studies would suggest that the decline in strength occurs at an even higher rate than reported in cross-sectional investigations (Bassey & Harries, 1993; Frontera, Hughes et al., 2000). Although a portion of these changes in strength can be attributed to disuse secondary to a more sedentary lifestyle, significant differences in muscle performance and strength are still noted between the young and very active and athletic older person (Enoka, 2002; Metter, Conwit, Tobin, & Fozard, 1997; Narici, Maganaris, Reeves, & Capodaglio, 2003; Ojanen, Rauhala, & Hakkinen, 2007) which suggest that ageing in itself is a factor in muscle and activity performance deficits.

Along with the decreases in strength, declines in muscle power appear to be even more significant with ageing. Bosco & Komi (1980) noted that when older men and women in their seventies were asked to perform a vertical jumping task, they produced 70% to 75% less power than men and women in their twenties. This reduction was higher than the noted 50% decline in average force between the two groups. A shift in the force-velocity curve has subsequently been demonstrated when comparing women in their thirties to women in their seventies (Harries & Bassey, 1990). These authors investigated and plotted torque produced at angular speeds up to 300º/s. And while both groups showed a decline in torque with increasing speed, when standardised against each group's own maximum voluntary contraction (MVC), there was a significant difference in rate of decline between the young and old women. This observation that with age comes a steeper decline of muscle power than muscle strength was also confirmed by Skelton, Greig, Davies & Young (1994) when testing isometric leg strength and handgrip as well as leg extensor power in men and women from 65 to 89 years of age and has been supported by others (Bassey et al., 1992; Lauretani et al., 2003; Metter, Conwit, Tobin, & Fozard, 1997). As such, age brings with it significant force and power deficits. The mechanisms behind these deficits will be discussed in the following sections.

2.3.2 Muscle architecture, size and shape

Reduced muscle size with increasing age has been demonstrated in a number of muscles and through a variety of techniques (Frontera, Hughes et al., 2000; Lexell, Taylor, &

Sjostrom, 1988; Narici, Maganaris, Reeves, & Capodaglio, 2003; Overend, Cunningham, & Paterson, 1992; Young, Stokes, & Crowe, 1985). Most results have pointed to muscle mass that is reduced anywhere from 15%-40% when comparing ages from 20 to 80 years. For instance, Lexell and colleagues (1988) studied cross-sections of autopsied whole vastus lateralis muscle from men between 15 and 83 years of age and found an average reduction in muscle area to be 40%. Ultrasound assessment of quadriceps was used to determine the quadriceps CSA in a young (25 years) and older (75 years) group of men by Young et al. (1985). They reported that the mean CSA of the older men's quadriceps was 25% smaller than that of the young men. Similarly, when computerised tomography (CT) was used to assess quadriceps CSA in young and older men, Overend et al. (1992) found it to be reduced by 23% in the older men. Frontera et al. (2000), also using CT, found that when seven men were first assessed at 65 years and then 12 years later at 77 years, quadriceps CSA decreased by approximately 15% over that time period. Magnetic resonance imaging (MRI) has also been used to evaluate muscle size (Kent-Braun & Alexander, 1999; Trappe et al., 2003). Kent-Braun & Alexander (1999) studied the ankle dorsiflexors of a group of young and older men and women and found a 17% difference in CSA between the groups. These findings were similar to those of Trappe et al. (2003) who also used MRI to determine CSA of the thigh in a young and older group and found a 15% reduction in the older group. Using CT and ultrasonography combined, Narici and colleagues (2003) investigated changes in aged muscle architecture by directly measuring ACSA as well as volume, fascicle length and pennation angle of the gastrocnemius muscle in young and older men. To limit any confounding factors, they were not only matched for height, body mass but also physical activity. All the variables were found to be significantly lower in the older group, confirming earlier studies and suggesting that with ageing comes a loss of sarcomeres not only in parallel, but also in series.

Since muscle force is proportional to CSA (Lieber & Bodine-Fowler, 1993; Roy & Edgerton, 1992), the reduction in muscle size would be expected to be related to a decrease in muscle force. However, as noted in earlier sections, the CSA measures used by investigators are often calculated by measuring ACSA. Since pennation angle and fibre length, used to calculate a more suitable measure of PCSA, have been found to decrease with ageing (Narici, 1999; Narici, Maganaris, Reeves, & Capodaglio, 2003), the validity of using CSA alone to estimate force may be in question. Additionally, increases in relative body fat make CSA a less valid measure as it may overestimate the

contractile capabilities of that muscle (Kent-Braun & Alexander, 1999; Lexell, Taylor, & Sjostrom, 1988; Overend, Cunningham, Kramer, Lefcoe, & Paterson, 1992). For example, Lexell et al. (1988) calculated that 70% of the muscle area was composed of muscle fibres in a young man's vastus lateralis. In contrast, this dropped to 50% in older individuals secondary to fat, connective tissue and blood vessels. Similarly, using CT scanning, Overend et al. (1992) found that the proportion of non-muscle tissue increased by 40% in the older group. And, while MRI showed that fat-free CSA dropped by 12%, actual total leg circumference was found to actually be larger in the older group (Kent-Braun & Alexander, 1999).

Nevertheless, declines in absolute concentric knee extensor muscle force have been noted in older men and women. A 30-40% reduction in force generation capabilities has been shown in cross-sectional and longitudinal isometric testing of various muscle groups by a number of authors (Grimby, 1995; Hakkinen et al., 1998; Klitgaard, Mantoni et al., 1990; Larsson, Grimby, & Karlsson, 1979; Lindle et al., 1997; Vandervoort & McComas, 1986; Young, Stokes, & Crowe, 1985). Isokinetic strength has also been shown to decline with ageing (Frontera, Hughes et al., 2000). These researchers tested knee extensor isokinetic strength at slow and higher speeds of men aged approximately 77 years and when they compared the findings to results obtained from the same men 12 years earlier, found a decline of between 24-30%. It also appears that these reductions are greater when tested at higher velocities (Candow & Chilibeck, 2005; Harries & Bassey, 1990). This was clearly demonstrated by Harries & Bassey (1990) who, when investigating the percentage of torque achieved against normalised MVC of knee extension of younger and older women, found that torque of the older group fell by 48% of MVC at higher angular velocities as compared to 30% for the younger group. Similarly, Candow & Chilibeck (2005) also noted greater declines in knee extensor torque at faster speeds between young and old men when testing with an isokinetic dynamometer (25% and 38% respectively).

However, these reductions in muscle performance appear to differ between the upper and lower limb with preservation noted in the upper limbs (Candow & Chilibeck, 2005; Frontera, Hughes, Lutz, & Evans, 1991). Frontera et al. (1991) found that in a group of 45-78 year old men and women, there was a greater decline of strength in the muscles of the lower extremities than those of the upper extremities, regardless of gender. This preferential strength loss was also noted by Candow & Chilibeck (2005) when they

compared upper and lower body muscle group strength of an older group against a younger one. They found reductions of 15% in elbow flexors as compared to 32% in knee extensors, even when expressed relative to muscle thickness. While the mechanisms behind this finding are not entirely clear, these differences may reflect the decreased mobility and leg use and concomitant increased arm use (ie. as with rising from a chair) with age, preserving the upper limb muscle mass.

In addition to upper body strength, eccentric actions also appear to be preserved in the older population (Klass, Baudry, & Duchateau, 2005; Porter, Vandervoort, & Kramer, 1997). Porter and colleagues (1997) investigated peak torques during concentric and eccentric actions of the ankle musculature of younger and older women. Using isokinetic equipment to test ankle plantarflexion and dorsiflexion at an angular velocity of 30º/s, they noted that while older women's concentric torque values were 74% and 89% of those of the younger women for dorsiflexion and plantarflexion respectively, eccentric values of the same actions were 97% and 100% of the values obtained from the younger women. Klass et al. (2005) tested concentric and eccentric ankle dorsiflexion, of young and old men and women, at several angular velocities and also noted age-related torque deficits of 38.6% during concentric actions but only 6.5% for the eccentric ones.

It is still under debate as to whether these losses in strength are purely caused by a loss of muscle mass and thus maintain a consistent specific tension or muscle quality, calculated as force per unit of muscle, or whether other factors have an influence. For example, Young, Stokes & Crowe (1984) demonstrated that isometric quadriceps weakness of women in their seventies, compared to women in their twenties, may be explained entirely by the smaller CSA of the muscle as measured by ultrasound. Although CSA did decrease with age, the consistency of whole muscle specific tension was also noted by Kent-Braun & Alexander (1999) and Overend et al. (1992). Conversely, Young et al. (1985) found that specific tension of older men was 19% smaller than that of younger men. Others (Hakkinen et al., 1998; Izquierdo, Aguado, Gonzalez, Lopez, & Hakkinen, 1999; Klitgaard, Mantoni et al., 1990; Macaluso et al., 2002; Morse et al., 2004; Vandervoort & McComas, 1986) have also found specific muscle strength to be decreased by as much as 40% in older men and women.

Frontera, Suh et al. (2000) addressed this discrepancy by assessing the specific tension of knee extensor whole muscle as well as that of biopsied FT and ST fibres of the same tested muscle to remove the confounding effects of neural influences, fibre architecture, and intramuscular connective tissue. Using CT to assess for whole muscle cross-sectional area, they noted no significant difference in force per CSA of whole muscle across ages. Absolute force of the isolated fibres was then assessed by mounting them on an experimental apparatus, attaching them to a force transducer and a lever system and exposing the fibres to a contracting solution. Fibre CSA was also measured. From these two variables, the authors calculated muscle fibre specific tension and found that it did differ significantly between age groups suggesting that the intrinsic properties of the muscle fibres are affected by age.

The number of sarcomeres in series has been shown to decrease by 10% in the gastrocnemius muscle when comparing a young group (35 years) to an older group (75 years) (Narici, Maganaris, Reeves, & Capodaglio, 2003) and confirms the animal findings of Hooper (1981). As such, maximum shortening velocity would also be expected to be reduced. This is supported by the work of Ochala, Lambertz, Pousson, Goubel & Van Hoecke (2004) who found that when testing ankle plantarflexors isometrically and isokinetically, men in their late sixties demonstrated a contraction velocity that was 17% slower than men in their early twenties. Additionally, Hakkinen & Hakkinen (1991) provided force-time curves to demonstrate that women in their thirties took 30% shorter time to reach maximum isometric muscle force levels with their knee extensors as compared to older participants aged in their seventies.

Therefore, older muscles produce less force and these forces decrease at higher velocities. As a result, an older person will only be able to generate the same absolute force of his or her younger counterpart by using a higher percentage of his or her maximum force and at a lower contraction velocity. As power is the product of both force and shortening velocity, these findings provide evidence that with ageing, power declines more than strength.

2.3.3 Non-contractile components

While age-related declines in force and power production can be attributed to muscle size and architecture changes, alterations in muscle-tendon unit mechanical properties have also been proposed as contributing to movement deficits. Age-dependent changes

to tendon mechanical and biochemical parameters had been investigated through in vitro studies of human and animal tissue. Noyes & Grood (1976) investigated human cadaver and amputation specimens from diseased and previously healthy men and women with ages ranging from 16 to 86 years. When grouped into a younger group (16-26 years) and an older group (48-86 years), these authors noted 30-60% decreases in strength, stiffness and elastic modulus as well as a reduced failure stress in the older group tissue. This is in agreement with results from studies using rat tail tendons. Vogel (1991) found that tensile strength of the tendon began to decline at twelve months (a third of their three year life span) and continued to decline up until the oldest age that was tested (thirty months). Along with the decreased tensile strength, this author also found that collagen followed a similar pattern of decline whereas elastin showed a non-significant increase. Similarly, Blevins, Hecker, Bigler, Boland & Hayes (1994) tested human patellar tendon specimens from donors aged 17 to 54 for tensile strength, elastic modulus and failure mode. Even in this relatively young cohort, age related changes were noted with a 25% decrease in elastic modulus over the age range examined.

More recently, real-time ultrasound protocols, despite their limitations, have allowed more concise examination of superficial human tendons under in vivo conditions (Narici & Maganaris, 2006). However, the studies of human calf muscle-tendon units have yielded some inconsistent results. For example, Kubo et al. (2003) studied the vastus lateralis of women aged from 20-79 years. Using ultrasonography to assess fascicle displacement relative to a superficially attached marker, they concluded that with age comes decreased maximal strain and decreased stiffness. Decreased stiffness was also found by Maganaris (2001) who studied gastrocnemius tendons in 6 young (20 years) and 6 older (70 years) men and found that older tendons were 15% more compliant than younger ones and that as force increased, tendons became more compliant. Similarly, Onambele, Narici & Maganaris (2006) assessed the displacement of the medial gastrocnemius distal myotendinous junction relative to a fixed skin marker and also reported decreased stiffness and decreased young's modulus in the older group of volunteers. However, Kubo et al. (2007) recently reported that in comparison to a 20 year old group, maximal strain of the Achilles tendon began to decrease in the 30 year group with further progression with increasing age and suggested that this change results in a decline in tendon extensibility. Confusion in the literature may be due to differing terminology and definitions. For example, strain, stiffness, extensibility,

strength, compliance, young's modulus have all been used without clear definitions and may have slightly different descriptions.

Furthermore, some confusion continues with regard to determining the mechanisms behind some of the above findings. For example, tendon diameter can influence its compliance and Strocchi et al. (1991) suggested that a decreased tendon diameter would result in a more compliant structure. Results from Nakagawa, Majima & Nagashima (1994) confirmed this view when they performed an in vitro study of older rabbit Achilles tendons and found that mean area and collagen fibril diameter decreased, as did the number of thick fibrils. Magnusson, Beyer, Abrahamsen, Aagaard, Neergaard & Kjaer (2003), on the other hand, found that the Achilles tendon CSA was actually larger in the tendons of older women as compared to younger women (79 years and 29 years, respectively). Furthermore, support for the decreased extensibility reported could result from increases in connective tissue and collagen cross-linking as proposed by Hamlin & Kohn (1971). However, this conclusion was based on testing on the very specialised human diaphragm muscle and as such, may be not applicable to limb muscles referred to in the other studies. Hence, the specific mechanisms and mechanical properties of ageing collagenous tissue remain an area of debate.

Nevertheless as the stretch shorten cycle relies in part on appropriate levels of stiffness created by the tendon at higher forces (Huijing, 1992; Komi, 1984), the level of stiffness in the tendon will influence the rate of force development behaviour of a tendon and will reflect the overall effect of the changes. This has been shown to be reversible as a result of resistance training. For instance, Reeves, Maganaris & Narici (2003) found that, after 14 weeks of high intensity progressive resistance training of the knee extensors by participants aged 65-81years, absolute tendon stiffness and stiffness relative to the tendon's dimension (Young's modulus), both increased significantly as did rate of torque development.

Some studies have examined the intramuscular connective tissue (IMCT) of epimysium and perimysium. IMCT of the extensor digitorum longus (EDL) and soleus muscle of older rats was compared to that of younger rats by Alnaqeeb, Al Zaid & Goldspink (1984). In these samples, the senile muscles were found to have a greater amount of both endomysium and perimysium with a resultant increased stiffness as evidenced by a steeper length-passive tension curve. These findings were related to an increased

collagen concentration. Similarly, in the work by Brown & Kautz (1999) who also used samples taken from aged rats, the relative stiffness per muscle mass increased, suggesting that the remaining tissue was stiffer per gram of tissue. In humans, Ochala et al. (2004) found higher in vivo musculotendinous stiffness in the plantarflexors of older (61-74 years) men compared to younger (19-24 years) men. They suggested that this may be as a result of the preferential FT atrophy and resultant increased relative area of ST fibres which have been found to be less compliant than FT fibres (Petit, Filippi, Exonet-Denand, Hunt, & Laporte, 1990; Toursel, Stevens, & Mounier, 1999).

The changes in both muscle and tendon stiffness will have implications on function and performance. An older muscle that is stiffer and has lost sarcomeres will need to adopt a new length-tension relationship (Macaluso & De Vito, 2004; Reeves, Maganaris, & Narici, 2003). A more compliant tendon will mean that for a concentric contraction, sarcomeres will need to shorten even more. As such, this will result in a less than optimal length for myofilament overlap which will lead to lower force production (Narici & Maganaris, 2006). This is in addition to the force deficits that occur from muscle and fibre atrophy. As well, contraction velocity will be affected by the increased tendon compliance since it will take longer for forces to be transmitted from muscle to bones. Therefore, changes to the non-contractile components may have notable effects on muscle performance. However, there is a need in the current research to standardise the nomenclature and terminology. Furthermore, the contrasting findings of in vitro and in vivo research make overall conclusions difficult to make.

2.3.4 Motor units

In respect to ageing, the repercussions on force, velocity and power production are due to not only reduced muscle mass and changes in non-contractile structures but also alterations in the motor units, comprising both muscle fibres and the accompanying motorneuron.

2.3.4.1 Muscle fibres

A reduction in muscle fibre number has been noted to begin as early as the twenty-fifth year with the decline accelerating after 50 years (Lexell, Henriksson-Larsen, Winblad, & Sjostrom, 1983). By 70 years of age, Lexell and colleagues (1983) estimated a 24% reduction in the mean number of vastus lateralis muscle fibres in men. While there is agreement on this age-related loss, there have been conflicting views on whether there is

actually a selective fibre-type reduction with ageing. Earlier histochemical analysis, on men aged between 20 and 65 years, suggested a drop in FT fibre numbers leading to an increased proportion of ST fibres in the vastus lateralis muscle (Larsson, Grimby, & Karlsson, 1979; Larsson, Sjodin, & Karlsson, 1978). Similar shifts were noted in the tibialis anterior of 70 year old volunteers (Jakobsson, Borg, Edstrom, & Grimby, 1988). However, in a similar cohort, findings from Grimby (1984) suggested no change in relative fibre composition in the knee extensors. The methodologies used for these earlier studies may account for this discrepancy since biopsy data was used and may not represent the human muscle as a whole. Subsequently, studies of whole muscle were performed by Lexell et al. (1988). Using whole muscle cross-sections taken from autopsy cases with ages ranging from 15-83yrs, these researchers classified each fibre within the vastus lateralis muscle. They concluded that there was a 39% reduction in total number of fibres affecting both main fibre types equally.

Recently, more advanced classification based on myosin heavy chain (MHC) isoform expression (Reiser, Moss, Biulian, & Greaser, 1985), has found that a much higher percentage of muscle fibres from older subjects may co-express MHC isoforms suggesting a transformation of fibre types that may not be reflected by histochemical analysis (Andersen, Terzis, & Kryger, 1999; Klitgaard, Zhou et al., 1990). For example, Klitgaard, Zhou et al. (1990) found that fibres had an increased co-expression of MHCI and IIA as well as MHCIIA and IIX in samples taken from 69 year olds and noted a general trend toward slow-type isoforms in the fibres studied. The trend towards co-expression was even greater in the work of Andersen, Terzis & Kryger (1999), who studied fibres from the vastus lateralis muscle of 88 year olds and noted that the majority of fibres co-expressed two or even three MHC isoforms, with the most common co-expressing MHCI and IIA. The amount of fibres expressing the fastest isoform (IIX) was very low. These authors suggested that there was not so much a change in the ratio between FT and ST fibres but more of a blurring of the borders between these fibre types and pointed to denervation and reinnervation, resulting in fibres of one type receiving conflicting neural input and eventually giving rise to a mixed expression of the MHC isoform. Therefore, in older muscle, the FT type fibres take on some ST expression, resulting in a ‘mixed’ muscle that will affect cross-bridge cycling rate and also power production (Faulkner, Claflin, & McCully, 1986).

Even with similar numbers of fibre types, many studies have demonstrated a preferential atrophy of FT fibres as compared to ST fibres (Grimby, Aniansson, Zetterberg, & Saltin, 1984; Larsson, Grimby, & Karlsson, 1979; Lexell & Taylor, 1991; Lexell, Taylor, & Sjostrom, 1988; Tomonaga, 1977). Lexell & Taylor (1991) noted that FT fibre area decreased by 35% with little change in ST fibre area when examining muscle cross-sections from cadavers aged 19-85 years. However, Frontera, Suh et al. (2000) and Trappe et al. (2003) found non-significant differences in FT fibre CSA when comparing age groups. This may be due to the differing ages of the young sample groups as Lexell & Taylor's group mean age was 27 years whereas Frontera and colleagues used a group with a mean age of 37 years. Of note is that when comparing results, the former study used whole muscle cross sections as compared to biopsy samples used in the latter studies. As Lexell & Taylor (1991) noted, there is variability within a muscle and intramuscular distribution of fibre types and diameters changes with age. As such, one would question whether a biopsy would be able to capture and reflect these changes. Nevertheless, with a reduction in size of even just the FT fibres, the relative contribution of FT fibres to force and power generation would be considerably more affected in older muscle.

Single muscle fibre contractile function has been tested in an attempt to determine if intrinsic fibre changes are behind the reduced contraction capabilities. The previously mentioned study by Frontera, Suh et al. (2000) found that when they normalised the force produced by vastus lateralis FT and ST muscle fibres to cross sectional area, specific tension values in the older group were reduced by 30% as compared to the younger group. This confirmed the similar findings of Larsson et al. (1997) who also noted that specific tension of the younger FT fibre types were 30% higher than those in the older control group.

Additionally, maximum unloaded shortening velocity (Vo) of muscle fibres has been examined. Younger muscle fibres appear to be able to contract at significantly higher speeds then older ones (Larsson, Li, & Frontera, 1997). Krivickas et al. (2006), using a similar methodology to that of Larsson and colleagues (1997), also noted that while the absolute values differed, Vo was generally reduced with age and the relative differences in Vo across fibre types was similar. However, there were differences in Vo between the older women and men groups suggesting that not only age, but also gender can affect muscle fibre properties in ageing. In addition, changes to the behaviour and

contractility of a given myosin isoform in aged muscle have been suggested by Hook, Sriramoju & Larsson (2001) who noted that actin sliding speed on MHC I is significantly slower in old age. These studies provide evidence that with age come changes to the mechanical and intrinsic properties of muscle fibres that, combined with decreased numbers and sizes of fibres, contribute to declines in force, velocity and power production.

However, recently, these findings have been challenged by Trappe et al. (2003) who reported no significant differences in specific tension, shortening velocity or peak power when comparing normalised muscle fibre function in 25 year old to 79 year old fibres of vastus lateralis. These authors found that while there were differences in whole muscle absolute power as well as relative power normalised to CSA between groups when tested isokinetically, this was not the case with individual muscle fibres. Muscle fibre segments were isolated and secured to troughs and were alternately transferred between relaxing and activating solutions. Force, length and velocity testing and subsequent power calculations showed that despite five to six fold differences between FT and ST fibres, when normalised to fibre CSA, there was no evidence of differences due to age. There were also no differences between groups in specific tension or shortening velocity. The authors suggest that despite comparable procedures to the previous studies, their considerable effort to control for physical activity levels and a larger sample size may account for the differences and point to loss of muscle mass (specifically loss of power generation from FT fibres) as the critical component in decreased muscle function.

2.3.4.2 Motorneurons

In addition to changes in muscle fibres, reductions in motor unit numbers also occur with ageing. Studies have consistently found reduced numbers and sizes of actual motorneurons as well as reduced numbers of functioning ones either by direct observation (Tomlinson & Irving, 1977) or by electrophysiological techniques (Campbell, McComas, & Petito, 1973). Campbell and colleagues (1973), in an attempt to establish the number of actual functioning motor units, compared maximally evoked muscle action potentials to average motor unit action potentials in the foot muscles in participants with ages ranging from 3 to 96 years. They noted that at 60 years of age began a dramatic decline in the number of motor units able to be recruited. These findings have been confirmed more recently in thenar muscles (Doherty, Vandervoort,

& Brown, 1993; Wang, De Pasqua, & Delwaide, 1999). However, less clear is whether or not there is a selective loss of the more rapidly conducting and larger motor axons with evidence being provided for (Wang, De Pasqua, & Delwaide, 1999) and against (Doherty & Brown, 1997) this notion. While calculating numbers of motor units in a group with ages ranging from 21 to 91 years, Wang et al. (1999) measured conduction velocity and reported a significant decrease in motor unit conduction velocity with increasing age and suggested that this is due to preferential age-related effects on the largest and fastest conducting motor units. However, the older participants in the work by Doherty & Brown (1997) appeared to preserve a full range of contraction speeds and while the motor units in the older group shifted toward a greater proportion exhibiting prolonged contraction and relaxation times, there was no evidence of actual preferential loss of FT motor units.

Along with a loss of numbers of motor units, changes to motor unit size and distribution have also been noted. Campbell and colleagues (1973), in their work estimating motor unit numbers, noticed that the amplitudes of the remaining motor units were actually greater than the young controls. This larger amplitude may be an indirect indication of an increased number of muscle fibres per motor unit and likely reflects the axonal branching of surviving motorneurons to re-innervate the orphaned muscle fibres and has been confirmed more recently in the tibialis anterior, vastus lateralis and thenar muscles (de Konning et al., 1988; Doherty & Brown, 1997). This process of denervation of FT fibres and subsequent reinnervation by axonal sprouting from adjacent ST motor units reflects a proposed motor unit remodelling which is also supported by the increased co-expression of MHC isoforms in aged muscle fibres reported earlier (Andersen, Terzis, & Kryger, 1999; Klitgaard, Zhou et al., 1990). Additionally, the shift from a random spatial distribution of a motor unit's fibres within a muscle to more of a clustered or grouped organisation has been shown by Lexell & Dowham (1991). These authors examined whole muscle cross sections of vastus lateralis from male cadavers aged between 15 and 83 years. They concluded that this finding further supported the evidence suggesting a continuous denervation and re-innervation process with increasing age, most likely due to an accelerating loss of functional motor units.

2.3.5 Activation

2.3.5.1 Motor unit coordination

Older muscle contraction is slower as demonstrated by prolonged twitch contraction and relaxation times (Doherty & Brown, 1997; Roos, Rice, Connelly, & Vandervoort, 1999; Vandervoort & McComas, 1986). For instance, Vandervoort & McComas (1986) tested twitch times of the plantarflexors and dorsiflexors of 111 men and women aged between 20 and 100 years. With increasing age, the contraction and relaxation times became significantly prolonged in both muscle groups. Doherty & Brown (1997), as presented earlier, also found a noticeable shift toward a greater proportion of motor units exhibiting prolonged contraction and relaxation times. Roos et al. (1999) in their work on quadriceps contractile properties of young (26 years) and old (80 years) men reported a leftward shift in the force-frequency curve reflecting how aged quadriceps muscle reached tetanus at lower frequencies of stimulation than younger muscles. This may reflect the fibre-type changes that occur and point to a functional adaptation since a prolonged contractile response enables fused contractions to occur at lower excitation frequencies and has been demonstrated in the adductor pollicis, vastus medialis and tibialis anterior (Connelly, Rice, Roos, & Vandervoort, 1999; Roos, Rice, Connelly, & Vandervoort, 1999). Possible mechanisms of this prolongation of contraction time may be found in the work by Klitgaard, Mantoni et al. (1990), who noted 35% reductions in the volume and concentrations of calcium-ATPase protein of the sarcoplasmic reticulum of FT units in older participants. This finding was confirmed by Hunter et al. (1999) who assessed electrically evoked contractile properties of quadriceps muscles of younger (25 years) and older (72 years) women and subsequently analysed biopsied fibres for maximal rates of sarcoplasmic reticulum calcium uptake as well as Ca-ATPase activity. They also reported reduced uptake and activity and significant associations between uptake and relaxation times and rates. Other factors such as alterations in the excitation-contraction coupling due to alterations in receptors in the sarcoplasmic reticulum membrane could lead to failure of the transduction of depolarisation by the sarcolemma. This would result in a decreased mechanical response and would also affect contractile speed (Payne & Delbono, 2004). Additionally, as reported in the preceding section, the reduced speed of cross-bridge cycling has also been proposed as a contributing factor (Hook, Sriramoju, & Larsson, 2001). Slow contraction speeds, would, therefore, compound the effects of an increased tendon compliance resulting in a longer time needed to generate force and stretch the tendon, with the consequence that less force will be produced over a given period of

time. All of these factors could contribute to the decline in the speed of rapid contractions with a subsequent reduction in the power output of that muscle.

2.3.5.2 Supraspinal drive

In addition to the musculoskeletal and neuromuscular changes in ageing that lead to declines in motor function, age-related changes have also been observed in the supraspinal centres. Cortical cell death in the primary motor cortex, while widely assumed to be concomitant with ageing and motor function, has not been substantiated (Haug & Eggers, 1991). Instead, it has been proposed that it is the reduction in white matter integrity that affects connectivity. A study by Eisen, Entezari-Taher & Stewart (1999) used electrophysiological testing to study discharges of single motor units from the extensor digitorum communis during randomly applied cortical magnetic stimulation in a group of volunteers aged 24 to 83 years. They found that by the age of 50 years, about 35% of the cortico-motorneurons were lost or non-functioning. This reduction is greater than has been reported in more peripheral spinal motor units (Campbell, McComas, & Petito, 1973; Tomonaga, 1977) which suggests that cortico-motorneurons commence their demise earlier than do spinal motor units (Eisen, Entezari-Taher, & Stewart, 1999). As such, compensatory strategies in an attempt to overcome these changes have been proposed (Madden, Whiting, Huettel, White, & Provenzale, 2004; Ward, 2006). In a number of studies using varying equipment and methodologies, older participants, in an attempt to maintain performance, demonstrate a more diffuse and greater overall cortical activation as well as more pronounced bilateral activation of sensorimotor regions while performing similar tasks to a younger group (Mattay et al., 2002; Sailer, Dichgans, & Gerloff, 2000).

Cortical inhibition and excitability in older adults has been studied using short and prolonged interstimulus intervals in paired-pulse TMS. However, these studies have demonstrated conflicting changes in motor cortical behaviour. In a middle-aged group (51 years), Peinemann, Lehner, Conrad & Siebner (2001) found reduced inhibitory mechanisms when comparing motor evoked potentials (MEP) of the first dorsal interosseus (FDI) muscle to that of a younger group. They suggested that this decreased inhibition indicated increased recruitment as a compensatory mechanism used to overcome the reduction in active neurons.

In contrast, Kossev, Schrader, Dauper, Dengler & Rollnik (2002), using a similar age group and paired-pulse paradigm but a different coil and muscle group (wrist muscles), found increased inhibitory mechanisms. As the authors point out, the differences in findings may in part be explained by the differences in waveform of magnetic stimuli since different stimulation types influence intracortical inhibition and facilitation (Corthout, Barker, & Cowey, 2001; Kammer, Beck, Thielscher, Laubis-Herrmann, & Topka, 2001). As such, comparisons between studies are difficult. Despite this, Kossev et al. (2002) noted that the older group's motor evoked potential (MEP) size was greater than the young group, even though the MEP threshold was the same, suggesting that a larger cortical area was involved in the activity, which is in agreement with the finding that with age comes more compensatory and diffuse cortical use (Ward, 2006).

Single-pulsed TMS has also been used to assess the excitability and integrity of the corticospinal projections to the first dorsal interosseous muscle of the hand by Pitcher, Ogston & Miles (2003). While these authors also found that MEP thresholds did not differ significantly among their group with ages ranging from 18 to 55 years, the older participants required increased stimulation intensities and time to reach 50% of the maximum MEP. These authors suggested that while the excitability of spinal motorneurons is relatively unchanged with age, as seen by similar MEP thresholds, alterations in firing behaviour such as cortical neuronal synchronisation or a loss of cortico-motorneurons may have explained the slower rate of MEP increases. Therefore, these findings would suggest that ageing brings a decline in cortico-motorneuron activity with a subsequent attempt at compensatory adjustment.

An alteration in the descending drive from supraspinal centres to the motorneurons has been purported to be one of the factors resulting in reduced motor unit discharge rates (Kamen, Sison, Du, & Patten, 1995), reduced specific tension (Frontera, Suh et al., 2000; Larsson, Li, & Frontera, 1997) and a possible decreased ability to voluntarily activate a muscle (Morse et al., 2004; Yue, Ranganathan, Siemionow, Liu, & Sahgal, 1999). However, the results from studies using twitch interpolation technique (TI) to assess voluntary drive in older adults are conflicting. Some indicate that old and young adults differ in their activation ability during an MVC (Morse et al., 2004; Yue, Ranganathan, Siemionow, Liu, & Sahgal, 1999) whereas others report that older adult voluntary activation does not differ from that in young adults (De Serres & Enoka, 1998; Kent-Braun & Alexander, 1999; Phillips, Bruce, Newton, & Woledge, 1992;

Roos, Rice, Connelly, & Vandervoort, 1999; Vandervoort & McComas, 1986). Perhaps the differences in results stem from differing definitions and methodologies since some studies allowed for practice and repeated attempts to achieve full activation (De Serres & Enoka, 1998; Vandervoort & McComas, 1986) while others didn't (Yue, Ranganathan, Siemionow, Liu, & Sahgal, 1999). Furthermore, some have used mean activation level (Yue, Ranganathan, Siemionow, Liu, & Sahgal, 1999) as compared to the highest level attained (De Serres & Enoka, 1998; Kent-Braun & Alexander, 1999; Phillips, Bruce, Newton, & Woledge, 1992).

Jakobi & Rice (2002) reflected on these discrepancies when they found that although both younger and older men could reach similar activation levels, the younger men reached peak levels more often and without need for practice. They also suggested that it was lack of practice, rather than impaired capacity of the nervous system to maximally activate muscles, that resulted in observed reductions in voluntary activation. As such, there are likely to be subtle differences in voluntary activation that have yet to be fully explored. As well, TI protocol may also have an effect since DeSerres & Enoka (2002) found differing results when using either tetanic stimulation or a series of twitches. And finally, as with younger people, differences may exist between muscles since Morse et al. (2004) concluded that the reduced torque seen in plantarflexors in older men is likely significantly affected by reduced voluntary activation while Kent-Braun & Alexander (1999) concluded no such influence in the dorsiflexors. When taken together, the information seems to suggest that while there may be differences in supraspinal descending control, they are likely not significant enough themselves to fully explain the weakness observed with ageing.

2.3.5.3 Muscle coordination

Voluntary activation of not only the agonist muscle but also the antagonist muscle seems to vary with ageing. While it is acknowledged that antagonist co-contraction occurs in high-velocity or explosive actions in the younger adult (Carpentier, Duchateau, & Hainaut, 1996; Marsden, Obeso, & Rothwell, 1983; Osternig, Hamill, Lander, & Robertson, 1986), this phenomenon appears to be even more apparent in the older adult (Hakkinen et al., 1998; Izquierdo, Aguado, Gonzalez, Lopez, & Hakkinen, 1999; Macaluso et al., 2002). Forty-two middle aged and elderly men and women underwent a series of assessments in a cross-sectional study by Hakkinen et al. (1998). Along with age-related decreases in absolute force, CSA and specific tension, these

authors also found that when performing explosive and powerful movements such as leg extension actions and squat jumps, the older group (70 years) demonstrated significantly higher integrated electromyographic activity (IEMG) in the antagonistic biceps femoris than that recorded for the middle-aged group (40 years). While age-related changes to the tissue (ie greater adipose tissue) may have increased the risk of cross-talk between EMG electrodes and therefore led to inaccurate assessment of co-contraction, this finding has been confirmed in both older men and women in other studies (Izquierdo, Aguado, Gonzalez, Lopez, & Hakkinen, 1999; Macaluso et al., 2002). Muscle activation of the antagonistic biceps femoris was again significantly greater in 65 year old men than 40 year old men during both isometric and dynamic activities (Izquierdo, Aguado, Gonzalez, Lopez, & Hakkinen, 1999). Macaluso et al. (2002) also used surface EMG to record vastus lateralis and bicep femoris activity of young and older women (23 year and 70 year respectively) during isometric torque testing. Co-contraction of the biceps femoris during knee extension was 42% higher in the older then younger women compared to the younger groups. Consequently, in the aged muscle with lower contraction force and speed capabilities, increased co-contraction may allow for joint stability without needing to recruit additional motor units even though this would also limit the full potential of the agonist muscle, particularly when speed is required.

2.3.6 Summary of changes with ageing

The mechanisms behind age-related changes in muscle performance are multi-factorial. Non-neural considerations such as reductions in muscle size and alterations in composition result in reduced muscle force. This is combined with a transformation in contractility of fibres that results in slower contractions. Additionally, variations to the musculotendinous units result in much less efficient force and speed of movements. Consequently, deficits in muscle power performance are seen. Along with these findings, neural changes such as a slower contraction along with a voluntary drive that may either be reduced, require more practice or more cortical excitation and be more variable appear with increasing age. These factors, along with any associated disuse will impact on the ability to activate muscles forcefully and quickly.

2.4 Changes after stroke

2.4.1 Introduction

Stroke, by its nature and pathophysiology, results in an inability or difficulty to voluntarily recruit skeletal motor units to generate force or movement leading to the observed muscle weakness that is commonly seen after this type of neurological lesion (Bourbonnais & Vanden Noven, 1989; Gracies, 2005; Ng & Shepherd, 2000). Since the incidence of stroke increases with increasing age, people who have suffered a stroke will likely already have had age-related changes in muscle performance and function which are now compounded. This section will present the consequences of stroke on muscle characteristics and on the performance of force, velocity and power.

Historically, impaired motor function after stroke was attributed more to spasticity, or muscle overactivity, than to weakness. This belief shaped a whole rehabilitation approach aimed at minimising or avoiding this overactivity (Bobath, 1985, 1990; Davies, 1985). However, more recent evidence suggests that muscle weakness is more of a significant limiting factor (Bohannon, 1986; Canning, Ada, Adams, & O'Dwyer, 2004; Davies, Mayston, & Newham, 1996; Harris, Polkey, Bath, & Moxham, 2001; Nadeau, Arsenault, Gravel, & Bourbonnais, 1999; Newham & Hsiao, 2001). As such, researchers and clinicians have begun to acknowledge and address this issue supported by evidence that strength training or effortful activities do improve strength with little to no exacerbation of spasticity (Ada, Dorsch, & Canning, 2006; D. Brown & Kautz, 1999; Sharp & Brouwer, 1997). Nevertheless, while not the primary cause of movement dysfunction, spasticity can still result in adaptive, structural and functional alterations in muscle that must also be acknowledged and considered (Gracies, 2005; Lieber, Steinmann, Barash, & Chambers, 2004).

People who have suffered a stroke are often more sedentary (Esmonde, McGinley, & Wittwer, 1997; Ng & Shepherd, 2000) and have additional co-morbidities (Roth & Green, 1996). As such, impairments that are seen after stroke may very well have their origin from not only the neurological lesion but also from factors such as disuse and disease. This should be kept in mind when considering the short and long term impact of stroke on muscle performance and mobility.

Additionally, while the observed deficits in strength and muscle control after stroke are typically contralateral to the affected cerebral hemisphere, there is evidence that the

ipsilesional side is also affected (Andrews & Bohannon, 2000; Harris, Polkey, Bath, & Moxham, 2001; Newham & Hsiao, 2001). Hence, care must be taken when reviewing literature that considers the 'unaffected' side appropriate for normal comparisons.

Muscle weakness after stroke includes more than insufficiencies in force production. Further deficits are seen in the rate of force development, and contraction velocity (Canning, Ada, & O'Dwyer, 1999; Clark, Condliffe, & Patten, 2006; McCrea, Eng, & Hodgson, 2003; Pohl et al., 2002). The mechanisms behind these deficits will be discussed in the following sections.

2.4.2 Muscle architecture, size and shape

Muscle CSA and composition of the involved and uninvolved legs after stroke have been assessed using computerised tomography (CT) by a number of authors (Metiko, Sato, Satoh, Okumura, & Iwamoto, 2003; Ryan, Dobrovolny, Smith, Silver, & Macko, 2002; Sunnerhagen, Svantesson, Lonn, Krotkiewski, & Grimby, 1999; Tsuji et al., 2004). Sixteen mildly affected hemiplegic volunteers demonstrated only insignificant differences in subcutaneous fat and muscle between legs in the study by Sunnerhagen, Svantesson, Lonn, Krotkiewski, & Grimby (1999). However, Ryan et al. (2002) found that midthigh muscle area was 20% lower in the paretic leg than the non-affected leg with a concomitant increase in intramuscular fat relative to muscle area in the affected limb. Whole muscle CT, performed by Metoki et al. (2003) and Tsuji et al. (2004), on the legs of a comparable group of stroke participants similarly yielded differences of 20%-23% in muscle CSA between legs. However, these studies using CT grossly evaluated areas involving a number of muscle groups, including antagonistic muscles and as such, cannot provide information about the changes in specific muscles. Ploutz-Snyder, Clark, Logan & Turk (2006), using magnetic resonance imaging (MRI) for its higher spatial resolution, found that while triceps brachii CSA showed similar deficits of 25% between arms in a very small sample of hemiplegics, biceps brachii CSA presented with considerably less differences (3%-4%) between sides, suggesting that atrophy changes may be not only lesion but also muscle specific.

Recently, ultrasonography has been used to assess pennation angle and fascicle length of the affected and unaffected brachialis in a small group of hemiplegics (Li, Tong, & Hu, 2007). These authors found increased pennation angles along with decreased fascicle lengths in the affected arm. The reductions in muscle size and increased

pennation angles as found by all these studies would consequently affect force production in the hemiplegic muscle (Lieber & Bodine-Fowler, 1993; Wickiewicz, Roy, Powell, & Edgerton, 1983). As well, since all of these studies used comparisons with only the lesser affected side, deficits would likely be even more significant when compared to those unaffected by stroke.

Despite some discrepancies in reports regarding the amount of muscle atrophy, there is a large and convincing amount of evidence confirming a loss of muscle strength after stroke. Researchers have noted isometric strength deficits of typically between 30-60% and even as high as 70% between leg muscles in people affected by stroke (Adams, Gandevia, & Skuse, 1990; Andrews & Bohannon, 2000). This is highlighted by the study from Adams and colleagues (1990) who measured eight muscle groups on both legs of moderately to severely affected hemiplegics. They found that the strength of the affected side was between 32% and 73% of the unaffected limb, with the distal muscles showing larger deficits than the proximal ones. Deficits of between 47% and 65% in isometric strength were also reported by Andrews & Bohannon (2000) who assessed several upper and lower limb muscle groups in a hemiparetic group. Similar findings have been reported by a number of other authors (Clark, Condliffe, & Patten, 2006; Newham & Hsiao, 2001; Ploutz-Snyder, Clark, Logan, & Turk, 2006). More recently, muscle training trials have used pre-training isoinertial assessments such as the one-repetition maximum (1-RM) to assess for muscle strength (Ouellette et al., 2004; Weiss, Suzuki, Bean, & Fielding, 2000). These studies have yielded deficits in knee extension 1-RM of 30-60% between sides, confirming the significant effect stroke has on muscle force capabilities.

One ultrasonographic study, as previously presented, has recently reported a decrease in hemiplegic fascicle length (Li, Tong, & Hu, 2007). Work on young healthy muscles has found that muscles with longer fibres will have faster shortening velocity (Edgerton, Roy, Gregor, & Rugg, 1986; Lieber & Bodine-Fowler, 1993). This documented loss of fascicle length in hemiparetic muscle confirms earlier activity-related investigations that have found deficits in contraction velocity after stroke (Bohannon, 1987; Canning, Ada, & O'Dwyer, 1999; Clark, Condliffe, & Patten, 2006; Lum, Patten, Kothari, & Yap, 2006). Using isokinetic testing, Bohannon (1987) found that while hemiplegic knee extension torque decreased with increasing knee extension velocities, the rate at which it decreased was not different to the stronger side. However, he did not make

comparisons to a control group unaffected by stroke. Clark et al. (2006), on the other hand, did use controls when assessing for velocity-dependent concentric torque and found significant velocity-dependent torque impairments in the hemiplegic group when compared to those without stroke. Therefore, the rate of torque decline with increasing speed appears to be greater in hemiplegic limbs indicating that post-stroke hemiplegia deviates from the normal force-velocity curve.

Rate of torque development has also been tested by isometric time to peak testing. Bohannon & Walsh (1992) used this approach to test knee extensor movement in acute strokes. When measuring time to peak and to 90% peak torque, they found the involved leg took about 1.5 times longer to reach the torque levels as compared to the contralateral limb. These findings were confirmed in the upper limb by Canning, Ada & O'Dwyer (1999) who studied rate of torque development and the time taken to achieve 90% of peak torque in hemiparetic and control subjects. They found that acute stroke subjects had time to 90% peak torque times that were even longer when compared to the controls (two to three times longer). Even in chronic stroke, McCrea, Eng & Hodgson (2003) found that time to develop 70% peak toque was impaired by 61% in the more affected arm and by 22% in the less affected arm of chronic hemiparetic participants, compared to a control arm. Taken together, these results point to significant time-dependent impairments with regards to force generation after stroke.

The deficits seen in force generating capabilities, combined with the added shortfall in speed of contraction would likely result in power deficits even greater than those of absolute strength. This has been recently confirmed by some authors (Clark, Condliffe, & Patten, 2006; LeBrasseur, Sayers, Ouellette, & Fielding, 2006). Peak knee extension power, when measured via a pneumatic resistance machine was found to be 45% lower in the involved leg as compared to the uninvolved leg in a group of thirty-one community dwelling stroke volunteers. 1-RM strength testing of the same sample yielded a 32% deficit between legs (LeBrasseur, Sayers, Ouellette, & Fielding, 2006). It also appears that power production after stroke is even more velocity dependant than those unaffected by stroke. Isokinetic knee extension testing at speeds ranging from 30º to 240º/s was carried out on a stroke and control group by Clark et al. (2006). While mean power was already 45% lower in the stroke group at speeds of 30º/s and reflects the findings of LeBrasseur and colleagues (2006), that discrepancy rose to 67% at faster speeds. As well, a plateauing of power output in the stroke group was noted at speeds

exceeding 90º/s, suggesting that people with stroke are limited to performing powerful contractions at slower velocities, as compared to those unaffected by stroke.

2.4.3 Non-contractile components

Efficient hemiparetic voluntary movement, including force and power production also appears to be affected by increased stiffness. Much attention has been focused on the factors involved in this stiffness. Spasticity, defined by Lance (1980) as a velocity-dependent resistance to stretch, suggests that the increase in stiffness is through a dynamic component involving a reflexive element. Exaggerated resistance to lengthening, termed hypertonia, on the other hand, involves changes in the non-neural passive mechanical properties of the musculotendinous unit in addition to the neural reflex changes seen with spasticity (Lieber, Steinmann, Barash, & Chambers, 2004; Patten, Lexell, & Brown, 2004). Numerous mechanisms are thought to contribute to the increased resistance to stretch seen in hemiparetic muscle with three main factors proposed: 1) passive muscle stiffness due to non-contractile elements; 2) neurally-mediated reflex stiffness from the balance between segmental monosynaptic reflex and descending inhibition and; 3) active or elastic muscle stiffness resulting from the state of crossbridge attachment (Foran, Steinmann, Barash, Chambers, & Lieber, 2005; Rydahl & Brouwer, 2004).

In an attempt to determine how these mechanisms contribute to the increased stiffness seen in spastic muscles, Sinkjaer & Magnussen (1994) compared the total stiffness of the ankle plantarflexors from both ankles of a mostly chronic hemiparetic group and one ankle of a control group. A four degree plantarflexion stretch was applied on top of either a voluntarily or electrically induced and maintained plantarflexion contraction at various levels of maximum contraction. The differences between the responses obtained provided estimates of both the reflex and non-reflex components of stiffness. They found that while spastic muscles showed significantly increased total stiffness compared to the control muscles, reflex stiffness of both legs of the hemiparetic group was not significantly different to the controls, nor was the intrinsic or active stiffness. The significant increase in stiffness in both limbs of the stroke group was due to greater passive mechanical stiffness. This was also found by Rydahl & Brower (2004) using a similar methodology on a group of chronic community dwelling stroke survivors. They noted that total ankle stiffness was 40% higher in the stroke group compared to the control group which was attributed to a significant increase in passive stiffness, and

insignificant increase in intrinsic stiffness. Reflex-mediated stiffness was similar for both groups.

However, some authors have suggested that the methodology employed by Sinkjaer & Magnussen (1994) and Rydhal & Brouwer (2004) risks underestimating the reflex contribution because of the changes in the intrinsic properties of passive tissues associated with eliminating the reflex response (Galiana, Fung, & Kearney, 2005). Therefore, these authors used a non-linear, parallel-cascade, system identification method to identify the neural and non-neural components of ankle stiffness in a group of chronic strokes and age and gender matched controls. They found that the majority (two-thirds) of hemiparetic volunteers showed no significant difference in reflex contribution, as noted by the other researchers. However, in one-third of those tested, an increase in reflex gain was found to contribute to the total stiffness seen.

Taken together, these findings would suggest that passive stiffness is a major contributor to hypertonia in spastic muscle. This passive mechanical stiffness has been attributed to increased collagen proliferation (Tabary, Tabary, Tardieu, Tardieu, & Goldspink, 1972; Williams & Goldspink, 1984; Witzmann, Kim, & Fitts, 1982), accumulation of intramuscular collagen and thickened perimysium and endomysium leading to fibrosis within the spastic muscle (Booth, Cortina-Borja, & Theologis, 2001; Jozsa et al., 1990), shortening of resting sarcomere lengths and increased modulus of elasticity (Friden & Lieber, 2003) and possibly changes to the titin isoform (Lieber, Steinmann, Barash, & Chambers, 2004). As well, the lack of significant contribution from neurally mediated reflex stiffness is supported by the finding that 1a facilitation has been shown to be similar in hemiplegics and controls during soleus H-reflex studies (Faist, Mazevet, Dietz, & Pierot-Deseilligny, 1994) reinforcing the suggestion that for the majority of hemiplegics, spasticity may not be the major contributor to ongoing hypertonia.

It is important to note, however, that none of the participants in these preceding studies had any significantly restricted range of motion at the ankle joints that were examined. The mechanisms contributing to hypertonia are interdependent and are further affected by adaptations in response to altered patterns of activity following stroke and, as such, can change over time (Ng & Shepherd, 2000). For example, prolonged mobility restriction could lead to contracture at the ankle joint which, in turn, could alter the

point in the range of motion at which the stretch reflex is initiated (Rydahl & Brouwer, 2004), changing the dynamics of the mechanisms behind hypertonia. Nevertheless, while these changes may reflect a compensatory adjustment so that hemiparetic muscles can be stiffer without requiring as much neural activation, the additional stiffness would also come at the expense of rapid muscle contraction, affecting power production.

2.4.4 Motor units

2.4.4.1 Muscle fibres

There is agreement that a reduction in CSA in muscle fibres occurs after stroke. However, some authors have suggested a predominance of FT fibre atrophy (Dattola et al., 1993; Dietz, Ketelsen, Berger, & Quintern, 1986; Edstrom, 1970; Hachisuka, Umezu, & Ogata, 1997; Scelsi, Lotta, Lommi, Poggi, & Marchetti, 1984) while others have reported no such selectivity (Frontera, Grimby, & Larsson, 1997; Jakobsson, Edstrom, Grimby, & Thornell, 1991). Early work by Edstrom (1970) investigating groups affected by either hemiplegia or Parkinsonism demonstrated that fibres with a high concentration of myofibrillar ATPase showed more atrophy than those with lower concentrations and suggested that these results reflect the selective disuse of high-threshold FT motor units secondary to paresis. Scelsi et al. (1984) studied anterior tibialis muscle biopsy specimens that were taken from a group of people with recent stroke (1-17 months). This sample was stratified into four groups reflecting the time since stroke. A progressive reduction in fibre CSA was found in both fibre types with a greater loss noted in the FT fibres. This alteration over time since stroke was also noted by Dietz et al. (1986), who also used histochemical and morphological examinations of bilateral gastrocnemius muscles from four hemiparetic patients. They too found muscle fibre atrophy that seemed to target the FT fibres. Similarly, Dattola and colleagues (1993), using biopsy specimens of the gastrocnemius muscle taken from a group of acute to recent hemiparetics, also showed that FT fibres demonstrated a high atrophy factor. These findings were more recently confirmed by Hachisuka et al. (1997). These authors studied vastus lateralis muscles of both subacute and chronic hemiplegics and controls. Their results showed, once again, that there was preferential atrophy of the FT fibre types and cited disuse as a major factor involved with this phenomenon. However, Jakobsson and colleagues (1991) investigated biopsy samples taken from tibialis anterior muscles of a group of chronic stroke volunteers as well as from young and older controls (whose combined mean age matched that of the hemiplegics). They found no prominent atrophy as had been reported by the previously presented studies.

Frontera et al. (1997) also concluded that while finding evidence of atrophy in ST and FT fibres in the paretic tibialis anterior muscle as compared to both the contralateral limb as well as controls, they did not observe any selective FT fibre atrophy.

There is also conflicting evidence regarding how stroke affects muscle fibre numbers and phenotype. In the study by Scelsi and colleagues (1984), the authors reported a progressive predominance of ST fibres over the time since stroke. Dietz et al. (1986) reported similar findings of an increased predominance of ST fibres as time since stroke increased. These results were later confirmed by Dattola et al. (1993). Their analysis revealed an increased proportion of ST fibres with a concomitant decrease of FT. In contrast to these findings, Jakobsson et al. (1991) found that the hemiplegic samples showed a significantly increased FT proportion, especially type IIB, as compared to the controls. Frontera and colleagues (1997) also tested for several muscle fibre properties on a similar chronic stroke group. They studied tibialis anterior biopsies taken from both sides of one 50 year old woman with chronic stroke and one age and gender matched control and reported that their hemiparetic sample demonstrated an increased prevalence of FT fibres compared to the normative data from the work of Jakobsson et al. (1991). When compared to their own control, however, these authors found little difference in FT proportion. This is not altogether surprising, given such a small sample size.

All authors suggest that the fibre type changes may reflect plasticity in response to changes in activity as well as altered motor unit firing patterns and rates that have been observed after stroke (Frontera, Grimby, & Larsson, 1997; Gemperline, Allen, Walk, & Rymer, 1995; Rosenfalck & Andreassen, 1980). What they are not clear about, however, are the details of this altered activity level and firing behaviour and how these factors relate to their findings. For example, activity may be slow and sustained or may involve high force and power levels of short duration. Sustained exposure to either of these situations would yield different fibre adaptations. Additionally, the results of lowered motor unit firing rates may result in less general submaximal muscle activity leading to less ST fibre use or it may result an inability to reach and activate high threshold units affecting the FT fibres more. Therefore, definite conclusions from the above studies are difficult.

Despite the conflicting evidence of fibre type changes based on histochemical studies, there appears to be more agreement around changes in muscle molecular phenotype based on MHC isoforms. Authors have reported a greater percentage of transformed isoforms with a shift towards fibres containing faster MHC in both lower and upper limb muscles (De Deyne, Hafer-Macko, Ivey, Ryan, & Macko, 2004; Frontera, Grimby, & Larsson, 1997; Jakobsson, Edstrom, Grimby, & Thornell, 1991; Ponten & Stal, 2007). Jackobsson and colleagues (1991) commented that 10% of their hemiparetic tibialis anterior samples demonstrated expression of both slow and fast isoforms of MHC. Frontera et al. (1997) also reported a higher content of fast MHC isoforms in their paretic samples. More recently, De Deyne et al. (2004) demonstrated a significant increase in the proportion of fast MHC in hemiplegic vastus lateralis muscle as compared to the non-affected side and suggested that this reflected a lack of chronic, regular submaximal activity. Similarly, in a cohort of young adults with spastic paresis including stroke, Ponten & Stal (2007) confirmed these findings in muscles of the upper limb. Biceps brachii samples were taken from spastic patients as well as age-matched cadavers. Fibres expressing fast MHC IIx were significantly greater while percentages of fibres expressing MHCI and MHC IIa respectively were less. These authors agree with De Deyne and colleagues (2004) and conclude that voluntary action and activity have a significant impact on the expression of MHC isoforms.

The functional implications of these fibre changes were also addressed by Frontera and colleagues (1997). In their study previously discussed, contractile properties of the same muscle fibres were investigated. The reduced fibre CSA would be expected to lead to reduced absolute force production. However, these authors found that the specific tension of the paretic muscle fibres was lower than that of the control fibres suggesting that the weakness observed may not only be due to fibre atrophy and subsequent reduction in number of cross-bridges, but also to a lower force generation per cross-bridge. These authors also found that maximal fibre contraction velocity (Vo) of the fibres expressing slow MHC was reduced by 15% as compared to the controls. However, they did not report on Vo of the FT fibres and did not comment on how the noted increase in fast MHC isoform content in paretic muscle impacted on contraction velocity. Regardless of the mechanisms, however, isolated muscle fibres reflect the demonstrated compromises in force and velocity production that are found at the whole muscle level.

2.4.4.2 Motorneurons

The deficits in motor performance after stroke can be attributed not only to changes in the structure and performance of muscle and its fibres as discussed, but, due to the cortical damage that is involved, also to the significantly affected capacity of the nervous system to activate the muscle through motor units. Reductions in the actual number of motor units that activate muscle fibres have been found to occur after stroke (McComas, Sica, Upton, & Aguilera, 1973). These authors estimated the number of functioning motor units by comparing electrically generated twitch potentials of single motor units to those of the entire extensor digitorum brevis (EDB) muscle in the hand of middle-aged hemiplegics of varying time since stroke. They noted that with increased duration (between 2 and 6 months after stroke), the number of motor units able to be elicited was halved. This reduction in motor unit function has been attributed to a loss of descending and trophic corticospinal input resulting in trans-synaptic changes and degeneration in the motorneurons (Dietz, Ketelsen, Berger, & Quintern, 1986; McComas, Sica, Upton, & Aguilera, 1973).

More recently, Lukacs (2005) addressed these claims by investigating motor unit changes on the hemiparetic side of volunteers with time since stroke between one month and one year. He found that in those with acute strokes, M wave amplitude was decreased, confirming previous authors' claims that the stroke's upper motor neuron lesion results in loss of synaptic input and a lack of activation of the lower motor neurons which become functionally inactive or undergo trans-synaptic degeneration. However, along with stroke recovery comes evidence of increased motor unit potentials of existing units as demonstrated by the recovery of M wave amplitudes as stroke chronicity increased (Lukacs, 2005) which further supports the idea of collateral sprouting and reinnervation (Campbell, McComas, & Petito, 1973; Dattola et al., 1993; Lukacs, 2005). Therefore, stroke recovery demonstrates significant compensatory mechanisms in response to motor unit loss.

2.4.5 Activation

2.4.5.1 Motor unit coordination

Compounded by the structural changes in motor units after stroke, there is evidence that muscle performance is further compromised by alterations in motor unit activation. Hemiparetic motor unit contraction and relaxation times have been shown to be prolonged. In the study by McComas and colleagues (1973), the surviving motor units

of the paretic extensor digitorum brevis demonstrated significantly slower twitch times when compared to the contralateral muscle. In an attempt to better explain these changes in relation to the different motor unit types, Young & Mayer (1982) classified and compared isometric contractions of single motor units in the first dorsal interosseous muscle in groups with short or long term hemiplegia and controls. They too found increased twitch contraction times as previously reported but noted that it was the FT units that demonstrated this significant change.

Motor unit firing behaviour during voluntary contractions would also contribute to the altered muscle function. Rosenfalck & Andreassen (1980) studied motor unit action potentials from single motor units recorded from the tibialis anterior muscle in a sample consisting of participants with several forms of upper motor lesions. As participants were performing voluntary dorsiflexion, these authors found that the spastic motor units fired at lower frequencies (5-13 Hz) as compared to normal controls (6-20 Hz). In addition, they also reported that this firing frequency fluctuated significantly resulting in participants being unable to maintain the isometric contraction at a constant torque. These findings were confirmed in the upper limb by Gemperline, Allen, Walk & Rymer (1995) who investigated motor unit discharge and recruitment rates in the involved and uninvolved biceps brachii muscle of hemiparetic participants. Although there was variability among the participants, they found a significant reduction in motor unit firing rates, as well as a failure to modulate, or increase firing rate with increasing force production. They also found that hemiparetic motor units were systematically recruited earlier as force levels increased that resulted in a narrowing of force levels at which they did fire. This was echoed by Frontera et al. (1997) who as well as finding reduced firing rates, noted that even at higher levels of muscle activity (25-50% maximum), hemiparetic tibialis anterior motor units did not increase their firing rate substantially, remaining under 15 Hz while the control units reached frequencies of 25 and 30 Hz.

Changes in neuromodulators as a consequence of stroke can shift the synaptic current-frequency relationship so that a cell can maintain a prolonged tonic firing rate following a brief period of excitation (Kiehn & Eken, 1998; McCrea, Eng, & Hodgson, 2003). This behaviour known as plateau potential results in a prolonged active state and would influence the regulation of motor unit firing rates. The lower firing rates seen in the preceding studies would suggest that muscles after stroke increase force by recruitment as opposed to rate coding which lead to weakness and increased voluntary effort and

subsequently fatigue. This, coupled with the loss of and difficulty in recruiting the remaining high-threshold FT motor units, would contribute to the difficulty hemiparetic muscles demonstrate in not only isometric force production but also larger deficits in torque production at higher velocities or powerful actions.

2.4.5.2 Supraspinal drive

By its nature, stroke affects the ability of the cortex and other higher centres to voluntarily produce descending commands needed to recruit and activate skeletal motor units to generate movement. However, these deficits improve in the weeks and months following a stroke (Twitchell, 1951). While recovery can vary widely, there is increasing understanding of brain plasticity and the mechanisms of neuronal reorganisation that involve the recruitment of pathways that are functionally similar but anatomically distinct from the damaged ones (Rossini, Calautti, Pauri, & Baron, 2003). Factors such as un-masking of existing but functionally silent cortico-cortical connections, or modulation of synaptic efficacy through long-term potentiation that involves down-regulation of local inhibitory circuits as well as stimulation of excitatory glutamate receptors, and the formation of new synapses have all been proposed as contributing to brain reorganisation after stroke (Cicinelli et al., 2003; Rossini, Calautti, Pauri, & Baron, 2003). This process is believed to occur in non-damaged tissue with evidence of increased activation of peri-lesional cortical areas and points to a re-adaptation of existing synapses that can adopt the function of the damaged neurons (Chollet et al., 1991; Seitz & Freund, 1997). Several neuro-imaging techniques have been used to assess for plastic cortical reorganisation after stroke and suggest that while initially there is excessive intensity and extent of activation in the cortical motor networks bilaterally, with effective recovery, a gradual normalisation and balancing of hemispheric activity occurs (Chollet et al., 1991; Cicinelli et al., 2003; Kim et al., 2004; Rossini, Calautti, Pauri, & Baron, 2003).

Evidence of initial and subsequent cortical reorganisation after stroke that comes from imaging studies must be considered along with functional studies to help understand how deficits in muscle performance are affected by altered supraspinal drive after stroke. While muscle atrophy is an acknowledged contributor to the muscle weakness seen in chronic stroke, supraspinal factors also affect the ability to generate force. Harris, Polkey, Bath & Moxham (2001) used twitch interpolation techniques in the first week after stroke and compared the knee extensors of the lesser affected leg of newly

hemiparetic subjects to a control leg. They reported significant differences in voluntary activation levels between the two (59% and 93% respectively) providing evidence that weakness and failure to fully activate a muscle is a primary consequence of stroke even before the effects of disuse compound it. Their results also show that the supraspinal effects of stroke result in deficits seen bilaterally. Newham & Hsiao (2001) tested sub-acute strokes upon admission to rehabilitation and then repeatedly over the next 6 months to understand the changes in deficits in central voluntary activation on primary muscle weakness. Assessments were performed bilaterally on hemiparetic patients and controls who performed maximal voluntary contractions of the knee extensors and flexors. They found significant differences in voluntary activation between the paretic limb (60%), the non-paretic limb (75%) and the control limb (93%) that persisted over the 6 month period. Similar deficits in hemiparetic activation levels were found in the upper limb by Riley & Bilodeau (2002) who investigated voluntary and stimulated maximum torque in the elbow flexors of 10 stroke participants. They found that maximal voluntary activation of the biceps brachii on the involved side was 66% as compared to 89% on the uninvolved side. This reduction in supraspinal input and voluntary activation has been suggested to be the main mechanism behind the reported loss of number of functioning motor units that occur with time after stroke as well as the reduction in discharge rates of voluntarily driven motor units in the paretic muscles (Gemperline, Allen, Walk, & Rymer, 1995; Gracies, 2005).

2.4.5.3 Muscle coordination

Despite improved knowledge about decreased agonist activation, there is continued belief that any apparent weakness is a consequence of excessive antagonist restraint. Some co-contraction is normal in tasks requiring accuracy or stability (Neilsen, Sinkjaer, Toft, & Kagamihara, 1994; Smith, 1981), in explosive actions (Carpentier, Duchateau, & Hainaut, 1996; Marsden, Obeso, & Rothwell, 1983; Osternig, Hamill, Lander, & Robertson, 1986) and appears to be increased in older people (Hakkinen et al., 1998; Macaluso et al., 2002). However, it is thought to have a much bigger impact on movement after an upper motor neuron lesion. Despite this perception, the evidence behind it is less than clear. Some authors have provided evidence of increased antagonistic recruitment with voluntary activation of an agonist muscle (Dewald, Pope, Given, Buchanan, & Rymer, 1995; El-Abd, Ibrahim, & Dietz, 1993; Kamper & Rymer, 2001; Levin, Selles, Verheul, & Meijer, 2000) while others have demonstrated little

additional co-contraction (Clark, Condliffe, & Patten, 2006; Davies, Mayston, & Newham, 1996; Newham & Hsiao, 2001).

For example, Kamper & Rymer (2001) measured EMG activity of flexor and extensor muscles of the fingers of eleven hemiparetic hands and compared this with a control group. They asked participants to flex and extend the fingers under isometric, isokinetic and no load conditions. In all conditions, they found significantly increased activity in the antagonistic muscle as compared to the controls. In fact, attempts by the hemiparetic subjects to create an extension torque typically resulted in the production of an inappropriate net flexion torque instead. However, they also found that while flexor EMG activity increased by 22% in isokinetic extension, there was a 48% drop in EMG recorded extensor activity, prompting them to agree that failure to voluntary activate the prime mover also contributes to the reduced net torque.

On the other hand, no such co-contraction was found by Clark and colleagues (2006). Knee extensor torque and EMG activity and concomitant hamstring activity was tested while hemiparetics and controls performed a knee extension action at differing velocities. At increased speeds, they noted that while the controls showed increased activity in the agonist, the hemiparetic agonist muscles did not show comparable increases. As well, it was actually the control group (who was age matched) who showed more antagonist co-contraction at higher velocities prompting these researchers to claim that the primary mechanism in movement dysfunction after stroke is impaired agonist activation. While apparently conflicting, these results may reflect the inter-muscle variation with regards to function and innervation. The studies supporting the presence of excessive co-contraction have all been performed on muscles of the upper limb. Whereas those proclaiming no inappropriate antagonist behaviour were performed on muscles of the lower limb. Upper limb muscles are inherently used for precise manipulation and as such, require fine-scale coordination and cortical control (Kuypers, 1978; Palmer & Ashby, 1992). This functional requirement may be reflected in increased cortical and segmental inhibitory reflexes to ensure accuracy. Lower limb muscle, however, have no such role and therefore, likely require less modulation or cortical control. Without supraspinal control, these differences between limbs may become even more apparent.

2.4.6 Summary of changes after stroke

The mechanisms behind stroke-related changes in muscle performance are numerous and complex. Ischemic or hemorrhagic insults to the parietal cortex lead to neuronal cell death or damage. This type of lesion results in an inability to excite descending corticospinal pathways in the same manner as would happen in a normally functioning system. It leads to a loss of motor units, subsequently affecting voluntary muscle activation. Resultant losses of force and contraction velocity capabilities are seen. Additionally, decreases in motor unit discharge rates and subsequent alterations in discharge patterns further result in inefficient muscle stimulation, particularly at high forces or speeds. Muscles affected by stroke are also stiffer and, while affected by reflex mediated stiffness, are significantly influenced by passive mechanical factors further resulting in contraction inefficiency. Abnormal muscle coordination may also occur and is likely largely influenced by the location and role of the muscle itself as well as the degree of voluntary activation. Consequently, muscle force and contraction velocity after stroke are significantly affected and lead to deficits in powerful performance.

2.5 Optimising power production and training parameters

2.5.1 Introduction

While power has traditionally been assessed and performed by athletes, power training or explosive resistance training has increasingly been incorporated in programmes for older adults. Increasing awareness of the role of muscle power after stroke would also suggest that rehabilitation aimed at improving physical performance might consider muscle power as part of a rehabilitation programme. However, there are inconsistencies in the recommended optimal load or training intensity to use in order to produce maximal power output. As well, while power is favourably linked to function, whether power training in the aged and stroke populations results in direct gains in mobility has not yet been shown. This section will review the different approaches and parameters in training for power in the younger and older adult, and discuss recent findings in relation to power assessment after stroke.

2.5.2 Optimising power in the younger adult

Training at the load that maximises power output appears to be recommended to improve muscle power production (Kaneko, Fuchimoto, Toji, & Suei, 1983; Wilson, Newton, Murphy, & Humphries, 1993). However, there continues to be debate

concerning the optimal load at which this maximum muscle power is generated with different authors advocating light, intermediate and heavy loads. Kaneko et al. (1983) investigated changes to elbow flexion performance after different groups of young men trained at a variety of loads ranging from no load to maximal isometric load. Maximum power output pre-training was generated at 30% of isometric force (Po) and increased most when training at 30% of Po but did have carry-over when tested at other loads. Peak mechanical power was also achieved, in isolated human muscle fibres, at one third of maximum shortening velocity and one third of maximum contraction force (Faulkner, Claflin, & McCully, 1986), confirming the findings of Kaneko and colleagues (1983). Similarly, Moss, Refsnes, Abildgaard, Nicolaysen & Jensen (1997) noted that when training elbow flexors in young active men, pre-training power peaked at 35% of 1-RM and that training at this load had the largest carryover in power generation. Furthermore, lighter loads (15-30% of 1-RM) have also been noted to optimise power output in concentric only bench press in young well-trained men (Newton et al., 1997). In the lower limb, Wilson, Newton, Murphy & Humphries (1993) compared a variety of training techniques including traditional progressive resistance training with higher loads, non-loaded plyometric depth jump training and dynamic jump squat training with lighter loads and higher speeds on three groups of young trained participants. Although the study design did not control for the differences in training technique or specificity or account for differences in training volume, the authors noted that leg power and jump performance were maximised when a 30% of maximum load, lifted at high speeds was utilised. Similarly, when assessing pre-training jump squat power with a Smith machine over a force plate and with a position transducer attached to the bar, McBride, Triplett-McBride, Davie & Newton (2002) found that power was highest when young athletic males performed the jump squats at loads of 30% of 1-RM as opposed to heavier loads of 55% and 80% of 1-RM. And despite slight differences in load calculations and using countermovement with their squat jumps, Cormie, McCaully, Triplett & McBride (2007) also recently reported that in a group of young male athletes performing jump squats on a force plate with the bar attached to two linear position transducers, peak power was achieved at loads equivalent to 30% of 1-RM.

On the other hand, others have reported power peaking when working against intermediate loads. Cronin, McNair & Marshall (2001) investigated how upper limb peak power was achieved across varying load levels. Several bench press type movements were performed by young untrained males using a modified Smith press

machine with a linear transducer attached. These authors found that while power was generally greater in actions involving projections (ballistic actions), peak power output was maximised at 50-60% of 1-RM, across all the bench press type actions. Similarly, when testing bench press-type throws in high level professional and semi-professional athletes on a plyometric power system, Baker, Nance & Moore (2001a) also found power was optimised at a range of 46%-62% of 1-RM, peaking at 55% of 1-RM. This research group also tested power in jump squats in groups of well-trained athletes that were either professional or semi-professional or college-aged. Again, a plyometric power system was used. They found that optimal load was equivalent to loads of 47%-63% of 1-RM, again peaking at 55-59% of 1-RM (Baker, Nance, & Moore, 2001b). While these results reflect the findings of young fit men, Thomas, Fiatarone & Fielding (1996) investigated leg power in young untrained women. These authors used a computer interfaced pneumatic double leg press machine to assess for peak power generated across a variety of loads. They also found maximum power occurred at intermediate to high loads in the range of 56-78% of 1-RM with it peaking at 68% of 1-RM.

Schmidtbleicher & Haralambie (1981) have recommended that even higher loads are needed for muscle to achieve maximum speed of movement. In an upper limb training study involving young active men, they compared 8 weeks of progressive resistance training at high loads (90-100% of maximum voluntary strength) to training at low loads (30% of maximal strength) to a control group. They found that while both training groups did show significant changes in maximal strength as compared to the control group, the high load training group demonstrated significant decreases in time to perform a push off movement without load whereas the low load group did not. The authors interpreted these findings to suggest that high load training resulted in better recruitment of FT fibres that would lead to greater power production (Faulkner, Claflin, & McCully, 1986). However, no mention was made regarding the speed at which each group attempted to perform the training. As well, from the data, both training groups did show significant increases in superficially measured minimal muscle twitch contraction time and half relaxation times, which make the results and their claims a little less clear.

The differences in the optimal load findings from above may reflect a number of factors. Firstly, the different muscle groups tested may influence the ultimate optimal

load. While findings from earlier upper limb work by Kaneko et al. (1983), Moss et al. (1997) and Newton et al. (1997) have suggested lower loads, other authors have found both higher optimal loads for upper limbs (Baker, Nance, & Moore, 2001a, 2001b; Cronin, McNair, & Marshall, 2001) and lower loads for lower limbs (Cormie, McCaulley, Triplett, & McBride, 2007; McBride, Triplett-McBride, Davie, & Newton, 2002; Wilson, Newton, Murphy, & Humphries, 1993). Between-subject variability among the studies would make for tricky comparisons. Seigel, Gilders, Staron & Hagerman (2002) used a Smith rack set up and compared peak power across differing loads in both an upper limb bench press and a lower limb squat performed by the same group of young men with weight training experience. They demonstrated that maximal muscle power in the upper limb was achieved at lower loads (40-60% of 1-RM) than the lower limb (50-70% of 1-RM) suggesting that smaller muscle groups use less force in power production.

Secondly, the different actions used in testing may explain the differences in optimal load findings. More traditional weight lifting actions, as well as projection-involving ballistic actions, have both been used to test power. Traditional weight lifting actions, even if performed quickly, require deceleration of the bar or load towards the end of the range of motion whereas ballistic actions involve a projection (of a load, of oneself) at the end of the movement and do not have a deceleration phase. As such, acceleration continues throughout the movement resulting in higher force and power generation at lighter loads. Consequently, peak power can be achieved at lower force or load levels (Cormie, McCaulley, Triplett, & McBride, 2007; Kawamori & Haff, 2004; Wilson, Newton, Murphy, & Humphries, 1993). Greater power output was reported by Cronin and colleagues (2001) in their study comparing traditional bench press to ballistic bench press movements. More recently, in the lower limb, these differing actions have been found by Cormie, McCaulley, Triplett,& McBride (2007) to produce significantly different optimal loads. These authors found that ballistic actions such as jump squats produced peak power at lower loads (equivalent to 30% of 1-RM) whereas the more traditional squats in which the feet did not leave the ground produced peak power at intermediate-high loads (equivalent to 70% of 1-RM).

Thirdly, the data collection systems vary among studies. As Dugan, Doyle, Humphries, Hasson & Newton (2004) and Cormie et al. (2007) argue, this may have implications in optimal load determination. Power has been calculated using only displacement,

accelerometry data or ground reaction force data or by a combination of displacement and ground reaction force data. When only limited direct data is collected, this data then needs to be further manipulated which can lead to accumulating error thereby reducing validity and reliability of the final power values (Dugan, Doyle, Humphries, Hasson, & Newton, 2004). Dugan and colleagues (2004) compared power output during jump squats over varying loads using three different data collection systems. They found differences in not only absolute power values between systems but also differences in optimal load, illustrating the influence the different systems would have on the results.

Fourthly, differences in optimal load may also exist depending on the strength of the study volunteers and their experience in performing powerful manoeuvres and hence their neuromuscular adaptations. For example, when Stone et al. (2003) investigated peak power during weighted jumps in young men, they noted that those with greater strength produced peak power at higher percentages of 1-RM as compared to those with less strength or experience (40% of 1-RM vs. 10% of 1-RM). These results would suggest that weaker subjects may produce power, in the absence of large CSA and great force production, through increasing velocity. As Stone and colleagues (2003) have suggested, the results would also imply that improving maximum strength could result in the improvement of peak power and at higher percentages of 1-RM. These findings seem to confirm other studies such as those performed on high level rugby league athletes by Baker et al. (2001a, 2001b) who also noted intermediate optimal loads in their trained and strong participants. The findings of Thomas et al. (1996) who reported that untrained (and generally weaker) young women actually produced peak power at relatively high loads (68% of 1-RM) do not seem to follow this argument until more attention is directed to the testing action and methodology. These authors tested power via traditional actions involving deceleration which, as noted above, may explain the relatively higher optimal percentage of 1-RM needed to produce power than has been found by those testing ballistically.

2.5.3 Optimising power in the older adult

The significant age-related changes reported in power and mobility (Bassey et al., 1992; Bean et al., 2003; Foldvari et al., 2000; Lauretani et al., 2003; Skelton, Greig, Davies, & Young, 1994; Suzuki, Bean, & Fielding, 2001) have led authors to investigate how muscle power can be maximised in the older adult. Despite numerous trials

investigating the effects of various training parameters, there have been limited studies that have expressly investigated optimal loads that maximise power output in this population.

Perhaps one reason for this finding is the testing methods and equipment used in this population. Jumping tasks have been commonly used in the younger population to assess leg power. In the older adult, concerns about safety, less skill base and balance as well as concerns regarding the presence of a floor effect have led to other means of testing lower limb power (Bassey & Short, 1990; Rantanen & Avela, 1997). A seated unilateral leg press rig, often referred to as the Nottingham rig, is one such device, incorporating a leg extension action on a lever to propel a flywheel from which angular velocity is obtained and subsequently, power is calculated (Bassey & Short, 1990). This system has been shown to be reliable and valid, with high correlations with jumping tasks (Bassey & Short, 1990; Bassey, Tay, & West, 1990). However, the nature of the equipment set up means that there is no means by which to know at which load, or percentage of 1-RM an individual's muscle power is being measured or maximised (Bean et al., 2003). More recently, a computer interfaced pneumatic seated leg press machine (Keiser) has been utilised. Thomas et al. (1996) compared this pneumatic device to the Nottingham rig and reported moderate correlation when power was expressed by kilogram of lean body mass. However, this pneumatic leg press machine showed only moderated to low correlations with vertical jumping or usual or maximal gait speed. Despite these issues, this apparatus does enable relative load to be measured and has been used in a variety of trials. Also of note is that these machines test non-projection actions. As such, when compared to those testing ballistic actions, results may yield different optimal loads.

Considerations related to training for power have been an emerging consideration in muscle training in the older adult. Some of the earlier studies investigating muscle power as an outcome were conducted by Skelton, Young, Greig & Malbut (1995) and Jozsi, Campbell, Joseph, Davey & Evans (1999). Healthy women aged 75 years and older underwent a 12 week training programme targeting muscles that are involved in activity performance (Skelton, Young, Greig, & Malbut, 1995). Pre- and post-training assessments were made on isometric knee extensor strength and leg extensor power. Power was assessed using a modified Nottingham rig. Strength improved by 27% and although there was an increase in power, it did not reach statistical significance. Most

of the activities tested also did not show significant improvements. A 12 week progressive resistance training programme on upper and lower body muscles was also performed by groups of younger (25 years) and older (60 years) volunteers (Jozsi, Campbell, Joseph, Davey, & Evans, 1999). Pre- and post-training assessments were performed on knee extension 1-RM and double leg press power using a Keiser pneumatic leg press machine. Results did demonstrate a power training effect in both men and women. Like many others, however, these authors applied the accepted training parameters for progressive resistance training and set relatively high resistance loads (80% of 1-RM) to bring about changes in muscle strength and size. And despite some increases in power, the authors conceded that the intensity was geared for strength and may not have been optimal for power production. Optimal load for ballistic or explosive power, as has been noted in the young and athletic literature, may well differ than that for force. It seems that this step of identifying optimal load in the process of optimising power has not been extensively addressed in the literature.

Earles, Judge & Gunnarsson (2001) examined power and explosive resistance training in healthy and high functioning adults with a mean age of 77 years. The power group followed a high velocity training programme using moderate to high loads and the control group carried out a walking programme. These authors collected power data measured unilaterally with a pneumatic leg press machine. At baseline, both groups demonstrated that power was maximised at loads of 30-40% of body mass, which, when re-calculated to their 1-RM, is closer to 45% of 1-RM. Interestingly, post training, the power group's maximum power shifted to 50% of body mass (or 74% 1-RM) whereas the control group did not. Peak power was reported to have increased by 22% in the power group and, when assessed at higher loads, leg press power improved by 150% while the control group demonstrated no significant change. These results would suggest that the older adult can improve power and would also agree with the theory that people with increased strength can contribute more force to power production resulting in higher power at higher loads (Stone et al., 2003).

Using the pneumatic leg press machine, Fielding et al. (2002) compared traditional high-load, low-velocity training to explosive high-load, high-velocity training in older women with self-reported activity limitations. Peak power was assessed via a bilateral leg press action across different relative intensities (40%, 50%, 60%, 70%, 75%, 80%, 85%, 90%) of the 1-RM and peak power at baseline was reported to occur at 75% of 1-

RM. However, it should be noted that the order of intensities never varied and always began with the lightest load. With only one attempt at each load, a training effect could have occurred over the 4 or 5 attempts. Both groups trained at high loads (70% of 1-RM) and both demonstrated significant gains in strength. However, only the explosive group trained at high speeds and was the only group to demonstrate significant change in muscle power. Therefore, even in a mobility-limited older population, explosive training can result in significantly greater power as compared to standard slower paced high-load training (97% vs. 45%). The authors noted that although the training intensity was set at 70% of 1-RM, peak leg press power values improved significantly at all loads tested, suggesting a carry over effect as also noted by Kaneko et al. (1983) and Moss et al. (1997).

Unlike the previous studies, Miszko et al. (2003) investigated higher but also lower load intensities as well as speed on power and activity performance. Using older adults with below average leg power, they compared two exercise groups performing either strength or power training with a control group. The strength group trained at higher intensities of 80% of 1-RM at a controlled pace. The power group, after 8 weeks of identical strength training to develop a base, trained for the remaining 8 weeks at lower loads of 40% of 1-RM performed as quickly as possible. Post training, both training groups demonstrated increased strength with no significant difference between the two suggesting that perhaps power training may be equally effective a producing gains in strength—an observation which has also been implied by Fielding et al. (2002). However, while Miszko and colleagues (2003) reported greater improvement in function in the power group, they found little difference in actual leg power production between the three groups. This could be explained by the fact that they used the 30-second Wingate anaerobic cycle test to test for power which likely did not validly measure the instantaneous muscle power action that was the focus of training.

Other studies assessing the effects of power training have used different training loads but have not systematically examined the effect of each. For example, Henwood & Taaffe (2005) investigated the effects of high-velocity training as compared to no training in adults with ages ranging from 60-80 years. The exercise group trained using upper and lower body exercises at all three intensities (35%, 55%, 75% of 1-RM) while performing explosive movements. While improvements in power and abilities were

noted in the exercise group, with this design, there is no way of determining which load, if any, was optimal in producing peak power or the change in power or function.

As can be seen from the above studies, despite work investigating methods of improving power, little attention has been paid to identifying optimal loads for producing and training for muscle power. de Vos and colleagues (2005) were the first to investigate explosive but non-projection training intensity to maximise gains in muscle power in the older adult. In a large cohort of healthy adults with an average age of 69 years, they assessed peak power in upper and lower limbs after training at one of three different loads (20%, 50%, 80% of 1-RM) using the pneumatic resistance machine. They found that power improved similarly after training at all three loads, reflecting the findings of Kaneko et al. (1983), Moss et al. (1997). All the training groups also made significant improvements in strength as compared to the control group which has also been reported by others (Earles, Judge, & Gunnarsson, 2001; Miszko et al., 2003). The greatest strength improvements were noted in the high load group, as would be expected. While these authors recommended high-intensity non-projection explosive resistance training for not only its strength and endurance effects but also power effects, it is still not clear how relevant these findings are when trying to ensure carry over into function as specific actions require specific contributions of both force and velocity. For example, Cuoco et al. (2004) concluded that power output at 40% of 1-RM explained more of the variability in habitual gait velocity than did power at 70% of 1-RM. Furthermore, actions requiring high velocities against light loads will require different power components to those that require high velocities against heavy loads (Cormie, McCaulley, Triplett, & McBride, 2007). As such, the velocity component of muscle power may be a more critical determinant of performance of lower-intensity tasks than of higher-intensity ones that are more strength related (Sayers, Guralnik, Thombs, & Fielding, 2005). Therefore, the identification of optimal loads that best relate to specific activities needs to be clarified.

2.5.4 Optimising power after stroke

The ability to perform powerfully is recognised as being compromised after stroke at both impairment and activity levels (LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Morris, Dodd, & Morris, 2004; van de Port, Wood-Dauphinee, Lindeman, & Kwakkel, 2007). Despite this knowledge, optimising power after stroke is still a very new concept and has not yet been explicitly considered. There has been work, however,

investigating the effects of progressive resistance training on strength and function (Ouellette et al., 2004; Teixeira-Salmela, Nadeau, McBride, & Olney, 2001; Weiss, Suzuki, Bean, & Fielding, 2000), with one of these studies also considering the effects of strength training on power (Ouellette et al., 2004).

A group of 70 year old stroke survivors participated in a 12 week lower limb strengthening programme for both the involved and uninvolved legs (Weiss, Suzuki, Bean, & Fielding, 2000). Hip and knee strength was assessed and trained using the Keiser pneumatic resistance machine. After 12 weeks, relative gains of 48% and 68% were found in the involved and uninvolved leg respectively. As well, improvements in chair standing times were also significant. However, all other activities measured, including gait speed and stair climbing speed, showed no significant changes. In a group of participants with similar age and function, Teixeira-Salmela et al. (2001) also noted that after a 10 week aerobic and progressive resistance training programme, knee extension strength, as measured isokinetically, increased by 18% and 46% at speeds of 30º/s and 60º/s respectively. Unlike Weiss and colleagues (2000), they did find improvements in both gait speed and stair climbing. These improvements, however, may have very well resulted more from task specific training as the aerobic training component incorporated both walking and stepping exercises. Without separating these two interventions, it is impossible to attribute an increase in function to increased strength. Ouellette et al. (2004) addressed not only force but also power after stroke. In their randomised controlled trial, a similarly aged and functional cohort either performed progressive resistance training of bilateral leg press, and unilateral knee and ankle flexion and extension over 12 weeks using a Keiser pneumatic machine or performed range of motion and flexibility exercises. All were assessed for leg press, knee and ankle 1-RM as well as knee extension power. While there were significant strength gains in the exercise group of between 16% and 38% as compared to the controls, there were variable power gains in both groups and no significant differences in any of the activities tested.

The findings from these strength training trials were confirmed in two recent systematic reviews that included meta-analyses and reported mostly a large effect for strengthening, but small or non-significant effects for functions such as gait (Ada, Dorsch, & Canning, 2006; Morris, Dodd, & Morris, 2004). In all three training programmes, exercise intensity was set at 70-80% of 1-RM and exercises were

performed at a slow and controlled pace that would not address the velocity component of power and would, therefore, limit carryover into activities that have lower force but higher velocity components such as gait (Cuoco et al., 2004; Sayers, Guralnik, Thombs, & Fielding, 2005).

Higher velocity training after stroke has indeed been investigated albeit with far more caution. Early concerns were that this approach would have detrimental effects on function in that the increased speed would exaggerate an abnormally large reaction to speed-dependent stretch reflex, resulting in increased spasticity. However, evidence from studies, including a meta-analysis (Ada, Dorsch, & Canning, 2006; Sharp & Brouwer, 1997) have shown that strength and speed training do not appear to have any significant adverse effects on spasticity. Isokinetic training studies, while potentially addressing the velocity component of power, have findings that are promising but limited. Sharp & Brouwer (1997) trained fifteen community dwelling stroke survivors, aged 67 years, isokinetically in knee extension and flexion over 6 weeks. While improvements in knee extension and flexion torque and gait velocity were observed, there were no significant gains in other measures such as stair climbing and timed up and go. As well, Kim et al. (2001) tested the differences between two groups of stroke survivors who were also community dwelling. One group underwent an isokinetic strength training programme and the other received passive range of motion exercises on the involved leg. While there was a trend towards increased strength in the exercise group, there were no significant differences in the changes in strength and function between groups. Interestingly, these researchers only trained at 60º/s as they reported their participants had difficulty generating faster movements. It has been noted that angular velocities in normal movements are considerable higher (MacDougall, Wenger, & Green, 1991; Perrine, 1986). As such, it is not surprising that at these slow training speeds, little training effect was noted.

Contrary to the above strength training studies, Moreland et al. (2003) found no significant differences in outcomes when comparing a group of hemiparetics who performed nine lower leg exercises against a resistance to a group performing the same exercises without resistance. The authors reported that the experimental group used loads that were progressively increased to ensure each participant was working moderately hard. At discharge, no differences in walking velocity, as measured by the two minute walk test, or in gross motor function were noted between the groups. The

selection of exercises, vague and questionable intensity of training, lack of objective strength assessment as well as lack of a control group make this study difficult to critically accept and consider along with the other strength training studies. However, the findings raise the question of whether, in this population, it is the resistance while performing an exercise or the movement practice itself that is responsible for the training effect and the improvements noted. Indeed, 'task specific training' while important for any population, takes on much more significance after a stroke in which so much more motor learning or motor re-learning needs to occur to optimise return of function (Carr & Shepherd, 2003). This approach to stroke rehabilitation and activity-based re-training is another entire intervention philosophy that is beyond the scope of this review and is currently being used and investigated by others (Ada, Dean, Hall, Brampton, & Compton, 2003; Dean, Richards, & Malouin, 2000). As such, after stroke, to optimise carry-over, it is important to appreciate that not only strengthening but also movement practice and, increasingly, power aspects of muscle function are useful components of a rehabilitation programme and should all be given consideration.

Muscle power after stroke has been specifically addressed recently (LeBrasseur, Sayers, Ouellette, & Fielding, 2006). In this cross-sectional study, the authors assessed muscle impairments, lower-body function, disability, quality of life and behavioural factors in a group of community dwelling stroke survivors. They measured unilateral knee extension power with the pneumatic resistance machines that has been used in studies in the older adult (de Vos et al., 2005; Earles, Judge, & Gunnarsson, 2001; Fielding et al., 2002) and assessed power at six relative intensities (40%, 50%, 60%, 70%, 80%, 90%) of the determined 1-RM as well as assessing habitual gait, stair climbing and chair rising times. They reported that the strength deficits noted between legs (>30%) were superseded by power deficits (>40%) and that involved leg extension power was significantly related to gait speed and explained nearly twice the variability in stair-climbing than did leg strength. They concluded with comments that efforts to optimise power may improve the performance of lower limb physical function, especially in tasks requiring a lower percentage of maximum strength such as walking and stair climbing. However, they did not report on the power profiles or the optimal load at which peak power occurred which leaves no clear direction on optimal training parameters for increasing muscle power and function after stroke.

2.5.5 Summary of optimising power production

While there are some promising results in training programmes in younger adults as well as older adults, optimal loads and training parameters for power are still unclear. It appears that many factors can influence determination of optimal load such as the action performed, the equipment used and the strength and skill level of those being tested. Power testing and ultimately training should be designed with the end physical performance activity in mind in order to ensure that the power components of force and velocity are targeted in a specific and relevant manner. In stroke rehabilitation, it is also encouraging to see that both strength and even speed training can be carried out without adverse effects, although more information about how muscle power is generated in this population will extend the knowledge in this area notably.

2.6 Muscle power and its relationship to activity performance

2.6.1 Introduction

The performance of activities such as gait speed have been recognised as indicators of self-perceived physical function, independence, ability and health (Cwikel, Fried, Galinski, & Ring, 1995; Engle, 1986; Luukinen, Koski, Laippala, & Kivela, 1995; Potter, Evans, & Duncan, 1995) as well as being strong predictors of mobility-related disability (Guralnik, Ferrucci, & Pieper, 2000). There has been a considerable amount of research devoted to optimising power output with the notion that it may have a larger influence on activity performance than muscle force alone. Hence, studies investigating the relationship of muscle power to activity performance will be discussed in this section. Firstly, the role of muscle power in the younger adult and in athletic performance will briefly be presented. Next, its role in older adult performance of three main tasks: gait, stair climbing and rising from a chair will be discussed. Finally, since muscle power in stroke is a relatively new consideration, the association of power as well as strength with the same three tasks will then be reviewed.

2.6.2 Muscle power in young and athletic performance

Investigations into leg power and its contribution to activity performance have yielded mixed results in the young and athletic literature. While some researchers have presented significant relationships with tasks such as jumping and sprinting, others have found that peak power was either not related, or no more related to performance than other measures such as force, and rate of force development. For instance, Young, McLean & Ardagna (1995) investigated the relationship between a number of strength

variables and sprint performance in track and field athletes. They found that while average jump squat leg power, normalised to body mass, showed significant correlation with both sprint starting ability and maximum sprinting speed, other measures such as relative maximum dynamic strength, absolute and relative maximum force and the relative force produced over the first 100ms of a leg extension action were also highly correlated with either or both starting ability or maximum speed in sprint performance. Similarly, Baker & Nance (1999) found that when power was normalised to body mass, jump squat maximal power was moderately associated with the initial 10m and final speed at 40m of a running sprint in a group of professional rugby league players, although to different extents, highlighting the different components of the sprint action (acceleration ability vs. maximum speed). As well, in a group of young weight-trained male athletes competing in power-type sports, Sleivert & Taingahue (2004) reported that relative peak and average leg power obtained while performing traditional and split jump squat values were also moderately correlated with 5m sprint times.

However, the significant association of leg power to sprinting performance has not been consistent. Thomas, Fiatarone, & Fielding (1996) found that in their young untrained female sample group, bilateral leg press power, as measured by the pneumatic resistance machine, was strongly correlated with vertical jump but only insignificantly with maximal gait speed and not at all with a 40 yard sprint. In addition, Murphy & Wilson (1997) found no clear relationships between training-induced changes in isoinertial and isokinetic muscle testing and the training-induced changes in sprint and cycle performance in a group of young active males, prompting the authors to conclude that changes in muscle function do not necessarily lead to changes in athletic performance. Finally, average leg extensor power per kilogram of body mass, as determined by jumping, was found to not have any significant association with either the initial acceleration or maximum speed obtained in a 30m sprint in a group of young male physical education students (Kukolj, Ropret, Ugarkovic, & Jaric, 1999).

The conflicting findings may, as Cronin & Sleivert (2005) argue, be due to a number of factors such as the interchanging terms, interpretations, and assessment techniques of variables such as rate of force development, explosive strength, impulse, and anaerobic cycling power which, in the various studies, all claim to assess muscle power. Additionally, the inconsistent use of body mass to normalise data may have an influence as it is known that body size has a positive relationship to muscle strength (Jaric, 2002)

and to various movement performances (Markovic & Jaric, 2004). Nevertheless, power is likely only one component involved in the performance of athletic manoeuvres and, as such, cannot fully explain or account for the ability to carry out such actions. Therefore, its carryover into activity performance remains unclear.

2.6.3 Muscle power in older adult activity performance

Numerous authors investigating leg power in the older adult have reported significant relationships between leg power and activity performance of tasks such as gait. An often cited paper by Bassey and colleagues (1992) reported strong correlations between summed unilateral leg power scores and gait speed in frail and elderly hospital residents when power was normalised to body mass and measured by the Nottingham rig. These findings prompted the authors to propose that the power rig measured a fundamental physical attribute needed for adequate function in old age and that there was a threshold of minimum power needed to perform unassisted walking. Other authors, using a variety of power assessment techniques such as bilateral leg press and unilateral ankle dorsiflexion and plantarflexion have found moderate correlations between power and gait speed at either habitual or maximal gait speeds. For example, Suzuki, Bean & Fielding (2001) reported moderate correlation between ankle power, measured isokinetically, and maximum gait speed in community dwelling women. Bean, Kiely et al. (2002) also reported moderate correlations between leg power and maximal gait velocity in a mobility limited American group of men and women over 65 years of age. This finding was again found in the work by Bean et al. (2003) in a large and similarly aged Italian group. Cuoco et al. (2004) noted that leg power had moderate correlations with habitual gait speed and explained a greater proportion of the variability than did 1-RM leg strength.

A relationship between stair climbing ability and leg power has been reported by some, but not all authors. Stair climbing ability and relative leg power was found to be highly correlated by Bassey et al. (1992) in a group of institutionalised elderly participants. In support of this, authors such as Bean, Kiely et al. (2002) also found moderate correlations between these two variables, claiming that there was a stronger association with stair climbing and bilateral leg press power than with stair climbing and leg press 1-RM. In contrast, Cuoco et al. (2004) reported no significant relationship between leg power and stair climbing. These authors proposed that this task requires a larger

amount of strength to perform and as such, would not be correlated with power values obtained at lower loads.

Leg power has also been found to have mixed associations with repeated chair standing ability. Moderate correlations between these variables were reported in the earlier discussed work by Bassey and colleagues (1992). When Skelton, Greig, Davies & Young (1994) used the Nottingham rig and also summed the leg press power scores and normalised them to body weight, they too found comparably moderate correlations. Similarly, Suzuki, Bean & Fielding (2001) reported that ankle dorsiflexion power was also moderately correlated with repeated chair rising, suggesting that this muscle group plays a significant role in initiating chair standing. However, a lack of association between leg power and chair rising has been reported by Bean et al. (2003) and Cuoco et al. (2004). These authors have suggested that the performance of this task is more related to force production than to the rate at which that force is produced. Repeated chair stands, they argued, may be more of a muscle and cardiovascular endurance test, an idea supported by others who have also questioned the validity of repeated chair stands as a measure of leg strength or power in the older adult (Netz, Ayalon, Dunsky, & Alexander, 2004). In assessing a group of women with a mean age of 72 years, these authors also found no significant correlation between isokinetic knee extensor strength and chair standing but did find a significant association between aerobic capacity and ten chair stands.

2.6.4 Muscle power in activity performance after stroke

The contribution of muscle power to mobility in people affected by stroke has not been extensively investigated. In one study, LeBrasseur and colleagues (2006) explored the relationship between involved knee extension power and habitual gait speed, stair climbing time and repeated chair standing time as well as the relationship between involved knee extension strength and the same three activities. These authors reported a significant association between residual impairments of both leg strength and power and activity performance of gait and stair climbing but not with repeated chair stands. They also found that muscle power was the strongest predictor of stair climbing time and explained nearly twice as much variability as muscle strength, suggesting that measures of power be included in impairment testing after stroke.

Associations between impairments of muscle strength and mobility have been more extensively tested in the stroke population. For instance, when assessing the contribution to gait, Bohannon & Andrews (1990) tested isometric involved knee extensor strength in a group of sub-acute hemiparetics and found it to be moderately correlated with self-selected gait speed. However, isokinetic strength, it has been argued, may be more appropriate in establishing relationships with activities which require force to be exerted through a large range of motion (Kim & Eng, 2003; Wilson & Murphy, 1995). Indeed, Nakaumra, Hosokawa & Tsuji (1985) reported higher correlations between gait and isokinetic measures as compared to isometric measures. More recently, Suzuki, Imada, Iwaya, Handa & Kurogo (1999) investigated isokinetic strength and its relationship to gait through a pre-training assessment in a group of stroke participants undergoing computer assisted gait training. They too demonstrated that involved isokinetic knee extensors had moderate correlation with maximal walking speed. Similarly, Flansbjer, Downham & Lexell (2006) assessed the relationship between bilateral knee strength and gait performance of participants mildly to moderately affected by stroke. These authors measured isokinetic knee extension and flexion strength at 60º/s and also reported moderate correlations between involved knee strength and fast gait but not uninvolved knee strength and fast gait, performed over a fourteen metre distance. They went on to report that involved knee strength explained 34% to 50% of the variance in gait performance. Milot, Nadeau, & Gravel (2007) used muscular utilization ratios (MUR)s, defined as an index of the percentage of maximal strength used by a given muscle group during a task such as gait, to assess the relationship between plantarflexors, hip flexors and hip extensors in self-selected and maximal gait in a group of independently functioning chronic hemiparetics. They found that when performing gait at maximal speed, hip flexor and extensor use approached the MUR of the highly active plantarflexors and suggested that in fast gait, hip musculature plays an increasingly important role.

Involved leg strength and its relationship to stair climbing have also been assessed. Bohannon and Walsh (1991) reported that the isometric strength of five involved muscle groups (hip flexors and extensors, knee flexors and extensors and ankle dorsiflexors) was significantly correlated with stair-climbing ability. In an attempt to be more precise, isokinetic assessment has been used to test the association of specific lower limb muscles. Kim & Eng (2003) noted a strong relationship between involved plantarflexor strength to stair climbing and a moderate one between knee flexion

strength and stairs. However, no significant association was noted between involved knee extensor strength and stair climbing. While in agreement with the finding of knee flexor contribution to stairs, Flansbjer et al. (2006) did report a moderate correlation between involved knee extension strength and ascending stairs.

Leg strength and its relationship to repeated chair stand time in people who have sustained a stroke is less clear. Ten chair stands may reflect more of a strength endurance test as opposed to the shorter-term power assessment as has previously been proposed (Bean et al., 2003). During a pre-training assessment of hemiparetic participants, Weiss et al. (2000) found a moderate correlation between the involved leg press 1-RM and time to perform chair stands when fewer were repeated. In support of this finding, more recent studies, such as the one by Lomaglio & Eng (2005) who used chair standing to test hemiparetic participants, have timed only one full manoeuvre. These authors assessed a single sit to stand action using two force plates, one under each foot, to determine movement onset and an optoelectric sensor attached to the acromion to determine movement termination. They found that involved knee extension strength, followed by ankle dorsiflexion strength, was highly correlated with a single sit to stand action.

Of further consideration in the discussion concerning the relationship between leg strength and chair standing is that it is difficult to exclude the skill factor involved in standing from a chair without the use of arms and the influence of compensatory movements. In a study by Alexander, Medell, Gross & Hoffmeyer (2001), participants showed different ways of performing a chair rising task such as tilting the torso forward to reduce the load on the lower extremities. Since the repeated chair stand test does not specify the need for controlling the torso, it would be difficult to determine the contribution of either the torso or the legs to chair standing performance.

The asymmetrical nature of stroke deficits and the bilateral nature of the sit to stand task would also suggest that when performing repeated chair standing actions, asymmetrical leg use compensations would occur. This has been confirmed by Cheng et al. (1998) who noted asymmetric body weight distribution, with much more weight directed onto the uninvolved side (approximately 75% of body weight), when they tested hemiparetics performing a single chair rise action using a force plate under each foot. In addition, Lomaglio & Eng (2005) reported significant positive relationships between

weight-bearing symmetry and chair standing times in their group suggesting that this is a another key factor that can influence the successful performance of chair standing in the hemiparetic population.

2.6.5 Summary of muscle power's contribution to functional performance

The ability to generate and transfer muscle power is a key component to successful performance of many athletic and everyday actions. While this has been found in investigations of athletic performance, it is becoming clear that power alone is not the only key component. Other variables such as force and rate of force development may be equally or even more important in certain movements. In the older adult, stronger associations between leg muscle power and certain tasks such as gait and stair climbing, and to a smaller extent chair stands have been observed. Similarly, in the stroke population, muscle power has been shown to be a stronger predictor of performance in some, but not all activities. Therefore, despite a clear link between muscle power and performance of activities, it is likely one of many factors that need to be considered when attempting to optimise mobility.

CHAPTER 3 – METHODS AND MATERIALS

3.1 Introduction

This chapter describes the method and procedures used to conduct a prospective cross-sectional study designed to assess muscle power after stroke and correlate it with the performance of specific activities. There are five sections in this chapter. Details on study design and participants will be followed by information on the instrumentation used. A detailed report of the procedure follows, while the final section describes the data and statistical analysis used.

3.2 Study design and participants

A cross-sectional design was used and involved participants who had suffered a stroke as well as age and gender matched controls. All testing was carried out at the Health and Rehabilitation Research Centre (HRRC) located at Akoranga Campus, AUT University in Auckland, New Zealand.

3.2.1 Power and effect size

Based on an effect size of 0.5, a power of 0.8, with an alpha level of 0.05, a pilot study was conducted using 10 participants to calculate sample size. For the main study, the variable in question was power as a percentage of one-repetition maximum (1-RM) (30%, 50%, and 70%). The sample size based on these criteria was set at n=60.

3.2.2 Participants

In accordance with the Auckland University of Technology (AUT) Ethics Committee requirements (Appendix A), community dwelling adults with and without stroke were invited to participate on a voluntary basis. They were recruited from local stroke clubs, the AUT Rehabilitation Clinics, notices in the local newspapers (see Appendix B for notice) and from existing participant databases. Information was presented at stroke club meetings and at the AUT Rehabilitation Clinic explaining the rationale and the aims of the study. A brief written study overview with contact details was also provided (Appendix C). Interested potential participants were later contacted and supplied with the Participant Information Sheet as additional information (Appendix D). Interested participants were contacted either by telephone or at a subsequent visit to schedule the first session.

3.2.2.1 Inclusion and exclusion criteria

To be included in the study, potential participants had to meet the following inclusion criteria:

- Having had a diagnosis of unilateral stroke no less than 6 months ago (or no stroke or any other neurological lesion for the control group);
- Residual motor deficits as a result of the stroke affecting at least the leg;
- Consenting to participate in this study;
- Independently ambulating with or without an assistive device for at least ten metres;
- Able to communicate, with an ability to understand and follow simple commands as determined by a Mini Mental State Examination score of 21 or above (this was further assessed at the initial session);
- Able to participate as per the general screening questionnaire.

They were excluded if they demonstrated:

- An inability to get onto or lift the minimal load on the modified leg press machine;
- An inability to perform the activities;
- A score of 3 or more on the Modified Ashworth Spasticity scale of the quadriceps or plantarflexor muscles on the affected leg.

Participants who met the eligibility criteria completed the following screening questionnaires at the beginning of the first session:

- a Standardized Mini-Mental State Examination (SMMSE) (Appendix E)
- a Physical Activity Readiness questionnaire (PARQ) (Appendix F)
- a Screening questionnaire (Appendix G)

The Participation Information Sheet was also reviewed. If all were completed satisfactorily, a Participant Informed Consent form (Appendix H) was signed prior to commencement of testing.

3.3 Instrumentation

3.3.1 Modified supine leg press machine

All muscle force and power testing occurred on a modified supine leg press machine located at the HRRC. Details of this machine have been reported previously by Cronin, McNair and Marshall (2003). Briefly, the supine leg press machine is a custom built machine (Fitness Works, Auckland, New Zealand) designed to allow participants to perform leg extensions and explosive and ballistic jumping actions in a horizontal direction, while being fully supported. Thus, it minimises the risk associated with such exercises in an upright position (Cronin, 2002). The body is supported on a padded platform that runs parallel to the ground. Once lying on it, the shoulders come in contact with 2 padded areas. The legs are flexed at the knees and hips so that the feet are placed on a large foot plate perpendicular to the padded platform and ground. Once pressure is applied to the feet and the legs extend, the shoulders press on the padded areas and the platform slides along 2 runners. There is a 300kg pin loaded weight stack that is attached to the platform so that resistance to extending the legs and moving the platform can be adjusted and recorded. See Figure 3.1 for more information.

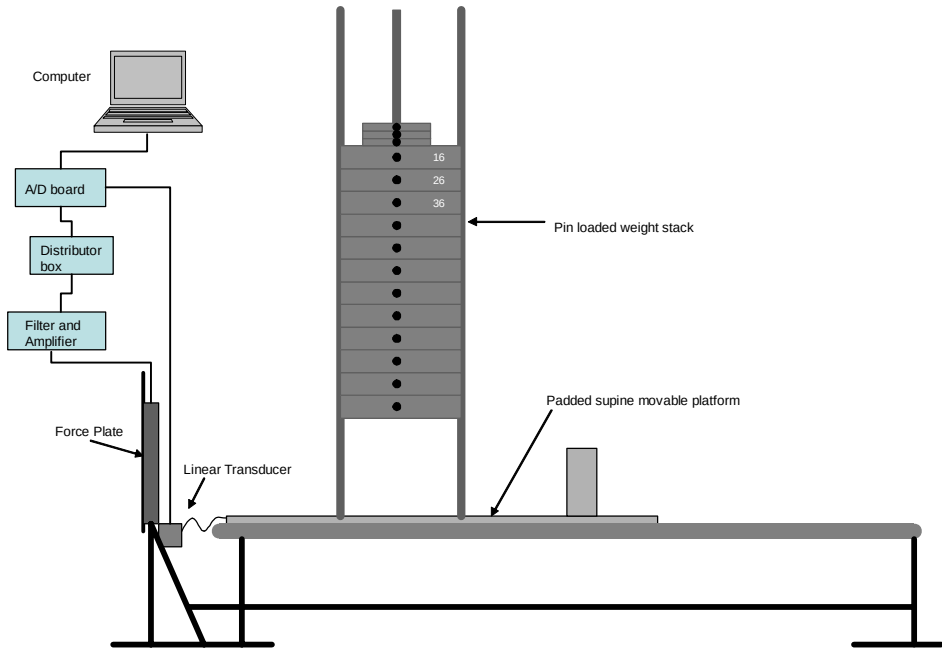

Figure 3.1
Schematic diagram of instrumentation set-up.

3.3.2 Force plate

A force plate (Advanced Mechanical Technology Inc., 179 Waltham Street, Watertown, MA 02172, USA) was mounted onto the foot plate of the modified leg press machine, perpendicular to the moving platform (see Figure 3.1). Force plate signals, orthogonal to the movement, were sampled at 1000 Hz, amplified and filtered with a 10.5 Hz low pass cut-off filter and relayed to a custom made data acquisition and analysis programme (SuperScope II, Version 3.0, GW Instruments, Boston, USA). Calibration of the force plate was undertaken before each data collection session.

3.3.3 Linear transducer

A linear transducer (P-80A, Unimeasure, Oregon) was attached to the platform of the modified leg press machine (see Figure 3.1). The transducer data, accurate to 0.1cm, was sampled at 1000 Hz by the above mentioned computer-based data acquisition and analysis program to provide displacement/time data. This system has been shown to be both valid and reliable in previous testing (Cronin, Hing, & McNair, 2004). It was calibrated to a known length before each testing session.

3.4 Testing procedure

3.4.1 Descriptive data

Demographic data for each participant were obtained and included age, gender as well as type, location and date of the diagnosed stroke. Additional physical measures such as participants' weight and height were also recorded. Afterwards, the Fugl-Myer Assessment (Appendix I) of the lower limb was administered to the stroke participants to assess and grade general hemiplegic lower limb motor recovery (Fugl-Meyer, Jaasko, Leyman, Olsson, & Steglind, 1975).

3.4.2 Procedure

Data was collected over two sessions separated by no less than two and no more than seven days.

The first session was used for screening and descriptive and demographic data collection. This session was also used to collect activity performance data and to determine each participant's 1-RM for each leg. See Figure 3.2 for more information.

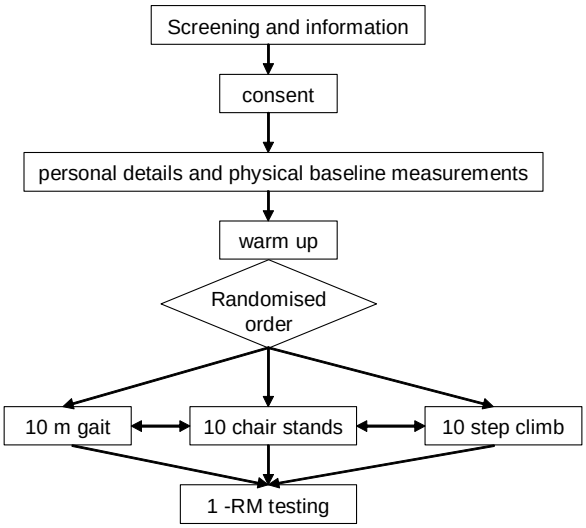

Figure 3.2
Flow diagram of the first session of data collection.

The second session was used for assessing muscle power performance at differing percentages of the previously determined 1-RM. See Figure 3.3 for more information.

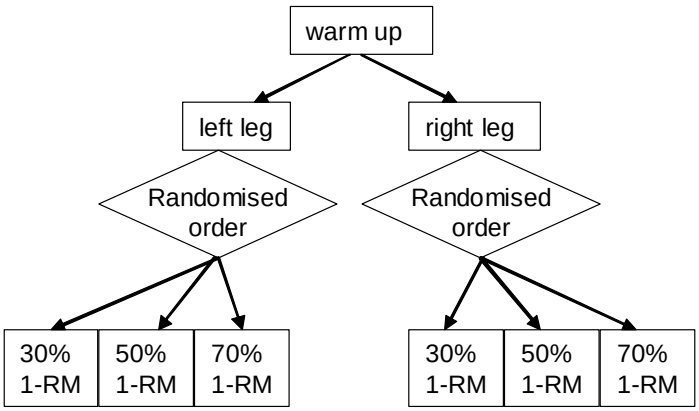

Figure 3.3
Flow diagram of the second session of data collection.

3.4.3. Activities

Following a standardised warm up involving 5 minutes of stationary cycling against no resistance, three activity-based tasks were performed. The order of the tasks was randomly assigned. Three repetitions of each task were performed. A one minute rest was taken between repetitions. The participant was asked to try to complete each task as fast as possible and the fastest repetition was used in subsequent data analysis. Verbal encouragement was included with each trial. All tasks were measured by a hand held digital stopwatch accurate to 0.01 second.

3.4.3.1 Gait speed

Gait speed was measured as suggested by Watson (2002), using a hard flooring area free from other obstacles and was measured over a ten metre distance. Participants were instructed to move as quickly and as safely as possible over this distance. The participants were timed from when the first foot moved to take a step, to when either foot first crossed the 10 metre line.

3.4.3.2 Stair climb

A standard staircase was used in assessing time taken to climb ten stairs. This is similar to the Continuous Scale Physical Function Performance Test (CS-PFP) (Cress, Buchner, Schwartz, & de Lateur, 1996). The steps measured 16cm high, 28cm deep, and rose at an angle of 60° and included a sturdy rail on either side. Participants were instructed to climb the ten steps as quickly as possible, placing only one foot on each step. Timing began when the leading foot left the ground and the stopped when the trailing foot contacted the last step.

3.4.3.3 Repeated chair stands

Repeated chair stands have been used to assess lower limb function (Jones, Rikli, & Beam, 1999). Participants were asked to rise from a chair ten times as quickly as possible without using their arms. The chair was of a standard height (43cm) and did not have any arm rests. Participants were positioned slightly forward in the chair at the start of the task. The task was timed from the moment that standing was initiated to the moment the participant's back contacted the back of the chair.

3.4.4 One-repetition maximum testing

Using the modified leg press machine, 1-RM was measured following Dowson's protocol (Dowson, 1999). This protocol takes into consideration some of the possible confounding variables in testing directly. The participant completed 1 set of 10 repetitions at a moderate load bilaterally to allow for familiarisation of the task and the loads. This was then progressed to 5 repetitions performed unilaterally. Once single repetitions of heavier loads began, a rest period of 1 minute and no more than 5 minutes between repetitions was permitted. The load was increased incrementally but enough to ensure 1-RM was reached within five attempts (Brown & Weir, 2001; Dowson, 1999; Morrow, Jackson, Disch, & Mood, 1995).

Participants were positioned on the supine leg press machine in a standardised position with the knee angle set at approximately 90° using a goniometer aligned to the lateral malleolus, lateral epicondyle of the knee and greater trochanter of the leg. The hip joint angle was also set at 90° using a goniometer aligned to the lateral epicondyle, the greater trochanter of the femur and the lateral midline of the pelvis (Norkin & White, 1985). This leg position is similar to earlier studies (Cronin, McNair, & Marshall, 2003; Earles, Judge, & Gunnarsson, 2001; Fielding et al., 2002; Newton et al., 2002) and is similar to the leg position adopted in many activities (ie chair rise, stair climb). The participant's untested leg was held secure via a supporting strap. See Figures 3.4 and 3.5 for more details.

Figure 3.4
Starting position for testing the left leg of the participant on the modified supine leg press machine.

Figure 3.5
Starting position for testing the right leg of the participant on the modified supine leg press machine.

The uninvolved leg in the stroke participants was tested before the involved leg while the dominant leg of the control group was tested. All orthotics were removed prior to testing.

3.4.5 Power testing

Following a period of at least two but no more than seven days, participants returned for power testing of each leg. This session involved pushing to extend the leg against 30%, 50% and 70% of the earlier determined 1-RM for that leg. After a standardised cycling warm up as previously discussed, participants were tested using the supine modified leg press machine and positioned as above.

A familiarisation session included three unilateral leg extensions against the tested percentage of 1-RM with the first two performed at half of maximum effort and the final repetition performed as hard and as fast as possible.

Thereafter, at each percentage of 1-RM, participants started from a statically held position and were instructed to push off the foot plate as hard and as fast as possible, as if trying to hop (see Figure 3.6). After each trial, the participants rested for 1 minute. Two repetitions at each load were performed. A rest of up to 3 minutes was provided when the loads were changed. Testing order of the three different loads was randomised prior to testing. Pilot testing demonstrated the reliability of these procedures (ICC: 0.91-0.97) over two testing sessions across no more than seven days.

Figure 3.6
Power testing with the participant pushing as hard and as fast as possible.

3.4.6 Adverse effects

Information on any adverse effects from the activity-based testing or the power testing was requested and recorded.

3.5 Power calculation

The collected measures of force and displacement were used to calculate power. The rate of change of displacement was used to calculate the velocity at which the platform moved and this was undertaken using a differentiation algorithm. Power was subsequently calculated as the product of force and velocity. The highest power observed across the two trials at each load was used in the statistical analysis.

3.6 Statistical analysis

Statistical analyses were undertaken using the Statistical Package for Social Sciences (SPSS) Version 11.0 (SPSS Inc., Illinois, USA). Descriptive data were assessed to determine the appropriateness of parametric analysis. Measures of skewness and kurtosis were checked for normality. Suspected outliers were assessed using the Grubb's test (Extreme Studentized Deviate ESD) (http://www.graphpad.com/quickcalcs/Grubbs1.cfm).

Control and stroke groups were compared for baseline characteristics of height, weight and body mass index (BMI) as well as for the activities of gait, stair climb and chair stands using an independent t–test or, if required, a Mann-Whitney U test.

Two 2x3 models of analyses of variance (ANOVA) were used to compare the involved and uninvolved legs over the differing loads (30%, 50%, 70% of 1-RM) and to compare the uninvolved leg and comparison leg over the same three loads. Departures from sphericity were accounted for with the Huynh-Fedlt epsilon value. Planned contrasts were examined using Bonferroni analyses to assess differences across loads (Field, 2000).

To quantify the relationship between muscle strength and activity performance and the relationship between muscle power and activity performance, Pearson Product-Moment Correlation Coefficients or, when necessary, Spearman's Rank Correlation Coefficients were calculated. For all statistical analyses, an alpha level of $p<0.05$ was set.

CHAPTER 4 - RESULTS

4.1 Introduction

This chapter is divided into sections that represent the main areas of investigation. The first section describes the participants and their characteristics. Results on activity performance are then reported, followed by results from the one-repetition maximum testing. Findings concerning peak power are then reported. Finally, the relationships between muscle strength and activity performance as well as muscle power and activity performance are presented.

4.2 Participants

A flow chart representing the participant recruitment, screening and testing is presented in Figure 4.1.

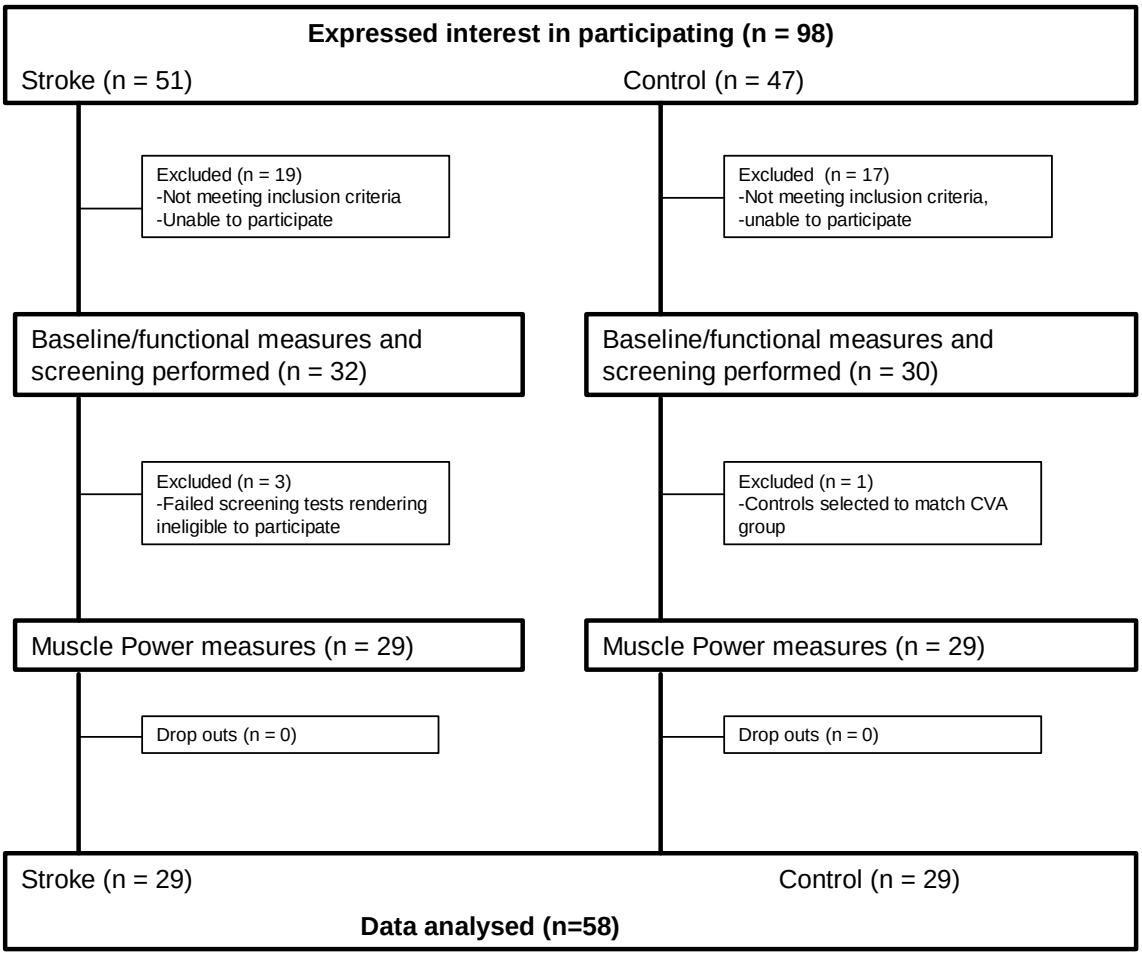

Figure 4.1 Flow diagram of participant progress through the study.

Ninety-eight people (51 having had strokes and 47 controls) expressed interest in the study. Of those, thirty-six were excluded initially because they did not meet the

inclusion criteria, were unable to travel to the HRRC, were away, or were unable to be further contacted. That left sixty-two participants (32 having had a stroke and 30 controls) who were initially screened and assessed at the first data collection session. Of these, three in the stroke group did not meet further inclusion criteria. The final group of 29 who had a stroke and 30 controls continued to the second data collection session. Of the 30 controls, 1 was left out to ensure the two groups were equally matched for gender, age, and body mass index (BMI). Therefore, the final total number of participants whose data were analysed was 58.

Baseline characteristics of the two groups of participants, including age, height, mass, calculated BMI are presented in Table 4.1.

Table 4.1 Descriptive statistics (M ± SD) for age, height, mass and calculated BMI for stroke group (n=29) and control group (n=29).

	Stroke		Control	
	M ± SD	Range	M ± SD	Range
Age (yrs)	64.6 ± 12.3	40.0 - 85.0	65.3 ± 12.2	38.0 - 84.0
Height (cm)	169.2 ± 8.5	154.0 - 185.0	170.1 ± 9.5	150.0 - 191.0
Mass (kg)	74.5 ± 16.3	47.0 - 104.0	73.3 ± 14.0	47.0 - 104.0
BMI*	25.9 ± 4.5	18.6 - 33.5	25.0 ± 3.3	20.1 - 33.1

*BMI= mass (kg)/height $(m)^2$.

Independent t-tests examining differences between the two groups across the baseline measures showed no significant differences ($p>0.05$). The male:female ratio was 17:13 for both groups.

For the stroke group, the average time (in months) since stroke was 66.8 months (SD=75.3, 6-360). The ratio of involved side (Left:Right) was 21:8 demonstrating a much larger representation of those with a left sided hemiplegia. The Fugl-Meyer Assessment lower limb score mean was 25 (SD=5.5, 16-33) out of a possible 34.

4.3 Activities

Descriptive data of the two groups performing the three activities are presented in Table 4.2.

Table 4.2 The results for activities (M ± SD) performed by the stroke group (n=29) and control group (n=29).

	Stroke M ± SD	Control M ± SD	p value
Gait speed (m/s)	1.0 ± 0.6	1.9 ± 0.4	p=0.000
Stair climb speed (steps/s)	1.1 ± 0.6	2.3 ± 0.5	p=0.000
Chair stand time (s)	44.4 ± 30.3	20.1 ± 6.1	p=0.000

There was a statistically signficant difference between the two groups across all three activities. It should be noted that the chair stand data was not normally distributed (Kolmogorov-Smirnov and Shapiro-Wilk tests) in the stroke group and hence a non-parametric statistical test was undertaken.

4.4 One-repetition maximum

Descriptive data of the 1-RM scores for the involved and the uninvolved legs of the stroke group and the comparison leg of the control group are presented in Table 4.3.

Table 4.3 One-repetition maximum (1-RM) values (M ± SD) of the involved and uninvolved legs of the stroke group (n=29) and the comparison leg of the control group n=29.

	Involved leg M ± SD	Uninvolved leg M ± SD	Control leg M ± SD
1-RM (kg)	46.4 ± 20.3*	60.1 ± 16.6*	70.9 ± 18.1*

* denotes statistical significance from other values $p<0.05$.

There were statistically significant differences in the leg press 1-RM between the involved and uninvolved leg of the stroke group as well as between the uninvolved leg of the stroke group and the comparison leg of the control group.

4.5 Peak power

Figure 4.2 presents peak power values obtained across limbs, irrespective of load. The control group limbs (Mean:708; SD:289 W) were significantly more powerful than the uninvolved limbs of stroke participants (Mean:461; SD:220 W). Furthermore, the uninvolved limbs of those with stroke were significantly more powerful than the involved limbs (Mean:220; SD:134 W).

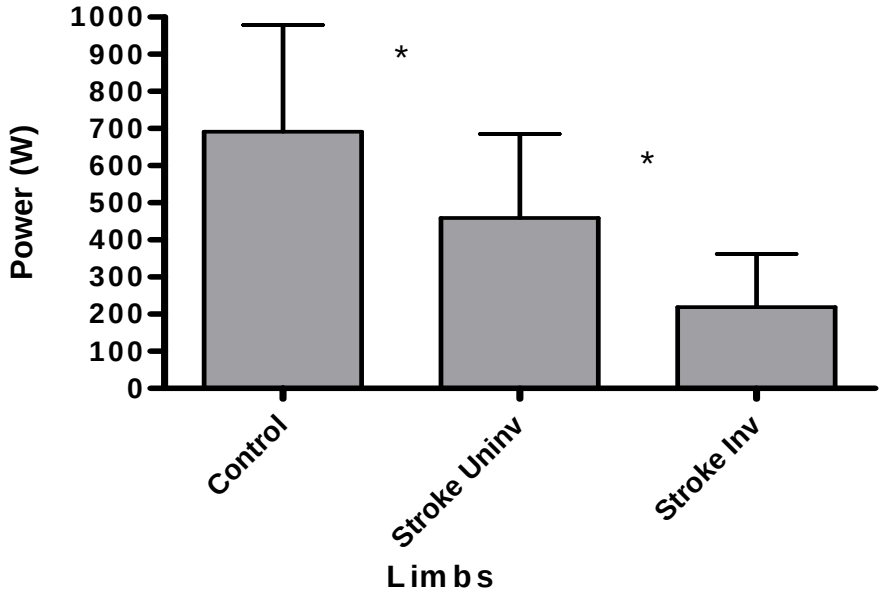

Figure 4.2 Peak power (W) and SD over three limbs irrespective of load. (Uninv = uninvolved limb, Inv = involved limb) *p<0.05.

Figure 4.3 shows peak power values across loads and limbs of the stroke group. In both limbs, power declined with increasing load. There was a significant interaction between limbs and load. In the uninvolved limb, peak power was significantly different across all three loads. In the involved limb, there was a significant difference between the 30% of 1-RM and 50% of 1-RM loads but not between 50% of 1-RM and 70% of 1-RM loads.

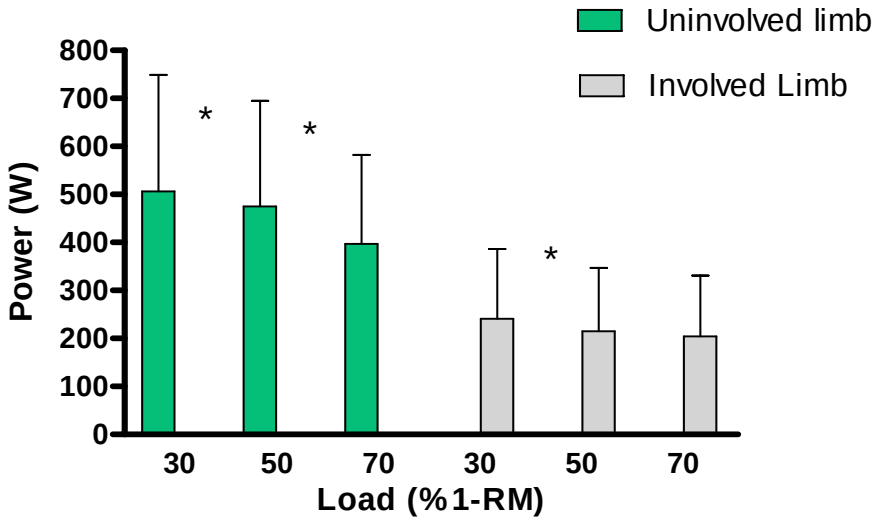

Figure 4.3 Peak power (W) over 30%, 50%, 70% of 1-RM for the uninvolved and involved limbs of the stroke group (*p<0.05).

Figure 4.4 presents peak power values across loads of the uninvolved limbs of the stroke group and the comparison limbs of the control groups. In both groups, peak power declined with increasing load and was significantly different across all three loads.

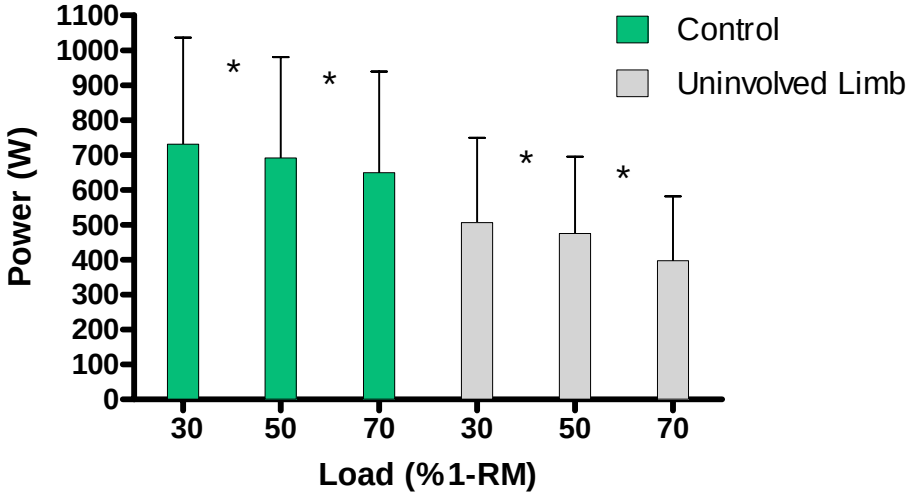

Figure 4.4 Peak power (W) over 30%, 50%, 70% of 1-RM for the comparison limb of the control group and the uninvolved limb of the stroke group (*p<0.05).

4.6 Correlations with activity performance

4.6.1 One-repetition maximum and activity performance

The relationship between 1-RM and the performance of all but one activity was calculated using the Pearson Product-Moment Correlation Coefficient. The stroke group's chair stand data were not normally distributed; therefore, the non-parametric Spearman Rank Correlation Coefficient test was used. Two correlation measures were calculated: 1) using absolute strength (1-RM) and; 2) using relative strength (1-RM/body mass) to account for differences in body mass across individuals. All results are included in Table 4.4.

Table 4.4 Correlation coefficients between absolute strength and relative strength and performance of activities for the involved and uninvolved legs of the stroke group and the comparison leg of the control group.

	Gait speed	Stair climb speed	Chair stands
Involved leg			
1-RM	0.66*	0.62*	-0.37
1-RM/body mass	0.72*	0.70*	-0.40*
Uninvolved leg			
1-RM	0.42*	0.34	-0.24
1-RM/body mass	0.48*	0.45*	-0.37*
Control leg			
1-RM	0.70*	0.60*	-0.56*
1-RM/body mass	0.47*	0.59*	-0.61*

* Correlation is significant at 0.05 (2-tailed)

There was a significant correlation between involved leg absolute strength and gait and absolute strength and stair climbing speed in the stroke group. There was no statistically significant correlation between absolute leg strength and chair standing ability. When strength relative to body mass was used, significant associations were found across all three activities.

The uninvolved leg of the stroke group also showed significant correlation between absolute strength and gait speed but not with the other tasks of stair climbing or chair standing. Relative strength was, however, significantly associated with all the activities.

In the control group, absolute and relative leg strength were both significantly correlated with all of the activities assessed.

4.6.2 Peak power and activity performance

The relationship between power and the performance of the gait and stair climbing tasks in both groups and power and the control group's chair standing task was calculated using Pearson Product-Moment Correlation Coefficient. The stroke group's chair stand data were not normally distributed; therefore, the Spearman Rank Correlation Coefficient test was used. Two correlations were examined: 1) using absolute values of peak power (W) and; 2) using peak power relative to body mass (W/kg) to account for differences in body mass across individuals. All results are shown in Table 4.5.

Table 4.5 Correlation coefficients of absolute and relative peak power across all three loads and performance of activities for the involved and uninvolved legs of the stroke group and the comparison leg of the control group

		Gait speed	Stair climb speed	Chair stands
Involved leg				
30% 1-RM	W	0.64*	0.64*	-0.15
	W/kg	0.69*	0.72*	-0.27
50%1-RM	W	0.63*	0.62*	-0.15
	W/kg	0.67*	0.69*	-0.15
70%1-RM	W	0.57*	0.56*	-0.20
	W/kg	0.62*	0.63*	-0.22
Uninvolved leg				
30%1-RM	W	0.01	0.07	0.15
	W/kg	0.04	0.11	0.07
50%1-RM	W	-0.01	0.06	0.13
	W/kg	-0.09	0.10	0.12
70%1-RM	W	-0.04	-0.02	0.21
	W/kg	0.00	0.03	0.13
Control leg				
30%1-RM	W	0.74*	0.64*	-0.57*
	W/kg	0.64*	0.68	-0.63*
50%1-RM	W	0.73*	0.63*	-0.56*
	W/kg	0.73*	0.64*	-0.61*
70%1-RM	W	0.67*	0.59*	-0.57*
	W/kg	0.66*	0.66*	-0.65*

* Correlation is significant at 0.05 (2-tailed)

In the stroke group, there were significant moderate correlations between absolute peak power generated by the involved limb and gait and stair climbing speed. There was no significant correlation between power at any level and chair stands. Relative power correlations were slightly higher and followed a similar pattern.

With respect to the uninvolved limb in the stroke group, no significant correlations were observed between absolute or relative peak power at any level with any activity.

In the control group, absolute peak power as well as relative peak power were both significantly correlated with gait, stair climbing speed and chair stands.

CHAPTER 5 - DISCUSSION

5.1 Introduction

This chapter discusses the current study's results. It is divided into sections that reflect the results of the main areas of investigation, namely, the participants, the three activities, the one-repetition maximum, peak power, optimal load and the correlations between strength and activity performance and power and activity performance. This will be followed by a discussion on the limitations of this work.

5.2 Participants

The baseline characteristics between the two groups in this study were similar. As well, the control group is comparable to a number of other studies using elderly participants (de Vos et al., 2005; Jozsi, Campbell, Joseph, Davey, & Evans, 1999; Newton et al., 2002). Of note is that although the mean age of the groups in the current study was 64 years, the range was quite broad with participants as young as 40 years. In fact, nearly one third of participants were aged under 60 years. When considering the physiological and functional changes associated with ageing, this has implications when comparing results to groups that have a higher proportion of older people. Comparing data from the current study to those other groups needs to take into account these differences as declines in power, strength and function occur with advancing age (Bassey et al., 1992; Foldvari et al., 2000; Frontera, Hughes, Lutz, & Evans, 1991; Harries & Bassey, 1990; Skelton, Greig, Davies, & Young, 1994).

Besides age, the additional functional inclusion criteria would suggest that the stroke group was of a comparable functioning level to other studies involving stroke participants. However, they were likely higher functioning when compared to the whole stroke population due to the nature of the inclusion criteria and as evidenced by the FMA leg scores which suggest that the current study's participants were mildly to moderately affected by the stroke (Duncan et al., 1994).

5.3 Activities

In the current study, measures of maximal gait speed, maximal stair climbing speed and fastest time to perform repeated chair stands were used for their functional relevance as well as their inclusion in other studies assessing leg power. All the tasks performed by the stroke group were significantly slower than the control group. This is not surprising

given the evidence regarding changes in muscle structure and function after stroke. Further changes due to inactivity would also be expected as the average time since stroke was considerable (66.8 months). Changes in not only muscle force and contraction velocity but also impaired voluntary activation and hypertonia could have, in any combination, contributed to differences in performance seen.

5.3.1 Gait speed

The maximal gait speed of the control group (1.9 m/s) was significantly higher than that of the stroke group (1.0 m/s). The current study's gait speed findings in the control group are comparable to maximal gait speeds found by other authors investigating the older adult. For example, Rantenen and Avela (1997) tested maximal gait speed in a group of 80-85 year old men and women and reported maximal speeds of 1.5 m/s. This slower speed likely reflects the twenty-year age difference between this and the current study. Suzuki et al. (2001), using participants with a mean age of 75 years, measured the middle five metres of a ten metre gait test, performed as quickly as possible and found a speed of 1.6 m/s. Using the same gait testing protocol, this speed was also reported by Sayers et al. (2003) when assessing a comparable sample group. Similarly, Bean, Kiely et al. (2002) reported 1.6 m/s as maximal gait speed, obtained after the first two metres in a ten metre test, in a group of mobility limited older people, aged 73 years. Henwood & Taaffe (2005) measured maximal gait speed over a 6 metre distance and found that in their cohort of 70 year old participants, maximal gait speed was 1.7 m/s. Taken together, these studies present an average maximal gait speed of 1.67 m/s for people with an average age of 76 years. Given that the current study's control group was more than ten years younger, a faster speed of 1.9 m/s is plausible.

The stroke group demonstrated a maximum gait speed of 1.0 m/s. Ouellette et al. (2004) and Polh et al. (2002), have reported maximum pre-training walking speeds of approximately 0.8m/s in intervention trials involving stroke participants of similar ages to the current study. More recently, Milot, Nadeau & Gravel (2007) reported maximal speeds of 1.26 m/s in their stroke sample, whose mean age was 60 years, when measured over a five metre distance. Therefore, these studies would suggest that there is a range of maximum gait speed in stroke participants. The speed of 1.0 m/s found in the current study falls within this range.

While none of these studies compared gait speeds between those with stroke and matched controls, results from all the previous studies would suggest that people with stroke are approximately half as fast in performing maximal gait tasks as those who have not had a stroke. The results from the groups in the current study agree with this finding.

5.3.2 Stair climb

Stair climbing speed was also assessed after instructions were given to reach the top of the ten stairs as fast as safely possible. The current study's results demonstrate differences between the two groups with the control group climbing the steps at a significantly faster rate (2.3 steps/second or s/s) than the stroke group (1.1 s/s). A regular flight of stairs was used in an attempt to ensure community relevance. Stair dimensions and subsequent distances used in other studies are rarely provided, however, making comparisons tricky. Despite this problem, when compared to other studies involving a similar set up of either eight or ten steps, the current study's control group speed was faster than other results. For example, Suzuki et al. (2001) found that in their group of older adults with a mean age of 75 years, stair climbing speed was 1.6 s/s. Similarly, Bean, Kiely et al. (2002) and Sayers et al. (2003) reported a stair climbing speed of 1.6 and 1.4 s/s, respectively; in groups whose mean age was 73 years. The younger age of the current study would most likely contribute to the faster stair climbing speeds noted in the current study.

The stroke group stair climbing speed of 1.1 s/s is also slightly faster than what has been reported by other researchers. Weiss et al. (2000) reported a speed of 0.8 s/s but had conflicting reports on the number of steps used, referring to eight and eleven steps, and also reporting that both stair ascent and descent were timed, making their results difficult to interpret. LeBrasseur et al. (2006) tested a stroke group with a mean age of 66 years and found that 10 steps were climbed in 14.8 seconds, for a speed of 0.8 s/s. However, these authors did not encourage stair climbing to be performed as quickly as safely possible, as was done in the current study. As such, direct comparisons with the current study's results may not be possible.

Taken together, however, these studies demonstrate that people with stroke are approximately two times slower going up stairs as compared to a similar group of people who have not had a stroke. This reflects the findings of the current study.

5.3.3 Repeated chair stands

Chair standing time was also significantly different between groups. The control group took 20 seconds to stand up and sit down ten times. Other authors have reported times ranging between 20 and 30 seconds in 70-80 year old non-stroke participants (Bean, Kiely et al., 2002; Earles, Judge, & Gunnarsson, 2001; Netz, Ayalon, Dunsky, & Alexander, 2004; Sayers et al., 2003; Suzuki, Bean, & Fielding, 2001). All of these studies used a similar chair height and participant starting position. However, in some of them, such as those by Suzuki et al. (2001) and Sayers et al. (2003), the average of two trials was taken and no mention was made of instructing the participants to move as quickly as possible. Therefore, this range of times may not all reflect the maximum power and effort possible during this task which was an aim of the current study. As well, the current study's chair stand times falling in the lower, or faster, portion of the range of times also likely reflects the younger age of the group as compared to other studies.

In the stroke group, the average time for ten chair stands was 44 seconds. This time from the current study was within the range of chair stand times obtained from other stroke sample groups tested (38-50 seconds) by LeBrasseur et al. (2006), Ouellette et al. (2004) and Weiss et al. (2000), when using a ten chair stand time. The data in the current study was positively skewed in that two recorded times were much higher than the rest. This likely reflects a floor effect in that the two most disabled participants struggled to complete the task. Floor effects have been noted by Jones et al. (1999) who proposed counting the number of chair stands performed over thirty seconds to overcome this problem. However, in agreement with the current study's predictions and in pilot testing, it was noted that, when using the testing protocol by Jones et al. (1999), participants paced themselves over the thirty second period and reflected the arguments of other authors who have suggested that this form of test is more of an endurance measure (McCarthy, Horvat, Hortsberg, & Wisenbaker, 2004). As the goal of the current study was to encourage movement that was as powerful as possible, an alternative protocol, recording the time taken to perform ten chair stands, was employed instead. Indeed, others have also used timed chair standing tests in stroke populations by using either five or ten stands (LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Ouellette et al., 2004; Weiss, Suzuki, Bean, & Fielding, 2000), although there is limited psychometric data available on this measurement protocol. Nevertheless, the results

suggest that people with stroke take about twice as long to stand up and sit down from a chair as a non-stroke group.

When comparing the stroke and the healthy older adult results in performing the three activities from all the presented studies, results from other authors are in agreement with the current study in that it appears that people with stroke are approximately 50-60% slower in performing gait, stair or chair standing tasks as compared to people of similar age who have not had a stroke.

5.4 One-repetition maximum

The current study's control group unilateral leg press 1-RM of 70.9 kg was significantly greater than the 60.1kg 1-RM and 46.4kg 1-RM of the stroke group's uninvolved and involved leg, respectively. This represents a deficit in strength of 15% between the control and uninvolved legs, 35% between the control and involved legs and a deficit of 23% between the legs of the stroke group. Although leg press 1-RM has been extensively tested in the older adult, straight comparisons with other studies are difficult due to the differing equipment, actions and set-up. Many authors have used the pneumatic leg press machine (Bean, Herman et al., 2002; Cuoco et al., 2004; de Vos et al., 2005; Fielding et al., 2002; Foldvari et al., 2000) which tests strength indirectly and uses a bilateral leg press action. Others have used stacked weights (Henwood & Taaffe, 2005; Miszko et al., 2003) or even an incline sledge (Rantanen & Avela, 1997). Bean et al. (2003) reported that the correlation coefficient between legs for power and strength in healthy volunteers was r=0.89-0.93, with both legs contributing roughly equally to the output. Therefore, for comparisons to the present study, the bilateral 1-RM leg press results have been halved to give an indication of a unilateral 1-RM. The resulting values vary with an average 1-RM of 63.6kg which is 7.6kg lighter than the results of the current study. However, the mean age of participants in these studies was 72 years. With almost a decade difference in age to the present study's mean age, it is not surprising that the current study's value is higher, given the known declines in strength with ageing (Frontera, Hughes, Lutz, & Evans, 1991; Hurley, 1995; Larsson, Grimby, & Karlsson, 1979). Cronin et al. (2003) assessed young men with a mean age of 23 years using a similar supine leg press machine to the current study and found a unilateral 1-RM of 127.1 kg. Although greater than the control group's results, the strength loss of approximately 15% per decade after the 4th decade (Frontera, Hughes, Lutz, & Evans,

1991; Hurley, 1995; Larsson, Grimby, & Karlsson, 1979) would suggest a 40% loss in strength is possible from 23 years to 65 years, justifying the current findings.

Although the reliability of the Keiser pneumatic leg press has been demonstrated (Thomas, Fiatarone, & Fielding, 1996), it is interesting to note that despite its consistent use among many researchers, as noted above, results from testing older adults varied considerably with calculated unilateral 1-RM values ranging from 46.5kg up to 108kg, even when other physiological measures, such as BMI, between participant groups were similar. In fact, one study that reported one of the higher 1-RM values (102kg) was taken from testing performed on a group of women all over the age of 70 years (Foldvari et al., 2000). Therefore, 1-RM results from studies using the pneumatic leg press machine should be considered with caution.

The deficit in 1-RM of 23% between the involved and uninvolved legs in the stroke group found in the current study is similar to reports by other researchers. Using a pneumatic leg press machine, Weiss et al. (2000) reported that hemiparetic leg press 1-RM was 34kg while that of the uninvolved side was 55kg. Unfortunately, two other studies reporting 1-RM values only tested knee extension (LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Ouellette et al., 2004). However, all authors have noted differences of between 29-39% between the legs, confirming the current study's results.

The 15% deficit between the uninvolved leg of the stroke group and the comparison leg of the control group was of note. This finding is not unexpected as many authors (Andrews & Bohannon, 2000; Hachisuka, Umezu, & Ogata, 1997; Newham & Hsiao, 2001) have provided evidence that the 'non-affected' side does also demonstrate changes after stroke, including reduced muscle activation. Indeed, the work of Harris et al. (2001) which compared the uninvolved leg in very acute hemiparetics to a comparison leg from a control group found significant deficits between legs (67%) providing further evidence of how much stroke can affect ipsilesional muscle performance. The participants in the current study, however, had suffered a stroke considerably earlier in time and would have undergone recovery and rehabilitation, improving the function of the uninvolved leg. However, a deficit of 15% still remained, likely resulting from both residual impaired ipsilateral activation as well as the effects of inactivity.

5.5 Peak power

There were significant differences in peak power output among all three legs tested regardless of the load at which they pushed. The stroke group's ability to produce peak power in the involved leg was significantly lower than that of the uninvolved leg which itself produced significantly less power than the comparison leg of the control group.

Absolute peak power of the current study's control limb was 757 W. Dugan et al. (2004) found that bilateral jump squat peak power, measured directly in younger men, was approximately 6675 W. When this value is halved to 3338 W, it can represent unilateral leg power. The current study's control group was considerably older. Since a 3-5% decline in power per year after the 4th decade has been demonstrated (Skelton, Greig, Davies, & Young, 1994), a significant decline in power would be expected when comparing the two groups. The comparison leg of the control group value of 757 W is approximately 75% lower than those of Dugan et al. (2004) and reflects the reported declines in power with increasing age. Cronin and colleagues (2003) used a supine leg press machine in their study with a similar set up, action and body position to the current study. Peak power values obtained at 50% of their younger participants' 1-RM (mean age 23 years) were slightly higher than those obtained at 50% of 1-RM in the current study's older group (932 W as compared to 706 W, respectively). Although the younger men's peak power value was greater, given the forty year difference in age between sample groups, a much larger difference would have been expected. This may be explained by the fact that in the work by Cronin et al (2003), while displacement of the load was measured directly, the force was not, which differs to the current study. These authors determined force by using the amount of load lifted and not a force plate, which may yield different force values and subsequently result in different power values. Dugan et al. (2004) noted that indirect power calculations led to a greater risk of accumulating error, subsequently reducing the validity and reliability of the derived power value. These researchers compared three different power data collection systems that included two procedures that collected only one variable and then used indirect calculations and one procedure up that measured both force and displacement directly. The system that directly measured the variables consistently led to significantly higher peak power values when compared to the other data collection systems. Therefore, when considering the above factors, the peak power value obtained in the current study is justified.

Despite the support from studies investigating power in the young adult, the absolute value of peak power found in the current study's control group is higher than values reported by a number of other researchers investigating power in the older adult. A number of factors were thought to contribute to this finding.

Firstly, the starting and ending body positions differed from those of the current study. In the work of Bassey & Short (1990), Bassey et al. (1992), Earles et al. (2001), de Vos et al. (2005) and Fielding et al. (2002), power was assessed with participants performing leg actions while in a sitting or semi-recumbent position. Because of the constant sitting position, the contribution of hip extensors would be compromised, possibly limiting full force or power potential. The current study assessed participants supine which allowed for greater excursion from the starting position of hip and knee flexion, resulting in greater contribution of all the extensors to the action.

Secondly, the action used in testing would have also influenced absolute force, velocity and subsequently power values. In both the Nottingham flywheel used by some authors (Bassey et al., 1992; Skelton, Greig, Davies, & Young, 1994) and the Keiser pneumatic machines used by others (de Vos et al., 2005; Earles, Judge, & Gunnarsson, 2001; Fielding et al., 2002), participants never lost contact with the foot plate. As Wilson et al. (1993), Cronin et al. (2001) and Siegel et al. (2002) argue, there are differences in power output depending on if the action is contained or if it involves a ballistic projection (of a bar, or of oneself). In the former set up, the action and effort stops at the end of range. In this situation, the foot plate decelerates over the latter part of the movement such that it will reach zero velocity at the end of the action. As a consequence, high force levels are only achieved in a small range with lower velocity leading to lower power values (Wilson, Newton, Murphy, & Humphries, 1993). In the current study's set up, participants pushed off the foot plate and projected themselves along the runners of the platform, thereby not having to decelerate. Consequently, larger accelerations, forces and velocities were exerted throughout the entire movement. High forces could be generated in lighter-load situations involving this ballistic action because of the high acceleration rates throughout the movements. Confirming this point, Cronin and colleagues (2001) also used ballistic allowing equipment and reported higher velocities in ballistic actions when compared to non-projection, more traditional actions which resulted in peak power that was 9.1% greater than when no projection was involved. Other authors, not using similar equipment but still involving projection

or ballistic actions have also reported higher power values than those involving traditional actions (Newton et al., 2002; Rantanen & Avela, 1997). In addition, this study's set up allowed a ballistic effort to be maximal since participants were fully supported on the platform and did not need to be concerned about stopping their motion which would encourage confidence to perform maximally.

Thirdly, the differing equipment used among studies would influence both power output as well as its recording. For example, the Nottingham flywheel power rig measures power indirectly and requires several calculations including the velocity of the flywheel and the amount of rotation, the moment of flywheel inertia together with its frictional energy loss per revolution, the force exerted by the return spring at the beginning and end of push, as well as the time taken to complete the push, to finally determine a power value (Bassey & Short, 1990). Peak power of the studies using this equipment ranged from 67W to 216W with a mean unilateral peak power of 111W. The Keiser bilateral pneumatic leg press machine requires slightly simpler calculations but still involves some indirect calculations to determine work and power (Thomas, Fiatarone, & Fielding, 1996). Unilateral peak power values from this system have been calculated to be approximately 159W. Thomas et al. (1996) used both of these data collection methods for assessing leg power in younger women and found that the flywheel system recorded unilateral leg power values that were over two times higher than the pneumatic machine, confirming that different equipment can yield different results.

Some authors have normalised peak power to body mass in the athletic and older adult populations (Baker & Nance, 1999; Bassey et al., 1992; Rantanen & Avela, 1997; Sleivert & Taingahue, 2004; Young, McLean, & Ardagna, 1995). The close matching in the current study for age, gender, mass, BMI and stature between groups resulted in no differences across groups. Therefore, when comparing peak power across legs, the need for this adjustment was not apparent in this portion of the data analysis.

Power deficits of 33-39% between the control group's comparison limb and the stroke group's uninvolved limb were seen across loads. The findings that power differed significantly between the uninvolved hemiplegic leg and that of the comparison leg reflects the pattern seen with strength (Andrews & Bohannon, 2000; Harris, Polkey, Bath, & Moxham, 2001) and is not surprising.

Of note is that the 33-39% difference in power between the stroke uninvolved and control limbs is greater than the earlier reported 15% difference in strength between the same limbs, suggesting that power is even more affected after stroke. No other studies to date have compared the uninvolved limbs of a stroke group to a comparison limb of a control group but the same pattern that shows greater deficits in power than in strength in the stroke population have also been demonstrated in the work of Clark et al. (2006) when comparing the involved limbs of a stroke group to the comparison limbs of a control group and in the work of LeBrasseur et al. (2006) when comparing the involved and uninvolved limbs of a hemiparetic group.

Peak power values of the stroke group's uninvolved and involved legs were 506W and 240W respectively. The involved leg of the stroke group produced 68% less power than the control group comparison leg. This finding is also supported by the work of Clarke et al. (2006) who found that, when testing knee extension power of the involved side in a stroke group and comparison leg of a control group through isokinetic methods, leg power deficits between the groups were 44.8%. The current study's results also demonstrate that the involved leg of the stroke group produced 53% less power than the uninvolved leg. Absolute values from LeBrasseur and colleagues (2006) and the current study differ due to the difference in tested actions (leg extension as compared to leg press) as well as the equipment differences as discussed previously. However, their values of 115W for the uninvolved leg compared to 65W for the involved leg, while lower than this study's, reflect a 43.5% deficit between legs. The power values from this study's stroke group across loads resulted in deficits of 49-55% between involved and uninvolved legs and are supported by previous findings.

The stroke group's reduced power from the current study reflects the physiological changes that occur as a result of the stroke. The cortical damage, particularly in the motor areas, leads to deficits in voluntary muscle activation (Andrews & Bohannon, 2000; Harris, Polkey, Bath, & Moxham, 2001; Newham & Hsiao, 2001; Riley & Bilodeau, 2002) and a subsequent loss of functioning motor units (McComas, Sica, Upton, & Aguilera, 1973). The remaining motor units demonstrate reduced discharge rates and prolonged contraction times (Frontera, Grimby, & Larsson, 1997; Gemperline, Allen, Walk, & Rymer, 1995; Rosenfalck & Andreassen, 1980). As such, the inability to fully activate existing motor units that are already compromised in recruitment and firing behaviour will affect both force and power generation. Furthermore, reductions

in both muscle CSA (Metiko, Sato, Satoh, Okumura, & Iwamoto, 2003; Ryan, Dobrovolny, Smith, Silver, & Macko, 2002) and in muscle fibre numbers (Frontera, Grimby, & Larsson, 1997; Hachisuka, Umezu, & Ogata, 1997) would further contribute to the force deficits seen. As well, the reduced fascicle lengths (Li, Tong, & Hu, 2007) and increased passive mechanical stiffness in the muscle (Rydahl & Brouwer, 2004; Sinkjaer & Magnussen, 1994) could contribute to decreased muscle excursion and further affect the velocity generating capabilities. Hence, the production of both muscle force and velocity will be compromised. These factors provide the likely mechanisms associated with the current study's findings of significantly reduced muscle power production capabilities after stroke.

5.5.1 Optimal load for peak power

The current study investigated peak power output at varying external resistances and the results demonstrated that peak power was significantly greatest at 30% of 1-RM, followed by 50% of 1-RM and then 70% of 1-RM in the control group as well as in the uninvolved leg of the stroke group. There is no known published literature to date on optimal loads for facilitating maximum muscle power output in the stroke population. Furthermore, there is conflicting information available in the younger and older adult literature regarding optimal load.

The results of this study agree with those of a number of authors investigating peak power production in the younger adult who have reported a light optimal load. This has been shown in both the upper limb in single and multiple joint actions (Kaneko, Fuchimoto, Toji, & Suei, 1983; Moss, Refsnes, Abildgaard, Nicolaysen, & Jensen, 1997; Newton et al., 1997) and in lower limb leg extension and jumping tasks (Cormie, McCaulley, Triplett, & McBride, 2007; Dugan, Doyle, Humphries, Hasson, & Newton, 2004; McBride, Triplett-McBride, Davie, & Newton, 2002; Wilson, Newton, Murphy, & Humphries, 1993).

However, not all studies agree with the current findings. Generally, authors investigating muscle power in the older adult have reported that power is maximised at higher percentages of 1-RM (65%-75% of 1-RM) (Bean et al., 2004; Bean, Kiely et al., 2002; Cuoco et al., 2004; Fielding et al., 2002; Foldvari et al., 2000; Sayers et al., 2003). All of these authors used participants in their seventies who were of similar ability to the current study's control group. All of them used a Keiser pneumatic leg

press machine, as discussed earlier, and tested for bilateral leg press power. While their results appear consistent, some factors must be considered. The non-randomised power testing protocol, for example, was similar in all the studies. It began testing at 40% of 1-RM with progressive increments of 10% of 1-RM and later of 5% of 1-RM until 90% of 1-RM was reached. Since there was no randomisation of load and only one trial at each load was performed, it is possible that there was a learning or familiarisation effect so that by 70% of 1-RM (ie 4 attempts), the participants had had the opportunity to practice and were optimising power production.

Another reason for the differences in optimal load may be due to the differences in action tested. The equipment set up used in the studies presented above had the participant extend his or her legs while remaining in contact with the foot plate during the entire action. The modified supine leg pres machine used in the current study enabled ballistic actions, allowing the participant to project him or herself off the foot plate, optimising velocity and acceleration. This system would therefore lead to peak power with greater velocity input (ie at lower loads) (Cormie, McCaulley, Triplett, & McBride, 2007; Newton & Kraemer, 1994; Wilson, Newton, Murphy, & Humphries, 1993). If the action required no deceleration, velocity would be contributing more to output so peak power would be achieved through higher velocity. High forces are generated in light-load situations with ballistic movements because of the high acceleration rates throughout the movements (Cormie, McCaulley, Triplett, & McBride, 2007). While Baker and colleagues (2001a, 2001b) have suggested that to move high loads, high forces and recruitment of high threshold motor units are required, it is possible to obtain high tension and recruitment of high threshold units even during actions with lighter loads, provided that acceleration is sufficiently high (Moss, Refsnes, Abildgaard, Nicolaysen, & Jensen, 1997).

Optimal load assessments using ballistic actions have been tested in both younger and older men and it has been demonstrated that power was maximised at intermediate percentages of 1-RM (30%-60% of 1-RM). Newton et al. (2002) measured power across a young and older group of men during a squat jump task. They found that when they measured power at loads of bar weight (17kg), 30% and 60% of 1-RM, power was highest at 60% of 1-RM. However, when these values were plotted, the power profiles of the young and old men differed markedly in that the young group (aged 30 years) produced considerably more power as the load was increased from 30% to 60% of 1-

RM. The older men (aged 61 years), on the other hand, showed only marginal increases in power between 30% and 60% of 1-RM, suggesting that older men had more difficulty in generating power at higher loads.

The difference in findings and optimal loading levels may, therefore, also be due to the different participant characteristics. It has been shown that weaker or untrained participants generally achieve peak power at lower loads (Baker, Nance, & Moore, 2001a, 2001b; Earles, Judge, & Gunnarsson, 2001; Stone et al., 2003). These power deficits would be even greater if 1-RM values were normalised to the stronger group. Age and stroke related decreases in muscle force are well documented (Adams, Gandevia, & Skuse, 1990; Andrews & Bohannon, 2000; LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Vandervoort & McComas, 1986; Young, Stokes, & Crowe, 1985) and propose that these people do not have the muscle cross sectional area, or cross-bridge availability to generate as much force. Therefore, with a lower amount of force generating capacity, they achieve lower absolute power values but do so by increasing the contribution of velocity. Consequently, although they may be able to perform powerful activities that require lower force intensities, albeit with declines in actual activity performance (ie speed in gait, climbing stairs), they may be even more debilitated in performing powerful tasks requiring a notable amount of force (ie chair stands, getting up from the floor). As well, with less functioning motor units in both the aged (Campbell, McComas, & Petito, 1973) and after stroke (McComas, Sica, Upton, & Aguilera, 1973) power is more likely generated via rate coding versus recruitment which, again, would suggest a dependence on a more velocity-related component. The findings of peak power generation at lower loads from this study would reflect these changes in muscle function.

Unlike the control limb and the uninvolved limb, peak power of the stroke group's involved limb did not significantly decline as the load increased from 50% of 1-RM to 70% of 1-RM. There is no literature to date that has explored power output against different loads in the stroke population that can be used to explain this finding. However, it may reflect a floor effect of the testing in this group. As these participants were significantly compromised in force production already, it is likely that, at loads of 50% of 1-RM, they were already approaching the lower limit of power production and increasing the load to 70% of 1-RM did not significantly alter this reduced power output.

5.6 Correlations with activity performance

Significant associations were found between peak power measures and the performance of the activities. Although all the associations were similar across all three loads of 1-RM, there was a trend for a stronger correlation being observed when using peak power values generated at the lowest load (30% of 1-RM). This finding is supported by others (Cuoco et al., 2004; Sayers, Guralnik, Thombs, & Fielding, 2005) who have noted that in the older adult, for tasks requiring lower percentages of maximum strength to perform, power produced against lower loads have stronger associations. Therefore, the results pertaining to correlations using peak power obtained at 30% of 1-RM will be the focus in the following sections.

Some authors have calculated correlations using strength and power relative to body weight (Baker & Nance, 1999; Bassey et al., 1992; Kukolj, Ropret, Ugarkovic, & Jaric, 1999; Skelton, Greig, Davies, & Young, 1994; Sleivert & Taingahue, 2004; Young, McLean, & Ardagna, 1995). Similar to some authors (Baker & Nance, 1999; Markovic, 2006), normalising for body mass did significantly change the strength to performance relationships. As Baker and Nance (1999) suggest, because acceleration is based on force/mass, participants who are strong per kilogram of body mass should theoretically perform timed tasks well. Power expressed relative to body mass was also found by Baker & Nance (1999) to produce stronger correlations than using absolute values in an athletic group. There continues to be some debate, however, as to the need for and the most appropriate normalisation method. The current study employed a ratio standard by using body mass. This method has been questioned and the use of an allometric formula for obtaining an index of muscle strength as well as an index of movement, both independent of body size has been advanced (Jaric, 2002). Nevertheless, in the current study, correlations using relative power were similar to those using absolute power values and will be presented in the following sections.

5.6.1 Gait speed

Both the stroke and control groups demonstrated significant associations between gait speed and peak power. The correlation of the control group's unilateral leg press peak power, generated at 30% of 1-RM, to maximal gait speed was r=0.74. This correlation is similar to the results of other studies that have also found moderate to strong associations between these variables in the older adult. Bassey and colleagues (1992) reported that unilateral leg power relative to body mass was highly correlated to

walking speed (r=0.80) in their cohort of very frail chronic care hospital residents. Rantanen & Avela (1997), whose power assessment system also involved ballistic actions, found that the mean correlation coefficient between peak power and maximal walking speed in their group of 80-85 year old men and women was r=0.66. Moderate correlations were also found between pneumatic machine measured bilateral leg press power and habitual and maximum gait speed by Bean, Kiely et al. (2002), Bean et al. (2003) and Cuoco et al. (2004) who reported r values of 0.59-0.67.

With regards to the stroke population, involved knee extension power and habitual gait speed has recently been shown to have a significant correlation of r= 0.35 (LeBrasseur, Sayers, Ouellette, & Fielding, 2006). This is lower than the findings of the current study (r=0.64-0.69). However, LeBrasseur and colleagues (2006) assessed only knee extensor power which would make comparisons to the current study's use of whole leg press power difficult. As well, they tested habitual gait speed and did not emphasise its performance be as fast as possible. Thus, their gait task may not have involved a large degree of power producing capabilities, making comparisons to the current results risky.

Uninvolved leg power was found to not be associated with maximal gait speed in the current study. Although part of their assessment, LeBrasseur et al. (2006) did not report on the correlation between gait speed and uninvolved leg power, making any comparison to the current findings difficult. However, these authors did report uninvolved leg 1-RM and its relationship to gait speed. The uninvolved side's 1-RM was found to have a fair correlation with gait speed (r=0.42-0.48). Previously published information regarding the contribution of the uninvolved limb to gait appears to be conflicting with non-involved knee extensors being shown to have either no significant correlation with gait speed (Flansbjer, Downham, & Lexell, 2006; LeBrasseur, Sayers, Ouellette, & Fielding, 2006; Nakamura, Watanabe, Handa, & Morohashi, 1988) or low correlations of r=0.33-0.42 (Kim & Eng, 2003; Suzuki, Imada, Iwaya, Handa, & Kurogo, 1999). Either way, taken together, these results would suggest that the strength and power of the uninvolved limb is much less an influencing factor on gait speed than the strength or power of the involved limb in hemiparetic participants.

5.6.2 Stair climb

Both the involved leg of the stroke group and the comparison leg of the control group had significant associations between stair climbing speed and peak power. The correlation coefficient of the control group's peak power to stair climbing speed of r=0.64-0.68 is supported by other authors (Bassey et al., 1992; Bassey, Tay, & West, 1990; Bean, Kiely et al., 2002; Bean et al., 2003). Bassey et al. (1990) and Bassey et al. (1992) found very high correlations between leg power and stair climbing in old and young participants respectively (r=0.81, r=0.86). Bilateral leg press power values have also been found to be significantly associated with stair climbing time with values ranging from r=0.52 to r=0.62 (Bean, Kiely et al., 2002; Bean et al., 2003) reflecting the moderate correlation found in the current study.

In contrast, other authors have reported no relationship between leg power and stair climbing (Cuoco et al., 2004). Stair climbing, because of the vertical rise component, has been suggested as requiring a greater percentage of maximal strength to perform and would, therefore, not demonstrate as strong a relationship with peak power obtained from a lower load using higher velocities (Cuoco et al., 2004). However, this reasoning would suggest that peak power at higher loads, with a heavier force component, would have stronger correlations. The results of the current study do not support this proposition since power and stair climbing correlations across all three loads were comparable, peaking at the lower load of 30% of 1-RM.

The stroke group showed good correlations (r=0.72) of involved leg peak power and stair climbing speed. That the strongest correlation occurred with power obtained at 30% of 1-RM suggests that this group relies on the contribution of velocity to perform the task as powerfully as possible. LeBrasseur et al. (2006) reported that in their cohort of hemiparetics, involved knee extension power was fairly associated with stair climbing ability (r=0.49). No mention was made as to whether the participants were instructed to climb the stairs as quickly as possible. Therefore, it is difficult to ascertain whether the differences in correlation coefficients between studies are due to methodological differences such as the action and muscle tested or the instructions given. Nevertheless, the significant associations do confirm the findings of the current study that leg power is an important factor in stair climbing ability in the stroke population.

5.6.3 Repeated chair stands

The control group had moderately negative associations between chair standing time and lower limb peak power (r=-0.57) while neither leg of the stroke group showed any significant association.

Bassey et al. (1992) also reported moderate associations (r=-0.65) between leg power and chair standing. Similarly, Skelton and colleagues (1994) also found that power influenced chair rise time. Using the Nottingham power leg extensor rig, these authors reported that unilateral leg power, when summed, had moderate correlations of ρ=-0.47 in older men and women between 65 and 89 years.

The present study's association of r=-0.57, while significant, is lower than the others found between power and gait (r=0.70) and power and stair climbing (r=0.64) which is a pattern also noted by Bassey et al. (1992). This may suggest that leg power is less of a determining factor in performance of chair stands than in the other tested tasks. This thought is supported by others who have noted a lack of association between bilateral leg power and repeated chair rising times (Bean et al., 2003; Cuoco et al., 2004). Chair standing has been argued as being more related to force production than velocity. As well, the repeated nature of the test may reflect more of an endurance test, negating the influences of shorter term differences in rate of force production between the participants (Bean et al., 2003). Indeed, the chair standing task took, on average, five times longer to complete than either of the other two activities and even longer than each of the power testing leg press actions. In fact, Netz et al. (2004) have reported significant associations between repeated chair standing and aerobic capacity as compared to isokinetic leg extension strength when tested at 180º/s. The employment of a single chair stand may have yielded a higher correlation.

In the work of McCarthy et al. (2004) in older women, the contribution of specific muscles to chair standing was investigated and these authors found that ankle plantarflexor strength contributed most to chair stand performance followed by hip flexor strength and only then knee extensor strength. In the current study, as well as many of the lower limb power studies in older adults, hip flexor strength and power was not measured. Incorporating this measure may have increased the association.

There were no associations between unilateral leg press power and chair standing time for either the involved or uninvolved leg of the stroke group. Likewise, LeBrasseur and colleagues (2006) also found that power of the involved or uninvolved limb was not associated with repeated chair stands. There are likely a number of explanations for this finding. Firstly, the mixed results from studies in a non-stroke population would suggest that this task is not highly correlated with leg power to being with. Secondly, standing from a chair, particularly without the use of arm support, is a skill that would have been very challenging to some of the participants who would not have had practice of this type of task in their daily life. As such, it is difficult to exclude the skill factor involved and the influence of compensatory movements. The work of Alexander et al. (2001) would suggest that leg use in chair standing performance may be influenced by movements of the torso. The repeated chair stand test only specifies the starting position and the non-use of arms with no mention of the trunk. Therefore, clear correlations between chair stands and leg function may be unlikely considering the possible confounding variables. Thirdly, in hemiparetic patients, it was shown that in addition to ankle plantarflexors and knee extensors, involved ankle dorsiflexors were also significantly involved in fast-paced chair standing (Lomaglio & Eng, 2005). This muscle group was not tested in the current study and could have influenced the correlations. Fourthly, it was difficult to control for the symmetrical use of both legs during this task. Due to the asymmetrical symptoms of stroke, it is unlikely that the hemiparetic participants used their affected leg as much as the unaffected leg. Cheng et al. (1998)'s findings of significant asymmetrical body weight distribution in hemiparetic participants performing sit to stand actions would support this proposition. The significance of this impairment was further highlighted by Lomaglio & Eng (2005) who found a significant correlation between asymmetry and fast-paced chair standing. Since, in this study, leg power was measured unilaterally, it is not entirely surprising that the values do not correlate with a bilateral task.

In interpreting all the correlational results from the current study, it is important to consider the level of ability of the participants. Both the control and stroke groups were at a reasonable level of ability given the inclusion criteria, ages, FMA scores and results in the tested activities. In a number of studies investigating the relationship between power and function, quadratic and logarithmic models explained more of the variance in gait speed, stair climbing speed and chair standing time than did the linear models (Bean, Kiely et al., 2002; Bean et al., 2003; Cuoco et al., 2004; LeBrasseur, Sayers,

Ouellette, & Fielding, 2006) supporting the proposition of a curvilinear association with impairments such as strength and power with activity performance. Therefore, there is likely some critical threshold of strength and power necessary for the performance of activities above which will demonstrate less association (Sayers et al., 2003). Indeed, in the study that has found the highest correlations between leg power and function (Bassey et al., 1992), participants were frail, mobility limited elderly hospital residents, most likely much closer to the threshold. Rantanen & Avela (1997) have suggested that the stronger *r* values seen in more debilitated men and women point to the fact they have less power and as such, are situated close to the critical power level needed to perform activities. For this study's control group participants, their amount of available leg power could have been above the power threshold and, as such, may have not be the limiting factor for the performance of the tasks. Additionally, for the stroke group participants, leg power alone would not have been the only limiting factor as the contribution of coordination, balance, and sensory deficits could have also influenced the performance of the tasks.

5.7 Limitations

Certain limitations need to be considered when viewing the results of this study. Differences in testing procedure, equipment and data collection systems, functional relevance and sample numbers must all be regarded and will now be discussed.

The moderate correlations found between strength and power and the performance of the activities may have been due to the nature of strength and power testing. Whole leg press 1-RM and power were tested. As such, the specific contribution of the different joints and muscles cannot be determined. Bean et al. (2002) and Suzuki et al. (2001) reported that distal power generation at the ankle and knee was more closely related to activity performance as it pertains to gait and balance. As well, the work of Nadeau, Gravel, Arsenault & Bourbonnais (1999) found that, for fast walking in a hemiparetic sample, involved hip flexor strength was a significant variable (r=0.88). This was most recently confirmed by Milot et al. (2007) who pointed to increased activity of the hip musculature in fast gait. Similarly, hip flexors and other muscles such as the knee flexors and ankle dorsiflexors have been found to be involved in stair climbing and repeated chair stands in people with or without stroke (Kim & Eng, 2003; McCarthy, Horvat, Hortsberg, & Wisenbaker, 2004). The current study did not allow for either kinematic analysis or specific assessment of these key muscle groups. Associations

may have been more robust had specific measures of ankle, knee and hip strength and power been included in the analysis. However, using a leg press action allowed the assessment of a movement pattern that was more generalisable to a number of everyday activities and was able to provide a good overview of general leg function.

The current study used a technique established in the younger and athletic samples to evaluate leg power in an older adult and stroke group. No previous studies have used this system. Therefore, comparisons between studies of similar participants are difficult and its reproducibility at other centres is limited. However, the system used in the current study allowed the direct measurement of force and displacement, and, through a differentiation algorithm to determine velocity, resulted in a simply calculated power value. As Dugan and colleagues (2004) have argued, direct measurement minimises accumulating error, thereby improving the validity and reliability of the measure. As well, particularly for the participants in the stroke group, this system ensured a safe environment that allowed them to produce maximum effort in an attempt to achieve their peak power potential.

The supine leg press machine had participants lie supine and allowed them to exert maximum effort and produce peak power under safe circumstances. However, position and posture in assessment and training should be specific to the goal. Wilson et al. (1996) found that after eight weeks of training, the improvements in performance were greatest in the tests involving postures that were used during training and recommended selecting exercises and assessments in which the posture closely resembles that of the movements in question. Therefore, the power values obtained in this study may not have been reflective of real-life activity-based power capabilities, especially when participants were upright and having to synergistically maintain posture, coordination, and balance all of which are significant issues in both of these populations.

Even though significant relationships were found between leg press power and the performance of the activities, it is important to remember that these do not confirm cause and effect. Therefore, it cannot be assumed that stronger or more powerful legs will directly result in improved function. Interventional trials addressing strength and even power training have found mixed results in the improvement of function (Bean et al., 2003; Ouellette et al., 2004; Sayers et al., 2003; Weiss, Suzuki, Bean, & Fielding, 2000) which would suggest that strength and power only partly contribute to activity

performance. Further work into this area will yield a better understanding of this relationship.

Finally, while the results of this study demonstrated significant differences in power output across the different loads, determination of even more detailed power profiles might be valuable. With measuring power at 30%, 50%, 70% of 1-RM, for example, the power response at 40% was missed, potentially leading to an incomplete result. However, data collection occurred over two sessions, taking at least ninety minutes. The addition of testing at further levels in the current study was beyond the ability of the participants and also the resources available for the study.

CHAPTER 6 – SUMMARY AND CONCLUSIONS

6.1 Study summary and conclusions

Stroke is the leading cause of disability worldwide. It often leads to mobility limitations resulting from deficits in muscle performance. While reduced muscle strength and even reduced speed of muscle contraction have been reported, little is known about the power generating capability of people after stroke and its relationship to mobility. Research in other populations has found that measures of muscle power may have a greater association to activity performance than do measures of muscle force alone. Consequently, in an attempt to optimise power, investigators have focused on identifying ideal parameters within which to train for power. One such parameter is the identification of the loading level at which maximal power is generated. Literature reporting optimal loads from both young athletic and healthy older populations has yielded mixed results, therefore, making the applicability to a hemiparetic population difficult.

The purpose of this study was to investigate muscle power performance at differing loads and to determine at what load muscle power is best elicited in hemiparetic and age and gender matched control sample groups. A secondary aim was to ascertain whether there is a relationship between the muscle power values obtained and the performance of activities including gait, stair climbing and standing from a chair.

Twenty nine hemiplegic volunteers and twenty nine age, gender and BMI matched controls were evaluated. Involved and uninvolved legs of the stroke group and a comparison leg of the control underwent testing. Leg press muscle power was measured using a modified supine leg press machine at 30%, 50% and 70% of a determined 1-RM load. Participants were positioned on the leg press machine and asked to push, with a single leg, as hard and as fast as they could. Data was collected via a mounted force plate and a linear transducer connected to a movable platform on which participants lay. From these variables, power was calculated. The activities were timed while being performed as fast as possible.

The results showed that peak muscle power values differed significantly within the involved, uninvolved and control legs. The control leg produced significantly more power than the uninvolved leg (33%) and the uninvolved leg produced significantly more power than the involved leg (53%). Peak leg power in all three leg groups was

greatest when pushing against a load of 30% of 1-RM and decreased as the load increased. Significant associations were found between involved leg peak power and gait speed and involved leg peak power and stair climbing whereas no correlation was found involved paretic leg peak power and chair stands. The control group leg peak power had significant associations with the performance of all three activities.

These findings have several clinical implications. Firstly, despite concerns for safety and capability, powerful muscle contractions and powerful mobility tasks can be safely performed by these populations as no adverse events were reported. However, it should be noted that while power assessments were performed successfully on these participants, they were screened before testing, ensuring the appropriateness of the participants to the tasks.

Secondly, while differences between the performance of the involved leg and that of the control leg are expected, it is important to note the significant differences between the 'uninvolved' limb and that of the control limb. This study provides further evidence that although stroke primarily affects the body asymmetrically, it still affects it bilaterally. This finding reinforces the need to question data that refers to the less affected contralateral limb in hemiplegia as a normal comparison and also confirms that strength and power assessment and training need to be undertaken on both sides.

Thirdly, the assessment method for power must be considered when examining the results of strength and power testing as inter-system measures and results have been shown to be quite variable. The dissimilar results in optimal load across studies likely reflect the methodological differences. Therefore, understanding the implications of ballistic as compared to non-projection actions as well as the implications of measuring variables directly as compared to deriving them from indirect measures is necessary to appreciate the findings from the differing studies.

Fourthly, even the best correlation coefficient value of r=0.7 obtained in this study, if converted to a coefficient of determination to interpret the meaningfulness of the relationship, would suggest that unilateral leg press power only explains about 50% of the performance of the three activities. Therefore, it is important to appreciate that, especially in these populations, many other factors are likely to be involved and need to be considered. In chronic and complex conditions such as stroke and ageing, several

additional psychological, emotional and physical impairments may impact on the ability to carry out an activity. For example, balance, sensory impairments, self-perceived ability and confidence, depression and pain could all substantially influence physical mobility. Of note is that self-efficacy has emerged as a strong predictor of measured activity performance, suggesting that interventions with goals of increasing activity and participation should recognise and include strategies aimed at addressing all the potentially limiting impairments.

Lastly, specificity must always be considered. Strength and power training are not simply a matter of using some generalised form of resistance training to produce adequate physical loading and muscle tension. The principle of specificity of training is central to the entire issue. Muscle power represents a continuum that has specific components (force and velocity) whose relative contributions make it more associated with specific tasks while still yielding similar 'power' values. What is critical is the ability to exert the appropriate force and speed characteristics for a given activity. As such, assessments should consider the individual as well as the task and its power requirements in order to reflect the demands of the task. For example, tasks requiring higher velocities against lighter loads such as gait, running, jumping and stair climbing will have different force and velocity contributions than actions requiring high velocities against heavy loads such as in rising from the floor and lifting heavy objects. As such, optimal load will depend on the task. Interestingly, many progressive resistance training programmes in the stroke population have not demonstrated large effects for improvement in activity performance while task specific training has shown better outcomes. This may be due, in part, to the fact that many activities have lower load and higher velocity components. Therefore, by encouraging participants to move and subsequently contract muscles at more appropriate and relevant speeds against lower loads, a task-specific form of power training actually does occur.

6.2 Recommendations for further research

Given the findings of the current study, it would be interesting to investigate several other areas. Firstly, the results suggest that, in the stroke and older adult populations, maximum power is produced at lighter loads. This proposition would need to be tested in an intervention trial. By assessing peak power outcomes after training at different loads, optimal training load in these populations can be confirmed.

Secondly, the current study's results also suggest that there are significant associations between peak power and performance of certain activities. Determining if power training results in not only improved power production but also in improved performance of mobility tasks would more fully explain this relationship. By combining these two recommendations, the notion of specificity could also be addressed by determining if training at lighter loads and higher velocities would yield different outcomes in a task such as gait as compared to training at heavier loads and slower velocities.

Finally, because of the expense and complexities of muscle power testing, work focussing on establishing a clinically applicable measure of leg power would improve its profile in the assessment and rehabilitation of people who have suffered a stroke.

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APPENDICES

Appendix A

MEMORANDUM

Student Services Group – Academic Services

To:	Peter McNair
From:	**Madeline Banda**
Date:	03 August 2004
Subject:	04/133 Muscle power after stroke

Dear Peter

Thank you for providing clarification and/or amendment of your ethics application as requested by AUTEC.

Your application is approved for a period of two years until 3 August 2006.

You are required to submit the following to AUTEC:

- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda
Executive Secretary
AUTEC

Cc: Verna Stavric

From the desk of ...
Madeline Banda
Academic Services
Student Services

Private Bag 92006, Auckland 1020
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Tel: 64 9 917 9999
ext 8044
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AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

MEMORANDUM

Academic Services

To:	Peter McNair
From:	**Madeline Banda**
Date:	14 October 2004
Subject:	04/133 Muscle power after stroke

Dear Peter

Your request for approval for amendments to your original application was considered by AUTEC at the meeting on 11 October 2004.

Your application is approved for a period of two years until 11 October 2006.

You are required to submit the following to AUTEC:

- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda
Executive Secretary
AUTEC
Cc: Verna Stavric

From the desk of ...
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Student Services

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Appendix B

Auckland University of Technology »

Faculty of Health

VOLUNTEERS REQUIRED

FOR

MUSCLE POWER AFTER STROKE STUDY

If you have had a stroke no less than 6 months ago and are walking for short distances, you are invited to participate in a study to determine how strong your leg muscles are.

The research will involve two sessions of no more than 2 hours in total. Both sessions will take place at the Physical Rehabilitation Research Centre, School of Physiotherapy at the Auckland University of Technology on Akoranga Drive. (Room AA 111).

Parking will be provided and assistance with travel costs will be offered.

To participate, you need to be able to walk for short distances and answer some simple questions.

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Appendix C

AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

Volunteers needed for Muscle Strength Study

Volunteers are needed for a study into the muscle strength of people who have had a stroke as well as people who have not had a stroke.

Verna Stavric, a physiotherapist based at AUT's Akoranga Campus in Northcote, is comparing how muscle changes seen after a stroke compare to those seen with the normal ageing process in people who haven't had a stroke. People who are between the ages of 60 to 85 are especially needed.

Once differences are pinpointed, better rehabilitation techniques may be developed to improve mobility and strength in people who have had a stroke.

Volunteers are needed to take part in several tests over 2 sessions at the Northcote campus.

During this time, a volunteer will be asked to perform the following:

- review the study and sign a consent form
- answer some questions about general health
- fill in a questionnaire about leg function
- perform 4 tasks: walk 10 meters, stand up from a chair, walk up a set of stairs and straighten the leg against a machine to test leg strength and speed

This first session should take about 1 hour to 1 ½ hours to complete. Individual results will then be compared to existing information about normal values for the age and gender and presented as an individualised report.

At the end of this first session, the final session will be scheduled for another day and time soon afterwards and would only last about ½ hour.

Travel compensation can be arranged for people who drive with petrol vouchers. For those who are unable to drive, a taxi will be arranged and paid for by taxi voucher.

Collection of information will continue until the end of the year and possibly into January 2005.

For more information, please contact Verna Stavric on 917-9999 ext 7320.

Appendix D

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Participant Information Sheet

Date Information Sheet Produced: 15 November 2004

Project Title Muscle power after stroke

Invitation

You are invited to take part in a research study to examine how muscles produce power after stroke. You have been selected for this study as you meet the entry criteria of having had a stroke and are living in the community. Or you may be suitable for comparison as you have not had a stroke.

What is the purpose of the study?

This study is part of a Master of Health Science thesis. The primary aim is to determine the optimum load for muscle, affected from stroke, to produce power. Secondary aims include: a) a comparison of power data with performance of simple functional tasks; b) a comparison of power data obtained from participants with age and gender matched controls.

How are people chosen to be asked to be part of the study?

Potential participants are recruited through notices placed in the AUT Neuro rehabilitation clinic, Akoranga Campus, in the local community paper and via the Stroke Foundation local support groups. Potential participants are asked to contact an administrator at the Physical Rehabilitation Research Centre for further information. Potential participants are then sent or given the information sheet and contacted by the researcher in not less than one week. At this point they are given the opportunity to ask any further questions about the study. If the potential participant agrees to take part, an initial session at the Physical Rehabilitation Research Centre at AUT is arranged at a time and date suitable to the potential participant.

What happens in the study?

This study involves two testing sessions carried out at the Physical Rehabilitation Research Centre at the Akoranga Campus of Auckland University of Technology.

In the first session, all potential participants will be informed about the study and tests and will be asked to complete a consent form. They will then be assessed to ensure that the inclusion/exclusion criteria are met and will undergo screening tests for cognitive function and physical activity readiness as well as a test for muscle spasticity. If appropriate to participate, their personal details such as age, date of stroke, type and location of stroke will be recorded. This will be followed by a questionnaire that asks about their leg function. As well, their weight and height will be measured as will their general leg function affected by the stroke.

Participants will warm up and then be asked to lie on a modified leg press machine and, with each leg separately, try to push and lift a heavy load. When the maximum load or weight that can be lifted only once is determined, this value will be used to calculate testing loads for the final session.

Physical mobility measures will then be tested by asking participants to carry out the following tasks as quickly as possible: walking 10 metres; climbing a set of stairs; and standing up from and sitting down on a chair. All of these tasks will be timed. This is the end of the first session.

In the final session (within 1 week of the initial session), participants will once again be asked to lie on the modified leg press machine. With each leg separately, participants will be asked to lift a percentage of the maximum load they could lift from the initial session. They will be asked to push against the platform as hard and as fast as they can at 30%, at 50% and at 70% of their maximum load. During this pushing motion, the leg, from the hip down, will be videoed. There will be a rest period of a maximum of 3 minutes between repetitions.

What are the discomforts and risks?

Maximal contraction of muscle can at times lead to muscle strain. If this occurs, there may be mild to moderate tightness or discomfort when trying to use the muscle soon after the task on over the next few days following the task. Participants may also experience emotions in reaction to being asked to perform physical tasks or in relation to their stroke.

How will these discomforts and risks be alleviated?

A proper warm up (ie by performing a standardised warm up) should reduce the chance of strain. As well, there will be a mandatory rest period between the lifting to allow the muscles to recover. Additionally, a registered physiotherapist will be doing the testing and is trained in identifying potentially hazardous situations. As well, the participants perform the leg movement in a voluntary manner (ie they will have full control of their movement) and will be reminded to stop with symptoms of pain. Each participant may communicate with researchers at all times during the testing procedures and may cease participation in the study at any time. Also, there will be first aid items on hand to be used as needed (ie ice, phone). Participants will be given contact details should there be any additional discomfort following either testing session. Should emotional needs be identified, participants will be put in touch with Health and Counselling services at Akoranga Campus.

What are the benefits?

Participants will learn about their muscle strength and more specifically about their muscle power. They will also learn about their functional mobility.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of participation in this study, a participant may be eligible for accident compensation legislation, with its limitations. They will also be encouraged to make contact with the researchers to inform them of their condition and to be able to access information and recommendations for management. This may include referral to the local physiotherapy clinic or the Health and Counselling Service at Akoranga Campus, Auckland University of Technology.

How will my privacy be protected?

Confidentiality will be maintained throughout this study in the following ways. No material which could personally identify you will be used in any reports about this study. Data collected in this study will be kept in a secure cabinet in a locked office and will be shredded on completion of this study.

How do I join the study?

If you are interested in joining, following learning of the study through local notices, advertorials and via local support group, you can contact and discuss your possible participation with the study supervisors. If you agree to take part, an initial session in the laboratory will be arranged at a suitable time to you.

What are the costs of participating in the project? (including time)
Participation in the study will not cost you anything, though you will be required to travel to and from the Physical Rehabilitation Research Centre two times within 1 week. Petrol or taxi vouchers may be provided to you for each session attended. As well, a parking space will be available during your session.
The entire duration of both sessions will be no longer than 90 minutes.

Opportunity to consider invitation
You have one week to decide whether you wish to take part in this study. You have a right to choose not to participate. If you do agree to take part, you are free to withdraw from the study at anytime, without having to give a reason.

Opportunity to receive feedback on results of research
The results of this study will be published in a rehabilitation journal and presented at scientific conferences. It is usual that a substantial delay between the end of the data collection and the publication or presentation of results may occur. The outcomes of this study will be available to you by discussion with the principal investigator if you wish.

Participant Concerns
Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, madeline.banda@aut.ac.nz , 917 9999 ext 8044.

Researcher Contact Details: Verna Stavric
Auckland University of Technology
Private Bag 92006
Auckland

917-9999 ext 7320

Project Supervisor Contact Details: Dr. Peter McNair
Auckland University of Technology
Private Bag 92006
Auckland

917-9999 ext 7146

Approved by the Auckland University of Technology Ethics Committee on 14 October 2004 **AUTEC Reference number** 04/133

Appendix E

Provided by the Internet Stroke Center — www.strokecenter.org

MINI-MENTAL STATE EXAMINATION (MMSE)

Patient Name: _____
Rater Name: _____
Date: _____

Activity	**Score**
ORIENTATION – one point for each answer	
Ask: "What is the: (year)(season)(date)(day)(month)?"	____
Ask: "Where are we: (state)(county)(town)(hospital)(floor)?"	____
REGISTRATION – score 1,2,3 points according to how many are repeated	
Name three objects: Give the patient one second to say each. **Ask the patient to**: repeat all three after you have said them. Repeat them until the patient learns all three.	____
ATTENTION AND CALCULATION – one point for each correct subtraction	
Ask the patient to: begin from 100 and count backwards by 7. Stop after 5 answers. (93, 86, 79, 72, 65)	____
RECALL – one point for each correct answer	
Ask the patient to: name the three objects from above.	____
LANGUAGE	
Ask the patient to: identify and name a pencil and a watch. (2 points)	____
Ask the patient to: repeat the phrase "No ifs, ands, or buts." (1 point)	____
Ask the patient to: "Take a paper in your right hand, fold it in half, and put it on the floor " (1 point for each task completed properly)	____
Ask the patient to: read and obey the following: "Close your eyes." (1 point)	____
Ask the patient to: write a sentence. (1 point)	____
Ask the patient to: copy a complex diagram of two interlocking pentagons. (1 point)	____
TOTAL	**(0–30):** ____

.

Reference: _____
Date: _____

Appendix F

Physical Activity Readiness Questionnaire (PAR-Q)

Yes	No	
☐	☐	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
☐	☐	Do you feel pain in your chest when you do physical activity?
☐	☐	In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	Do you have a bone or joint problem that could be made worse by a change in physical activity?
☐	☐	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
☐	☐	Do you know of any other reason why you should not do physical activity?

Appendix G

Screening Questionnaire

Reference:_____
Date _____

1.Is your general health good? ☐ Yes ☐ No
If no, what problems do you have?_____

2.Are you currently taking any medication? ☐ Yes ☐ No
If yes, please specify:_____

3.Do you have any current conditions such as diabetes, hearing or vision problems? ☐ Yes ☐ No
If yes, please specify (e.g. where?):_____

4.Do you have uncontrolled high or low blood pressure? ☐ Yes ☐ No

5. Do you have a heart condition? ☐ Yes ☐ No

6.Have you ever had a stroke? ☐ Yes ☐ No

7.Have you had any falls in the last month? ☐ Yes ☐ No
If yes, please specify_____

8.Do you have uncontrolled epilepsy? ☐ Yes ☐ No

9.Do you have any open wounds, rashes or skin conditions? ☐ Yes ☐ No
If yes, please specify:_____

10.Do you have any pain stopping you from putting weight through your legs? ☐ Yes ☐ No
If yes, please specify:_____

11.Have you had radiotherapy or chemotherapy in the past 6 weeks? ☐ Yes ☐ No
If yes, please specify:_____

12.Do you have any bladder or bowel problems? ☐ Yes ☐ No
If yes, please specify:_____

13.Do you have osteoarthritis in your legs? ☐ Yes ☐ No
If yes, please specify:_____

14.Have you had any physiotherapy treatment in the past 3 months? ☐ Yes ☐ No
If yes, please specify (e.g. what for?):_____

Appendix H

Consent to Participation in Research

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This form is to be completed in conjunction with, and after reference to, the AUTEC Guidelines (Revised January 2003).

Title of Project: **Muscle power after stroke**

Project Supervisor: **Dr. Peter McNair**

Researcher: **Verna Stavric**

- I have read and understood the information provided about this research project (Information Sheet dated 15 November 2004.)
- I have had an opportunity to ask questions and to have them answered.
- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- I agree to take part in this research.
- I wish to receive a copy of the report from the research.

Participant signature: .....

Participant name: .....

Participant Contact Details (if appropriate):

.....

.....

.....

.....

Date: .....

Approved by the Auckland University of Technology Ethics Committee on 14 October 2004 **AUTEC Reference number** 04/133

Note: The Participant should retain a copy of this form.

Reference: _____

Appendix I **Fugl-Meyer Assessment** Date: _____

Lower Extremities			
Reflex Activity Position: **Supine**			
Achilles	0 No reflex activity		2 reflex activity
Patellar	0 No reflex activity		2 reflex activity
Active Movements ("flexor synergy") Position: **Supine** (does hip abduct and outwardly rotate? Knee flexors active?			
Hip Flexion	0 no motion	1 partial motion	2 full motion
Knee Flexion	0 no motion	1 partial motion	2 full motion
Ankle Dorsiflexion	0 no motion	1 partial motion	2 full motion
Resisted movements ("extensor synergy") Position: **Supine**			
Hip extension	0 no motion	1 weak motion	2 almost normal
Adduction	0 no motion	1 weak motion	2 almost normal
Knee extension	0 no motion	1 weak motion	2 almost normal
Ankle plantarflexion	0 no motion	1 weak motion	2 almost normal
Active movements Position: **Sitting**			
Knee flexion beyond 90	0 no motion	1 slight flex to 90	2 flex beyond 90
Ankle dorsiflexion	0 no motion	1 slight df vs opp	2 normal df vs opp
Active movements Position: **Standing, Hip at 0** (bend your knee to 90 keeping hip still)			
Knee flexion	0 no knee w/o hip	1 knee first then hip, angle < 90	2 full motion
Ankle dorsiflexion	0 no motion	1 slight df vs opp	2 normal df vs opp
Normal reflexes			
Knee flexors	0 2/3 markedly hyperactive	1 1/3 hyperactive, 2/3 lively	2 no more than 1/3 lively
Patellar			
Achilles			
Coordination/Speed Position: **Supine** **Heel to Opposite Knee (5 repetitions)**			
Tremor	0 marked tremor	1 slight tremor	2 no tremor
Dysmetria	0 pronounced	1 slight or syste	2 no dysmetria
Speed	0 >5 s slower N	1 2-5 s slower N	2 <2 s difference

Total Score: _____

SPSS

Appendix J **Muscle power after stroke**
Data Collection and Checklist Sheet

Reference: _____
Date:_____

First Session

Consent obtained

Date of Birth | Age | Gender (1 / 2) M/F

Ethnicity

CVA (2 / 1) Y/N

Type | Location (1 / 2) R/L hemi | Date | Time since CVA mos

Screening Par-Q completed? | General completed? | Proceed?
MMSE Score | Proceed?
Mod Ashworth Quads pf | Proceed?

Testing
LLTQ Completed?
Height cm | Weight kg | BMI
Fugl-Myer lower extremity score /34

5 min warm up

Test Order

	10 m				Fastest time
	Stairs				Fastest time
	Chair				Fastest time
	Strength:				

	Leg Order		Left 1 -RM	kg		Right 1 -RM	kg
			30 %			30 %	
			50 %			50 %	
			70 %			70 %	

Adverse Effects reported: msk pain msk stiffen other

Comments

Next session date | Vouchers given | T / P | Signed

Contact between sessions?

Second Session

Date **Days since 1st session**

Leg Order		% of 1 -RM tested				Report given	Vouchers given
	R leg	1st		2nd		3rd	
	L leg	1st		2nd		3rd	

Adverse Effects reported: msk pain msk stiffness other

Comments