2.1  **Introduction**

This chapter consists of 4 sections. The first section briefly outlines the clinical benefits of NMES induced exercise for people with SCI. While clinical outcomes were not a focus of this thesis, this section has been included to highlight the initial motivation for conducting this study.

The second section of this chapter introduces the reader to previously published designs of cycle ergometers for use with SCI individuals. Important features have been identified that must be included in the design of an ergometer for SCI individuals. This section also discusses the methods that have previously been utilised to choose NMES firing angles for cycling. This outlines the initial reasoning for the use of computer simulations to predict suitable firing angles.

The focus of this thesis is on the development of computer simulations to predict motor performance. The third section of this chapter gives a brief introduction to the field of forward dynamic modelling, outlines the history of simulation methods used to investigate voluntary cycling performance, and finally reviews the literature of simulation methods that have been applied to the study of muscle performance by individuals with SCI using NMES.

The final section of this chapter reviews literature related to the development of muscle models. Each component of a muscle is reviewed within this section to justify the modelling decisions made in Chapter 4.

2.2  **Benefits of Electrical Stimulation Exercise**

Secondary complications associated with spinal cord injury are a continuing source of health problems after the immediate traumatic effects of the injury (Davis, 1993; Faghri et al., 1992; Petrofsky et al., 1983). DeVivo (1997) estimated the total cost to the American health care system to have been more than $950,000 over the lifetime of a spinally injured person.
compared with approximately $218,000 in the first year following injury. Some secondary complications arising from spinal cord injury include increased risk of cardiovascular disease (Davis, 1993), pressure sores (Ferguson, 1992), bone fractures resulting from osteoporosis (Pacy et al., 1988), muscle atrophy (Block et al., 1989), limited range of motion in affected joints (Rodgers et al., 1991), increased incidence of skin complaints (Petrofsky and Smith 1988), urinary tract infections (Braun et al., 1985) and respiratory system disorders (Petrofsky and Smith 1988).

The use of NMES to provide leg muscle exercise has been used as therapy for many of these secondary complications to SCI. The aim of this thesis is to investigate the mechanical actions of leg muscle contractions using NMES and not to determine whether these are effective therapies for SCI individuals. The therapeutic benefits of NMES are therefore discussed here only briefly for the purpose of explaining why people with SCI use this type of exercise.

**Cardiorespiratory Fitness**

Spinal cord injury often leads to an increasingly sedentary lifestyle that reduces cardiorespiratory fitness and predisposes SCI individuals to a greater risk of cardiovascular disease (Davis, 1993). Upper body exercise utilises only a relatively small muscle mass and hence the load on the cardiorespiratory system during upper body exercise is often insufficient for effective development of cardiovascular fitness (Faghri et al., 1992). Exercise of the leg muscles has been proposed as a mechanism of increasing fitness in SCI individuals because it enables use of the larger muscles in the legs and hence offers the potential for greater overload of the cardiorespiratory system (Davis, 1993; Faghri et al., 1992; Figoni et al., 1991; Franek et al., 1988; Petrofsky and Stacy, 1992). Leg extension exercises have been shown to raise oxygen consumption and cardiac output in SCI individuals (Figoni et al., 1991; Rodgers et al., 1991), however Figoni et al. found that increases in oxygen uptake came more from improved peripheral oxygen extraction than from increased cardiac output. Furthermore, the greater venous return produced by contracting leg muscles meant that increases in cardiac output came predominantly from larger stroke volume rather than heart rate; and thus was not likely to significantly stress the heart for fitness purposes. Cycling exercise has been shown to produce larger cardiorespiratory loads than knee extension exercise because cycling involves a larger amount of muscle mass (Faghri et al., 1992; Glaser et al., 1989; Petrofsky and Stacy, 1992). Heart rate increases of 100% above resting levels have been found for SCI subjects
cycling at 40 W (Petrofsky and Stacy, 1992) in comparison to only 6% increase for subjects performing knee extension work by lifting a load of 15 kg (Figoni et al., 1991).

Regular training using NMES cycling exercise has been shown to produce increases in cardiovascular fitness among SCI injured persons (Faghri et al., 1992; Petrofsky and Stacy, 1992). Both these studies found decreases in heart rate and blood pressure during exercise after a period of training using NMES cycling. Faghri et al. (1992) also demonstrated decreases in resting blood pressure for paraplegic subjects, although resting heart rate was unchanged after training. Interestingly, Faghri et al. found that resting heart rate and blood pressure rose in quadriplegic subjects after training. It was noted that these variables are normally suppressed in quadriplegic persons owing to decreased sympathetic drive; and that resting heart rate and blood pressure increased towards more normal levels after training.

In addition to the limitations imposed by small muscle mass, venous pooling in the legs due to inactivity of the venous muscle pump limits the cardiac output of SCI individuals in upper body exercise while the legs are inactive (Davis et al., 1990). A number of studies have shown that lower limb NMES can enhance venous return from the legs and thus increase stroke volume during upper body exercise (Davis et al., 1990; Figoni et al., 1988; Raymond et al., 1999); although it has not been demonstrated that this will result in a higher maximum work output from the upper limbs.

**Decubitus Ulcers**

Decubitus ulcers (pressure sores) over the ischial region are a major cause of health problem for SCI persons and result from continuous pressure applied to insensitive regions where circulation is reduced (Ferguson et al., 1992; Petrofsky and Smith, 1988; Stefanovska et al., 1993). Direct application of electrical stimulation to the buttocks has been shown to increase blood flow to the skin (Levine et al., 1990a; Mawson et al., 1993) and to change the shape of the buttocks while sitting, which would result in changes in pressure distribution across the buttocks (Levine et al., 1990b). Treatment of decubitus ulcers with electrical stimulation therapy results in significantly faster healing of the ulcers for paraplegic patients (Griffin et al., 1991; Stefanovska et al., 1993) and several authors have suggested that such treatment is likely to have a preventative effect (Levine et al., 1990b; Mawson et al., 1993). It has been reported that NMES cycling reduces the incidence of decubitus ulcers (Pacy et al., 1988;
Petrofsky and Smith, 1988) however this has not been demonstrated by controlled clinical trials.

**Bone Loss**

Prolonged inactivity through spinal cord injury results in severe reduction in bone mineral content with a corresponding decrease in bone strength and increased risk of fractures (Pacy et al., 1988; Rodgers et al., 1991). It is commonly suggested that NMES exercise may halt or reverse this trend (Braun et al., 1985; Pacy et al., 1988; Petrofsky and Smith 1988; Rodgers et al., 1991), however few studies have directly investigated this effect. Pacy et al. (1988) trained four SCI individuals for an average of 42 weeks using knee extension and cycling exercise and found no significant change in femoral bone density during this time. Similarly, BeDell et al. (1996) trained 12 SCI individuals over 34 weeks and found no improvement in bone density across a number of sites within the femur and lumbar vertebrae. Sloan et al. (1994) measured bone density, again from the femur and lumbar vertebrae, in two SCI individuals after 6 and 12 months of cycle training. Once again, no change in mineral density was recorded.

Rodgers et al. (1991) trained 12 SCI individuals over 12 weeks using cycling exercise and found that tibial bone density declined over this period, although at a rate slower than they expected. Although Rodgers et al. concluded that NMES exercise appeared to decrease the rate of tibial bone loss, this result should be treated very cautiously because the rate of bone loss they considered normal was based on an equation derived from a cross sectional study of SCI persons with different numbers of years since the time of injury, rather than a longitudinal study of actual rates of bone loss. Seven of the nine subjects in Rodger et al.’s study had initial bone densities higher than was predicted from the equation so they would have been expected to show a lower rate of bone loss than that predicted, even without the exercise intervention. Without the use of a sedentary control sample, it is hard to draw firm conclusions from the bone mineral loss data of Rodgers et al.

There have been studies showing limited improvements in bone mineral density following NMES cycle training. Mohr (1996) trained 10 SCI individuals for three sessions per week over one year and found improvements in density of the proximal tibia. Femoral neck density, however, was unchanged over this period. A further six months training by these subjects with only one session per week resulted in a subsequent decline in density back to pre-
training levels. It appears, therefore, that NMES cycling may improve tibial density; while the femur is not significantly affected. Furthermore, a threshold level of training is required to produce benefits. The finding of a training threshold was also reported by Bloomfield et al. (1996). NMES cycling over a six month period resulted in improved mineral density at the proximal tibia and distal femur for a group of four subjects that trained at 18 W or greater for a period of at least three months. Other subjects within the same study that did not achieve 18 W training levels did not demonstrate significant changes in mineral density.

**Reflex spasticity**

Hypersensitivity of the muscle stretch reflexes often results in increased muscle spasticity in people with SCI (Robinson et al., 1998a). While electrical stimulation has been used as a therapy to reduce this spasticity since the late nineteenth century (Duchene, 1855, cited by Douglas et al., 1991), the responses of spasticity levels to NMES training have not been consistently reported in the literature.

Literature is consistent in suggesting that electrical stimulation produces short term decreases immediately following stimulation (Robinson et al., 1988a; Vodovnik et al., 1984). This decrease in stiffness may be attributed either to an inhibition of spinal reflex arc (Douglas et al., 1991) or to simple fatigue of the muscles being reflexively activated (Robinson et al., 1988a).

Reported changes in spasticity over longer time frames are more variable. Franek et al. (1988) used either 12 or 18 days continuous electrical stimulation therapy and found spasticity to be “permanently” reduced in half their sample of 44 subjects, for a period of up to “a few months” after stimulation therapy ceased. Similarly, Granat et al. (1993a) found that six months gait training using FES reduced spasticity levels in six SCI individuals. In this study, spasticity measures were taken at least 24 hours after the most recent training session. Other authors have reported increases in spasticity with NMES training (Douglas et al., 1991; Robinson et al., 1988b). Both these studies measured spasticity after one to two months training. Robinson et al. measured spasticity at least 24 hours subsequent to electrical stimulation while Douglas et al. did not report the delay. Sloan et al. (1994), while not providing quantitative measurements of spasticity, reported that reflexive spasms increased with FES cycling training and that some subjects were unable to continue training because of the increase in spasticity.
All studies have reported significant variation in spasticity levels; both between individuals and within single individuals over subsequent tests. Furthermore, the responses are likely to vary between different levels of spinal injury and with the initial degree of spasticity (Vodovnik et al., 1984). While the changes in spasticity with training are not consistent, there is evidence that at least some SCI individuals will benefit from NMES training. Other individuals may find that spasticity increases with training, providing a contra-indication to training for these people.

2.3 **Design of ergometers for NMES induced cycling**

A number of different ergometers have been used for electrical stimulation induced leg cycling for SCI individuals. Many laboratories have built their own ergometers by modifying a Monark ergometer (Block et al., 1989; Pacy et al., 1988; Petrofsky & Stacy 1992). The ergometer used by Petrofsky & Stacy (1992) was developed as a commercially available ergometer, the Regys (Therapeutic Alliance, Fairborn, Ohio), and used by studies such as BeDell et al. (1996). Further development of this ergometer resulted in a more advanced design, marketed as the Ergys 2, and described in Chapter 1 of this thesis. The strong design qualities and wide availability of this ergometer has seen it used in a large number of studies for SCI individuals (eg Bloomfield et al., 1996; Faghri et al., 1992; Sinclair et al., 1996).

While the commercial ergometers allow ease and safety of use, they are limited in flexibility for use as a research tool. For this reason, a number of laboratories have continued to design and build their own ergometers (Bremner et al., 1992; Chen et al. 1997; Mulder et al., 1989; Petrofsky et al., 1983; Pons et al., 1989).

All ergometers for use in NMES cycling have similar requirements. They must have satisfactory bracing systems to hold the feet on the pedals and prevent excessive lateral motion of the legs. The ergometers must monitor crank angle and use this information to apply NMES to suitable muscles at appropriate crank angles to generate forward propulsion. Lateral bracing of the legs can be achieved by use of a calliper type brace arising from the
pedals to embrace the calves (Bremner et al., 1992; Pons et al., 1989). Alternatively, the Ergys and Regys ergometers have a two-linkage stabiliser bar attaching to the thigh that allows movement only in the sagittal plane. This provides a more stable support to the legs, however requires more substantial engineering of the brace to be rigid enough, yet not heavy or restrictive of movement. All ergometer designs reviewed within this chapter have utilised rigid ankle orthoses that effectively prevent any movement at the ankle joint. This constrains the cycling action to a single degree of freedom where the crank angle effectively defines the joint angles at the hip and knee.

Reviewing the choice of muscles stimulated reveals nearly as many different muscle combinations as there are ergometers that have been developed. The Ergys and Regys ergometers utilise the quadriceps, hamstring and gluteus maximus muscles. Other combinations include the quadriceps and hamstrings (Chen et al., 1997); quadriceps and gluteus maximus (Petrofsky et al., 1983); quadriceps and iliacus (Petrofsky et al., 1984); quadriceps, hamstrings, gastrocnemius and gluteus maximus (Pons et al., 1989) and the quadriceps alone (Mulder et al., 1989). While it seems likely that greater benefits would come from an increased number of muscles being recruited, choice is practically limited by the number of available stimulation channels as well as the time taken to apply electrodes before training. To date, there has been no experimental measurement of the effectiveness with which each individual muscle group can generate external work on a cycle ergometer.

All ergometer designs reviewed to date have utilised a rigid ankle orthosis to control movement of the leg in both sagittal and frontal planes. Voluntary cyclists, however, plantarflex their ankle while pedalling to provide a significant proportion of their power output from the triceps surae muscles (Ericson et al., 1986). While it is impossible with the bracing systems used on current ergometers, an ankle orthosis permitting flexion/extension, together with stimulation of the triceps surae muscles, may provide a way to increase the work done by SCI individuals while cycling.

To control the onset and cessation of stimulation for each muscle, all ergometers utilise a rotary sensor to measure the angle of the crank in real time. Monitoring of crank angle by a microprocessor allows closed loop control of the muscle stimulator. In addition, some systems monitor crank velocity, either by differentiating the angle signal or using a separate velocity sensor, and use this as feedback for the level of stimulation current (Ergys and Regys
ergometers; Pons et al., 1989). By adjusting the stimulation current, the microprocessors for these ergometers were able to maintain a pre-set cycling cadence.

The arrangement of ergometer seat and crank positions affects the range of crank angles through which stimulation of a particular muscle will result in positive work being done on the pedals (Schutte, 1992). Therefore, the range of suitable stimulation firing angles differs for every ergometer in use. Different methods have been employed in the past to select stimulation firing angles. Petrofsky et al. (1983) and Petrofsky et al. (1984) selected firing angles through intuitive consideration of each muscle’s anatomical action. Other studies have utilised EMG analysis of able-bodied individuals to design stimulation patterns (Bremner et al., 1992; Mulder et al., 1989; Popp, 1986). Popp (1986, p93), however, noted that stimulation patterns based on EMG analysis did not result in “useful movement”. He therefore designed stimulation patterns initially from intuition which were then modified “on a trial and error basis”. Pons et al. (1989) note that the finally selected stimulation patterns had a poor correlation to the EMG patterns produced by able-bodied cyclists.

Not only must stimulation firing patterns differ between ergometers, but it has been suggested that optimal patterns may differ between individuals because of individual anthropometric characteristics (Schutte et al., 1993). Forward dynamic models have been developed by Schutte et al. (1993) and by Gächler and Lugner (2000) to predict optimal stimulation patterns for individual subjects. To date, however, there have been no reported studies that have experimentally tested the ability of models to generate individual stimulation firing patterns.

### 2.4 Forward Dynamic Simulations

#### 2.4.1 Why perform simulations?

Contraction of skeletal muscle is the means by which movements are produced. Consideration of the forces involved with skeletal muscle contractions furthers the understanding of motor control of the human machine. Analysis of a task performed by human subjects assists the

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1 Pons et al. (1989) is a published version of the Masters thesis written by Popp (1986).
understanding of how that movement was performed. Studying the sequence and force of muscle contractions enables one to learn about the determinates of skilled performance, how to assess the skill level of an individual and what injury potentials exist within a movement. The actual muscle forces present during most movements, however, can only be estimated. Standard techniques of inverse dynamics allow the calculation of nett moments across joints using a linked-segment modelling (Winter, 1990). These joint moments cannot, however, be directly decomposed into individual muscle forces owing to the degree of redundancy present in the system. Many muscles cross each joint in the body and, without direct measurement, it is impossible to know exactly what force is present in each muscle. While direct measurement has been used both on animals (Fowler et al., 1993; Herzog and Leonard, 1991) and humans (Fukashiro et al., 1993; Gregor et al., 1991; Schuind et al., 1992), these techniques are quite invasive and therefore unsuitable for general usage. Furthermore, there is some question as to the accuracy of in-vivo calibration for these devices in situations where the calibration cannot be performed post-mortem (van den Bogert, 1994).

Inverse dynamics, coupled with an appropriate model of the musculature and a decision algorithm to partition the measured torque between the available muscles, can be used to estimate forces in individual muscles (Pierrynowski and Morrison, 1985). While there are varying degrees of complexity between models, the aim of such modelling is share the load between synergistic muscles using some objective function such that the overall joint moment equals that measured by inverse dynamics. Common methods to partition force between muscles include equalising the force or stress between synergistic muscles (eg Chadwick et al., 2000; Pedotti et al., 1978), or more complex functions estimating energy consumption (Boichut and Valentini, 1983). Models may simply allocate load without considering how the dynamics of individual muscles affects their ability to generate force (eg Crowninshield and Brand, 1981; Glitsch and Baumann, 1997; Seirig and Arkivar, 1975). Alternatively, the dynamics of individual muscles may be included to calculate their ability to generate force and use this information when partitioning loads between muscles (Happee, 1994).

Methods for partitioning loads between muscles are vigorously debated (eg Hatze 2001, Ait-Haddou et al., 2001) and it is not the intention of this thesis to resolve this debate. Rather, the intention is to point out that there are alternatives available and none of them are universally accepted. Forward dynamic techniques will be used within this thesis; hence, discussion of inverse dynamic techniques will not be dealt with further.
Analysis of tasks that have already been performed use inverse dynamics to estimate muscle forces, utilising recorded movements as inputs to a computer model of segmental dynamics. An alternative type of modelling, forward dynamics, uses muscle forces as inputs to determine movement outcomes. Rather than examining recorded movements, forward (or direct) dynamics enables the researcher to investigate novel tasks without the need for direct experimentation. This has a number of advantages over inverse dynamics in reducing the need for costly and time consuming experimentation, enabling the investigation of tasks that could be dangerous for human participants, and increasing the number of conditions that could be tested beyond that possible for direct experimentation.

Forward dynamic simulations provide a unique advantage in developing an understanding of human motor control systems. Muscle actions in multi-segmental movements, particularly of those spanning more than one joint, are complex and not intuitive (Zajac, 1993). For example, the muscle gastrocnemius has the direct action of knee flexion, but may have the opposite effect in some movements. When the foot is fixed on the ground, the ankle plantar-flexion action of gastrocnemius may result in extension of the knee in some circumstances (Zajac and Gordon, 1989). Computer simulations enable these different actions to be calculated in specific circumstances.

Another example of how forward dynamic modelling contributes to an understanding of motor control comes from van Soest et al. (1994). In this study, a model was used to predict optimal muscle activation patterns for vertical jumping from different starting positions. While each starting position had a unique activation pattern that produced optimum performance from that position, van Soest and colleagues found a single pattern that enabled close to optimal performance from all starting positions. This finding gives important insights to how motor patterns are stored within the brain. While it seems unlikely that optimum patterns for every unique movement can be stored, it is much more reasonable that the brain learns patterns that are useable across a large range of similar movements.

Discussion has taken place on the necessary complexity of models. Hatze (1980) has criticised others for over simplification. His argument was that, unless a model reflects the inherent complexity of the system being modelled, then it cannot produce an accurate simulation of that system. Khang and Zajac (1989a) produce the counter argument that, if a
model is too complex, it cannot be used because of the computation time needed for its implementation. Linear simplifications must be used in order to make a model useable. Obviously, a suitable model must be a compromise between being simple enough to implement while still being complete enough to provide reasonably accurate predictions. Certainly one must avoid the trap of choosing algorithms for mathematical convenience rather than for any physiological reasoning (Sepulveda et al., 1993).

Audu and Davy (1985) made a number of simplifications to a model from Hatze (1976) in order to investigate the effect of complexity on model performance. They found that modest simplifications, using simpler component equations that still predicted the same general relationship between variables, greatly increased numerical stability and decreased processing time. A simpler model, however, was able to generate similar predictions of performance. Gross simplifications, however, that no longer retained the original relationship between variables (eg the use of a linear force-length relationship), produced much larger changes in model outcomes and were not considered appropriate.

2.4.2 Simulation methods used with cycling

What is optimum cycling technique?

A number of researchers have looked at the profiles of forces applied to the pedals during cycling. The general pattern has been similar to that illustrated in Figure 2.4.2.1.

![Figure 2.4.2.1](image)

**Figure 2.4.2.1** Typical pattern of force application to pedals during upright cycling. From Ericson and Nisell (1989), p120.
Only the component of this force that is perpendicular to the angle of the crank provides propulsion to the bicycle (Gregor et al., 1991). The component parallel with the crank places tension or compression on the crank but does not do work on the crank because there is no movement in that direction. A number of researchers state that this non-propulsive component indicates an ineffective application of force to the pedal (Davis and Hull, 1981; Ericson and Nisell, 1988; Lafontaine and Cavanagh, 1983; Sanderson, 1991). They suggest that cyclists would be better to reduce the magnitude of this non-propulsive force, thus applying more of the total force in the direction of travel of the pedal.

A number of researchers have calculated an "index of effectiveness" to describe how well the cyclist pushed in the direction of travel of the pedal (eg Davis and Hull, 1981; Ericson and Nisell, 1988; Lafontaine and Cavanagh, 1983; Sanderson, 1991). While the terminologies, and indeed the calculation formulae, vary slightly between papers, the concept involves a ratio of the tangential force component to the total force magnitude. This ratio varies throughout pedal cycle, usually between values of 1 and -1 (Figure 2.4.2.2).

**Figure 2.4.2.2** Typical pattern of Performance Index throughout one complete revolution in cycling. From Ericson and Nisell (1988), p121.
While the foot is moving down it actively pushes to drive the pedal. The index of
effectiveness approaches one at 90 deg because the total pedal force is almost entirely in the
direction of travel at this time. The index is positive throughout the entire downstroke. During
the upstroke, however, the foot does not pull up on the pedal but is pushed up by the pedal
This does not imply that the leg flexors do not withdraw the leg at all; merely that they do not
provide enough force to completely unload the pedal. The performance index is therefore
negative throughout most of the upstroke. This finding has been consistent for both skilled
and unskilled cyclists, with and without cleated pedals (Davis and Hull, 1981, McLean 1989,

Ericson and Nisell averaged the index across the entire pedal cycle. The index curves
approximated a sine curve (Figure 2.4.2.2) so that the average index came out to be very close
to zero. What is inappropriate about this method is that, although the index approaches one on
the downstroke and negative one on the upstroke, the magnitudes of the forces during these
phases are very different. Sanderson (1991) suggests that it is inappropriate to average the
index over an entire revolution because this places undue importance on the relatively small
forces during the upstroke. Rather, the index should be quantified over different segments of
the revolution as in Figure 2.4.2.1 so that only relevant portions of the revolution are
analysed.

Inherent in the above measurements of cycling effectiveness appears to be the assumption that
pushing on the pedal in the direction of movement is an appropriate technique. It appears to
the present author not to be an ideal method, however, since no cyclist has ever been
measured to apply forces at all similar to this. Rather, it seems likely that the muscular forces
required to pedal in this fashion would be much higher than those required to pedal in the
manner shown in Figure 2.4.2.1. Cyclists obviously find it easier to lift the leg up by pushing
down with the contra-lateral leg than to completely lift the ipsi-lateral leg. If this were not so,
then at least some cyclists would do this. Kautz and Hull (1993) demonstrated that much of
the non-tangential component of pedal forces comes from accelerating the limbs against
inertia. Actively contracting muscles to eliminate this radial component would require
additional muscle effort and thus would reduce the efficiency of cycling. Furthermore, Gregor
et al. (1991) suggest that clearly inappropriate riding technique (eg too high a seat height)
may sometimes increase measurements of pedalling "effectiveness", indicating the
unsuitability of this as a measure of good technique.
An appropriate test of cycling effectiveness needs to be based on "effort" required from the muscles rather than simply forces applied to the pedals. Measurement of "effort" is clearly the difficulty with this concept. Computer modelling enables technique to be analysed by considering the loads of muscles rather than simply forces on the pedals. Such models may investigate technique either by considering the nett moments about each joint or forces within individual muscles. A brief review of how these processes have been applied to cycling will be considered below.

**Modelling in Cycling**

A number of computer simulation models have been developed to investigate the mechanics of cycling. Redfield and Hull (1986) developed a cycling model with 11 muscles in each leg. Little detail was given regarding the development of the muscle model, particularly in regard to the activation parameters of the muscles. A cost function minimising the sum of moments about the hip, knee and ankle enabled reasonable predictions of pedal forces, however the agreement between measured and modelled joint moments was not as close. A cost function based on minimising the stresses in each of the 11 muscles, however, produced a better match to both pedal forces and joint moment data.

Subsequent modelling by Hull and Gonzalez (1988) and Gonzalez and Hull (1989) used a minimisation of joint torques cost function to optimise cycling parameters such as cadence, crank length, seat height and seat tube angle and position of the feet on the pedals. Similar modelling was performed by Yoshihuku and Herzog (1990) using a more detailed muscle model, but with no ankle joint. These papers demonstrate the potential for modelling to contribute to cycling technique. Without further validation of the models, however, it is by no means certain that the modelling process would produce a better result than trial and error selection by an experienced athlete.

Schutte et al. (1993) and Gföhler and Lugner (2000) developed similar models to optimise stimulation patterns for NMES cycling by SCI individuals. The muscle models for these studies were more detailed than those of the earlier studies cited above, however, there was again no validation of the model outcomes. The simulation methods look promising, but require experimental validation before the results could be accepted with confidence.
More recent modelling has provided both detailed muscle models and experimental testing of the model outcomes. Raasch et al. (1997) predicted both pedal forces and muscle EMG patterns using a model with 15 muscles controlled via 9 independently controlled muscle groups. The outcome of this study was similar to that of Redfield and Hull (1986), however the more recent results were more quantitatively validated, the muscle model more thoroughly described and based on established physiological principles.

A comparable model by Neptune and van den Bogert (1998) was similarly capable of predicting pedal forces. When modelled muscle work was compared against physiological oxygen cost, however, there was little correlation between measured and modelled energy expenditure. This indicates that a model’s ability to predict pedal forces does not completely validate its performance and therefore the ability to optimise external variables such as crank length and cycling cadence may be compromised. The process of computer simulation provides insights into the mechanisms of cycling, however the ability of models to predict optimal technique has not yet been well established.

van Soest and Casius (2000) have used a detailed model to explain the relationship between pedalling cadence and power output. Muscle force – velocity relationships are commonly used to explain the high pedalling cadence that maximises power output, however the results of this model indicate that muscle’s activation timing plays an equally significant role. Unless muscles are given sufficient time to switch on and off, they cannot generate significant forces and hence power declines. This study shows that appropriate modelling can both provide accurate predictions of performance as well as to help explain the mechanisms underlying physiological measurements.

2.4.3 Computer simulation methods used with NMES

Computer modelling offers a number of important advantages in the development of effective stimulation patterns for use by SCI individuals. Firstly, there are relatively few SCI individuals available for experimental procedures requiring trained subjects. Furthermore, the subjects available in any given location will vary in level of lesion and completeness of injury, further exacerbating the problem of finding a relatively homogenous sample. Computer simulations enable researchers to advance their understanding of FES control
systems, while minimising the demands made on those SCI individuals available as experimental subjects. Simulated experiments enable researchers to learn the essential requirements of tasks, such as which muscles to stimulate and what levels of strength are required from potential subjects. This would be particularly useful for movements such as standing and walking that carry a risk of injury from falling.

Much of the literature regarding FES control has concentrated on system control parameters, with no inherent muscle models. A number of these studies have demonstrated strategies to control a freely swinging leg using quadriceps stimulation (eg Ezenwa et al, 1991; Hausdorff and Durfee, 1991; McNeal et al, 1989) or to stand an SCI individual using isometric contractions of the quadriceps muscles (eg Jaeger et al, 1989, Mulder et al., 1992). In light of the present study, only those employing a muscle model in the control system will be discussed here.

One of the earliest studies to apply simulation techniques to FES was by Trnkoczy et al. (1976). A simple planar model of the ankle joint was developed with muscle force inputs from two groups: dorsi and plantar flexors. A simple model was developed using torque generating elements, rotary springs and visco-elastic elements. Model elements were measured from tetanic stimulation of the gastrocnemius and tibialis anterior muscles of between one and ten able-bodied individuals. Modelled torque and angle data were then compared with ankle torque and angle measurements from a single subject performing isometric and dynamic contractions of the ankle muscles. Measured torques always rose in advance of the modelled data, indicating a limitation in the model's activation response. Measured responses also exhibited significant variability that was not present in the modelled data.

Employing more sophisticated muscle models, more recent modelling by Riener et al. (1996) and by Dorgan and O'Malley (1997) has managed to improve the match between measured and modelled responses. The model of Dorgan and O'Malley (1997) was based on earlier work by Hatze (1977) and was able to adapt to changes in stimulation parameters during tetanic contractions. Riener et al. (1996) based their model on that of Delp et al. (1990), however they included an additional function accounting for changes in force amplitude with fatigue and recovery. The parameters for this fatigue function were individually fitted for each of five SCI subjects. This model was able to provide a close match to measured responses to a
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A wide range of isometric and free swinging knee extension exercises utilising a variety of stimulation patterns and durations. A comparison between performance of these three models (Dorgan and O'Malley, 1997; Riener et al., 1996; Trnkoczy et al., 1976) highlights the benefits of more contemporary, physiologically based muscle models on the ability to predict mechanical responses to NMES.

Bajd and Trnkoczy (1979) used the model developed by Trnkoczy et al. (1976) to generate ankle muscle NMES parameters for walking. Stimulation patterns were developed for a single, hemiplegic individual with the aim of returning ankle joint kinetics to a more normal pattern. Although the modelling and stimulation was applied only to a single joint, it differs from the models described above in that it is incorporated into a complex movement like walking. After development of the model, a within-shoe heel switch was used to trigger events within the gait cycle. Resulting ankle joint angles and moments were altered towards more normal patterns, although there were still considerable deviations from the desired joint moments. These differences were prescribed primarily to the inability of the stimulated muscle to generate required amounts of force because their stimulator was not able to fully recruit all fibres within the muscle. Bajd and Trnkoczy recommended either the introduction of a muscle strengthening program or the use of implanted stimulation electrodes to activate more fibres within the muscle. They concluded that their model appeared to be performing appropriately, however improvements in muscle strength or stimulator hardware were required to test this further.

A number of authors have developed simulation models for complex, multi-muscular movements such as standing (Jaeger, 1986; Khang and Zajac, 1989a, 1989b), walking (Tashman et al., 1995; Yamaguchi and Zajac, 1990) and cycling (Gföhler and Lugner, 2000; Schutte et al., 1993). These models vary in complexity, particularly with regard to the physiological detail within the muscle model. Jaeger (1986) used simple torque generators about the hip, knee and ankle, with no feedback of joint angle or velocity on joint torque. This simple model was sufficient, however, to provide information about the degree of control necessary to support standing. The other models cited above used “Hill type” muscle models similar to that used within the present thesis. This type of model enables more general physiological parameters to be included, more easily modified to account for differences between muscles.
Chapter 2

No experimental testing has been conducted on the validity or performance of these complex standing and walking models (Khang and Zajac, 1989a; Tashman et al., 1995; Yamaguchi and Zajac, 1990). Yamaguchi and Zajac (1990) state that a number of factors limit the ability to implement their model until further technological enhancements take place (eg the ability to provide fine coordination of multiple muscles and to control the effects of fatigue). These simulations, therefore, were performed with the aim of furthering the understanding of NMES control systems. They were not undertaken with the intent to provide stimulation patterns that would be used immediately.

Schutte (1992) developed a musculoskeletal model based on the methods of Delp et al. (1990). The model consisted of four separate muscles; gluteus maximus, rectus femoris, a generic vastus muscle and a generic hamstring muscle. A detailed Hill type model of each muscle enabled the simulation of cycling for SCI individuals using NMES initiated muscle contractions. Schutte et al. determined optimal stimulation patterns for each muscle, and examined the effects of changing seat position on performance. Simulations were made to represent a single subject pedalling an ERGYS ergometer (see Section 1.1), and the muscle groups modelled were those stimulated by this ergometer. Schutte compared the velocity after 7 s of pedalling with that predicted from simulations performed under a number of conditions (eg seat reclined, seat forward, resistive load increased). While the velocities measured experimentally matched quite well to those predicted from simulations, there was no further validation of the model. No kinetic measurements were taken to confirm these predictions. No experimental results at all were included in the externally published report of this project (Schutte et al., 1993).

Gföhler and Lugner (2000) conducted simulation of NMES cycling that was similar in intent to Schutte et al. (1993). More muscles were included in the simulation, and optimum stimulation patterns were examined. Gföhler and Lugner reported more detail on the kinetic outcomes of their simulations (eg drive torques resulting from the stimulation of individual muscles), however, once again no experimental measurements were taken to confirm these predictions.

Pedalling a cycle ergometer is a much simpler activity than walking, with fewer muscles required to be individually controlled for successful performance. Furthermore, the consequences of unsatisfactory stimulation parameters are less catastrophic when seated on a
cycle ergometer with no risk of falling. It is surprising, therefore, that more experimental
evidence has not been collected to support the modelling performed by Gföhler and Lugner
(2000), and Schutte et al. (1993). One of the main aims of the current investigation was
therefore to provide this experimental verification in order to more thoroughly test kinetic
predictions by the model.

2.5 Muscle Modelling

2.5.1 Muscle lengths and moment arms

Methods to measure muscle length and/or moment arm

Modelling the torque generated by a muscle about a joint requires estimates of the length and
moment arm of that muscle about the joint in question. Changing joint angles will alter the
muscle’s length and moment arm, requiring equations to model this relationship. The two
functions are related, and therefore dealt with here in the same section, because muscle length
changes are often calculated by integrating moment arm over joint angle (van Soest et al.,
1993).

Modelling muscles as straight lines or curves between known points in three-dimensional
space provides a way to estimate muscle lengths and moment arms (Brand et al., 1982; Frigo
and Pedotti, 1978; Seireg and Arkivar, 1973). Locations of the origins and insertions of each
muscle modelled must be known before this method can be used. Brand et al. (1982)
performed a very detailed study of the lower limb anatomy. In this study they dissected three
cadavers then measured the co-ordinates of the origin and insertion of each muscle in three
dimensional space. The co-ordinate locations of each muscle were reported with respect to
local axes defined by the hip, knee and ankle joints. Using these co-ordinates, and modelling
the muscles as straight lines between origins and insertions, it is possible to calculate the
moment arms of each muscle as well as to predict changes in muscle length which would
result from joint angle changes. For those muscles wrapping around joints, “effective” origins
and insertions were given so that the muscle moment arms could be more accurately
calculated. The data set from Brand et al. has been used by a number of computer modelling
studies for determining muscle lengths and moment arms (eg Delp et al., 1990; Glitsch and Baumann, 1997; Hoy et al., 1990; Mansour and Pereira, 1987). Hawkins and Hull (1990) used this data set to provide generic regression equations that can be applied to calculate muscle lengths given segment lengths and joint angles. Other studies have generated similar data sets (Boichut and Valentini, 1983; Keppele et al., 1998; Pierrynowski and Morrison, 1985; White et al., 1989), however that of Brand et al. (1982) remains the most commonly used in generic muscle modelling.

Using origin and insertion co-ordinate locations for modelling has the advantage that three-dimensional models can easily be developed that predict muscle lengths and moment arms for any given joint position. The assumption of muscles forming a straight line, even if an effective origin is used, remains a significant limitation of this method (RA Brand, personal communication, August 6, 1991).

Alternative methods, based on experimental measures, have been used to determine muscle moment arms. A number of studies have measured muscle length changes from direct measurements taken on cadavers (eg Bu ford et al., 1997; Grieve et al., 1978; Spoor et al., 1990; Visser et al., 1990). Each of these studies have moved cadaver limbs through a range of joint angles and measured the length of muscles at each joint angle. An equation defining the moment arm of a muscle as a function of joint angle may be calculated from the first derivative of the equation defining muscle length as a function of joint angle (Bobbert et al., 1986). These studies using the tendon excursion method have an advantage over the previous straight line modelling approach in that direct measurements are involved, with no assumptions regarding axes of rotation and lines of pull, leading to greater confidence in their accuracy (Spoor et al., 1991b). There are limitations in this approach, however, in that the measurements are taken from cadavers and may differ for active muscle contractions.

Lieb and Perry (1968) hung weights off individual quadriceps tendons and hence calculated relative moment arms between each of the four muscles in human limbs recently amputated because of peripheral cardiovascular disease. Unfortunately, only the relative size of moment arms between the muscles was reported, rather than actual values. A similar study performed by Grood et al. (1984) measured the moment arms of the quadriceps muscles by directly measuring the force applied to cadaver tendons to produce a known torque. These studies appear to have high face validity because they involve the measurement of forces during knee
extension, however, they still have the disadvantage of not being performed on living, active muscles.

Moment arms have been measured directly from in-vivo imaging systems (eg Kellis and Baltzopoulos, 1999; Koolstra et al., 1989; Nemeth and Ohlsen, 1985; Smidt, 1973; Spoor and van Leeuwen, 1992). Spoor and van Leeuwen (1992) found errors of up to 30% in measures of knee moment arm from a single cadaver when measured in-vivo using MRI and compared to the tendon excursion method described by Grieve et al. (1978). Spoor and van Leeuwen attributed this error to an effect from uneven force distribution within tendons having a finite thickness, as well as uncertainties in the measurement of instantaneous centres of rotation. If the anterior or posterior portion of a tendon took more force, then this would change the moment arm of the muscle, but would not be visible from photographic techniques. The importance of axis of rotation has been emphasised by Mikosz et al. (1988) who found that more accurate measures of the instantaneous axis significantly improved the performance of their model. While the accuracy of imaging techniques has been questioned, these have the distinct advantage of being measured in-vivo from living subjects during active muscle contractions.

Once a muscle’s moment arm has been defined as a function of joint angle, muscle length changes can be determined by integrating the moment arm equation (Hoy et al., 1990). This method, however, can only be applied to the specific ranges of angles about a single axis through which moment arms have been measured. This is because rotations about one axis can significantly change the moment arm of a muscle about other axes (Murray et al., 1995). Determining muscle lengths through moment arm integration is therefore applicable only for two-dimensional models. Three-dimensional models employing rotations about multiple axes within a single joint tend to rely on straight line modelling between effective origin and insertion co-ordinates (eg Delp et al., 1990). The present model under construction is two-dimensional; consequently, only flexion/extension moment arms and joint angles will be described further in this section.

**Moment arms for the quadriceps about the knee**

The quadriceps muscles all act through a single patella tendon to exert torque about the knee. For this reason, a single moment arm - knee angle relationship is frequently assumed for the four separate muscles. Tendon excursion experiments have shown slightly different
relationships for the individual muscles (Buford et al., 1997; Visser et al., 1990), however the
differences between quadriceps muscles are relatively small when compared to the different
values found between studies.

Figure 2.5.1.1 illustrates the variation in moment arm values found between studies. For those
studies that have reported different moment arm relationships for individual quadriceps
muscles, these have been combined together for illustration in Figure 2.5.1.1. The resulting,
combined moment arms were calculated by considering the relative cross-sectional area of
each muscle to determine a weighted average (see Section 2.5.5). Most studies report that the
quadriceps moment arm peaks between 30 and 60 deg of flexion. The two exceptions to this
from Figure 2.5.1.1 are Visser et al. (1990) and Hawkins and Hull (1990). Both of these
studies derived moment arm equations by differentiating a quadratic length - angle
relationship, consequently their moment arm equations had to be linear.

![Figure 2.5.1.1](image)

**Figure 2.5.1.1** Comparison between moment arms from various sources for the quadriceps
femoris muscles about the knee.
Moment arms from Kellis and Baltzopoulos (1999) have been chosen as representative moment arms for the quadriceps muscles about the knee for this study. The variety of results and methodologies from Figure 2.5.1.1 make it unsuitable to calculate an average or “best” set of data. Moment arms from Kellis and Baltzopoulos have been chosen because the study is recent, based on in-vivo measurements from active contractions and reasonably representative of the other studies. The suitability of this choice will be explicitly tested in Chapter 6.

**Moment arms for rectus femoris at the hip**

The number of studies reporting moment arms for rectus femoris at the hip is more limited than for the knee. Visser et al. (1990) and Meijer et al. (1998a) reported rectus femoris moment arms calculated from cadaver tendon excursion experiments using six and four legs respectively. In addition, Hawkins and Hull (1990) have reported moment arms calculated from a straight line model based on the data of Brand et al. (1982).

![Comparison between moment arms from various sources for rectus femoris about the hip.](image)

**Figure 2.5.1.2** Comparison between moment arms from various sources for rectus femoris about the hip.

The present cycling model covers hip angles flexed up to 130 deg (Section 7.4.1) requiring extrapolation of data from Figure 2.5.1.2. This extrapolation would clearly be inappropriate for the function produced by Visser et al. (1990). While extrapolating data from the other
sources can not be justified either, there are no alternative data sources available and, at least, the extrapolations provide useable moment arm functions. Therefore, in the absence of further experimental data, an average of the constant moment arms provided by Hawkins and Hull (1990) and Meijer et al. (1998a) has been used for the present study.

Moment arms for the hamstring muscles about the knee

It has been a common practice to provide a single moment arm function for all hamstring muscles about the knee (eg Hoy et al., 1990; van Soest et al., 1993). While this practice will be adopted by the present study as well, it must be acknowledged that there is greater variability in moment arms between the various hamstring muscles than there is for the quadriceps. Buford et al. (1997) measured moment arms for individual hamstring muscles using the tendon excursion method. Moment arms for the semitendinosus, semimembranosus, long head of biceps femoris and short head of biceps femoris peaked at respectively 4.8, 3.7, 3.0 and 2.2 cm. Care should therefore be used when using moment arms measured from a single hamstring muscle. Figure 2.5.1.3 demonstrates moment arms reported by a number of sources. Data representing moment arms from a single hamstring muscle (eg Visser et al., 1990) have been excluded from Figure 2.5.1.3. As for the quadriceps muscles, a single moment arm function has been derived by taking averages across all muscles, weighted according to the relative cross sectional area of the muscles.

![Figure 2.5.1.3](image)

**Figure 2.5.1.3** Comparison between moment arms from various sources for the hamstring muscles about the knee.
Once again, there is little reason why one moment arm function should be chosen above another. The data of Buford et al. (1997) has been chosen for further modelling within the present study because it comes from a recent study that approximates the mid-range of the other data sources. A further advantage of Buford et al.’s data is that it covers a wider range of measured knee angles than most other sources.

**Moment arms for the hamstring muscles about the hip**

Like rectus femoris, there are few studies measuring moment arms for the hamstring muscles about the hip. The tendon excursion experiment of Visser et al. (1990) provides data for biceps femoris. Hawkins and Hull (1990) again provide equations to calculate individual hamstring moment arms using the data set of Brand et al. (1982). In addition to these two sources that have been used for other muscles, Nemeth and Ohlsen (1985) measured moment arms for the hamstring muscles from CT scans. Measures were taken by projecting straight lines of action onto the CT images and measuring perpendicular distances to the centre of the head of the femur.

Figure 2.5.1.4 illustrates the range of hamstring moment arms across the hip reported by these studies. Each differs substantially from the others in both magnitude and shape. While Nemeth and Ohlsen (1985) believe their moment arms to be correct within 1 mm, this level of confidence is hard to justify. While their measurements may be accurate to this level, uneven distribution of force within the thickness of the muscle may result in the mid-line not accurately representing the line of force (Spoor and van Leeuwen, 1992). Furthermore, if the centre of rotation was not coincident with their calculated centre of the femoral head, particularly in the anterior-posterior direction, this would introduce error into their moment arm estimations (Delp and Maloney, 1993).
Figure 2.5.1.4 Comparison between literature moment arms from various sources for the hamstring muscles about the hip.

For the purposes of this study, the moment arms of Visser et al. (1990) will be used to represent the hamstring muscles about the hip. The moment arm equation must be extrapolated out to 130 deg of hip flexion. While there are no data available for these high angles of flexion, it does not appear likely that either the constant moment arm of Hawkins and Hull (1990) or the rapidly diminishing moment arm of Nemeth and Ohlsen (1985) is likely to be satisfactory.

**Moment arms for gluteus maximus about the hip**

Very limited information is available for moment arms of the gluteal muscles. As well as reporting hamstring moment arms, Nemeth and Ohlsen (1985) also measured values for the gluteal muscles. This was more difficult than for the hamstrings because of the broad origin of the muscle and because the muscle wraps around the ischium. To overcome this, the line of action of the muscle was represented by two straight segments when measuring the moment arm. This difficulty in straight line representation may be why Hawkins and Hull (1990) chose not to include gluteal muscles in their otherwise complete set of lower limb muscles modelled using the data of Brand et al. (1982). Other models have utilised this data set (eg Delp et al., 1990; Hoy et al., 1990) without reporting the values of the moment arms derived.
The model developed by Schutte (1992) adapted the model of Delp et al. (1990) to include a cylindrical surface for gluteus maximus to wrap around rather than a single point. This modification was performed in order to allow better model performance at the flexed hip angles used in recumbent cycling. Figure 2.5.1.5 illustrates the moment arm – hip angle relationships determined by Nemeth and Ohlsen (1985) and Schutte (1992).

![Figure 2.5.1.5](image)

**Figure 2.5.1.5** Comparison between literature moment arms from various sources for gluteus maximus about the hip.

The choice of moment arms functions between these two sources was not immediately obvious. It will be established in Section 6.2.2, however, that the moment arms of Schutte (1992) are required for the present model to function adequately.

### 2.5.2 Series Elasticity

Skeletal muscle is made of a series of individual muscle fibres joined in series with connective tissue between the muscle’s origin and insertion. The series elastic component of pennate muscles is comprised of external tendon as well as intramuscular aponeurosis (internal tendon). Figure 2.5.2.1 illustrates a typical arrangement of fibres and connective tissue.
As the fibres develop force, this force is transmitted through the series elastic component which lengthens in response to the force. The series elastic component is comprised of those structure in series with the contractile elements of the muscle that lengthen in response to force developed within the contractile element. Most muscle models consider only the series elastic element located external to the fibre; that is the tendon and aponeurosis (Zajac, 1989). Muscle cross bridges may also contribute to the series elasticity of the muscle, however most authors consider the effect of this to be insignificant when modelling the behaviour of whole muscles (eg van Ingen Schenau, 1984). Ettema and Huijing (1993) estimated that approximately 15% of the series elastic compliance of rat muscles was located within the cross bridges. The compliance of this component changed, however, with the level of force, the number of attached cross bridges and the type of contraction (eg dynamic vs isometric). Furthermore, they suggested that inter-species differences may prevent the generalisation of animal measurements to human models. Consequently, only structures external to the fibres will be considered in further discussion of series elasticity.

The lengthening of the series elastic component during the development of force results in the fibres shortening as they develop tension, even under isometric conditions (Ito et al., 1998). The amount of fibre lengthening is determined by the force exerted as well as the length and stiffness of the tendon (both internal and external).
The strain of the intramuscular aponeurosis has been treated differently by different authors. Zajac (1989) suggests that, for all parts of the series elastic component to experience equal strain (both intramuscularly and external), the cross sectional area of the aponeurosis must increase proportionally with proximity to the external tendon. This is because that part of the aponeurosis closer to the external tendon has the force of more fibres passing through in series.

Experimental verification of the relative amount of strain between external tendon and aponeurosis, however, is not conclusive with quite a variety of responses being observed. Rack and Westbury (1984) found stiffness of the external tendon component of cat soleus to be similar to that measured for the internal and external components combined. Lieber et al. (1991), however, found external tendon stiffness to be approximately four times higher than the intramuscular aponeurosis for frog semitendinosus muscle. Ettema and Huijing (1989) examined stiffness of the external tendon and aponeurosis of rat extensor digitorum longus muscle during isometric contractions at different muscle lengths. Their results demonstrate that the stiffness of the external tendon was unaffected by muscle length, however aponeurosis stiffness changed with muscle length. Zuurbier et al. (1994) found inconsistencies in the behaviour of different segments of the aponeurosis for rat gastrocnemius medialis, with the distal portion being more compliant than the proximal or middle portions. Herbert and Gandevia (1995) noted that intramuscular tendon (aponeurosis) did not experience similar length changes to extramuscular tendon. Indeed, in some cases intramuscular tendon was observed to shorten under muscle contraction while tendon lengthened in response to applied force.
Scott and Loeb (1995) suggest that changes in aponeurosis stiffness may be measurement artefacts owing to epimysium sliding along aponeurosis as muscle length changes. Sliding of the epimysium would mean that different results could be found depending upon how markers were attached to the muscle during measurements. Scott and Loeb (1995) found that, once corrections are made to account for this sliding, the measured stiffness of the aponeurosis was similar to that of the external tendon.

The best way to model tendon and aponeurosis stiffness for human muscle is not yet clear. The aponeurosis can be treated as a rigid structure with no compliance (eg Huijing and Woittiez, 1984), it can be given the same compliance as the external tendon (eg Zajac, 1989) or the aponeurosis can be modelled with a different compliance to the external tendon (Lieber et al., 1992). Neither of these alternatives are likely to be completely accurate given the range of compliance values available from published literature. The data of Scott and Loeb (1995), however, do at least provide some justification for the second alternative.

There are a number of values presented for tendon stiffness in the literature (eg Butler et al., 1984; Loren and Lieber, 1995; Proske and Morgan, 1987). In a major review of available literature Zajac (1989) found measurements of maximum stress to vary between 18 and 84 MPa coinciding with strains between 2 and 9%. Clearly, there is not a single value that can be prescribed with confidence for all muscles. As tendon compliance is not expected to have a major effect on the present simulations, however, a single value will be used for numerical convenience. Zajac (1989) recommended assuming that:

a) all muscles have tendons with cross sectional areas proportional to their maximum force and
b) all tendons experience a strain of 3.3% at maximum force.

Given these two assumptions, the stiffness of each tendon in N/m may be calculated given nominal values for the maximum force and the tendon slack length.
The use of a linear tendon stiffness would not be satisfactory if the primary aim of this research was to investigate the amount of tendon elongation generated by a musculo-skeletal model. Tendon stiffness is relatively constant at high forces, however there is a “toe region” at low force levels where stiffness is much less than within the linear region (Butler et al., 1978). Because forces are likely to be low during NMES cycling, all forces may be expected to be within this toe region. Furthermore, the effect of chronic muscle immobilisation due to spinal cord injury on tendon parameters is unknown. In the absence of more definitive information about series elastic stiffness for paralysed human muscles, the above stiffness parameter will be used and the model outcomes specifically tested for sensitivity to this assumption.

Equation 2.5.2.1 suggests that the amount of tendon elongation is directly proportional to the resting (slack) length of a tendon. Tendon length is also important in setting the length of muscle fibres at particular joint angles and thus affects a muscle's torque-angle relationship (Hoy et al., 1990). Slack length, however, cannot be measured directly owing to the difficulty in establishing a length without tension on the series elastic component (Hoy et al. 1990). Rather, slack length is usually estimated during the development of a muscle model in order ensure the model produces maximum torque at an appropriate joint angle. This process will be described in detail in Section 6.1.2.
2.5.3 Angle of pennation

Angle of pennation

Fibres in many muscles are arranged at an angle to the tendon. The degree of angulation is known as the angle of pennation (sometimes pinnation).

\[
\theta
\]

Figure 2.5.3.1 Illustration of the angle of pennation of muscle fibres.

Since forces are generated parallel to the fibres, any angle of pennation will reduce the amount of fibre force transmitted along the line of the tendon such that from Figure 2.5.3.1, such that

\[
F_{\text{tendon}} = F_{\text{fibre}} \cos(\theta)
\]

Equation 2.5.3.1

Where \( F_{\text{fibre}} \) is the force in a fibre
\( F_{\text{tendon}} \) is the force transmitted along the line of the tendon \( \theta \) is the angle of pennation.

Angle of pennation is also significant in that it changes the amount of fibre shortening necessary to produce a specific change in whole muscle length. The explanation of this will be discussed below as it depends on the particular model of pennation used. Briefly, however, the change in fibre length is usually considered to be less than the corresponding change in muscle length. This allows the fibres to operate within a range closer to their optimum fibre length (Legreneur et al., 1997; Muhl, 1982).

A third effect of muscle pennation is that it enables more fibres to be packed into a given muscle volume to increase the physiological cross sectional area, (Rutherford and Jones, 1992). This increases the force output of muscles, as force production is proportional to the number of sarcomeres in parallel. Although pennation reduces the proportion of fibre force
aligned parallel to the tendon, the increase in force from more parallel sarcomeres more than compensates for alignment decrease. This results in increased force production from pennate muscles (Alexander and Vernon, 1975).

Angle of pennation changes as a function of muscle length, with angles becoming more acute as the muscles shorten (Fukunaga et al., 1997; Herbert and Gandevia, 1995; Narici et al., 1996; Rutherford and Jones, 1992). Furthermore, increasing tension within a muscle increases pennation angle (Fukunaga et al., 1997; Herbert and Gandevia, 1995; Narici et al., 1996). An ideal muscle model should account for these changes in pennation.

The angle of pennation has been ignored by many models of muscle dynamics (eg Bobbert et al., 1986; Cole et al., 1996; Lieber et al., 1992) because, with angles of pennation typically less than 15 deg, the cosine of the angle is still greater than 96%. There are, however, quite a number of sources suggesting that ignoring pennation angle can introduce significant errors into a model by affecting the length of fibres corresponding to a particular change in overall muscle length (Fukunaga et al., 1997; Scott and Winter, 1991; Spoor et al., 1991a). Notably, if constant pennation angle were assumed, then fibres would have to shorten over a relatively greater length in order to produce a particular range of shortening of the whole muscle. This is contrary to both experimental findings (eg Muhl, 1982) and to model simulations where pennation increases with muscle shortening (eg Legreneur et al., 1997). A changing angle of pennation will therefore be included in the present model.

There are a variety of approaches in modelling changes in pennation angle as a function of muscle fibre length. The most common method is to assume a parallelogram of constant thickness that must therefore change angle as it shortens (eg Maganaris and Baltzopoulos, 1999; Pandy et al., 1990; Pierrynowski and Morrison, 1985).
T is thickness
lf is fibre length
lf₀ is resting fibre length
θ is angle of pennation
θ₀ is resting pennation angle.

Figure 2.5.3.2 Illustration of pennation change with fibre shortening.

If thickness is constant then from Figure 2.5.3.2:

\[ T = \text{lf}_0 \times \sin(\theta_0) \]

\[ \theta = \arcsin\left(\frac{T}{\text{lf}}\right) = \arcsin\left(\frac{\text{lf}_0 \times \sin(\theta_0)}{\text{lf}}\right) \]

Equation 2.5.3.2

Alternate illustrations of fibre arrangement to Figure 2.5.3.2 have been provided by Spoor et al. (1991a). Assuming constant muscle thickness in both models, Spoor et al. gave equations for fibre length and pennation angle changes with the bipennate model to be identical to that described above. The unipennate model was more complex owing to the within muscle aponeuroses not being parallel to the line of pull of the muscle. Calculations of pennation angle, fibre length and muscle force as functions of muscle belly length gave very similar results for both models, however. This led Spoor et al. to conclude that “differences between bipennate and unipennate models are so small that the choice between them seems unimportant. The bipennate model with its simpler analysis may be preferred for practical reasons” (Spoor et al., 1991a, p158). Figure 2.5.3.1 will be used to illustrate the modelled fibre architecture for the current model. While Figure 2.5.3.3a is more correct for unipennate muscles, the advice of Spoor et al. (1991a) will be followed and the simpler model used.
While Huijing and Woittiez (1984) have questioned the assumption of constant muscle thickness, these authors used a different definition to that illustrated by Figure 2.5.3.1. They measured the distance perpendicular to the line of pull, rather than the perpendicular distance between tendon plates. More recently, ultrasound techniques have permitted the in vivo measurement of muscle thickness for human muscles during both active and relaxed states; and as joint angles change. Maganaris and Baltzopulos (1999); Narici et al. (1996) and Ichinose et al. (1995) found no significant change in muscle thickness for respectively tibialis anterior, gastrocnemius medialis and vastus lateralis, as muscles went from fully relaxed to MVC. Maganaris et al. (1998) also found no significant change for gastrocnemius medialis. Their gastrocnemius lateralis and soleus muscles, however, significantly increased their thickness during MVC compared to rest. Three of these papers examined a range of joint angles and found muscle thickness to remain constant across all muscle lengths (Ichinose et al. 1995; Maganaris et al. 1998; Maganaris and Baltzopulos 1999).

Equation 2.5.3.2 predicts that pennation will increase as a muscle develops tension, because of the increased length of the series elastic component leading to fibre shortening. For those muscles where thickness changes with activation, the equation is likely to produce erroneous results. Therefore, Maganaris et al. (1998) tested Equation 2.5.3.2 using fibre length changes during activation to predict changes in pennation. Predicted pennation angles were always within 4 deg of those measured for gastrocnemius medialis, which demonstrated no significant change in thickness. For gastrocnemius lateralis and soleus, which did increase in thickness during activation, the formula underestimated pennation angles by between 9 deg and 14 deg during MVC. Maganaris and Baltzopulos (1999) repeated this finding for tibialis
anterior, demonstrating no change in thickness and consequently measurable pennation angles that matched those predicted by Equation 2.5.3.2.

Hypertrophy of a muscle has been shown to increase pennation angle (Kawakami et al., 1993; Rutherford and Jones, 1992). While Henriksson-Larsen et al. (1992) did not find such a relationship, Kawakami et al. suggested this may have been owing to the relatively small variation in muscle sizes studied by Henriksson-Larsen et al. The effect of hypertrophy is relevant to the present study because the SCI individuals exhibit greatly reduced muscle bulk from normal levels. Most of the data available on fibre pennation comes from elderly cadavers however, hence SCI individuals may be no more different from this population than healthy young individuals who are commonly modelled using the same data.

Scott et al. (1993) measured pennation angles along the length of muscles vastus medialis and semimembranosus and found pennation to increase almost linearly from proximal to distal. Assigning a single angle of pennation to all fibres is therefore a simplification, but is necessary when modelling a whole muscle based on a single, representative fibre (see Chapter 3).

There are a number of sources giving normative pennation angles for many muscles within the human lower limb (eg Friederich and Brand, 1990; Wickiewicz et al., 1983). Pierrynowski (1995) has compiled these studies into one complete source, although the exact source of each datum has been lost in the compilation. The studies from which these data were compiled included measurements taken from cadaveric muscles. Table 2.5.3.1 compares the cadaveric data from several sources including Pierrynowski (1995) with more recent data measured in living subjects. The in-vivo studies, while comprising smaller data sources than that compiled by Pierrynowski (1995), give data sources for angle changes resulting from muscle length and activation changes. Note the large variation in reported values for some muscles. Such variation between studies should not be unexpected given the small sample sizes typically used in each study. Henriksson-Larsen et al. (1992) illustrate this variation by reporting vastus lateralis pennation angles for 10 different individuals ranging between 11 and 23 deg.
Table 2.5.3.1  Pennation angles for quadriceps, hamstring and gluteus maximus muscles.

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<thead>
<tr>
<th>Muscle</th>
<th>VL</th>
<th>VM</th>
<th>VI</th>
<th>RF</th>
<th>SM</th>
<th>ST</th>
<th>BF(L)</th>
<th>BF(S)</th>
<th>GM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pierrynowski (1995)*</td>
<td>11</td>
<td>10</td>
<td>6</td>
<td>10</td>
<td>15</td>
<td>4</td>
<td>7</td>
<td>15</td>
<td>1.5</td>
</tr>
<tr>
<td>Friederich and Brand (1990)</td>
<td>13</td>
<td>7</td>
<td>2.5</td>
<td>14</td>
<td>16</td>
<td>6</td>
<td>7</td>
<td>15</td>
<td>3.3</td>
</tr>
<tr>
<td>Wickiewicz (1983)</td>
<td>5</td>
<td>5</td>
<td>3.3</td>
<td>5</td>
<td>15</td>
<td>5</td>
<td>0</td>
<td>23.3</td>
<td></td>
</tr>
<tr>
<td>Cutts (1988)</td>
<td>12.7</td>
<td>14.3</td>
<td>16.7</td>
<td>16.7</td>
<td>17</td>
<td>5</td>
<td>3.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fukunaga et al. (1997) #</td>
<td>18</td>
<td>(21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rutherford and Jones (1992)** #</td>
<td>8</td>
<td>(14)</td>
<td>8</td>
<td>(16)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Henriksson-Larsen et al. (1992)#</td>
<td>15</td>
<td>(22)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pennation angles are given in degrees.

Muscles are identified using the following abbreviations: vastus lateralis (VL), vastus medialis (VM), vastus intermedius (VI), rectus femoris (RF), semimembranosus (SM), semitendinosus (ST), biceps femoris long head (BF(L)), biceps femoris short head (BF(S)), gluteus maximus (GM).

# Measured in vivo using ultrasound. All other measurements performed directly on cadavers. Numbers in brackets were measured in vivo during maximum voluntary contractions. All other measurements were measured relaxed (or dead).

* Data collated from various sources including Friederich and Brand (1990) and Wickiewicz (1983)

** Measurements taken with the knee at 90 deg relaxed and fully extended during MVC.
Pennation angles measured in the relaxed state in vivo do not differ significantly from those measured directly on cadavers. As there is no obvious reason for choosing one data source over another at present, the pennation angles from Pierrynowski (1995) will be utilised for the present model as it has been compiled from a larger sample of data.

Note that this data source is for relaxed muscle in the anatomical position. Active pennation angles will be calculated using Equation 2.5.3.2. There are a number of limitations that will prevent angles for all subjects being predicted as accurately as was done by Maganaris and Baltzopoulos (1999). Firstly, not all muscles maintain constant thickness during activation; hence, calculations of pennation change during activation are likely to be in error. Secondly, pennation is not uniform throughout the muscle, so any single value will be an approximation. Finally and specifically to the current project, people with spinal cord injury will certainly differ from the normal population in terms of muscle strength, cross sectional area and tendon compliance. The exact effects of these differences on fibre pennation are unknown. Despite these limitations, it is anticipated that providing best estimates of pennation will be better than assuming no pennation at all.

### 2.5.4 Optimum fibre length

Chapter 3 outlines the development of a force-length relationship for a single sarcomere. Within that chapter, it will be suggested that the length of each muscle fibre can be modelled as being proportional to the length of a single sarcomere, provided that all sarcomeres in the fibre change length proportionally. Furthermore, the force-length relationship of the whole muscle will be proportional that of a single sarcomere. The optimum length of a fibre is determined by the number of sarcomeres in series for each fibre (Ettema and Huijing, 1994). If the optimum sarcomere length is known and all fibres in a muscle are assumed to have the same number of sarcomeres in series, then the optimum fibre length for a muscle is simply the product of sarcomere number by sarcomere length.

Gordon et al. (1966) demonstrated that single frog muscle fibres reached a plateau of maximum force at sarcomere lengths between the 2.0 and 2.2 \( \mu \text{m} \). These lengths coincided with length of the actin filaments and the length of the actin filaments plus the width of the H zone for frog muscle. Filament lengths vary between species with humans having a thin filament length of 2.64 \( \mu \text{m} \) (Walker and Schrodt, 1974) and a similar width H zone of 17 \( \mu \text{m} \).
Optimum sarcomere length should therefore be between 2.64 and 2.81 μm with a mid-point of 2.73 μm (Spoor et al, 1991a). Gordon et al. (1966) reported the slack length of a sarcomere to fall about the middle of the plateau region (2.1 μm for frog muscle) providing further justification for using the mid point of the plateau as optimum sarcomere length. Note that, while previous studies have used the same principles and filament lengths, there has been some minor variation in the single value used for optimal sarcomere length (eg Bobbert and van Ingen Schenau, 1990: 2.9 μm; Hoy et al., 1990: 2.8 μm).

Numbers of sarcomeres in series for individual fibres have been reported by Wickiewicz et al. (1983). Although Wickiewicz et al. reported fibre lengths based on an optimal sarcomere length of 2.2 μm (more suitable for frogs than human), the primary measure of sarcomere number is important and the total fibre length can be easily scaled. Herzog et al. (1990) and Meijer et al. (1998a) have also used this method on a smaller number of muscles (See Table 2.5.4.1).
Table 2.5.4.1  Optimum fibre lengths for quadriceps and hamstring muscles.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Wickiewicz et al. (1983)*</th>
<th>Meijer et al. (1998a)</th>
<th>Herzog et al. (1990)**</th>
<th>Calculations from data of Friederich and Brand (1990) and Cutts (1988) ***</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>7.7</td>
<td>7.8 (7%)</td>
<td>8.6 (9%)</td>
<td>7.7 (4%)</td>
</tr>
<tr>
<td>VL</td>
<td>8.2</td>
<td>11.3 (13%)</td>
<td>11.2 (18%)</td>
<td>10.1 (2%)</td>
</tr>
<tr>
<td>VM</td>
<td>9.6</td>
<td>11.3 (12%)</td>
<td>11.7 (14%)</td>
<td>10.4 (4%)</td>
</tr>
<tr>
<td>VI</td>
<td>8.6</td>
<td></td>
<td></td>
<td>10.6 (2%)</td>
</tr>
<tr>
<td>SM</td>
<td>7.2</td>
<td></td>
<td></td>
<td>6.9 (5%)</td>
</tr>
<tr>
<td>ST</td>
<td>15.6</td>
<td></td>
<td></td>
<td>8.2 (3%)</td>
</tr>
<tr>
<td>BF(L)</td>
<td>9.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BF(S)</td>
<td>12.9</td>
<td></td>
<td></td>
<td>13.4 (10%)</td>
</tr>
</tbody>
</table>

Fibre lengths are reported in cm.
Numbers in brackets refer to coefficient of variation of fibre length.

* Sarcomere number has been multiplied by an optimum length of 2.73 µm instead of the 2.2 µm originally used by Wickiewicz et al.

** Sarcomere numbers from the three portions of rectus femoris measured separately have been averaged prior to multiplying by the optimum sarcomere length.

*** Method for calculation described in text. Coefficients of variation come from Friederich and Brand’s data.

Because Wickiewicz et al. (1983) provide the only source of optimum fibre lengths for many muscles, an alternative measure was sought for comparison. Spoor et al. (1991a) used an alternate method of calculating fibre optimum length. They measured sarcomere length and fibre length in the anatomical position, then used the optimal sarcomere length as above to scale anatomical fibre length to optimal fibre length. This method assumed that pennation angle did not change significantly during fibre shortening. While Spoor et al. only investigated muscles crossing the ankle, Table 2.5.4.1 uses this method to calculate optimum fibre lengths based on anatomical fibre lengths reported by Friederich and Brand (1990),
together with sarcomere lengths from Cutts (1988). While the combination must be treated with caution as the data comes from different subjects, the resulting calculations give remarkable agreement with other measures of optimum fibre length (see Table 2.5.4.1). Furthermore, Tables 2.4.4.2 and 2.4.4.3 demonstrate that the data of Friederich and Brand (1990) and Cutts (1988) do not differ greatly from other sources.

**Table 2.5.4.2** Anatomical fibre lengths for quadriceps and hamstring muscles.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>6.6</td>
<td>7.3</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>VL</td>
<td>6.6</td>
<td>7.9</td>
<td>8.0</td>
<td>9.67</td>
</tr>
<tr>
<td>VM</td>
<td>7.0</td>
<td>8</td>
<td>7.8</td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>6.8</td>
<td></td>
<td>7.6</td>
<td></td>
</tr>
<tr>
<td>SM</td>
<td>6.3</td>
<td></td>
<td>6.4</td>
<td></td>
</tr>
<tr>
<td>ST</td>
<td>15.8</td>
<td></td>
<td>9.0</td>
<td></td>
</tr>
<tr>
<td>BFL</td>
<td>8.5</td>
<td></td>
<td>7.3</td>
<td></td>
</tr>
<tr>
<td>BFS</td>
<td>13.9</td>
<td></td>
<td>11.2</td>
<td></td>
</tr>
</tbody>
</table>

Fibre lengths are reported in cm.
Table 2.5.4.3  Anatomical sarcomere lengths for quadriceps and hamstring muscles.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Herzog et al., 1990</th>
<th>Cutts (1988)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>2.3</td>
<td>2.146</td>
</tr>
<tr>
<td>VL</td>
<td>1.92</td>
<td>2.173</td>
</tr>
<tr>
<td>VM</td>
<td>1.86</td>
<td>2.048</td>
</tr>
<tr>
<td>VI</td>
<td></td>
<td>1.97</td>
</tr>
<tr>
<td>SM</td>
<td></td>
<td>2.541</td>
</tr>
<tr>
<td>ST</td>
<td></td>
<td>2.987</td>
</tr>
<tr>
<td>BFS</td>
<td></td>
<td>2.281</td>
</tr>
</tbody>
</table>

Sarcomere lengths are reported in µm.

The only muscle from Table 2.5.4.1 to produce markedly different estimates between sources is semitendinosus; which both Wickiewicz et al. (1983) and Friederich and Brand (1990) report as having two separate compartments. Wickiewicz et al. cite Bodine et al. (1982) as finding very different fibre lengths in each compartment. While Wickiewicz et al. did not find greatly different semitendinosus fibre lengths in their own subjects, Friederich and Brand (1990) report semitendinosus as having the largest variability in fibre length from the muscles they studied.

Friederich and Brand (1990) reported fibre length to change in proportion to muscle size. Neither fibre lengths nor muscle lengths, however, showed a consistent difference between subjects, even though they differed significantly in standing height (168 cm vs 183 cm). This suggests that, although fibre length may be related to muscle length, there is no evidence to suggest scaling fibre lengths between individual subjects. Hoy et al. (1990) point out that both fibre length, tendon slack length and muscle moment arms determine joint angles that coincide with maximum joint torques. They indicate that, when modelling different subject sizes, either optimal fibre length, tendon slack length or both must be modified in order to maintain the observed constancy between individuals in optimum joint angle for torque production. The results of Friederich and Brand (1990) would appear to suggest that, with
present knowledge, assumed fibre lengths should be kept constant between individuals and tendon slack length modified to adjust optimum joint angle. This also reflects the practice observed by Meijer et al. (1998a). Similarly, keeping moment arms constant between individuals would preserve the joint angle at which greatest torque would be produced. Maintaining these three variables constant for all individuals is equivalent to scaling each variable linearly with segment length and therefore preserving their relative values.

The interaction between parameters such as fibre length, moment arm and tendon length complicates attempts to derive best estimates of these parameters from different sources. Meijer et al. (1998a) emphasise that these are likely to co-vary between individuals. For example: an individual with unusually short fibre lengths would be expected to have longer tendon lengths to compensate. Also, an individual with larger moment arms would be expected to have longer fibre lengths in order to accommodate the increased range of shortening that would result. This interaction between variables will be addressed in more detail later in Section 6.1.7.

Rack and Westbury (1969) presented results indicating different optimum fibre lengths when cat muscle was stimulated at different frequencies. High stimulation frequency produced maximum tension at shorter muscle lengths as well as producing greater peak values. They suggested that at short fibre lengths, there was some inhibition of the contraction process as well as the generally accepted reduction in force due to compression of the myosin filaments. This result has been reproduced by Stephenson and Wendt (1984) for skinned fibres but has not yet been incorporated into major muscle models

### 2.5.5 Muscle cross-sectional area

The amount of force a muscle is capable of generating is determined by both the volume of the muscle (determining the amount of contractile material) and the arrangement of fibres within the muscle (Lieber, 1992). The relationship between force, volume, fibre length and pennation angle may be quantified using the physiological cross sectional area (PCSA) of the muscle according to Equation 2.5.5.1.
The maximum force able to be produced by a muscle is generally considered to be proportional to this PCSA (Lieber, 1992). When estimating the relative strength of muscles for modelling purposes, it is therefore necessary to know the relative PCSA of each muscle.

PCSA has traditionally been measured from cadavers by directly measuring the pennation angle of the most superficial fibres; measuring fibre length by considering the number of sarcomeres in series and taking into account the optimum sarcomere length of a muscle; and using the mass of each muscle to calculate volume using a known muscle density (Friederich and Brand, 1990; Wickiewicz et al., 1983). Cadaveric methods were also employed by Cutts and Seedhom (1993), although in this study muscle volume was calculated from a geometric relationship using length and anatomical cross sectional area and fibre length was taken directly from Wickiewicz et al. (1983). Taylor and Kandarian (1994) suggest that replacing volume in Equation 2.5.5.1 with a direct measure of the quantity of myofibrillar protein produces even higher correlations to muscle strength, however this method has not been applied to human muscles.

PCSA has been measured in-vivo using MRI scans (Fukunaga et al., 1992; Kawakami et al., 1994; Narici 1999). These studies have the advantage of taking measurements from young, healthy subjects, however there is less precision in the measurements and there are, as yet, no available in-vivo measurements on many lower limb muscles. While PCSA is much smaller in cadavers owing to the hypotrophic effects of age, the relative PCSA between muscles is similar between measurements from young in-vivo muscles and cadavers (Cutts and Seedhom, 1993). Cutts and Seedhom therefore conclude that cadaveric data is suitable for use when the aim is to determine the relative PCSA between muscles, even for young athletic subjects. They caution that their results are less conclusive for hamstring muscles than they are for quadriceps, however they believe this difference to result from measurement difficulties with hamstring muscles, rather than preferential wasting of particular muscles with age.
The amount of force generated by a muscle, normalised by the PCSA is known as the specific tension (Lieber, 1992). Specific tension values for human muscles have been reported between 20 and 100 N cm\(^{-2}\) (Narici, 1999). Narici suggests that much of this variance results from incorrect measurements of PCSA. Powell et al. (1984) suggests that failure to measure force at fibre optimum length contribute further to this variance. Precise measurements from animals in-vitro provide a more consistent measure of specific tension between 15.7 and 29.4 N cm\(^{-2}\) with 22.5 N cm\(^{-2}\) being a commonly found value (Powell et al., 1984). The actual value for specific tension is not important for the present study, because the model’s force output for each muscle group will be adjusted to match values measured experimentally. The significance of specific tension is for estimating the relative amount of force generated by muscles within the same muscle group that are stimulated simultaneously (eg quadriceps).

Specific tension varies somewhat with muscle fibre type (Bodine et al., 1987). While some early work suggested that the force generated by a muscle could be explained just from the architecture of the muscle with no effect from fibre type (eg Spector, 1980), more recent studies suggest that slow twitch fibres have specific tensions approximately 30% less than fast fatigable fibres (Bodine et al., 1987; Bottinelli et al., 1996; Powell et al., 1984). The effect of fibre type on relative force between muscles will not be considered further, however, because of the relatively homogenous composition of paralysed muscles containing mostly fast twitch fibres (Crameri et al., 2000; Round et al., 1993). Bottinelli et al. (1996) has found no difference in specific tension between categories of fast twitch fibres from skinned human muscle fibres, hence there would not be any significant variation in specific tension expected between paralysed muscles. This point will be discussed further in Section 2.5.6 with regard to the maximum shortening velocity of a fibre.

Quadriceps muscles

A number of sources quote values for the relative cross sectional area of the individual quadriceps muscles (Table 2.5.5.1). With the exception of Narici et al. (1992), these studies are remarkable in their consistency of measurement. The different measurements from Narici et al. are likely to have resulted from a slightly different formula for PCSA. They used muscle thickness and the sine of pennation angle rather than the fibre length term from Equation 2.5.5.1. These terms are geometrically equivalent if muscle thickness remains constant (see Figure 2.5.3.2 and associated discussion), however errors may accumulate if the assumption of constant muscle thickness is violated.
### Table 2.5.5.1 Relative cross sectional areas of quadriceps muscles expressed as a percentage of the total muscle group.

<table>
<thead>
<tr>
<th>Source</th>
<th>VL</th>
<th>VM</th>
<th>VI</th>
<th>Sum of Vastii</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cadaver Studies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Friederich and Brand (1990)</td>
<td>27%</td>
<td>26%</td>
<td>31%</td>
<td>84%</td>
<td>16%</td>
</tr>
<tr>
<td>Wickiewicz et al. (1983)</td>
<td>35%</td>
<td>26%</td>
<td>25%</td>
<td>86%</td>
<td>14%</td>
</tr>
<tr>
<td>Meijer et al. (1998a)</td>
<td></td>
<td></td>
<td></td>
<td>83%</td>
<td>17%</td>
</tr>
<tr>
<td>Cutts and Seedhom (1993)</td>
<td>32%</td>
<td>25%</td>
<td>27%</td>
<td>84%</td>
<td>16%</td>
</tr>
<tr>
<td><strong>In-vivo Studies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cutts and Seedhom, 1993</td>
<td>36%</td>
<td>26%</td>
<td>21%</td>
<td>84%</td>
<td>16%</td>
</tr>
<tr>
<td>Narici et al., 1992</td>
<td>21%</td>
<td>23%</td>
<td>30%</td>
<td>75%</td>
<td>24%</td>
</tr>
<tr>
<td><strong>Review Article</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pierrynowski, 1995</td>
<td>34%</td>
<td>26%</td>
<td>23%</td>
<td>83%</td>
<td>17%</td>
</tr>
</tbody>
</table>

Setting the relative PCSA of the vastii muscles at 83.4% of the total quadriceps group (Pierrynowski, 1995) and a nominal force output of 1 for rectus femoris gives a relative peak force for the vastii of 5.04 times the rectus femoris force. This value will be used in future modelling when partitioning the relative force of the quadriceps between the rectus femoris and vastii muscles.

#### Hamstring muscles

Table 2.5.5.2 lists relative cross sectional areas of individual hamstring muscles from various sources. Again, there is high consistency between reported values, particularly for the cadaver studies. The relative areas of Pierrynowski (1995) will be used for further modelling to
maintain consistency with the quadriceps muscles. The only time these numbers will be used within the present model is to consider the relative importance of each muscle when calculating fibre lengths and pennation angles for a single, generic hamstring muscle.

Table 2.5.5.2  Relative cross sectional areas of hamstring muscles expressed as a percentage of the total muscle group.

<table>
<thead>
<tr>
<th></th>
<th>BF</th>
<th>SM</th>
<th>ST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadaver Studies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Friederich and Brand (1990)</td>
<td>39%</td>
<td>45%</td>
<td>16%</td>
</tr>
<tr>
<td>Cutts and Seedhom (1993)</td>
<td>39%</td>
<td>46%</td>
<td>15%</td>
</tr>
<tr>
<td>Wickiewicz et al. (1983)</td>
<td>41%</td>
<td>47%</td>
<td>12%</td>
</tr>
<tr>
<td>In-vivo Studies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cutts and Seedhom (1993)</td>
<td>40%</td>
<td>37%</td>
<td>24%</td>
</tr>
<tr>
<td>Review Article</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pierrynowski (1995)</td>
<td>45%</td>
<td>45%</td>
<td>11%</td>
</tr>
</tbody>
</table>

2.5.6  Force - velocity relationship

Decreased force produced by muscles contracting concentrically with increased velocity is a phenomenon that is well known and researched (Hill, 1970). While there have been suggestions for alternate models (eg Hatze, 1977; Ma and Zahalak, 1991), the hyperbolic equation first proposed by AV Hill in 1938 is still used most frequently by researchers performing complex simulations of muscle performance (Huijing, 1998). The Hill equation was originally proposed as a measure of energy expenditure, with \( E = (P + a)v \) (Hill, 1970). While the physiological predictions from the equation are no longer considered valid (Hill, 1970), the equation still provides a remarkably good prediction of the relationship between force and velocity for concentric contractions.

A major criticism of Hill’s equation has been that it assumes that the force of contraction of a muscle is dependent only upon the velocity of contraction and the isometric force at a given
muscle length (Zajac, 1989). Controlled studies have indicated, however, that contraction history affects the force generated during subsequent contractions (Herzog and Leonard, 1997). Eccentric contractions enhance the force generated by a muscle during subsequent isometric contractions (Sugi and Tsuchiya, 1988) while concentric contractions have been shown to depress force during subsequent contractions (Herzog and Leonard, 1997). Mechanisms have been proposed to account for these history dependant changes in force (eg Herzog, 1998) and models proposed to account for history dependence under limited circumstances (eg Meijer et al., 1998b). To date, however, no satisfactory model has been developed to predict force over a range of conditions (Huijing, 1998). Consequently, contraction history will be not be included within the present model.

Gordon et al. (1966) present the classic Hill's equation given in Equation 2.5.6.1.

$$V = \frac{(P_a - P)b}{P + a}$$

Equation 2.5.6.1

The instantaneous length of a fibre affects the maximum velocity of shortening in unloaded muscles. As fibre length declines below optimum, the maximum speed of shortening decreases (Gordon et al., 1966) and this must be accounted for in Equation 2.5.6.1. Gordon et al. quote Abbott and Wilkie (1953) as saying that Equation 2.5.6.1 can predict maximum speed at lengths less than optimum provided that \( P_0 \) is given as the maximum isometric force for a particular muscle length, rather than just the maximum force at the optimum length. The data of Gordon et al. (1966), however, suggest that this equation overestimates shortening speed for short lengths. Stern (1974) quotes Bornhorst and Minardi (1970) as giving adjustments to constant \( a \) to correct this error. Stern went on to say, however, that “the effects of these alterations were quantitatively very small and of no significance with regard to the general pattern of results or their interpretation” (p413). Certainly, visual inspection of the data from Gordon et al. (1966) suggests that the equation appears to fit quite well; and only percentage errors at short fibre lengths are likely to be significant.

At fibre lengths greater than optimum, Gordon et al. (1966) demonstrated that maximum velocity of shortening was not affected by length. Stern (1974) therefore adjusted Hill’s constant \( a \) so that, when fibre length was greater than optimum, constant \( a \) decreased in
proportion to the decline in isometric force. The effect of this was to cancel out the effect of isometric force and maintain a constant peak shortening velocity. van Soest and Bobbert (1993) also used the methods of Stern (1974) with regard to the effect of fibre length on Hill’s equation.

Muscle activation may also affect the force-velocity equation, although there are conflicting results between studies. A number of studies have shown that maximum velocity of shortening is not affected by activation level in isolated muscle fibres (Bigland and Lippold, 1954; Edman 1979). Other studies using whole muscle preparations have found that the maximum velocity of shortening is reduced at low levels of activation (Chow and Darling, 1999; Phillips and Petrofsky, 1980). Chow and Darling (1999) suggest that the difference may be due to the mix of fibre types present in whole muscles. Type 1 muscle fibres are known to have a lower maximum rate of shortening (Bottinelli et al., 1996). Because type 1 fibres are preferentially recruited at low activation levels, the muscle’s force-velocity relationship would be biased towards these muscles at low activation levels.

This effect of activation level changing Vmax by altering the relative percentage of fibre types will be ignored in the present model for electrically stimulated muscles of SCI individuals. SCI individuals have almost exclusively type II fibres (Crameri et al., 2000; Round et al., 1993) and hence the mix of fibre types would not change significantly with increasing levels of activation. Phillips and Petrofsky (1980), although using electrical stimulation to activate the muscles of cat gastrocnemius, used a special stimulation technique to try and recruit slow fibres first in a physiological manner (maximally stimulating the nerve, but with an anodal voltage block below the level of stimulation to block certain fibres). This may therefore have caused their results to fit with the hypothesis that Vmax increases with activation because, at higher activation levels, a higher percentage of fast fibres were being recruited.
Length and activation level interact to further complicate the force-velocity relationship. For fibre lengths greater than optimal, Stern (1974) increased constant $a$ in proportion to the decline in $P_o$ in order to maintain constant $V_{max}$. Stern wrote equations giving force as a function of fibre velocity and activation.

\[ p = \frac{(P_o + a) b \times \text{activation}}{V + b} - a \times \text{activation}. \]

Equation 2.5.6.2

When rearranged to calculate velocity, this gives

\[ V = \frac{(P_o \times \text{activation} - P)b}{P + a \times \text{activation}}. \]

Equation 2.5.6.3

Equation 2.5.6.3 produces a constant $V_{max}$ independent of activation level.

van Soest and Bobbert (1993) used the same formula as Stern to account for fibre length, but multiplied $V$ by an additional term FACTOR, defined as Min(1, 3.33*activation).

\[ V = \frac{(P_o - P)b}{P + a} \times \text{FACTOR}. \]

Equation 2.5.6.4

When multiplied by the velocity of shortening, FACTOR has the effect of producing a constant $V_{max}$ for activation levels above 33.3% and a linear decline in $V_{max}$ for activations below this.

Petrofsky and Phillips provided regression equations to predict constants $a$ and $b$ from measurements of length, activation and temperature. While these equations provide an excellent example of how each variable affects the force/velocity equation, it is not known how well the regression equations translate to other muscles and methods of activation. These equations cannot, therefore, be applied to the present study.
The current model does not contain intermediate levels of activation, except during the transition from stimulation off to on, and vice versa. The choice of whether activation level affects fibre velocity will affect model performance during these transition times, but not as much as will the choice of activation time constants. Therefore, Equation 2.5.6.3 will be used for subsequent modelling of the force-velocity relationship. Po will be adjusted to instantaneous muscle length, but the effect of activation on maximum velocity will not be included. Activation constants will be fitted using experimental data collected from SCI individuals to ensure the model provides appropriate predictions during the periods where activation levels are changing.

Constant $a$ in Equation 2.5.6.3 determines the degree of curvature in the force-velocity curve (Baratta et al., 1995). The ratio of $a/Po$ differs between fibre types, with type I fibres having a ratio of approximately 0.2 and type II fibres (both fatigue resistant and fast fatigable) having ratios of 0.35 (Pierrynowski and Morrison, 1985). For mixed muscles, Hill constant $a$ has shown to vary proportionally with the percentage of slow twitch fibres (Baratta et al., 1995). Constant $b$ determines the maximum velocity of shortening and is usually expressed relative to the length of a muscle fibre. Constant $b$ also varies with fibre length; with Pierrynowski and Morrison, 1985 reporting values of 0.40 and 2.25 times the fibre length for fibre types I and II respectively. Pierrynowski and Morrison used the same constant for all type II fibres, although more recent work by Bottinelli et al. (1996) found a higher maximum velocity of shortening for type IIB fibres than for type IIA. For muscles with mixed fibre types, Baratta et al. (1995) found that velocity of shortening could be predicted from a linear regression of percentage slow twitch fibres and the length range of active fibre shortening.

Hill’s formula for the force-velocity relationship was originally defined only for concentric contractions (Hill, 1970). During muscle lengthening, muscle force can rise to levels above isometric force (Cook and McDonagh, 1995; Joyce et al., 1969). Increasing rates of muscle lengthening result in increased force, up to a plateau, where further velocity increases no longer result in increased force. Quite a large variance is present in the reported levels of maximum force during eccentric contractions. Example values for the plateau force level include approximately 125% for cat soleus muscle (Joyce et al., 1969), 140% for human quadriceps group (Dudley et al., 1990) and 170% for rat soleus muscle (Krylow and Sandercock, 1997). Reasons for these differences are difficult to establish, but may be related to the amplitude of stretch (Cook and McDonagh, 1995) or the type of muscle being recruited.
Fitting an equation to the eccentric portion of the force – velocity curve requires a solution that generates the same magnitude of force and slope of the curve at zero velocity, in order to generate a continuous function when moving from eccentric to concentric contractions. Pierrynowski and Morrison provide such an equation (adapted from Fitzhugh, 1976) that uses Hill’s constants within an alternative equation to match the pattern of force production during eccentric contractions. Using Hill’s constants within this equation ensures that the slope of the function is continuous for both concentric and eccentric equations at $v = 0$.

$$P = P_{\text{ecc}} - \frac{D (P_{\text{ecc}} - P_o)}{D - V}$$

Equation 2.5.6.5

Where

$$D = \frac{b (P_{\text{ecc}} - P_o)}{P_o + a}$$

$P$ is the force of the muscle at a particular velocity of lengthening

$P_{\text{ecc}}$ is the plateau level of maximum eccentric force

$V$ is the velocity of lengthening

Suitable values for Hill’s constants have not yet been measured for contractions of SCI individuals using NMES. Chapter 6 will therefore use experimental data to verify the choice of constants used within the present model.

**2.5.7 Rate of force development**

When a muscle is stimulated, the rise in force is not instantaneous. Electrical stimulation acts on the motor nerves (Mizrahi, 1997). Between neural stimulation and force development, the following events take place:

<table>
<thead>
<tr>
<th>Table 2.5.7.1</th>
<th>Events in the development of muscle force in response to NMES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Stimulation of motor nerve</td>
</tr>
<tr>
<td>2.</td>
<td>Conduction of action potential along nerve</td>
</tr>
</tbody>
</table>
Chapter 2

3. Transmission of action potential across neuromuscular junction
4. Transmission of action potential across muscle membrane and down T-tubules
5. Release of calcium from sarcoplasmic reticulum
6. Diffusion of calcium through the cell
7. Binding of calcium to troponin molecules
8. Change in conformation of troponin
9. Binding of actin to myosin as muscle develops force
10. Lengthening of series elastic components in response to increase in muscle force.

Typical values for electromechanical delay (EMD)

Events 1-5 from Table 2.5.7.1 cause a delay between stimulation and force development. If the fibre is slack before force is developed then there may be a delay while the fibre shortens to the length of the muscle. Therefore all the above processes may be involved in the delay between stimulation and force development.

Duration of this delay may vary between 10 and 120 ms depending on the contraction conditions (Zhou et al., 1995). EMD has been shown to differ depending on the force of contraction, rate of force development and the muscle fibre type (Zhou et al., 1995). Krajl and Grobelnik (1973) investigated electromechanical delay of one or two paraplegic subjects performing quadriceps contractions using NMES. Full details of the experiment were not reported, however EMD was reported as being within the range of 40-60 ms.

Typical values for rate of force rise in response to stimulation

Events 6 and 7 from Table 2.5.7.1 determine the rate of force development. Rate of force development is determined by the rate of cross bridge attachment and modulated by the concentration of calcium in the fibre (Bagni et al., 1988; Stein et al., 1988; Wahr and Rall, 1997). The rate of cross bridge attachment differs between muscle fibre types with fast (type II) fibres having a greater rate of force development (Fitts and Widrick, 1996).
Rate of force development by muscles is often studied by investigating muscle response during a single twitch. Rochester et al. (1995) report that, as muscles rarely reach full activation during a single twitch, the rates measured from tetanic contractions will differ to single twitches. This review will therefore concentrate on activation rates measured from tetanic contractions as this is specific to the current project.

The number of papers quantifying the rate of force development in response to tetanic contractions is small compared to the number investigating single twitches. The results of these papers, however, tell a consistent story.

Backman and Henriksson (1988) studied the response of adductor pollicis muscles of children from three different age groups (15, 12 and 9 years). Table 2.5.7.2 shows force of contraction, contraction time and contraction rate. Values for 9 and 12 year olds are expressed as a percentage of the elder group for comparison. It can be seen that while the force of contraction and half time of contraction were quite different between groups, the contraction rate varied between groups as a similar percentage of the 15 year value to peak force. For this set of data, contraction rate appeared to vary as a function of peak force.

<table>
<thead>
<tr>
<th>Age</th>
<th>Force</th>
<th>Rise Rate</th>
<th>Rise Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>42.7 N (100%)</td>
<td>125 N s⁻¹ (100%)</td>
<td>171.0 ms (100%)</td>
</tr>
<tr>
<td>12</td>
<td>28.7 N (67%)</td>
<td>76.5 N s⁻¹ (62%)</td>
<td>187.5 ms (110%)</td>
</tr>
<tr>
<td>9</td>
<td>21.2 N (50%)</td>
<td>55.2 N s⁻¹ (44%)</td>
<td>192.2 ms (112%)</td>
</tr>
</tbody>
</table>

* Contraction time was reported as the time taken to achieve half the peak force.

** This column was calculated from the other data using \( \text{Rise Rate} = \frac{\text{Force}/2}{\text{time}} \)

Miller (1979) studied the first dorsal interosseus muscle of subjects with partial denervation of that muscle and compared results with a normal control group (Table 2.5.7.3). Miller’s data also suggest that rise rate may be proportional to peak force.
Table 2.5.7.3  Effect of nerve denervation on activation dynamics from Miller (1979)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Force</th>
<th>Rise Rate</th>
<th>Rise Time *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>2.01 kg (100%)</td>
<td>16.2 g ms⁻¹ (100%)</td>
<td>124 ms (100%)</td>
</tr>
<tr>
<td>Denervated</td>
<td>0.97 kg (48%)</td>
<td>8.68 g ms⁻¹ (54%)</td>
<td>112 ms (90%)</td>
</tr>
</tbody>
</table>

* Calculated from the other data using \( \text{time} = \frac{\text{Force}}{\text{Rate}} \)

Klein et al. (1988) investigated elderly subjects (66 years) and young subjects (24 years) undergoing tetanic contractions of the triceps surae muscles. Their results from contractions stimulated at 20 Hz are shown in Table 2.5.7.4.

Table 2.5.7.4  Effect of age on activation dynamics from Klein et al. (1988)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Force</th>
<th>Rise Rate *</th>
<th>Rise Time **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>832 N (100%)</td>
<td>830 N s⁻¹ (100%)</td>
<td>1.0 s (100%)</td>
</tr>
<tr>
<td>Elderly</td>
<td>649 N (78%)</td>
<td>450 N s⁻¹ (59%)</td>
<td>1.4 s (143%)</td>
</tr>
</tbody>
</table>

* Klein et al reported rate as % peak per ms. I have multiplied this by peak force to give a value in Newtons per second.

** Calculated values from the other data using \( \text{time} = \frac{\text{Force}}{\text{Rate}} \)

Data from Klein et al. (1988) do not support the hypothesis that the rate of force development is proportional to the peak force as well as the previous two studies. Bearing in mind that rate of force development is dependent on muscle fibre type, and that peak force is dependent on physiological cross section (Fitts and Widrick, 1996), it is not surprising that there are a variety of results present. Fibre type ratios differ between young and elderly subjects (Hunter
et al., 1999), so perhaps rate of force development is proportional to peak force only for comparisons where fibre type is kept constant.

Beelen et al. (1995) studied the rate of force development of the quadriceps muscles of healthy, able-bodied men cycling an isokinetic cycle ergometer using NMES elicited contractions. Crank torque due to muscle activity was calculated by subtracting crank torque measured during passive cycling from torque values during muscle stimulation. This situation is very close to the conditions of the present study, so the results are pertinent with the exception that they relate to able bodied subjects rather than paraplegics. Values for crank torque were not reported in absolute terms; only as a percentage of unfatiged values. Peak torque and rate of torque development were reported for eight subjects at varying stages of muscle fatigue. Values for peak torque and rate of torque development across all trials were highly correlated with an R² of 0.97². This suggests that, for a given subject contracting at different force levels, rate of force development will change proportionally with peak force.

**Typical values for rate of force decline in response to stimulation**

Backman and Henriksson (1988) also reported relaxation rate following tetanic stimulation for three age groups. Results shown below indicate that relaxation time was very similar for each age group. As peak force was quite different between groups, relaxation rate was varying proportionally with force to maintain constant relaxation time.

**Table 2.5.7.5**  Effect of age on relaxation dynamics from Backman and Henriksson (1988)

<table>
<thead>
<tr>
<th>Age</th>
<th>Force</th>
<th>Relaxation Rate *</th>
<th>Relaxation Time **</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>42.7 N (100%)</td>
<td>297 N s⁻¹ (100%)</td>
<td>144 ms (100%)</td>
</tr>
<tr>
<td>12</td>
<td>28.7 N (67%)</td>
<td>192 N s⁻¹ (67%)</td>
<td>149 ms (103%)</td>
</tr>
<tr>
<td>9</td>
<td>21.2 N (50%)</td>
<td>147 N s⁻¹ (69%)</td>
<td>144 ms (100%)</td>
</tr>
</tbody>
</table>

* Relaxation rate reported as % force lost after 100 ms. I have converted units to N s⁻¹

** Calculated from the other data using \( \text{time} = \frac{\text{Force}}{\text{Rate}} \)
Equations to model muscle activation

There have been a number of attempts to model muscle activation using equations. Hatze (1977) used a detailed model to estimate muscle activation. This was not a simple black box model. Rather, each step was justified using physiological events comprising the muscle contraction. While this approach provides face validity of the resulting equations, the complexities involved have been described as suffering from “colossal computational costs and a large degree of uncertainty and are therefore rendered of little practical value” (Baratta and Solomonow, 1992, p422).

While complex equations such as Hatze (1977) may be useful for investigating mechanisms controlling the rate of contraction, they are not necessary to give an excellent fit to the shape of muscle activation dynamics. The following equation was used by Pandy et al. (1990):

\[
\frac{da(t)}{dt} = \text{If } u(t) = 1, \quad \text{Then } \frac{1-a}{\tau_{\text{rise}}}, \quad \text{Else } \frac{a_{\text{min}} - a}{\tau_{\text{fall}}}
\]

Equation 2.5.7.1

where \(a(t)\) is activation of muscle

\(\tau_{\text{rise}}\) = rising time constant

\(\tau_{\text{fall}}\) = falling time constant

\(a_{\text{min}}\) = minimum activation level

\(u(t)\) = stimulation on (1) or off (0)

This equation was derived from that reported by Zajac (1989) for on-off type control systems. Controlling the rise and fall time constants alters the rise and fall time, allowing the equation to fit the contraction dynamics of different muscle types. Pandy et al. (1990) used rise and fall

\[\text{Correlation calculated by the present author from all data presented by Beelen et al. (1995).}\]
time constants of 20 ms and 200 ms respectively, indicating that the muscle would develop force after activation ten times faster than it would relax when switched off.

Schutte et al. (1993) used another modification of the equation from Zajac (1989) using quite different rise and decay times derived from the work of Krajl and Grobelnik (1973). Schutte et al. used the same time constant for both rise and fall (70 ms) giving rise and fall times for a typical muscle contraction of 175 and 150 ms. The calculated rise and fall times were slightly different, even though the time constants were identical, owing to the non-linear contraction dynamics of their cycling model. While the work of Krajl and Grobelnik (1973) is directly relevant to contractions elicited by NMES of paraplegic subjects, only very limited data was presented to justify these time constants.

The activation equation of Pandy et al (1990) uses a non-zero minimum activation level in order to avoid “problems associated with inverting the force-velocity curve of muscle at low activation levels”. This approach was taken after the work of Hatze (1977) and is essential to maintain mathematical stability of a model during periods of muscle inactivity.

2.5.8 Summary of muscle modelling parameters

Tables 2.4.8.1 to 2.4.8.3 summarise the constants used for all future modelling. The derivations of these constants have been described throughout the present chapter. These tables, therefore, provide a summary of this chapter.
### Table 2.5.8.1  Summary of model constants common to all muscles

<table>
<thead>
<tr>
<th>Model Parameter</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force/Length Eqtn</td>
<td></td>
<td>Delp et al. (1990)</td>
</tr>
</tbody>
</table>
| Hill’s Constants                 |\begin{align*}
    \text{brf} &= 2.25 \times \text{llorf} \\
    \text{arf} &= 0.35 \times \text{fforf}
\end{align*} | Pierrynowski and Morrison, (1985)               |
| Tendon Stiffness                 |\begin{align*}
    \text{ktrf} &= \text{fforf} / \text{sltrf} / 0.033
\end{align*} | Zajac (1989)                                   |
| Activation Time Constants        |\begin{align*}
    \text{amin} &= 0.01 \text{ s} \\
    \text{risdelay} &= 0.05313 \text{ s} \\
    \text{tris} &= 0.04802 \text{ s} \\
    \text{tfall} &= 0.04819 \text{ s}
\end{align*} | Measured in Section 6.1.8 of the present study. |
| Maximum Eccentric Force #        |\begin{align*}
    \text{eccrf} &= 1.25
\end{align*} | Pierrynowski and Morrison (1985)                |

# Maximum eccentric force is given as a ratio to maximum isometric force.

Note: the suffix rf is applied to some of these variables indicating how they were calculated for the rectus femoris muscle. These formulae were applied similarly to other muscles.
### Table 2.5.8.2  Summary of model constants for the quadriceps muscles

<table>
<thead>
<tr>
<th>Model Parameter</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee Moment Arms*</td>
<td>$drfk[t] := 0.0347 - 0.01469 * qk[t] - 0.0084857 * qk[t]^2 \text{ m}$</td>
<td>Kellis and Baltzopoulos (1999)</td>
</tr>
<tr>
<td>RF moment arm at hip*</td>
<td>$drfh[t] := 0.0259 \text{ m}$</td>
<td>Average of Hawkins and Hull (1990) and Meijer et al. (1988a)</td>
</tr>
<tr>
<td>Muscle Neutral Length*</td>
<td>$lmtrf0 = 0.4317 + 0.02594 * qh[0] - 0.03473 * qk[0] + 0.007346 * qk[0]^2 + 0.007591 * qk[0]^3 \text{ m}$</td>
<td>Hawkins and Hull (1990)</td>
</tr>
<tr>
<td></td>
<td>$lmtv0 = 0.2020 - 0.03473 * qk[0] + 0.007346 * qk[0]^2 + 0.007591 * qk[0]^3 \text{ m}$</td>
<td></td>
</tr>
<tr>
<td>Optimum Fibre Length**</td>
<td>$lforf = 0.07452 \text{ m}$</td>
<td>Wickiewicz et al. (1983)</td>
</tr>
<tr>
<td></td>
<td>$lfov = 0.085161 \text{ m}$</td>
<td></td>
</tr>
<tr>
<td>Pennation angle</td>
<td>$pennangorf = 0.175 \text{ radians}$</td>
<td>Pierrynowski (1995)</td>
</tr>
<tr>
<td></td>
<td>$pennangov = 0.157 \text{ radians}$</td>
<td></td>
</tr>
<tr>
<td>Relative muscle cross sections (and hence force)</td>
<td>$ffov = 5.04 \times fforf$</td>
<td>Pierrynowski (1995)</td>
</tr>
<tr>
<td>Tendon Slack Length</td>
<td>$sltrf = 0.351034 \text{ m}$</td>
<td>Fitted to experimental data in Section 6.1.2 of the present study.</td>
</tr>
<tr>
<td></td>
<td>$sltv = 0.155585 \text{ m}$</td>
<td></td>
</tr>
</tbody>
</table>

* Lengths from the literature were adjusted proportionally to a thigh length of 0.39 m. When thigh length was not available, 23.2% of standing height was used as an estimate (Plagenhoef et al., 1983).

** Fibre lengths from Wickiewicz et al. (1983) were scaled up to use an optimum sarcomere length of 2.64 µm (see Section 3.2).
Table 2.5.8.3  Summary of model constants for the hamstring muscles

<table>
<thead>
<tr>
<th>Model Parameter</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee Moment Arms*</td>
<td>( dhk[t] := 0.0226961 - 0.0171859 * qk[t] - 0.00957207 * qk[t]^2 ) m</td>
<td>Buford et al. (1997)</td>
</tr>
<tr>
<td>Hip moment arms*</td>
<td>( dhh[t] := 0.0712255 + 0.0160201 * qh[t] ) m</td>
<td>Visser et al. (1990)</td>
</tr>
<tr>
<td>Muscle Neutral Length*</td>
<td>( lmt0 = 0.3920 + 0.0226961 * qk[0] - 0.0171859 * qk[0]^2/2 - ) m</td>
<td>Hawkins and Hull (1990)</td>
</tr>
<tr>
<td></td>
<td>( 0.00957207 * qk[0]^3/3 - 0.0712255 * qh[0] - 0.0160201 * qh[0]^2/2 ) m</td>
<td></td>
</tr>
<tr>
<td>Optimum Fibre Length**</td>
<td>( lfoh = 0.0963 ) m</td>
<td>Wickiewicz et al. (1983)</td>
</tr>
<tr>
<td>Pennation angle</td>
<td>pennangoh = 0.1955 radians</td>
<td>Pierrynowski (1995)</td>
</tr>
<tr>
<td>Tendon Slack Length</td>
<td>( slth = 0.385 ) m</td>
<td>Fitted to experimental data in Section 6.2.1 of the present study.</td>
</tr>
<tr>
<td>Normative data for setting slack lengths</td>
<td></td>
<td>Knapik et al. (1983)</td>
</tr>
</tbody>
</table>

* Lengths from the literature were adjusted proportionally to a thigh length of 0.39 m. When thigh length was not available, 23.2% of standing height was used as an estimate (Plagenhoef et al., 1983).

** Fibre lengths from Wickiewicz et al. (1983) were scaled up to use an optimum sarcomere length of 2.64 \( \mu \)m (see Section 3.2)
### Table 2.5.8.4  Summary of model constants for gluteus maximus

<table>
<thead>
<tr>
<th>Model Parameter</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip moment arms*</td>
<td>(d_{gh}[t] := (0.05319 + 0.018876 \cdot q_{h}[t] + 0.003431 \cdot q_{h}[t]^2) m</td>
<td>Schutte (1992)</td>
</tr>
<tr>
<td>Muscle Neutral Length*</td>
<td>(l_{mtg0} = 0.1419 - 0.05319 \cdot q_{h}[0] - 0.018876 \cdot q_{h}[0]^2/2 - 0.003431 \cdot q_{h}[0]^3/3) m</td>
<td>Nemeth and Ohlsen (1985)</td>
</tr>
<tr>
<td>Optimum Fibre Length</td>
<td>(l_{fog} = 0.18) m</td>
<td>Hoy et al. (1990)</td>
</tr>
<tr>
<td>Pennation angle</td>
<td>(pennangog = 0.059) radians</td>
<td>Hoy et al. (1990)</td>
</tr>
<tr>
<td>Tendon Slack Length</td>
<td>(s_{tg} = 0.05) m</td>
<td>Fitted to experimental data in Section 6.2.2 of the present study.</td>
</tr>
<tr>
<td>Normative data for setting slack lengths</td>
<td></td>
<td>Nemeth et al. (1983)</td>
</tr>
</tbody>
</table>

* Lengths from the literature were adjusted proportionally to a thigh length of 0.39 m. When thigh length was not available, 23.2% of standing height was used as an estimate (Plagenhoef et al., 1983).