Quantification of Abnormal Muscle Tone Following Spinal Cord Injury: A Clinical Perspective

Fiona Margaret Douglas Barr

A thesis submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the University of London

Institute of Orthopaedics University College London

The copyright of this thesis rests with the author & no quotation from it or information derived from it may be published without prior written consent of the author.

ProQuest Number: 10797844

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10797844

Published by ProQuest LLC (2018). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code

Microform Edition © ProQuest LLC.

ProQuest LLC. 789 East Eisenhower Parkway P.O. Box 1346 Ann Arbor, MI 48106 – 1346

Abstract

Abnormal muscle tone is a frequent complication following traumatic spinal cord injury with subsequent upper motor neurone paralysis. Treatment of this complex problem presents many difficulties for the clinician and appropriate assessment is a vital pre-requisite when devising a pertinent treatment regimen. The purpose of this study was to examine a variety of measures and to create a comprehensive assessment programme to quantify abnormal muscle tone in this patient group which was compatible with use in the clinical environment.

The study examined the reliability of the five measurement techniques: therapist rating, patient rating, electrogoniometry and dynamometry augmented by polyelectromyography, prior to implementing the combined assessment programme in a group of spinal cord injured and neurologically intact subjects. The relationships between the findings of each assessment were examined and the suitability of the combined assessment programme was considered for routine use in the clinical environment.

All the measures examined in the study proved to be reliable, able to differentiate between the spinal cord injured and neurologically intact subjects and practical for implementation within the clinical environment. The lack of correlation between the findings of the singular components of the assessment programme supports the hypothesis that it is necessary to include all the measures investigated if the clinician wishes to undertake a comprehensive evaluation of the individual person presenting with abnormal muscle tone following spinal cord injury.

Acknowledgements

The nature and the duration of this study have elicited the interest and cooperation of many people without whose contribution this work would not have come to fruition.

I would like to acknowledge the advice and support of my two supervisors, Dr Frederick Middleton FRCP & Professor Giles Brindley FRS and their contribution to the planning, experimentation and documentation stages of the study.

Many of my colleagues from the Royal National Orthopaedic Hospital Trust have assisted in the study with the development of equipment, provision of advice - scientific, statistical and practical - and several gave hours of their valuable time to participate in the experiments. Particular thanks should go to Dr Graham Phillips for his technical and programming expertise, the staff of the Centre for Disability & Research & Innovation, and to Professor Roger Woledge from the Institute of Human Performance for his enthusiastic support of the original concept and his invaluable assistance in the analysis of the vast amount of data collected in this study.

My thanks also to Dr Duncan Wood from the Department of Medical Physics & Bioengineering, Salisbury District Hospital for his part in the provision of analysis software.

The generous financial support of the Sir Jules Thorn Trust which enabled the purchase of equipment for the study and the loan of the Kin Com dynamometer from the Chattanooga Corporation (UK) for the duration of the experimental stages were vital to the completion of this work. My thanks also to colleagues at Digitimer for their patience and persistence in the design and production of the specialised equipment required to undertake the study.

I would like to thank my family and friends for their confidence in my ability to complete the study and their practical assistance in helping me to accomplish this.

Finally I would like to dedicate this thesis to my dear father who was always so supportive of my work but sadly did not see the completion this study.

Contents

7	•	•	2	1	ĸ	
		1	1	۲.		Ω
		E	н	ы		L

Abstract		1
Acknowledg	gements	2
Contents		4
Index of Ta	bles	6
Index of Fig	ures	9
Index of Ap	pendices	10
Chapter 1	Introduction	12
1.1	Spinal Cord Injury	18
1.1.1.	Anatomy	19
1.1.2.	Pathology	21
1.1.3.	Classification	25
1.1.4.	Incidence	31
1.1.5	Treatment & Prognosis	34
1.2	Spasticity	43
1.2.1.	Muscle	44
1.2.2.	Spasticity in Spinal Cord Injury	56
1.2.3.	Treatment of Spasticity	59
1.3	Measurement of Abnormal Muscle Tone	70
1.3.1.	Principles of Measurement	70
1.3.2.	Measurement techniques	77
1.3.3.	Subjective Measures	79
1.3.4.	Objective Measures	87
Chapter 2	Development of Method	104
2.1.	Introduction	104
2.2.	Method	110
2.2.1		110
	Subjects	112
2.2.2	Instruments	117
2.2.3	Procedure	11/
2.3	Results	128
2.3.1	Subjects	128
2.3.2	Subjective Measures	128
2.3.3	Objective Measures	130
2.4	Discussion	136
2.5	Conclusion	138

Contents - Continued

Chapter 3	Method	140
3.1	Subjects	140
3.2	Instruments	141
3.3	Procedure	143
3.3.1	Subjective Measures	145
3.3.2	Objective Measures	146
Chapter 4	Results	152
4.1	Subjects	152
4.2	Subjective Measures	153
4.2.1	Therapist Rating	153
4.2.2	Patient Rating	154
4.3	Objective Measures	156
4.3.1.	Pendulum Test	156
4.3.2.	Electromyography	161
4.3.3.	Dynamometry	167
Chapter 5	Discussion	171
5.1	Method	171
5.2	Results	177
5.3	Limitations of Study	190
5.4	Suggestions for Further Study	192
Chapter 6	Conclusion	194
References		198
Appendices		220

Index of Tables

Chapter 1		
Table 1.1	ASIA Impairment Scale	27
Table 1.2	Clinical Syndromes	28
Table 1.3	Sensory Classification	29
Table 1.4	Motor Classification	30
Table 1.5	Ashworth Scale	79
Table 1.6	Modified Ashworth Scale	80
Table 1.7	Electrophysiological Testing for Spasticity	88
Chapter 2		
Table 2.1	Modified Ashworth Scale	112
Table 2.2	Numerical Rating Scale	113
Table 2.3	Neurolog Settings	122
Table 2.4	Neurolog Input/Output Settings	123
Table 2.5	Electrode Tests	125
Table 2.6	Ashworth Scores	129
Table 2.7	Patient Rating Scale – P values	130
Table 2.8	Patient Rating Scale – R values	130
Table 2.9	Intra-Tester Measurement of Knee Extension	132
Table 2.10	Electromyograph Calibration	133
Table 2.11	Skin Impedance With Different Preparation Techniques	134
Table 2.12	Electrode Comparison	135

Index of Tables - Continued

Chapter 3		
Table 3.1	Procedural Order	145
Table 3.2	Electrode Positions	148
Chapter 4		
Table 4.1	Correlation Coefficients of Patient Rating	155
Table 4.2	Correlation Coefficients of Modified Ashworth Score and Patient Ratings	156
Table 4.3	Pendulum Test Intra-test Reliability	157
Table 4.4	Correlation Coefficients of Modified Ashworth Score And Relaxation Index	158
Table 4.5	Correlation Coefficients Between Patient Rating Scores & Relaxation Indices	159
Table 4.6	Change in Electrode Impedance – P Values – Right Leg	161
Table 4.7	Change in Electrode Impedance – P Values - Left Leg	161
Table 4.8	Intra-test Repeatability – Average EMG Activity - P values	163
Table 4.9	Inter-test Repeatability – Average EMG Activity P values	164
Table 4.10	Correlation Coefficients Between EMG Signal from Test 1 & Test 6	165
Table 4.11	Correlation Coefficients Between Torque & Relaxation Indices	168
Table 4.12	Correlation Coefficients Between Torque & Modified Ashworth Score	169

Index of Tables - Continued

Chapter 4		
Table 4.13	Correlation Coefficients Between Torque & Patient Rating of Spasm Strength	169
Table 4.14	Correlation Coefficients Between Torque & Patient Rating of Spasm Frequency	170
Table 4.15	Correlation Coefficients Between Torque & Patient Rating of Impairment of Function	170

Index of Figures

Chapter 1		
Figure 1.1	Anatomy of Spinal Column & Cord	19
Figure 1.2	Transverse Section of Spinal Cord	20
Figure 1.3	Afferent & Efferent Pathways	46
Chapter 2		
Figure 2.1	Visual Analogue Scale	113
Figure 2.2	Marked Visual Analogue Scale	113
Figure 2.3	Kin Com Dynamometer	114
Chapter 5		
Figure 5.1	Data Output – Velocity - 200°/second – Neurologically Intact Subject	180
Figure 5.2	Data Output – Velocity - 0°/second – SCI Subject	180
Figure 5.3	Data Output – Velocity - 200°/second – SCI Subject	183

Index of Appendices

Chapter 1		
Appendix 1.1	ASIA Key Sensory & Motor Testing Points	221
Chapter 2		
Appendix 2.1	Technical Specification for Neurolog Polyelectromyograph	222
Appendix 2.2	Project Information Sheet	224
Appendix 2.3	Consent Form	225
Appendix 2.4	Patient rating Scale Record Sheet	227
Appendix 2.5	Pilot Study Subjects	228
Chapter 3		
Chapter 3		
Appendix 3.1	MathCad Analysis Programmes	22 9
Chapter 4		
Appendix 4.1	Subject Details	233
Appendix 4.2	Spinal Injury Subjects – Level of Injury	234
Appendix 4.3	Subjective Rating Scores	235
Appendix 4.4	Relaxation Indices – SCI Subjects	237
Appendix 4.5	Relaxation Indices – Neurologically Intact Subjects	238
Appendix 4.6	Electrode Impedance – SCI Subjects	239
Appendix 4.7	Electrode Impedance – Neurologically Intact Subjects	240
Appendix 4.8	Relationship Between EMG & Torque – Phase 1 PAUSE – Vastus Medialis	242
Appendix 4.9	Relationship Between EMG & Torque – Phase 1 PAUSE – Biceps Femoris	243

Index of Appendices – Continued

Chapter 4

Appendix 4.10	Relationship Between EMG & Torque – Phase 1 PAUSE – Tibialis Anterior	244
Appendix 4.11	Relationship Between EMG & Torque – Phase 1 PAUSE – Gastrocnemius	245
Appendix 4.12	Relationship Between EMG & Torque – Phase 2 Extension to Flexion - Vastus Medialis	246
Appendix 4.13	Relationship Between EMG & Torque – Phase 2 Extension to Flexion – Biceps Femoris	247
Appendix 4.14	Relationship Between EMG & Torque – Phase 2 Extension to Flexion – Tibialis Anterior	248
Appendix 4.15	Relationship Between EMG & Torque – Phase 2 Extension to Flexion – Gastrocnemius	249
Appendix 4.16	Relationship Between EMG & Torque – Phase 3 PAUSE - Vastus Medialis	250
Appendix 4.17	Relationship Between EMG & Torque – Phase 3 PAUSE – Biceps Femoris	251
Appendix 4.18	Relationship Between EMG & Torque – Phase 3 PAUSE – Tibialis Anterior	252
Appendix 4.19	Relationship Between EMG & Torque – Phase 3 PAUSE – Gastrocnemius	253
Appendix 4.20	Relationship Between EMG & Torque – Phase 4 Flexion to Extension - Vastus Medialis	254
Appendix 4.21	Relationship Between EMG & Torque – Phase 4 Flexion to Extension – Biceps Femoris	255
Appendix 4.22	Relationship Between EMG & Torque – Phase 4 Flexion to Extension – Tibialis Anterior	256
Appendix 4.23	Relationship Between EMG & Torque – Phase 4 Flexion to Extension – Gastrocnemius	257

Chapter 1 Introduction

Traumatic spinal cord injury is a catastrophic event in the life of an individual. The consequent impairment of such an injury is loss or reduction of muscle activity, sensory deprivation and disruption of autonomic function, related to the anatomical level & severity of the cord damage. The goals for rehabilitation in this patient group are to minimise the impairment, optimise physical and psychological function, reduce the incidence of secondary complications and promote reintegration of the individual back into the community environment of their choice (Bedbrook 1980).

Patients suffering insult to the cord resulting in an upper motor neurone paralysis will present with spasticity, a velocity dependent increase in tonic stretch reflexes with exaggerated tendon jerks, and involuntary spasms to a lesser or greater degree in all the muscles below the cord lesion. The presence of an excessive level of spasticity or severe muscle spasms in patients with complete or incomplete paralysis may cause secondary complications such as contractures, pain, pressure sores and a reduction in functional ability, all of which may affect the quality of life for an individual (Westgren 1998).

Treatment of spasticity and muscle spasms post spinal cord injury may be conservative or surgical. Drug therapy via oral, intramuscular or intrathecal administration modifies tonic activity by acting centrally or directly on the central nervous system at spinal level or at the neuromuscular junction (Priebe 1997, Katrak 1992, Pierson 1996). In addition to reducing abnormal muscle tone, these medications often result in other systemic reactions such as

drowsiness, respiratory depression and a reduction in available voluntary muscle function such that the treatment is unsuitable.

In patients with a severe degree of spasticity, more radical surgical procedures, myotomy, tenotomy and neurotomy have been used to reduce activity in key muscles. Posterior and anterior rhizotomy will eradicate activity in the spastic muscle through disruption of the reflex pathways. This extreme measure is unsuitable for patients with incomplete neurological loss as any residual sensory or motor preservation will be lost (Edgar 1992). Epidural stimulation of the posterior columns of the spinal cord has been shown to reduce spasticity and spasms in spinal cord injury but the invasive nature of the intervention and lack of evidence for long term clinical efficacy has led clinicians to favour other modalities (Midha 1998).

Physiotherapy provides a non-invasive means of treatment of abnormal muscle tone. Techniques include passive stretching and standing (Knutsson 1981, Bohannon 1993), re-education of normal movement patterns (Bobath 1978, Carr & Shaped 1998), cryotherapy (Price 1993), hydrotherapy (Bedbrooke 1985) and electrotherapy (Bajd 1985, Alfieri 1982, Shindo & Jones 1987, Robinson et al 1988). Studies demonstrating successful outcomes with each of these treatments have been conducted in a variety of patient groups using subjective and objective methods of quantifying spasticity in both the laboratory and clinical environment.

In order to establish the most appropriate regime of treatment to reduce abnormal muscle tone, it is essential to perform a comprehensive and holistic assessment of the patient. Quantification of abnormal muscle tone presents many problems to the rehabilitationist. In addition to the complexity of the mechanisms involved in the regulation of muscle tone, changes in the spastic state occur due to systemic factors (presence of infection, pressure sores, emotional status), environmental changes (temperature, pressure surface) and training effects during the procedure. Diurnal variation may also occur due to these and other factors.

Different methods, both subjective and objective, of quantifying abnormal muscle tone have been reported. Subjective rating scales have frequently been employed in clinical practice (Ashworth 1964, Bohannon 1987) to grade levels of spasticity from absent to severe. Such scales lack inter-tester reliability and show a cluster effect around the middle grades. Indices of function examining activities of daily function such as the Barthel Index (Mahoney 1965) or Functional Independence Measure (Granger 1986) have been applied, relating the level of spasticity to impairment of function but these again lack sensitivity as a gross difference in spasticity must occur for a global change in the functional capacity of the patient to be observed.

Electrophysiological measurement techniques, examining motor and sensory reflex responses, have been employed to assess muscle and nerve pathology and establish correlations with the degree of spasticity (Delwaide 1984, Katz 1989). Surface and needle electromyography have been employed to examine

individual motor unit function and gross muscle activity following spinal cord injury and cerebro-vascular accident (Campbell et al 1991, Sköld 1998). The equipment required for such analysis is not however readily available in the clinical setting and therefore these methods for quantification of spasticity have, to date, been restricted to the laboratory.

Various biomechanical tests have been devised to examine different components of muscle tone within both the clinical and laboratory environment. Bajd et al (1982) further developed an electrogoniometer to examine the range and rate of angular change in the knee to assess spasticity in the quadriceps and hamstrings. This method shows good reproducibility but is confined to only two muscle groups of the lower limb and does not exclude the involvement of soft tissue changes. Measurement of the torque generated during passive or active movement of the limb and velocity dependence or change in electromyographic activity have also been examined to create an index of spasticity (Knutsson 1980, Katz 1989). These studies have again been conducted within the laboratory environment.

More recently, the increased presence of dynamometers in the physiotherapy clinic has enabled the clinician to record the moment generated about the joint when passive motion of the limb is performed (Bohannon 1989). Early work at the Royal National Orthopaedic Hospital (Barr 1989) using a dynamometer to record torque generated during passive movement of the lower limb at pre-set velocities was considered to give an overall assessment of the resistance to

passive movement but the test did not differentiate between resistance related to soft tissues & the degree of activity in individual muscle groups.

In an attempt to describe this complex phenomenon, recent studies have combined assessment techniques to add a broader perspective to the examination of abnormal muscle tone (Bohannon 1987, Sköld 1998, Lamontagne 1998). This has demonstrated relationships between a small number of variables but the techniques employed remain limited when considering the holistic management of the patient.

The purpose of this study was to devise a comprehensive assessment programme of objective and subjective measures to quantify abnormal muscle tone in patients with spinal cord injury which was appropriate for routine use in the clinical environment. The study evaluated the reliability of five techniques for the assessment of abnormal muscle tone, examined the relationship between the findings of each assessment technique and considered the compatibility of the assessment programme with the clinical environment.

The hypotheses tested were:-

- 1. Dynamometry is a reliable method of measurement of muscle tone in the lower limbs of patients with complete spinal cord injury.
- 2. Surface electromyography is a reliable method of measurement of muscle activity in the lower limbs of patients with complete spinal cord injury.

- 3. Electrogoniometry is a reliable measure of lower limb muscle tone in spinal cord injury patients.
- 4. There is a correlation between electromyographic activity & resistance to passive movement of the lower limb in patients with complete spinal cord injury.
- 5. There is a correlation between therapist rating of spasticity and resistance to passive movement.
- 6. There is a correlation between patient rating of spasticity and resistance to passive movement.
- 7. There is a correlation between the therapist and patient rating of spasticity.
- 8. Dynamometry, electromyography, electrogoniometry, therapist and patient rating are all valid assessment techniques for the measurement of abnormal muscle tone following spinal cord injury in the clinical environment.

1.1. Spinal Cord Injury

Spinal cord injury, through trauma or disease, has been described by clinicians as one of the most catastrophic conditions in all of medicine (Stover 1986).

Injury to the cord may result from bony or soft tissue intrusion or compression of the cord following disruption of the vertebral canal. Damage to the cord may also be sustained in the absence of bony injury whereby sufficient force has been applied to disrupt the vascular supply to the cord. The consequent impairment - 'any loss or abnormality of psychological, physiological or anatomical structure or function' (W.H.O 1980) - of such an injury is loss or reduction of muscle activity, sensory deprivation and disruption of autonomic function, related to the anatomical level & severity of the cord damage. These in turn may lead to long term disability - 'any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being' (W.H.O 1980) – eg. inability to walk, feed or dress oneself. A further consequence may be handicap - 'a disadvantage for a given individual, resulting from an impairment or disability, that limits or prevents the fulfillment of a role that is normal (depending on age, sex and social and cultural factors) for that individual' (W.H.O 1980) for the individual who will require specialised rehabilitation post injury and life long strategic management.

1.1.1. Anatomy

The spinal cord is a cylindrical extension of the cerebrospinal axis, running from the foramen magnum at the base of the skull, down the length of the vertebral column in the vertebral canal, terminating at the conus medullaris between the level of the first and second lumbar vertebrae. Extending caudally from the conus is a slender filament, the filum terminale, which attaches to the posterior aspect of the coccyx. The segmental levels of the cord are classified into four areas, cervical, thoracic, lumbar and sacral as shown in Figure 1.1.

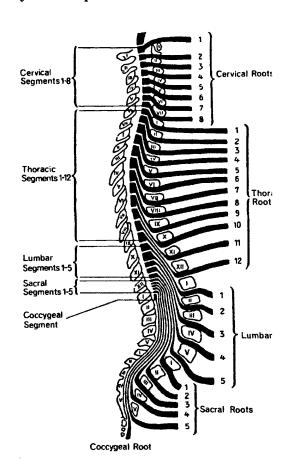
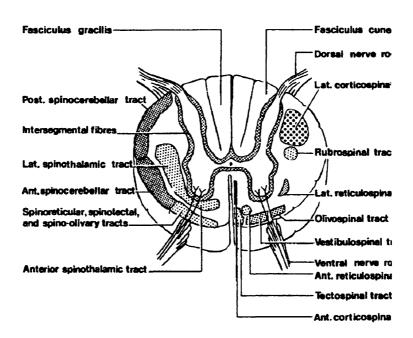


Figure 1.1: Anatomy of the Spinal Column & Cord

(From Haymaker W 1969 Bing's local diagnosis in neurological diseases, 15th edition. Mosby, St. Louis)

The cord contains the ascending and descending sensory and motor tracts that connect the cerebral cortex, cerebellum, medulla oblongata, and pons with the peripheral organs and limbs (Mitchell 1977). Transection of the cord reveals that each half of the cord comprises four columns - anterior, posterior, lateral and postero-median (see Figure 1.2). The outer white matter contains longitudinal medulated fibres running between the brain and the periphery, blood vessels and neuroglia (connective tissue). The grey matter, distinguished by its colour and anterior and posterior horn formation, contains transverse medulated fibres and neuroglia and provides the connection between the efferent sensory pathways and the afferent output to the motor organs.

Figure 1.2: Transverse Section of Spinal Cord



(From Mitchell 1977 Essentials of Neuroanatomy 3rd edition, Churchill Livingstone. Edinburgh)

The cord is surrounded by three protective membranes. The dura mater is the outer tough fibrous sheath; the pia mater, a delicate vascular structure that

surrounds the cord directly; and between the two lies the arachnoid membrane, a fibrous membrane which is connected to the pia mater by slender filaments. In the space between the arachnoid and pia mater flows the cerebrospinal fluid, the primary function of which is to protect the cord from concussion from external forces (Mitchell 1977).

Attached to the cord at every vertebral level is a pair of spinal nerves, one serving each side of the body. Each spinal nerve has a posterior and anterior nerve root. The posterior root enters the posterior horn of the cord and divides into an ascending and descending branch. The descending branches synapse with other nerve cells in the posterior horn, traversing the grey matter to synapse with the alpha motor neurone cells in the ipsi or contralateral anterior horn. Fibres from the ascending branch pass up the cord and terminate in the gracile and cuneate nuclei in the medulla oblongata of the brain. The anterior nerve roots are a continuation of the axons of alpha motor neurones from the anterior horn.

1.1.2. Pathology

Definition of the pathological changes following damage to the cord has relied predominantly on observation of post-mortem evidence (Hughes 1984, Kakulas & Taylor 1992). Kakulas & Taylor (1992) reported from a series of three hundred and eighty five traumatic spinal cord injured cadavers that changes occur in three stages, early, intermediate and late. Control of the time between injury and examination, to determine the time scales for pathological change, was difficult as it was dependent on the length of survival of the patient post

injury. In a review of several hundred post mortem cases, Hughes (1984) reported that changes in the early stage relate to damage or severance of the nerve fibres but both post mortem studies (Kakulas & Taylor 1992) and magnetic resonance imaging (Silberstein 1992) have shown that the extent of neurological damage is not dependent on the degree of skeletal damage. The inflammatory response to trauma causes the axon of the nerve and the myelin sheath surrounding it to swell and disintegrate. Swelling (oedema) and haemorrhage also occur within the inter-cellular tissues. This increase in volume of the cord in the confined space of the vertebral canal may cause additional neural compromise above and below the lesion (Kakulas & Taylor 1992).

Any significant injury to the cord below the mid pons will result in 'spinal shock'. The vascular and biochemical changes occurring in the first hours post injury will affect synaptic transmission and receptor function, causing impaired sensory, motor and reflex activity (Bach-y-Rita 1993). The patient will present clinically with flaccid, a-reflexic limbs below the level of the injury. The duration of spinal shock varies considerably, from minutes to hours and in the more severe injuries for several weeks post injury.

As resolution occurs, there is an altered level of reception to stimuli: stimuli must be strong or prolonged but this changes to a state of hypersensitivity whereby small stimuli can elicit a large muscle response. Resolution is also indicated by the staged return of reflex activity in a recognised order (Bach-y-Rita 1993): Firstly, the knee jerk, followed by the crossed extensor reflex and

extensor thrust and finally the stretch reflex, as described by Sherrington (1932).

The physiology of spinal shock and the development of spasticity and muscle spasms as the spinal shock resolves remains a challenge to the scientists. In a comprehensive review of work undertaken in both animal and human models over a twenty year period, Bach-y-Rita and Illis (1993) hypothesised that the recovery of reflexes is related to the regulation of receptors at the synapse and on the surface of partially denervated spinal cord cells. This causes an increased sensitivity to neurotransmitters which are released at the surviving synapses or elsewhere and may be transported in the extracellular fluid resulting in nonsynaptic transmission. Anecdotally, clinicians observe patterns of abnormal muscle tone evolving in specific patient groups: spasticity appears much earlier and in a more severe form in patients with incomplete cord transection (Stover et al 1995). To date these observations have not been examined with respect to history, pathology and severity of the injury. Bach-y-Rita & Illis (1993) correctly point out that provision of 'a convincing explanation' of the process of recovery of reflexes would greatly enhance the clinicians understanding of the ongoing pathological and reparative processes which may in turn contribute to more appropriate clinical management of patients in this crucial phase post injury.

Two to three weeks after the initial injury occurs, the intermediate or reparative phase begins. The oedema and smaller haemorrhages have been reabsorbed but the larger haemorrhages may lead to the formation of longitudinal cysts (syrinx)

in the cord. This intermediate stage can extend to as long as three years but symptoms from the cysts may not present until after that time (Williams 1990). In a review of thirty one cases, Williams (1990) was unable to identify predictive factors to identify patients at risk of post traumatic syringomyelia but proposed the continuation of prospective studies, utilising MRI scanning to monitor cord anatomy in patients undergoing either surgical or conservative management to guide the future treatment regimens for patients.

In the late stage, traumatic scar tissue will form, adhering the meninges to the spinal canal. Grossly damaged areas are replaced with fibrous tissue which again will contribute to adverse neural tension. Also during this stage, the cell bodies in the posterior root regenerate central processes into the region of the scar tissue. These will ramify into the connective tissue but do not penetrate into intact nervous tissue (Hughes 1984).

Each of these physiological changes manifest in clinical symptoms. The hypersensitivity and excessive response to efferent stimulation present as spasticity and involuntary muscle spasms which persist throughout life. Neurological improvement may be observed during the first year and some minor changes may be observed up to three years post injury, reflecting the intermediate stage, after which deterioration or alteration of neurological function, an increase in spasticity, muscle spasms and pain are often reported. These observations reflect the end effect of the damaged spinal cord but to date there is no conclusive evidence available to demonstrate whether the neuroanatomy and physiology remain the same or are subject to change.

1.1.3. Classification

The international community involved in the management of spinal cord injury (SCI) advocated the development of a recognised standard for the classification of skeletal damage, motor and sensory deficit following spinal cord injury.

The standards for neurological classification of impairment following SCI developed by the American Spinal Injuries Association (ASIA) in 1982 were considered by a panel of experts from the International Medical Society of Paraplegia (IMSOP) and amended to improve sensitivity and reliability. The revised standards were endorsed as the recognised international standard by IMSOP in 1992. The ASIA format provides a standardised vocabulary which includes the two terms, tetraplegia and paraplegia, that are most frequently used to describe the impairment following spinal cord injury.

Tetraplegia

'This term refers to impairment or loss of motor and/or sensory function in the cervical segments of the spinal cord due to damage of the neural elements within the spinal canal. Tetraplegia results in impairment of function in the arms as well as in the trunk, legs and pelvic organs. It does not include brachial plexus lesions or injury to peripheral nerves outside the neural canal.'

Dittuno 1994

Paraplegia

'This term refers to impairment or loss of motor and or sensory function in the thoracic, lumbar or sacral (but not cervical) segments of the spinal cord, secondary to damage of neural elements within the spinal canal. With paraplegia, arm functioning is spared, but depending on the level of injury, the trunk, legs and pelvic organs may be involved. The term is used in referring to cauda equina and conus medullaris injuries, but not to lumbosacral plexus lesions or injury to peripheral nerves outside the neural canal.'

Dittuno 1994

Within the ASIA method of classification of impairment, it is important to differentiate between the neurological level and the skeletal level of injury, as these may often be different. The neurological level refers to

'The most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body.'

Dittuno 1994

The skeletal level refers to

'The level at which, by radiographic examination, the greatest vertebral damage is found.'

Dittuno 1994

One further common term is the zone of partial preservation (ZPP).

'This term refers to those dermatomes and myotomes caudal to the neurological level that remain partially innervated. When some impaired sensory and/or motor function is found below the lowest normal segment, the exact number of segments so affected should be recorded for both sides as the ZPP. The term is used only with complete injuries.'

Dittuno 1994

Classification of the sensory and motor neurological impairment resulting from injury to the spinal cord has been modified from the Frankel Scale and is shown in Table 1.1.

Table 1.1: ASIA Impairment Scale

Grade	Classification
	Complete. No sensory or motor function is preserved in the sacral
A	segments S4-5
	Incomplete. Sensory but no motor function is preserved below the
В	neurological level and extends through the sacral segments S4-5
	Incomplete. Motor function is preserved below the neurological
C	level, and a majority of key muscles below the neurological level
	have a muscle grade less than 3
	Incomplete. Motor function is preserved below the neurological
D	level, and a majority of key muscles below the neurological level
	have a muscle grade greater than or equal to 3
E	Normal. Sensory and motor function is normal

Dittuno 1994

In addition to the complete and incomplete cord lesions, the impairment classification also includes the description of five clinical syndromes, relating to damage at particular levels of the cord, as described in Table 1.2.

Table 1.2: Clinical Syndromes

Syndrome	Description
	A lesion occurring almost exclusively in the cervical
Central Cord	region, that produces sacral sensory sparing & greater
	weakness in the upper limbs than in the lower limbs
	A lesion that produces relatively greater ipsi-lateral
Brown-Séquard	proprioceptive and motor loss and contra-lateral loss of
	sensitivity to pain & temperature
	A lesion that produces variable loss of motor function and
Anterior Cord	of sensitivity to pain and temperature while preserving
	proprioception.
	Injury of the sacral cord (conus) and lumbar nerve roots
Conus Medullaris	within the neural canal, which usually results in an a-
	reflexic bladder, bowel & lower limbs. Sacral segments
	may occasionally show preserved reflexes eg. Bulbo-
	cavernosus & micturition reflexes.
	Injury to the lumbosacral nerve roots within the neural
Cauda Equina	canal resulting in a-reflexic bladder, bowel & lower limbs

Dittuno 1994

Strict guidelines are provided for sensory and motor examination to ensure correct classification and a training pack with video and standardised paper work for recording classification are provided, in order to maximise the reliability of this measure. Surface marking points for dermatomes ('the area of

skin innervated by sensory axons within each segmental nerve root') and key muscle groups for testing of myotomes ('the collection of muscle fibres innervated by the motor axons within each segmental nerve root') are shown in Appendix 1.1. Two elements of sensory testing are applied; sensitivity to light touch and pin prick. Clear guidelines for each sensory testing procedure are given. The classifications for sensory impairment are as shown in Table 1.3.

Table 1.3: ASIA Sensory Classification

Grade	Classification
0	Absent
1	Impaired
2	Normal
NT	Not testable

Dittuno 1994

The limited number of grades within the sensory grading scale is aimed at maximising reliability but has a reduced sensitivity as a result.

Table 1.4: ASIA Motor Classification

Grade	Classification
0	Total paralysis
1	Palpable or visible contraction
2	Active movement, full range of movement with gravity eliminated
3	Activity movement, full range of movement against gravity
4	Activity movement, full range of movement against moderate resistance
5	Activity movement, full range of movement against full resistance
NT	Not testable

Dittuno 1994

The ASIA motor assessment (Table 1.4) looks at key muscles for each neurological level. Assessment of these muscles alone may prevent detection of neurological deficit in other muscles supplied by the relevant nerve root. A full neurological examination of all muscles should be performed on a patient immediately on admission to hospital and at regular intervals thereafter. This precaution will rectify any insensitivity of the ASIA scoring.

The ASIA assessment procedure was designed to be performed in supine lying as this is a position that most spinal injury patients can be placed in, but it is important to include the category of 'Not testable' for both sensory and motor testing as there may be occasions when supine lying is not possible or the dermatome or muscle to be tested is unobtainable due to other injuries or covering over the dermatome testing area by dressings, splints or other

equipment.

The existence and proven reliability of classification systems for international use (Dittuno 1994) has enabled the accumulation of data to provide clinicians with quantifiable evidence on which to base their treatment plans and to predict long term outcomes in spinal cord injury (Stover et al 1995). Silberstein (1992) sought to combine the clinical assessment (Frankel score), the stability of the injury, degree of intrusion into the spinal canal and degree of cord contusion as an index to determine selection for decompression surgery. The small subject numbers and wide variance in the nature of impairment (bony and neurological) render the findings of this study interesting but inconclusive. Waters et al (1994) demonstrated a strong correlation (p < 0.001) between the initial ASIA motor score post injury and eventual walking ability. Again the patient group was disparate in relation to neurological level of injury but the time post injury for assessment was well controlled. The patient numbers in both these studies (Silberstein 1992 & Waters 1994) were too small to be conclusive but the work merits replication as it is indicative of the predictive potential and clinical importance of the ASIA classification as a measurement tool.

1.1.4. Incidence

Many demographic studies have been published from specialist spinal injury units world-wide, describing patients and treatment outcomes (Stover et al 1995, Chen et al 1997, Exner & Quine 1997, Suyama et al 1997). These studies report the characteristics of patients admitted to the specialist centres but the method and accuracy of reporting the incidence was not discussed in depth. The

largest data set was reviewed by Stover et al (1995), examining over fourteen thousand new cord injuries admitted to model spinal cord injury centres in the United States. Accurate figures for the incidence of spinal cord injury (SCI) in the United Kingdom are difficult to obtain as SCI is not a notifiable condition (as opposed to the countries where the above studies were undertaken) and there is presently no national database. The probable incidence of spinal injury resulting in significant cord damage has remained constant over the last five years and is estimated as 10 - 15 per million of the population per year (personal communication – Middleton 1999). Higher figures (up to 40 per million) are reported in the United States (Stover et al 1995) and an increased prevalence has been reported in Australia (Blumer 1996). The most frequent cause of spinal cord injury internationally is road traffic accidents. Other common causes include falls (accidental and intentional), violence, sporting accidents (diving, riding, contact sports) and injuries in the work place (Stover et al 1995). It is interesting to note that the cause of injury varies with geographic location. It is also important to note that these figures are calculated on the number of patients treated in specialist centres and not the total incidence in the countries stated.

Correlations between incidence, level and severity of injury have been identified with age and sex. In a retrospective study of all spinal cord injured admitted to a regional spinal unit over a nine year period, Spivak et al (1994) observed a greater incidence of cervical injury in patients over 65. The most frequent cause of injury in this age group was falling but the cause of injury in the other age groups was not reported. In the under 40 age group, a greater

proportion of cervical injuries were male (4:1) which was not reflected in the older group (1:1). The younger group were also more likely to sustain complete cervical lesions. Kiwerski (1993) reported 43% of cervical injuries admitted to a specialist unit over a 6 year period had complete (Frankel Grade A) injuries. The least common level of injury in this group was C1 -C3 and the most frequent was C5 - T1 (58%). This is logical as survival at the scene of the accident is less likely for the higher level injuries, as ventilatory function may be compromised due to involvement of the phrenic nerve which is supplied by the 3rd, 4th and 5th cervical nerve root. The age and cause of injury were not reported in this study.

A review of patients admitted to the Spinal Injuries Unit at the Royal National Orthopaedic Hospital Trust (Barr 1998) has shown a significant change in neurological deficit. In 1984, 85% of patients admitted to the Unit presented with complete injuries (Frankel Grade A) as compared with 37% in 1998. This is probably a reflection of advances in in-car safety, improved emergency care at the scene of the accident (immobilisation during lifting & handling, rapid transport from accident to trauma centre, appropriate medication) and early referral and admission to specialist spinal units. Sadly, little objective data are available to support these hypotheses; the Department of Transport statistics relating to road traffic accidents (HMSO Publications) demonstrate a reduction in deaths and serious injury but do not categorise the serious injuries to any extent. National statistics are also not available from all the spinal units in the United Kingdom to compare the speed of referral to a specialist unit, the nature of treatment interventions and the eventual clinical outcome. The benefits of

collecting such statistics have been demonstrated internationally where resources and facilities have been adapted to meet the changing needs of the cord injured population (Blumer 1996). However, data collected from only the specialist units would still fail to include those patients receiving acute and rehabilitation management in centres not specialising in spinal injuries.

1.1.5. Treatment & Prognosis

Management of spinal cord injury should begin with prevention. Education and awareness of mechanisms of injury, legislation relating to road and in-car safety, health and safety at work, development of protective clothing for work and leisure activities and changes in rules for many contact sports have raised public awareness and subsequently reduced the incidence of this devastating injury. In a review of spinal cord injury sustained through playing rugby in South Africa, New Zealand and the United Kingdom, Silver et al (1994) analysed the mechanism of injury, resultant neurological deficit (using the ASIA impairment scale) and standard at which the player was operating (amateur, professional, local or international). From their findings they were able to provide advice and guidelines to the relevant sporting bodies to reduce the incidence of cord injury. To date this study has not been repeated to compare data.

When primary prevention fails and a spinal cord injury occurs, the goal must be that of secondary prevention (Bedbrook 1980). Objectives for management should be to minimise damage at the time of injury, through prevention of further spinal misalignment and maintenance of vascular supply and

oxygenation of the cord (American College of Surgeons). Improved methods of stabilisation and lifting and handling at the scene of the injury have no doubt had an effect on the neurological outcome although this remains an anecdotal opinion and has not as yet been substantiated in the literature. The effects of chemotherapy on the pathological processes occurring within the cord have attracted much interest. The administration of steroids within eight hours of injury has been shown, through a large randomised controlled clinical trial involving human subjects in the United States (Bracken et al 1990), to improve eventual outcome of neurological deficit. Despite criticism of the reliability of the assessment tools used in this study and minimal improvements made, immediate administration of steroids is now recognised as standard practice in some countries.

In order to be able to establish the biological and clinical benefits of these treatments, it is essential that the appropriate measurement techniques are developed. Recent advantages in magnetic resonance imaging (Silberstein 1992), and electrophysiological monitoring (Tosi 1993) have served as useful adjuncts to patient assessment and treatment planning by identifying occult lesions below the diagnosed level of injury. Silberstein (1992) examined MRI images of a small group of acute spinal cord injured and concluded that that compression of the cord in excess of 40% was an indication for surgical decompression. The numbers in the study, however, were extremely small and the extent of the neurological impairment varied such that the recommendations should not be considered conclusive. Tosi (1993) combined conventional neurological examination with neurophysiological examination of patients with

long standing cord injury resulting in an apparent upper motor neurone paralysis. Patients with obvious lower motor neurone compromise were excluded from the study. The neurophysiological assessment comprised examination of electromyographic activity in three lower limb muscles, lumbosacral somatosensory evoked potentials, bulbocavernosus reflex, H reflex & F wave from stimulation of the tibial and peroneal nerve. The methodology and criteria for identification of pathological status were clearly outlined in the study, in which Tosi classified 14% of the thirty five subjects to have atypical clinical signs. He further tried to hypothesize the progress of the pathological process within these five subjects. His theories were based on one assessment session in a very small group of subjects which does not seem viable to support any of his conclusions.

The decisions regarding a treatment plan should be based on obtaining neurological improvement, stabilising the injured spine, minimising the period of immobilisation whilst not increasing the risk of fatality or neurological deterioration (Kiwerski 1993). The debate surrounding the choice of surgical or conservative management remains unresolved. Kiwerski (1993) proposed that the advantages of surgical management are early mobilisation, prevention of further neurological deficit due to bony instability and correction or prevention of bony deformity but advocated that the purpose of surgical management is not to ensure neurological recovery.

Kiwerski compared the outcome of surgical and conservative management in a group of 1761 cervical spinal cord injuries admitted to a specialist spinal injury unit within two weeks of injury. Allocation to the surgical or conservative management regime was not randomised but based on type of injury, degree of severity, general condition of the patient and accompanying injuries. The study demonstrated a better outcome for the surgical group, comparing neurological improvement and mortality. Sixty percent of the surgical group showed neurological improvement as compared to forty percent in the conservative group. The mortality rate was also significantly different between the two treatment approaches; twenty percent of the patients managed conservatively died whereas only eight percent died in the surgical group. These findings would appear to provide useful clinical indicators for choice of management and to support a preference for surgery. However, other confounding variables have not been considered. The subjects were categorised into degrees of completeness (Frankel Grade A - D) and outcomes presented within these categories. No indication was given as to the levels of injury within the surgical and non-surgical group which could have a significant effect on the mortality rate. The subject groups compared can therefore not be considered as homogeneous. It has been reported that up to 84% of the high cervical injuries (C1 – 4) will experience respiratory complications and therefore have an increased risk of mortality due to compromised ventilatory function (Jackson 1994). Furthermore, no detail was given of associated or accompanying injuries. These data are essential in order to draw sound conclusions from the study.

Kiwerski (1993) also put forward a non-clinical reason for selecting the surgical option of treatment which relates to the shorter duration of the in-patient

episode. Specialist units are constantly under pressure for bed availability, therefore shortened lengths of stay enable a greater number of patients to benefit from treatment in a specialist centre.

When Sir Ludwig Guttmann first pioneered the treatment of spinal cord injury, following the Second World War, the mortality rate was between eighty and ninety percent (Guttmann 1973). A majority of tetraplegic patients died and paraplegics rarely survived, succumbing to respiratory and urinary tract infection or septicaemia as a result of appalling pressure sores. Guttmann's aim was to reduce mortality and rehabilitate those injured to fulfil a useful life role. In order to fulfill this goal, he advocated a specific regime (Guttmann 1978): The supervision of a dedicated unit by a physician or surgeon; a sufficient number of skilled nursing and paramedical staff to cover all aspects of the patient's physical and emotional care; adequate laboratory, training and workshop facilities to enable physical and vocational rehabilitation through education, exercise and re-training in activities of daily living and personal care; and finally a life long commitment to regular follow-up post discharge.

Guttmann's principles were followed internationally (Bedbrook 1985, Stover et al 1995). Such was the success of rehabilitation in this patient group that the survival rate increased substantially. The average life expectancy for paraplegic patients is 84% of normal and a tetraplegic may expect up to a 30% reduction in normal life expectancy (Stover et al 1995, Yeo et al 1998). These figures, however, are based on the survival of people injured in the 1960's at which time management of spinal cord injury had not advanced to its present status. Acute

management was predominantly conservative, fewer specialist units were in existence and structured life long follow-up was not readily available for this patient group. The number of patients lost to follow-up is also not considered in these figures. Frankel et al (1998) performed a large follow-up study of all patients admitted for the acute management in one specialist centre and achieved follow-up data on 92% of the patients. Their findings concurred with those of Stover et al (1995). In an attempt to account for the change in management of spinal injury, Frankel et al divided their study group into two bands and found that in both groups the survival rate of tetraplegic patients was still higher but that in general the survival rate had improved in the latter group to the levels quoted by Stover et al (1995). It is therefore reasonable to hope that the next set of figures to be calculated will show a further increase in life expectancy.

Emphasis in spinal cord injury rehabilitation today has subsequently progressed from the restoration of physical function and the prevention of secondary complications to focus on the reintegration of the individuals back into their previous social environment and life role (relationships, employment, finances, leisure activities) and the optimisation of quality of life (Bedbrook 1985, Krause 1998, Westgren 1998). The increasing population of long term survivors of spinal cord injury will provide a wealth of information to steer the rehabilitation and reintegration programmes for themselves and future patients. Several studies have already been undertaken to examine the long-term outcomes and problems experienced by this patient group. Krause (1998) used the Life Satisfaction Questionnaire (LSQ) to determine the dimensions of well-

being and devise a new scale. His sample was large (1032 subjects) but was drawn from two rehabilitation centres in separate geographic locations, in an attempt to establish a broad range of responses from different cultural settings. The selection of subjects within the centres was not however randomised as the researchers sought to ensure representation of injuries at all levels. Krause (1998) concludes that the identification of further sub-scales for the LSQ enabled improved 'conceptualisation of subjective well-being' and the reliability of the instrument but correctly called for revalidation of the measure in other patient groups.

Westgren (1998) surveyed the total population (353 patients) under the care of a specialist spinal unit with the Swedish SF-36 Health survey (Sullivan 1995), a self-administered questionnaire, to assess quality of life. The SF-36 examines eight dimensions ranging from physical function and health status to social and emotional role. The method of administration of the questionnaire (postal or face to face) was not stated but the response rate was extremely high (92%), suggesting face to face delivery. The classification of clinical problems were clearly defined and criteria were well expressed for each. The only criticism of the methodology of the study is the use of parametric statistical analysis (2 tailed Student 'T' test) to compare the level of quality of life (ordinal level data) with the medical characteristics or symptom.

When compared with a normal population, the respondent cord injured group (320) scored significantly lower in all sub-scales, the greatest difference being in physical role and function. Surprisingly, level and severity of injury did not

correlate with quality of life, except within the physical domain. Age at time of injury was a significant factor, the younger age group (20 years of age or less) scored higher in the domains of physical and physical role function, bodily pain and social function. In subjects experiencing medical problems, the numbers of which are not stated in the paper, neurogenic pain, bladder problems and spasticity were associated with a lower quality of life. Conversely, the time since injury had a positive correlation with quality of life, despite the development of complications secondary to the injury. This is no doubt a reflection of the adaptive process surrounding adjustment to life with a spinal cord injury, as described by Buckelew (1991).

One further element which cannot be excluded from this review of present and future management of spinal cord injury is that of restorative medicine. Since the pioneering work of Aguayo et al (1981), who sought to determine the appropriate environment to permit regeneration of central nervous system axons, many groups have endeavored to determine the mechanisms involved in the prevention of regeneration of the spinal cord (Fawcett 1998). Techniques to reduce the inhibitory environment by blocking inhibitory molecules (Schwab 1993) and removing inhibitory cells (Kierstead et al 1995) have been developed in cord injured mice where restoration of some functional muscle activity was achieved although detail of the nature of the functional activity is not given. Implantation of Schwann cells (Xu et al 1995), nerve grafts (Berry et al 1996) and implantation of ensheathing cells (Li et al 1997) to increase the regeneration potential of nerve axons has also shown promising results in animal models. Comments in a review by Hughes (1984) that the age of the

animal at the time injury is also significant in the regenerative process and the fact that all these studies were performed in a highly controlled laboratory environment and with acute cord injuries, should be taken into account when considering extrapolation of these findings. To date, no trials have been undertaken in humans but it is predicted that the first human treatments will be attempted during the next decade (Fawcett 1998).

It is acknowledged that regeneration of the cord will be achieved to a lesser or greater degree in humans but predictions as to the functional outcomes are more speculative. Appropriate and novel connections between tracts in the cord may occur, testing the neuroplastic capacity of the central nervous system to the maximum. Restoration of the conductive pathways may also serve as a precursor to less welcome activity, such as pain and spasticity, often symptoms of disorganisation within the central nervous system. This hypothesis, together with the increased longevity of patients, indicate that secondary problems following spinal cord injury will remain a major issue in management of this patient group.

In an excellent summary of the basic science of spinal cord repair, Fawcett (1998) comments that in order for restorative therapies to be successful, basic scientists, clinical scientists and clinicians must all work together - a sentiment that is echoed by this author, not just in relation to the regeneration of spinal cord function but in all areas of spinal cord injury management to ensure that all individuals are given the opportunity to achieve their maximum potential.

1.2 Spasticity

Spinal cord injury gives rise to lower motor neurone dysfunction at the level of the cord lesion and an upper motor neurone syndrome below that level. There are both negative and positive features of the upper motor neurone syndrome. The most prominent negative features are muscle weakness, paralysis and impairment of selective control of movement. Positive features include exaggerated deep tendon reflexes; spread of reflexes from one muscle to another or from one limb to another; involuntary muscle spasms in response to cutaneous or visceral stimuli or without any previous stimulus; clonus (sustained oscillating joint motion) and rigidity (Mayer 1997). Amongst these positive features of the upper motor neurone syndrome is spasticity. Defined as:-

"A motor disorder characterised by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting in hyperexcitability of the stretch reflex as one component of the upper motor neurone syndrome"

Lance 1980

extensive reviews of large patient numbers by Edgar (1992) and Stover (1995) support the view that spasticity should not always be viewed as a complication of the upper motor neurone syndrome, as it may sometimes offer functional and psychological benefits to the patient.

In order to discuss the theories surrounding the mechanisms responsible for spasticity, it is necessary to review the structure and function of normal muscle,

1.2.1. Muscle

The human body contains two types of muscle - striated and smooth. The smooth muscle remains under the control of the autonomic nervous system and as such is not under the direct control of the cerebral cortex. Cortical control of striated or skeletal muscle however develops in the growing infant. The skeletal muscles in the human body are numerous and varied in their shape, size and function. The muscles are joined to bones, cartilage, ligaments and skin, either directly or via tendons or aponeuroses and their primary function is to produce movement or stabilisation of joints (Jones & Round 1992).

Each muscle contains thousands of muscle fibres, which themselves are made up of bundles of thick filaments forming myofibrils. The myofibrils are primarily (80%) made up of two contractile proteins, actin and myosin which are arranged in a parallel structure giving the muscle it's striated appearance under the electron microscope. Each myofibril is enveloped in sarcoplasmic reticulum, a membranous bag which forms a branching network running across the whole muscle fibre and contacting every myofibril (Lieber 1986).

Every muscle in the body contains different muscle fibre types which can be categorised into three main groups. Type 1 fibres are slow to generate force but can sustain the force over a longer period than the type 2 fibres, which can develop force rapidly but fatigue quickly. The third type, 2A are also capable of generating force rapidly but are fatigue resistant, due their oxidative

metabolism (Jones & Round 1992). Normal muscle will contain a mixture of all these fibre types, with a ratio of type 1 to type 2 fibres dependent on the function of the specific muscle, e.g. the soleus muscle in the calf is a postural antigravity muscle which requires sustained muscle activity and therefore contains a predominance of slow twitch type 1 fibres (Jones & Round 1992).

In the event of trauma or disease, muscle atrophy will occur and the fibre type characteristics of the muscle are seen to alter (Walton 1981). These patterns are not standardised and may even vary within syndromes. Biopsy data from patients with upper motor neurone lesions as a result of cerebrovascular incident and spinal cord injury show specific atrophy of type 2 and type one respectively. Round et al (1996) first observed a greater predominance of type 2 fibres in the quadriceps muscles of complete paraplegics. The findings in this study were significant as the biopsy samples were of high quality but were only representative of the area of muscle sampled. The sample numbers were small and the nature of the procedure was not pleasant for the patients such that replication of the study is merited but with a larger patient population rather than repeated in the same patient group. The absence of type I fibres was consistent with the rapid onset of muscle fatigue observed in this patient group, following electrical neuromuscular stimulation (Barr et al 1989).

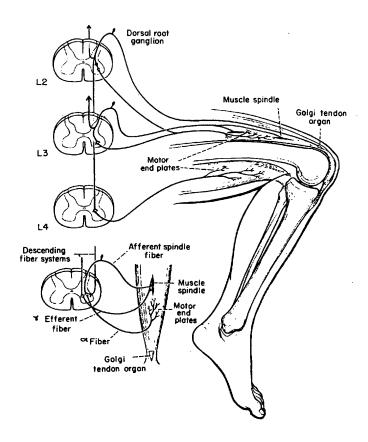
The motor neurones innervating skeletal muscle originate in the anterior horn of the grey matter in the spinal cord, pass down through the peripheral nerves and separate into axonal branches which terminate at the neuronuscular junction. In healthy muscle, one motor neurone will supply a number of muscle

fibres scattered throughout a muscle. All the muscle fibres and the motor neurone which supplies them form a motor unit. Within a single muscle there is a range in size of motor unit. The fast conducting motor neurones support large motor units and the slower motor neurones support smaller motor units. The motor units follow an ordered recruitment, smaller units firing first during low tension muscle contractions whilst the larger, faster units are recruited when stronger forces are required (Henneman et al 1965).

To produce a muscle contraction, voluntary or reflex, the alpha motor neurone is stimulated in the anterior horn. In the case of a voluntary action, the stimulus will arise in the cerebral cortex, travel down through the cord in the corticospinal tracts to synapse with the anterior horn cell at the relevant spinal level. Reflex muscle activity occurs as a result of afferent stimulation from any receptor organ. The action potential enters the cord through the posterior root at any level and travels to the appropriate spinal level to synapse with the target anterior horn cell. These pathways are shown in Figure 1.3.

The action potentials travel down the lower motor neurone to the axon termination, causing an influx of calcium into the presynaptic membrane. This causes the vesicles containing acetyl choline to release their contents into the synaptic cleft which produces a depolarisation of the muscle fibre membrane. Provided the extent of the depolarisation is sufficient, the action potential will spread along the length of the muscle fibre, causing the actin and myosin filaments to slide over one another and the muscle fibre length to shorten (Round & Jones 1992).

Figure 1.3: Efferent & Efferent Pathways



From. MB Carpenter. Core text of Neuroanatomy. 3rd edition. Pub. Williams & Wilkins

There are several different types of muscle contraction. An isotonic muscle contraction indicates that the muscle changes in length but exerts the same force although the speed of the movement may vary through the range of movement. When a muscle contracts isotonically, it may work concentrically whereby the origin and insertion of the muscle draw closer together as the muscle shortens or the muscle attachments are drawn further apart as it works to oppose the action of a force which is greater than that of its own contraction. This is known as eccentric muscle work.

Under normal conditions a muscle never works in isolation to produce movement or provide stability of a joint. The agonist muscle will contract to produce a force to bring about movement in a direction. Subsequently, the activity in the opposing muscle group or antagonists will be inhibited such that they relax and lengthen permitting movement to occur. When both agonist and antagonist muscle contract with the same amount of force, an isometric contraction results where the muscles remains the same length, usually to provide a stabilising force around a joint to permit activity in other muscle groups.

Scattered throughout the muscle, lying parallel to the muscle fibres, are sensory receptors known as muscle spindles which are responsible for detecting change in length of the muscle fibres (extrafusal fibres). The spindles consist of highly specialised muscle fibres (intrafusal fibres) which are contained within a collagenous capsule. There are two types of intrafusal fibre; the smaller chain fibres which form a central chain in the spindle and attach to the larger 'bag' fibres which extend beyond the capsule and attach to the extrafusal muscle fibres. Afferent (sensory) innervation of the muscle spindle is through Ia primary afferent fibres which terminate around the central portion of the chain and bag fibres and smaller secondary II afferents which synapse with the outer regions of the bag fibres. The efferent (motor) innervation of both types of intrafusal fibre are the gamma (fusimotor) motorneurones. Stimulation of the gamma system will cause the intrafusal fibres to contract, causing a stretch on the central portion of the intrafusal fibres where the primary afferent nerve endings are situated. Stimulation of these afferents will then fire the alpha motor neurone causing the extrafusal fibres to contract and shorten. This in turn removes the stretch on the intrafusal fibres and reduces the afferent signal.

Tension of the extrafusal fibres will also be detected by the golgi tendon organs which are encapsulated endings of the large myelinated axons (Ib afferent fibres) located at the musculo-tendinous junction. The information from the golgi tendon organs acts via an inhibitory neurone to reduce the excitability of the alpha motor neurone supplying the extrafusal fibres and the gamma motor neurone supplying the muscle spindles. The excitatory influence of the Ia and II afferents on the alpha motor neurone is further modulated by the Renshaw cells, small interconnecting neurones that receive collateral input, by descending inhibitory pathways from the cerebral cortex and cerebellum and by other afferent stimuli from receptor and nocioceptor organs throughout the body. This complex mechanism maintains a fine balance between the excitatory and inhibitory mechanisms from cortical to spinal reflex level which control normal muscle tone.

Walsh (1992) provides an excellent historical review of the phenomenon of muscle tone which has long been a source of fascination for scientists. Galen of Pergammon first demonstrated the balance of muscles by severing flexor muscles and observing the unopposed force of the antagonist muscles pulling the joint into extension. The 19th century belief that muscle tone was a result of continuous muscle activity was supported by observations of elongation of frog muscle following damage to motor nerves. Liddell & Sherrington (1924) revised this theory when they first demonstrated the stretch reflex in muscle and implied that muscle tone is a result of reflex activity only; a constant motor

output in response to a steady barrage of afferent input. Their theory was further supported by observations of the imbalance of muscle tone following damage to the intact nervous system, which are now described as the symptoms of upper motor neurone syndrome.

Basmajian (1957) then demonstrated through needle EMG studies that normal muscle exhibits no electrical activity (an indication of membrane depolarisation induced by nervous excitation) when at rest. This refutes the theory that tone of completely relaxed muscle can be attributed to activation of the alpha and gamma motor contractile mechanism. Liddell & Sherrington also showed in an animal model that relaxed muscle exhibited considerable resistance to stretch after severing the nerve supply in order to disrupt the reflex mechanism. In his review, Walsh (1992) proposed that such muscle tension could only be attributed to the mechanical properties of muscle itself - inertia, spring stiffness, viscosity or thixotropy. Lehmann (1989) measured the total stiffness (elastic, spring and viscous) in the ankle joint in a group of patients with spastic paresis and normals, by moving the joint in a sinusoidal pattern through a free range of movement stiffness. He clearly demonstrated an increase in stiffness in the spastic group, using a well controlled experimental design.

In another thorough and well controlled experimental study, Lakie, Walsh & Wright (1984) introduced the concept of three mechanical properties of relaxed muscle - resting tension, non-linear resistance to imposed stretch & dependence of this resistance on history of movement. The resting tension of a muscle can be demonstrated by observing the angular position adopted by a joint in a limb

at rest. This will relate to the relative amounts of tension in the flexor & extensor muscles working over the joint. Lakie (1984) also observed that when an external force was applied to a joint, movement occurred. When the force was removed, the joint referred back to a consistent resting position. No EMG activity was observed during the experiment therefore it was assumed that the agonist & antagonist muscles possess a low level of resting tension not attributable to nervous excitation. When the torque applied to the joint was increased in a linear fashion, the resulting displacements were not directly proportional to magnitude of torque i.e. when the torque was low, displacement was disproportionately small.

In his review of muscle function, Rothwell (1987) described two components of the tension response of muscle; the phasic response which is dependent on the velocity of the stretch applied to the muscle and the tonic response which is related to final static degree of stretch. Each of these responses will be modified according to the state of excitation of the afferent and efferent system of intra and extrafusal fibres.

Three characteristic responses can be observed following electrical stimulation, as summarised by Lehman & Ritchie (1970) - the M (motor) wave, the H (Hoffman) reflex and the F (following) response. The M wave is the direct muscle response to the stimulation of the motor nerve. The H reflex is a monosynaptic spinal reflex response to stimulation of the afferent fibres of a mixed nerve, which produces a late compound action potential (H wave) which occurs after the M wave. The stimulus threshold is lower than that required for

the M response and the maximal H wave is also smaller in amplitude than the maximal M response. The F response is evoked by a supramaximal stimulus to the nerve, producing a small amplitude wave which occurs after the M wave. The F wave is not a reflex but represents the direct motor response to afferent stimulation of the cord. These responses have been used to measure the function of the afferent and efferent conductive pathways (Lenman &Ritchie 1970).

Insult to the central nervous system, through trauma or disease will upset the delicate balance between the inhibitory and excitatory neuronal control of muscle tone, resulting in the onset of spasticity and involuntary muscle spasms. There has been much debate surrounding the pathophysiology of spasticity, and whether this symptom of the upper motor neurone syndrome, resulting hyperexcitability of the anterior horn cell, is a result of fusimotor hyperactivity or deficient inhibition of the anterior horn cell. Fusimotor hyperactivity would lead to an increase in sensitivity of muscle spindles to stretch and a subsequent increase in the Ia response to stretch which would produce the exaggerated stretch reflex. Assessment of fusimotor function to determine its role in hypertonicity presents several methodological problems. Vallbo et al (1979) examined the activity in the muscle spindles in anaesthetised patients with spasticity and normals using microneedle electrodes and found no evidence of increased Ia discharge or increased sensitivity in the muscle spindles. Fusimotor hyperactivity is thought to be the target for injection of alcohol or phenol into the nerve or nerve root. Local anaesthetic blocks are however not this selective and effect both the small and large afferent fibres in the locality

of the injection but have little effect on the smaller fibres further away from the site. To date, therefore, there has been no evidence to support the theory that spasticity results from increased fusimotor drive on the muscle spindles.

The more favoured theory is that of reduced inhibition— presynaptic, post synaptic and recurrent - involving the Ia afferents from the muscle spindles, Ib afferents from the golgi tendon organs, and the Renshaw cell. Vibration of the tendo-achilles in normals is shown to produce strong Ia afferent discharge (Burke et al 1976), producing a decrease in the H reflex, indicating presynaptic inhibition of the alpha motor neurone activity (Delwaide 1985). When vibration is applied to spastic muscle, a smaller reduction is noted in the H reflex, indicating that Ia input is not entirely responsible for inhibition. The reduction in H reflex response also shows no correlation with the increase in exaggeration of the stretch reflex in these patients (Delwaide 1985). These findings have been demonstrated in well controlled experimental models in both human and animal subjects.

Input from the II afferents of the muscle spindles reach the alpha motor neurone via interneurones and produce excitatory and inhibitory effects in corresponding agonist flexors and antagonist extensor muscle groups. Selective exploration of II fibre afferents is not currently possible but indirect evidence is available from the effects of tonic stretching of a spastic agonist muscle (quadriceps) which demonstrates a decrease in stretch reflex of the elongated muscle with a corresponding increase in stretch reflex in the opposing antagonist muscle group (Burke & Lance 1973).

The Ib fibres from the golgi tendon organs are both facilitated and inhibited by the descending tracts in the cord. Removal of the supraspinal influence as in the decerebrate cat will depress Ib activity and result in rigidity (Pierrot-Deseilligny 1985). Renshaw cells are also facilitated and inhibited by supraspinal control. Removal of this influence results in inhibition of the Renshaw cells which will subsequently prevent recurrent inhibition of the alpha motor neurone, as demonstrated by an exaggerated stretch reflex response.

In normal subjects, voluntary contraction of agonist muscle results in reciprocal inhibition of the antagonist muscle, via the Ia interneurones, as shown by measurement of the H reflex. In the spastic patient, a stretch reflex also occurs in the antagonist muscle during voluntary contraction. Pierrot-Deseilligny (1985) hypothesised that this may result from a malfunction of the reciprocal Ia inhibition, which would normally inhibit the antagonist stretch reflex. The evidence in the literature therefore supports the notion that spasticity is a culmination of several factors; the absence or decrease of suprasegmental inhibition, the absence of pre-synaptic inhibition of the phasic gamma motor system and the enhanced affect of sensory input upon a disinhibited spinal cord segment as identified by Edgar (1992) in his review.

Many studies have considered spasticity as a single entity, performing analysis on subjects with upper motor neurone syndrome but making no distinction regarding the site of the lesion. Herman et al (1988) explored the effects of repeated sinusoidal stretching on calf muscles, in SCI and hemiplegic patients.

The SCI group showed a slow rise of reflex activity as compared to the hemiplegic group. He summarised that the cord lesion removed inhibitory influences on the polysynaptic segmental pathways such that the primary afferent discharges from the muscle spindle were transmitted through the multisynaptic chains in the interneuronal pool. The rapid build up in the hemiplegic group indicated that the transmission of primary ending spindle discharges occurred through a monosynaptic pathway. The experimental methodology in the study was well controlled but the subject numbers were small and comprehensive clinical details were not given for either patient group, indicating the need for replication of the study with carefully selected patients in each group.

Chapman & Weisendanger (1982) proposed from their comprehensive review that cerebral and spinal spasticity gives rise to different patterns of muscle activity: cerebral spasticity causing an increase in tone in the antigravity muscles (flexors of the arm and leg extensors) and spinal spasticity presenting as a marked increase in tone of flexor muscles of the leg. They associated this with the dynamic and static gamma systems controlling fusimotor activity, the sensitivity of the dynamic system depending largely on the spinal mechanisms. Meinck et al (1985) refuted these findings reporting that the change in flexor reflexes is the same in cerebral, brainstem and spinal lesions. Gildenberg et al (1985) also observed similarities in a small but well controlled comparative study of patients with spinal cord injury and brain injury; a large EMG response was elicited by the passive movement and the tonic vibratory response was present in both groups. He did however note a greater exaggeration in the

tendon jerks of the SCI patients and marked difference in the patterns of resistance to passive movement of the lower limbs using an isokinetic dynamometer. Although the patient numbers were extremely small, the differences and similarities were so clearly marked that Gildenberg concluded 'similar suprasegmental mechanisms may be responsible for hypertonia' in both groups of patients.

1.2.2. Spasticity in Spinal Cord Injury

As discussed in Chapter 1.1, spasticity is not present immediately after injury, due to spinal shock but symptoms will present to some extent following all spinal cord injury resulting in upper motor neurone paralysis within days or weeks of injury.

Barolat & Maiman (1987) and Cioni et al (1995) both reported from large case reviews that over 90% of SCI patients experience muscle spasms, of which 25% complain of severe spasms that fail to respond to treatment. Sköld et al (1998) reported a 68% incidence of spasticity in a population of 353 SCI patients, of which 41% reported spasticity to be a problem. Stover et al (1995) reported a gender related incidence of problematic spasticity, 43.5% of SCI males and only 38.9% of females in a group of 15,000 patients reviewed at one year post injury. These data were collected on the National Spinal Injuries database in the United States by the Model Spinal Injury Units using standardised assessment protocols, supporting the reliability of the data set. Stover (1995) also recorded that there was a higher incidence of problematic

spasticity in tetraplegic patients (53.5%) than paraplegics (38.9%). The paraplegic group however also contained those patients with lumbar lesions, which result in lower motor neurone paralysis and will therefore not present with spasticity at all.

Patients with spasticity in a state of hyperexcitability will experience involuntary muscle spasms which occur in response to some afferent input signal, usually a noxious stimulus e.g. sudden movement, infection, a distended bladder, extremes of temperature, stress or noise (Mayer 1997). This may present problems for the SCI patient when performing activities of daily living e.g. an increase in resistance to movement will hinder manipulation of the limbs when transferring to and from a wheelchair or attaining a position to perform self catheterisation or maintain personal hygiene. Powerful muscle spasms may also cause general fatigue and skin pressure areas can occur through friction or sustained pressure on the skin and underlying soft tissues. Increased muscle tone may also prevent full ranging of joints such that joint contractures occur. These, in turn, will all create noxious stimuli to trigger a further increase in spasticity and muscle spasms, creating a vicious cycle. It is therefore not surprising that the presence of spasticity has repeatedly been identified as a moderate to strong negative influence on well being and quality of life in the spinal cord injured population (Krause 1998, Westgren & Levi 1998). Krause (1998) reviewed self reported problems in a large spinal cord injured patient group (over one thousand), taken from two model spinal injury centres in the United States, to establish reliable scales to examine the underlying dimensions of self well-being. The response rate in the study was high (89 & 84%) but the subject selection was not identical in the two centres as Krause chose to review consecutive admissions to one unit but stratify his sample from the second unit in order to represent the ethnic and female population. Westgren (1998) chose a different approach in a survey of a large but smaller patient group (over three hundred) to examine quality of life using the SF-36 Health survey. He compared the medical data already available about this patient group through the national database but failed to state the time span between the accumulation of the medical data and the patient survey, which is of vital importance to validate the data for comparison.

The intensity of spasticity is known to vary in relation to the spinal cord impairment and is greater in patients with incomplete cord lesions. The extensive case review by Stover (1995) reported that 67.9% of patients with Frankel grade B lesions and 64.4% of patients with Frankel grade C had undergone treatment for spasticity during their first year post injury, as compared with 59.5% of patients with Frankel grade A (complete) lesions. Stover does not discuss the indications for treatment but the presence of spasticity and involuntary muscle contractions in this sub-group will affect control of preserved voluntary muscle activity, presenting as reduced muscle power and impaired co-ordination of movement. In these cases, it is therefore essential that the appropriate treatment is determined in order to reduce abnormal tone and optimise preserved voluntary muscle activity as much as possible.

1.2.3. Treatment of Spasticity

The presence of spasticity following insult to the central nervous system should not always be viewed as a complication but if present in excess, spasticity does become a difficult and complex problem to manage. The goal of any treatment plan following insult to the central nervous system should therefore be to prevent or minimise the development of abnormal muscle activity. Edgar (1992) reports several strategies for this in his review which include ranging and stretching of joints and muscles (Knutsson & Odeen 1981), cryotherapy (use of cooling – Price et al 1993), appropriate positioning of the patient in bed, sitting and standing (Bobath 1978), weight bearing and optimising any preserved muscle function in patterns of normal movement (Carr & Shepherd 1998), appropriate nursing care to ensure tissue viability, correct bladder management and prevention of any other complications (infection, joint dislocation) which may serve as noxious stimuli to the already hypersensitive afferent system. These treatments are based only on anecdotal evidence and small case series observations which often combine patients with varied pathology, in the absence of substantive comparative studies.

Once present, reduction of abnormal muscle tone in itself is not a treatment goal, unless there is functional benefit to be gained, for example, improvement in ability to perform activities of daily living or a reduction in pain. Management strategies may include a combination of several treatment modalities and outcome and progression of treatment should be reviewed on a regular basis. Price et al (1993) examined the application of ice to the lower legs of a small group of patients with spasticity following stroke, traumatic

brain injury and spinal cord injury, selected on the basis on ankle range of movement. The study demonstrated a reduction in ankle stiffness twenty minutes after application but this returned to baseline after sixty minutes from which Price concluded that the application of ice was useful as an adjunct or precursor to other therapies but this hypothesis was not tested during the study. Although the methodology of the study was otherwise well controlled, the author failed to provide substantial clinical details and the experiment was only performed on one occasion.

Treatment regimens should be based on the chronicity, severity and distribution of spasticity and devised on an individual basis with the patient, their carers where appropriate, and the multi-disciplinary rehabilitation team. Secondary causes of spasticity, such as infection, pain and extreme temperature changes should also be investigated, as treatment or removal of any noxious stimuli may result in a reduction in spasticity. Treatment of minimal spasticity in the acute phase after onset may reduce the abnormal balance between agonist and antagonist muscle groups, delaying or preventing contracture, whilst greatly assisting the functional achievements of the patient. Gormley et al (1997) proposed that this is a result of bombarding the central nervous system with appropriate stimuli at a time of maximum plasticity. Edgar (1992) reports in his review that mild spasticity often responds well to exercise, splinting and oral medication. However, Katrak et al (1992) failed to demonstrate significant improvement when examining the efficacy of Dantrium in the early recovery stage following cerebrovascular accidents, using a double blind cross over design. Subject selection was well controlled

but the subject numbers were small with an 18% drop out rate and assessment of outcome was valid and comprehensive but reliability data was not reported. The study also reported a reduction in muscle power in the unaffected limb and increase in lethargy, neither of which are conducive to functional improvement. Severe spasticity does not, however, respond well to these treatments but poor results with conservative treatment are not an automatic indication to progress to more aggressive interventions, which are often also unsuccessful in such cases, as described by Midha & Schmitt (1998) in their review of a small group of patients with epidural stimulators, as discussed later in this chapter.

Management of spasticity in the case of spinal cord injury is usually conservative. Medication may be used to modify tonic activity by acting at spinal level in three ways: By blocking small calibre gamma afferents and efferents from the skin and viscera to diminish proprioceptive and exteroceptive reflex excitability; by producing an inhibitory effect on the interneurones which modify the mono or polysynaptic proprioceptive reflex or by directly inhibiting the anterior horn cell by exerting a post-synaptic effect on the membrane (Katz & Rymer 1989).

Administration of drug therapy may be oral, intramuscular or intrathecal. Oral administration of centrally and peripherally acting drugs such as benzodiazepines, baclofen and dantrolene sodium, is less invasive but may produce systemic side affects; drowsiness, nausea, muscle weakness and ataxia (Gautier-Smith 1976, Katrak et al 1992). Benzodiazepines (e.g. Diazepam) inhibit neurotransmission but the sedative effect limits use to night time, to

enable the patient to sleep, reducing the general fatigue during the day. Baclofen is also an inhibitor of neurotransmission, although the exact site of action is still under debate, and the sedative effect is recognised to be less marked than with benzodiazepines, as discussed in the comprehensive reviews by Nathan (1997) and Edgar (1992) respectively. Despite it's other side effects, nausea, fatigue and muscle weakness, Baclofen is the drug which is in most common use with the spinal cord injured population (Edgar 1992). Other drugs in common use with this patient group are Dantrium (dantrolene sodium) and gabapentin. Dantrolene sodium inhibits the excitation-contraction mechanisms within the muscle and has been shown to reduce spasticity in patients with upper motor neurone syndrome resulting from different pathologies. The effects of the drug through oral administration are not selective but diffuse to all the skeletal muscle in the body, causing a reduction in preserved muscle function which may present additional problems to the patient such that any benefit is lost (Katrak et al 1992). This may negate its use in the management of the high level tetraplegic who may suffer respiratory problems if accessory muscle and diaphragmatic function is reduced.

Anti-spasmodic medication has been the subject of several experimental trials, as this is required prior to licensing and the introduction of a specific drug as a treatment modality. In a well-controlled double blind cross over trial, Brar et al (1991) found the combination of oral baclofen with stretching to produce a significant reduction in resistance to passive movement. The subject group again was small and they failed to assess the affect on active muscle activity which could be so important to the patient. Likewise, Priebe et al (1997)

demonstrated a significant reduction in tone after forty eight hours following administration of Gabapentin in a double blind cross over trial. They provide excellent detail of the methodology but the patient group was extremely small, the range of time post injury large (twenty one years) and the degree of paralysis ranged from Frankel grade A - D, indicating that the subject group was not homogeneous.

Intrathecal introduction of Baclofen, a physiological neuroinhibitor, by means of a lumbar puncture will depress monosynaptic and polysynaptic reflex activity. Administration in this way may only provide a temporary relief of symptoms but it enables the patient to experience the effects of reduced spasticity. If this causes a significant reduction in functional capacity, the patient may elect to continue on an oral regime or explore other treatment modalities. If found to be successful, permanent implantation of an intrathecal baclofen pump may be performed. In a review of the studies exploring this procedure, which only examine small numbers of patients, Teddy (1995) highlights the invasive nature of the procedure and subsequent risks of infection and also mechanical breakdown, which are not uncommon.

Introduction of sclerosing agents such as phenol into the intrathecal space through lumbar puncture (Edgar 1992) will produce demyelination of the anterior and posterior nerve roots and therefore disruption of the spinal reflex arc. The destructive nature of this procedure renders it an unpopular choice for patient and clinician alike.

The distribution of the spasticity will also dictate the treatment approach. Common patterns of muscle action and co-activation are often observed which may often result in joint contracture (Nathan 1996). Classification of patterns of spasticity and identification of the balance in muscle strength between agonist and antagonist muscle groups may indicate treatment of specific muscles.

Continuous imbalanced muscle activity often results in deformity. Patterns of muscle activity have been described in relation to specific deformities in a review by Nathan (1997) which provide useful guidelines for clinicians when considering the use of local nerve blocks to reduce activity in spastic muscle. Injection of Guanethadine or Botulinus toxin into the region of the neuromuscular junction has produced significant clinical improvement in patients with spinal cord injury, cerebral palsy, multiple sclerosis, cerebrovascular accidents and brain injury (Simpson 1997). The Botulinus has the additional advantage of acting only the motor pathways, preserving any sensory function when present. Pierson et al (1996) examined the use of Botulinus Toxin in a small series (thirty nine) of patients with acquired spasticity. The experimental trial compared placebo and actual treatment but the paper failed to provide comprehensive details of the patient's clinical background or the methodology employed. The treatment goals were to reduce tone, improve motor function, management of personal hygiene, positioning, tolerance to bracing and relieve pain but detail of outcome measurement is only provided for subjectively assessing resistance to passive movement (Ashworth Scale) and change in active and passive range of movement. Pierson found

Botulinus Toxin to be effective but advocated careful patient selection to achieve optimum outcome but failed to discuss this further or give any guidance to assist with selection.

In another small trial (nine subjects), Hesse et al (1992) injected Botulinus Toxin into the upper limb muscles of hemiplegic patients. The subjects were divided into two groups and a different number of muscles were injected in both groups. Hesse failed to discuss how the patients were allocated to the groups but reported an improvement in three of the patients who had five muscles injected. Omission of subject selection and allocation and an increase in subject population should be addressed before the required replication of this study, in order to draw conclusive evidence. Despite the successful outcomes with Botulinus toxin, it is not in frequent use in the United Kingdom due to poor availability and cost.

In patients with a severe degree of spasticity, myotomy, tenotomy and neurotomy have been used to reduce activity in a specific spastic muscle, e.g. obturator neurotomy has been used to reduce activity in the adductor muscles of the lower limb. Anterior rhizotomy will eradicate activity in the spastic muscle but this is unsuitable in patients with incomplete paralysis as any residual voluntary muscle power will also be lost. Posterior rhizotomy has been used to interrupt the reflex arc activity but this again is unsuitable for patients with incomplete cord lesion because of the sensory loss (Gautier-Smith 1976). The invasive and destructive nature of each of these procedures, as with the chemoneurotomy, reduces their popularity with clinicians and patients, but

again no large case series have written up and evidence remains anecdotal.

Epidural spinal cord stimulation has been found to relieve spasticity and muscle spasms in SCI patients (Edgar 1992). It is hypothesised that the stimulation inhibits the hyperexcitable spinal circuits in the absence of modulation from the higher centres but the actual mechanisms of action is uncertain (Midha & Schmitt 1998). In a telephone and face to face interview follow-up of patients with epidural stimulators, Midha & Schmitt (1998) were only able to trace 59% of their spinal injury patients who had received epidural stimulators, of which 50% no longer had the stimulators in situ. The reasons for removal were infection, lack of symptom relief or increase in spasticity and pain. Midha (1998) failed to give extensive clinical details of the group studied but found only one subject in their sample who reported a successful outcome. Midha also failed to report or discuss any factors that could predict a successful outcome with the stimulator and concluded that the invasive procedure, the poor results and expense incurred rendered the implantation of an epidural stimulator as non cost-effective. Gybels & Van Roost (1991) were also of this opinion, observing that a useful reduction in spasticity was only obtained in 20 - 40% of patients in their case review.

Physiotherapy techniques provide a non-invasive means of treatment for spasticity and include passive stretching and standing (Knutsson & Odeen 1981), cryotherapy (Price et al 1993), reflex inhibitory positioning and reducation of normal movement patterns (Bobath 1978) and hydrotherapy (Bedbrooke & Jackson 1985). Substantive evidence to support these treatments

remains anecdotal or has been collected from small case series observations, as previously discussed in this chapter.

More recently, different techniques of application of electrical stimulation have been described - stimulation of the spastic muscle group with high or low frequency current (Robinson et al 1988), stimulation of the antagonist muscle group to the spastic group (Seib et al 1994) or reciprocal stimulation of the agonist and antagonist muscle group (Benton et al 1981, Shindo & Jones 1987). Stimulation of the appropriate dermatome has also been reported to cause a reduction in the level of spasticity (Bajd et al 1985). The methods employed in all these studies require careful consideration before the results are extrapolated to a larger population. No randomised controlled trials have been undertaken, the subject numbers were small but selection criteria nominal and the selection of stimulation parameters was not standardised within each study. Sieb et al (1994) did control the stimulation parameters but had a disparate patient group (brain injury and spinal cord injury), only repeated the experiment on one occasion and failed to examine voluntary muscle power which would have been extremely relevant in their subject sample. Petersen & Klemar (1987) went to the other extreme and stimulated patients for thirty minutes, twice a day for one year. The patient numbers in the study were greater (twenty two) but again of mixed pathology and the stimulation parameters were not constant for all subjects. It is interesting to note that subjects in these studies have usually experienced but not responded to several other forms of treatment, prior to testing electrical stimulation (Peterson & Klemar 1987).

Electrical stimulation provides a non-invasive technique which can be incorporated as an adjunct to current therapeutic modalities. It does not cause systemic side effects and may be conducted by the patient in their own home. In addition to reducing spasticity which may cause the patients considerable anxiety, fatigue and discomfort, stimulation has been shown to promote peripheral circulation (Reger et al 1990) and enhance cosmesis of the lower limbs due to an increase in muscle bulk (Barr et al 1989). This may in turn reduce the risk of varicous ulcers pressure areas in the lower limbs, a further source of noxious afferent stimuli to increase muscle tone.

In certain circumstances, management of the consequences of increased muscle tone, rather than the increased tone itself should be the goal of the treatment plan. Reviews by Edgar (1992) and Stover (1995) report that many patients learn to utilise the presence of increased muscle tone to assist them in functional activities such as transfers from wheelchair to bed or toilet and in extreme cases enable the patient to stand for short periods but objective evidence to support this is not readily available. Reduction of muscle tone may remove this ability which would cause a significant reduction in their level of independence, which would not be acceptable to the patient.

It is clear that the treatment of abnormal muscle tone must be conducted on a selective and individual basis. Abnormal muscle tone can produce direct and indirect problems for the patients and therefore requires a broad spectrum management strategy. In order to determine and monitor the most appropriate treatment plan, it is essential to perform a comprehensive assessment of the

individual to evaluate abnormal muscle tone at both a physiological and human level.

1.3. Measurement of Abnormal Muscle Tone

Measurement of abnormal muscle tone remains a source of interest and frustration to both scientist and clinician. This chapter reviews the broad spectrum of literature surrounding the development and implementation of numerous techniques for the measurement of abnormal muscle tone, with respect to the recognised principles of measurement and discusses the application of such measures within the clinical situation.

1.3.1. Principles of Measurement

The importance of standardised measures to assess patient status and treatment outcome within rehabilitation is well recognised (Wade 1992). A measure should have 'the capacity to define the severity of the problem, detect any changes which occur over time and the effect of a treatment' (Pierson 1997). In order to select the appropriate outcome measure, it is essential to have a clear definition of the question to be answered and that the measure is pertinent and capable of answering the question posed.

When selecting an outcome measure, it is important to review the reliability and validity of the tool. A tool is deemed reliable if it gives repeatable results on every occasion of use. In addition to the reliability of the instrument itself, the intra (within) and inter-tester (between different testers) reliability must be ascertained. Failure to demonstrate the reliability of the measurement tool will render any form of comparison within or between subject groups meaningless.

The implementation of outcome measures in the rehabilitation environment

should follow several basic principles. In order to ensure that the measurements are performed correctly and when required, it is essential that the measures are simple and standardised, to optimise intra and inter-tester reliability and prevent ambiguity in the interpretation of results. Inexpensive measurement techniques, which are quick and simple to carry out, will improve the compatibility with the clinical environment where encouragement may be required to conduct the measurement in the standardised conditions required. The validity of a measure must also be considered within the clinical environment, in conjunction with the specificity and sensitivity. These will both impact on the reliability of the measure.

The validity of an instrument indicates that the tool measures that which it is deemed to measure. For example, the Functional Independence Measure (Granger 1986) was validated as a measure of independence in 18 specific activities of daily living in several patient groups but would not be considered as a valid measure of social reintegration.

There are three aspects of validity that are often discussed;

Construct validity -

'the extent to which results obtained by the measure concur with the results predicted from the underlying theoretical model'

Wade 1992

Criterion related validity -

'the testing of the measure against external criteria'

Wade 1992

and Content validity -

'the component items should not only relate to the construct being measured, but also cover all aspects of that construct'

Wade 1992

Each of these aspects must be considered when selecting the pertinent measure for a particular rehabilitation programme. It is also important to note that a measure validated in one patient group should not automatically be considered valid in another patient group. For example, the Kurtzke M.S Rating Scale, shown to be reliable as a measure of impairment in patient suffering from multiple sclerosis by Noseworthy et al (1990), examines pyramidal, cerebellar, brain stem cerebral, visual, bladder and bowel function. The patient is classified by the degree of failure in a number of systems. Such a scale would be considered invalid for patients with spinal cord injury as the cerebral, cerebellar and brain stem functions of this patient group are not affected.

Goldberg (1991) when reviewing measurement of outcomes in children with cerebral palsy, suggested four parameters by which outcome measures should be evaluated - technical accomplishment; functional outcomes; patient satisfaction and cost effectiveness. The technical outcome of a particular procedure, such as tenotomy to improve range of movement, can be clearly quantified using objective measurement techniques to measure joint range of

movement before and after the procedure. Functional outcomes reflect the impact on the patient themselves; these may include measurement of physical activities e.g. specific motor functions, activities of daily living and personal care, but also measures of psychological function. Many indices of physical and psychological function have been developed for use in a variety of patient groups (Wade 1992) and these will be discussed in Section 1.3.2.

Goldberg (1991) advocated in his review that patient satisfaction should be ascertained, not only in relation to the outcome of the treatment but also with respect to the way in which the health care was delivered. He did not discuss the varied methods available to obtain this information - questionnaires and interviews (structured or semi-structured) or the methods for their administration (postal or personal delivery of questionnaires). Both tools may include closed questions requiring categorised responses or open questions which explore specific points in greater detail. The choice of assessment tool should relate to the available access and size of the study group and the nature of the information required. In addition to reflecting the nature of the tool used, the response of the patient may be biased by their attitude towards the researcher, the outcome of their treatment in relation to their expectation s and their experiences whilst undergoing the treatment. Goldberg's review (1991) fails to draw attention to all these issues which are fundamental to conducting sound research.

The final and perhaps most controversial measure proposed by Goldberg (1991) is that of cost effectiveness. Many clinicians would argue that this

cannot be reviewed in isolation but must take the other three principles into consideration i.e. those of technical and functional outcome and perhaps most importantly patient satisfaction. Failure to achieve the latter may be considered to negate the technical and functional outcomes.

Data collected from outcome measures falls into four categories; nominal, ordinal, interval or ratio (Wade 1992). Ratio level data is considered to be the highest and most robust level for statistical analysis but the nominal and ordinal level data collected through qualitative measures also provide invaluable information in rehabilitation research. In an attempt to address the problem of the level of data collected in qualitative research, the technique of Rasch analysis has been applied (Harvey & Jellinek 1981). Ordinal level data can be converted to interval level, thus enabling parametric statistical analysis. In order to convert the ordinal data from functional rating scales to interval level, the equal intervals required are calculated on a basis of the probability of a person with a given level of ability to perform a specific function. True interval data is ordinal with the additional feature of the intervals between any two points being identical in real terms. The technique of Rasch analysis for the manipulation of data can therefore be criticised as it is dependent upon the use of an averaging process and the ability to determine and recognise the interval points.

The measurement of the symptoms of the upper motor neurone syndrome present several methodological problems to the rehabilitationist. In addition to the complexity of the mechanisms involved in the regulation of muscle tone, changes in the state of muscle tone will occur due to systemic factors (presence of infection, pressure sores, emotional status), environmental factors (temperature, support surface), positioning of the patient during testing and training effects during the test procedure. Guttman (1973) observed these phenomena in his patients as did Edgar (1992). Both authors observed many spinal cord injury patients in rehabilitation but no formalised research was conducted to explore this further and Edgar fails to quote substantive references to confirm his observations in his review. It is also not uncommon for a pattern of diurnal variation to emerge due to these and other factors. This must be considered when examining the test-retest capacity of the measure, which may quite accurately indicate a significant difference in the measurements taken on several occasions. These may be representative of the changed status of the patient baseline rather than an unreliable measurement tool and technique.

As discussed in the previous chapter, there are many facets to the presence of abnormal muscle tone. These present difficulties when considering the content validity of any measure. It is therefore important to clearly define the question that is being posed, in order to select the most appropriate measurement tool. i.e. which particular component of abnormal muscle tone is to be assessed. The focus of the question will be determined by the individual posing the question clinician or scientist, the location of the assessment and the nature of the treatment intervention.

Haas et al (1994) conducted a postal survey of one hundred and fifty six health

care professionals (physiotherapists, occupational therapists, nurses and physicians) in rehabilitation settings to establish current practice in the measurement of spasticity. 51% of the questionnaires sent out were returned. Incomplete or corrupted questionnaires were excluded from the final analysis, reducing the response rate to only 35%, of which 43% were completed by physiotherapists. These factors must be taken into account when considering the findings of the study as the sample was not representative of the professions surveyed.

Methods of spasticity measurement most frequently recognised by the respondents were electromyography and tendon jerks. Testing tendon jerks was the modal method by occasional and frequent assessors of spasticity. These responses were reviewed across the whole study population and not differentiated within the individual disciplines. All doctors and nurses surveyed believed that it was important to measure the level of spasticity in patients with neurological conditions. Surprisingly, three respondents (one occupational therapist and two physiotherapists) did not feel that this was necessary. Conversely, 94% of the respondents identified the physiotherapist as being the most appropriate professional to measure spasticity. The findings of Haas were also disappointing to this author in that less than 50% of the physiotherapists surveyed believed that measurement was integral to the management of spasticity.

1.3.2. Measurement Techniques

Numerous studies have been performed in the search for a reliable method of quantification of muscle tone (Bajd & Vodovnik 1984, Bohannon 1987, Lehmann et al 1989, Priebe et al 1996, Perell et al 1996, Lamontagne et al 1998, Sköld et al 1998). Some of these studies have favoured patients with cerebrovascular accidents resulting in hemiplegia (Bajd & Vodovnik 1984, Bohannon 1987) thus providing one limb as a control but often studies combine patient groups, the most frequent being hemiplegia, traumatic brain injury and spinal cord injury. This combination could be criticised for including intra-cranial pathology and spinal cord pathology. Bohannon (1987) examined reliability of the Pendulum Test in thirty patients with intra-cranial lesions (22 with cerebrovascular accidents and 8 with traumatic brain injury). He demonstrated high reliability (Intraclass correlation = 0.96) despite the large age range and time post injury. Bajd & Vodovnik (1984) also examined reliability of the Pendulum Test and compared a group of spinal cord injury and hemiplegic patients. They too found a greater consistency in the measures performed on the hemiplegic group but failed to indicate the number of patients in each group or to give additional clinical details ie. level and degree of paralysis in the cord injured group. Lehman et al (1989) also failed to give this clinical information in an extremely small study of only eight cord injured subjects and five subjects with traumatic brain injury. Their conclusions however, related to the comparison of the spastic subjects with neurologically intact subjects and did not seek to compare the two pathological groups in depth.

In studies where a single patient group is examined ie. spinal cord injury, often the sample is still not homogeneous as it includes subjects with complete and incomplete paralysis. Subjects with incomplete paralysis may have residual sensory function and a range of motor preservation such that comparison is invalid. Priebe et al (1996) and Lamontagne et al (1998) examined cord injured patients with complete and incomplete paralysis but neither author stated the degree of sensory or motor function in the incomplete group and only Lamontagne controlled for time post injury, which may in turn also affect the amount of voluntary motor function. Sköld et al (1998) however, in her study to compare two methods of measurement, did control for extent of paralysis and time post injury but only examined a small number of subjects (fifteen) on one single occasion. The small subject numbers is an additional criticism of most of these studies. Priebe et al (1996) examined one of the largest populations (eighty five spinal cord injured patients) but failed to give adequate clinical information as previously, discussed.

Wade (1992) provides a comprehensive listing of impairment, disability and handicap measures for both subjective and objective quantification of spasticity in various patient groups but the literature supports the opinion that no single instrument or technique will provide a global measure of this multi-faceted phenomenon in any one patient group.

Subjective Measures

The Ashworth Scale (Ashworth 1964), was first developed as a measure of spasticity to evaluate the use of medication in multiple sclerosis. The patient is placed in a standardised position & the limb (upper or lower) is moved passively through a full range at a fixed speed. The resistance to movement felt by the tester is then graded from 0 to 4. Descriptions of each classification are shown in Table 1.5.

Table 1.5: Ashworth Scale

Grade	Description			
0	Normal muscle tone			
1	Slight increase in muscle tone, "catch" when limb is moved			
2	Marked increase in tone, but limb easily flexed			
3	Considerable increase in muscle tone			
4	Limb rigid in extension or flexion			

Ashworth 1964

Whilst grading at the extreme ends of the scale i.e. 0 & 4 is reliable, intra and inter-tester reliability around the middle grades has been shown to less. Bohannon & Smith (1989) criticised the numerical rating scale for the cluster effect around the middle values and subsequently modified the scale, adding the Grade of 1+ (Table 1.6). Reliability of the modified scale was examined in two testers (Bohannon & Smith 1989), evaluating the resistance to passive movement in thirty patients with a variety of upper motor neurone problems on only one test occasion. Intra-tester reliability was not reported but a correlation between the scores of the two assessors of 0.847 (Kendall's Tau) was

demonstrated, indicating high inter-tester reliability.

Table 1.6: Modified Ashworth Scale

Grade	Description		
0	No increase in muscle tone		
1	Slight increase in muscle tone, manifested by a catch & release or by minimal resistance at the end of range of motion when the part is moved in flexion or extension/abduction or adduction		
1+	Slight increase in muscle tone manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of movement		
2	More marked increase in muscle tone through most of the range of movement but the affected part is easily moved		
3	Considerable increase in muscle tone, passive movement is difficult		
4	Affected part is rigid in flexion or extension (abduction or adduction)		

Bohannon & Smith 1989

Further criticism of both scales relates to the subjectivity and lack of control over the velocity of movement. The test is also considered to give a global picture of reflex muscle activity and soft tissue contracture, rather than assessing one particular entity. In an attempt to address the non-specific nature of the measure, Sköld et al (1998), in a small but well-controlled group of chronic spinal cord injury patients (fifteen with complete motor paralysis) recorded a score for the movement through flexion and separate score for the movement through extension and found a significant difference. The experiment did not however vary the order in which the movement was performed, ie. flexion was always performed and scored first. The attraction of the intra and inter-tester reliability of the Modified Ashworth Scale and its validity in a variety of patient groups is such that it is now recognised measure for simple and rapid use in the clinical environment.

Several authors have utilised indices of physical and psychological function ie. measures of disability, as a representation of the impact of increased muscle tone on an individual. These scales are numerous and devised for use within different patient groups (Wade 1992) to address the issue of validity. Wade (1992) categorises assessments into focal disability ie. examination of specific activities, for example timed walking tests, specific upper limb tasks and more generalised activities of daily living.

One of the most frequently recognised scales is the Barthel Index (Mahoney & Barthel 1965) which examines the level of independence of the individual in

ten activities of daily living, relating to mobility and personal care. The scoring varies for each item of the test, ranging from 0 to a maximum of 3. The index is scored by the clinician, obtaining information from the patient or carers if necessary about the patients actual rather than potential ability to conduct the activities examined. The Barthel Index can be criticised for its insensitivity, as compared with the Functional Independence Measure - FIM (Granger 1986) which comprises eighteen items, offers a standardised range of scores from 0 to 7 and clearly differentiates between independence with and without assistive devices. The FIM is also performed by the clinician and scores the actual, not the potential ability of the patient. Both the Barthel and the FIM have been used in various patient groups (Wade 1992).

Other scales with a smaller range in scores have also been shown to be valid and reliable in a range of patient groups. The Rivermead ADL Index (Whiting & Lincoln 1980) was developed for use with hemiplegic patients and provides clear explicit guidelines for assessment and a score range of 1 to 3, 3 indicating independence with or without an assistive device. This scale examines self care and household tasks but fails to address bladder and bowel management, which features in the FIM. The Northwick Park Index of Independence in Activities of Daily Living (Benjamen 1976), also developed for use with hemiplegic patients and accompanied by clear guidelines for assessment and provides a three point score range (0 - 2) which aids reliability but again reduces sensitivity to change.

The Fugl-Meyer Scale (Fugl-Meyer et al 1975), used in a comparative study of

different assessments of muscle tone (Katz et al 1992), is specific to the hemiplegic patient group. It measures the degree of motor function (ie. impairment) rather than specific functional activities of daily living (ie. disability) and differentiates between upper and lower limb activity. Katz et al (1992) demonstrated a high correlation between this scale, the Modified Ashworth Scale and biomechanical measures of spasticity, supporting the construct validity. The assessments were carried out on ten subjects on three occasions by one investigator but the reliability of the tester and the measurement tools were not quoted in the paper.

Interpretation of data from such scales must be approached with caution. Use of the total scores for the index function will facilitate comparison of patients within and between studies but further analysis of the individual dimensions of the index may be required to improve the sensitivity and specificity of the evaluation of treatment outcome.

Objective assessment of specific functional activities has also been used to assess muscle tone. Gait analysis has been used to evaluate the impact of spasticity on locomotion in spinal cord injury subjects with incomplete paralysis. Krawetz & Nance (1996) studied temporal and kinematic parameters during walking in a mixed group of cervical, thoracic & lumbar cord injured subjects with upper motor neurone paresis and preserved lower limb function (Frankel D) and reported that the level of injury correlated with alteration in specific parameters of gait. Several methodological issues cloud the study. The number of subjects in each group was small (n = 8, 8 and 11 respectively) and

the range of time post injury was from six months to forty nine years. They recruited a control group of ten for age, sex and anthropometrically matched normals but did not indicate how these related to the individual patient groups. The gait analysis relied on data obtained from reflective markers placed on the patient but no reference was made to the intra or inter-tester reliability or the reliability of the equipment. Despite the shortcomings of the methodology, the findings of the study merit further replication of the study in a larger subject group and demonstration of reliability of the assessors and equipment.

It is important to review the findings of a functional measure in the context of the individual patient's circumstances. Indices of function examine the disability of the patient but may not differentiate between the causative impairments or the handicap to the individual. When analysing the impact of a treatment on functional abilities, it may be appropriate to review any change in status in relation to the specific impairment. For example, a significant reduction in muscle spasms further to implantation of a baclofen pump in a spinal cord injured patient may not be represented by a significant increase in functional ability in a high level tetraplegic, who will continue to achieve a low score due to the level of assistance required in activities of daily living, regardless of the level of abnormal activity. The benefits from the reduction in tone may be considered no less advantageous by the patient and therefore alternative outcome measures will be required to demonstrate change.

The patient perspective of abnormal muscle tone and the subsequent consequences have been addressed through self reporting instruments and form

an important contribution to the evaluation of any treatment or rehabilitation programme (Midha & Schmitt 1988, Penn et al 1989). Again interpretation of the findings must be considered in light of other supporting information. Cognitive impairment may affect the patients ability to complete such an exercise accurately or correctly, invalidating the information. The emotional status and attitude to disability at the time of testing may also influence their scores (Buckelew 1991).

Measures of psychological status may also be used to evaluate the consequences of abnormal muscle tone for an individual. These should again be viewed with caution as many factors can contribute to the mood state of an individual and any change in psychological status may not be attributable to a single impairment. As with the numerous indices of function, many psychological assessments have been developed for specific patient groups (Wade 1992) but several generic assessments also exist. The Beck Questionnaire (Beck et al 1961) is a self reporting assessment which has been validated and shown to be reliable for the evaluation of the effects of antidepressant medication but is not regarded as a valid diagnostic measure. Wade (1992) suggests in his review that it may also be used as a measure of distress rather than depression in patients with a disability. The Hospital Anxiety & Depression scale (Zigmond & Snaith 1983) is also a generic self report measure that is short and simple to administer, which accounts for its frequent use, but the scale still requires further work for validation and demonstration of reliability with patients with traumatic brain injury. The General Health Questionnaire (Goldberg 1972) is another well recognised index which

examines somatic symptoms, anxiety, depression and social dysfunction using a four point Likert Scale. The limited scoring will improve the reliability of the instrument but as patients do not complete the GHQ themselves, its utility may be limited and also influenced by the relationship between the patient and clinician completing the questionnaire.

The location and method of data collection for self assessments may bias or distort the data. Face to face administration of the measurement tool may facilitate patient understanding and completion of the assessment, providing a more reliable data set. This method may not always be appropriate for the parameter under evaluation. For example, spasm diaries are often used to record patterns of abnormal muscle activity over long periods of time. This method relies on the diligence and compliance of the patient: It is not inconceivable that a patient may complete the daily diary at the end of the day rather than at regular intervals throughout the day, as may be required. This will undoubtedly affect the reliability of the information. Similarly, if the patient is desperate to seek an alternative method of management to reduce their spasms, the records of spasm frequency for example may be adjusted by the patient to achieve that goal. Midha & Schmitt (1998) used a 0 -10 rating scale in a telephone survey of SCI patients to rate the effect of epidural stimulation on the patient's spasms. The paper gives no indication that the reliability or validity of such a measure have been considered yet draws substantive conclusions from their results.

Objective Methods

Objective measures are divided into two main categories - mechanical and physiological.

Electrophysiological measures examining the H reflex, M response (Delwaide1984) and lumbosacral evoked responses (Katz & Rymer 1989) have been reported to assess the excitability of the motor nucleus and presynaptic responses in the dorsal horn respectively. Characteristics of the tonic stretch reflex have also been examined and compared in patients with upper motor neurone syndrome resulting from different impairments (Gildenberg 1985).

A summary of the electrophysiological measures examining different components of the central and mechanisms governing muscle tone are shown in Table 1.7. (Seghal & McGuire 1998).

Table 1.7: Electrophysiological Testing for Spasticity

Mechanism	Test	Normal Values	Values in spasticity
Alpha motor	Hmax/Mmax	5-35%	Increased
neurone	Tmax/Mmax	5-40%	Increased
excitability	F response	1-5% of M amplitude	Increased
	-	_	amplitude/frequency
Presynaptic	Vibratory index	40% in relaxed subject	Acute - no change
inhibition		(wide variability)	Chronic - significant
			reduction
Reciprocal	Conditioned H	H reflex inhibited	Decreased
Inhibition	reflex by		Inhibition/facilitation
	antagonist		of H reflex
	contraction		
Recurrent	H responses to	H reflex inhibited	Variable results
(Renshaw)	double collision		
inhibition			
Polysynaptic	Tonic vibration		Abnormal
activity	reflex		
	H reflex recovery		Abnormal
	cycle		
	Flexor reflexes		Wide spread response

Seghal & McGuire 1998

Little correlation was shown between the H/M reflex ratio and the degree of severity of spasticity, giving little clinical application to this method of evaluation, but a strong correlation was seen with presynaptic responses in the dorsal horn. In a small group (ten) of hemiplegic patients, Katz et al (1992) examined repeated measures of the H reflex. Despite rigorous control of positioning subjects and both stimulating and recording electrodes, he showed poor intra-subject repeatability of this technique and poor correlation with the clinical assessment. The equipment required for such analysis is also not readily available in the clinical setting and therefore such methods of quantification of spasticity have, to date, been restricted to the laboratory.

Electromyography (EMG) is a technique by which action potentials of motor units (the motor neurone, its axon and all the muscle fibres innervated by it) are recorded and displayed. This methodology can provide information about the structure and function of the muscle. (Aminoff 1980).

The characteristics of the EMG signal which are considered for quantification are the frequency, duration, amplitude and shape of the signal. De Luca (1997) refers to the firing rate (the number of repetitions of action potentials within a fixed time pocket) as opposed to firing frequency which implies a regularity in the intervals between action potentials. Analysis of the sequence of action potential waveforms reveals that the motor units do not fire synchronously and demonstrate a range of firing frequencies. The average of these firing rates may then be calculated (mean—firing rate). The firing rate gives an indication of central nervous system function whereas the shape of the motor unit dictates

the fibre type characteristic i.e. change in firing rate will represent the state of the upper motor neurone and change in the shape is indicative of lower motor neurone problems (Aminoff 1980).

Analysis of the EMG power spectrum (plotting amplitude of the signal against frequency) also reflects a difference between normal and pathological muscle. In normal human muscle, the peak amplitude is found between 100 - 200Hz during maximal contraction. In the presence of a myopathy, the peak amplitude will shift to a higher frequency (Aminoff 1980). In a review of methodological problems with electromyography, Turker (1993) reported that the frequency band width appropriate for all EMG signals is 20 - 10,000Hz, although it is possible to filter at 500Hz without detecting a significant change in the signal. The band width of the signal is inversely proportional to the distance between the electrodes therefore the electrode type will be reflected in the band width of the signal. The band width will also be affected by the level of muscle contraction. These issues should be recognised in any experiment with repeated measures.

The method for analysis of the EMG signal must be considered in light of the question posed. Two methods of processing have been utilised in previous studies. Calculation of the root mean squared value is performed by squaring the signal, averaging it and then taking the square root of it. De Luca (1997) advocated the use of the mean rectified signal as more correct. It examines the amplitude by rectifying signal and averaging it. Both methods require averaging over fixed period of time. The sample interval should be sufficient to

produce smoothing of the signal (200 - 300ms/min) but not so long as to smooth out the pattern altogether. Measurement of the mean amplitude of action potentials cannot be used as a universal measure as the values depend on the technique of recording and would require a level of duplication of methodology that is not possible in the clinical environment (Turker 1993). A different electrode position will change the signal, which presents difficulties with test/retest reliability. This problem may be addressed by normalising the data. Measurement of the mean duration of action potentials can be viewed across subjects and this technique has been used to establish age related normal values and also ranges of values for specific pathological conditions, provided the methodology remains constant (Aminoff 1980). This technique requires the exact marking of the start and finish of the action potential using strictly defined criteria. Such precision is time consuming and relies on a skilled operator and therefore is perhaps not the most compatible with a busy clinical practice.

The choice of recording medium, surface or intramuscular electrodes, is dependent on the nature of the investigation. Neurologists need to examine the properties of individual motor units and determine voluntary motor unit recruitment in order to diagnose neuromuscular disorders (Tosi 1993). Needle electrodes are therefore the more appropriate medium in this instance. There are two types of needle electrodes - concentric or coaxial electrodes and monopolar electrodes. Concentric needle electrodes consist of an outer stainless steel cannula through which runs a single wire that is exposed at its tip. The inner wire serves as a recording electrode and the outer cannula serves

as a reference electrode and the potential difference between the two is recorded. A separate ground electrode is used with this system. The concentric needle will record from a smaller area than the monopolar electrodes (Aminoff 1980). In an attempt to address this problem, Campbell et al (1991) recorded activity from ten sites in four quadrants of three lower limb muscles, using concentric electrodes in a small group (nine) of spinal cord injured patients to compare activity within muscles. The purpose of the study was to compare patients with acute and chronic cord injuries and the patients served as their own controls but the assessment was only performed on one occasion and no discussion is presented regarding differences between the ten different sites examined.

Monopolar needle electrodes are solid steel covered with insulation except at its tip. This electrode serves as the active recording electrode: a second reference electrode (either surface or needle) is placed nearby. The potential difference between the two is measured. A separate ground electrode is also required for this system. Variability in the position of the reference electrode and changes in the recording area on the needle tip can affect the motor unit potential characteristics with monopolar recordings (De Luca 1997). However, the monopolar electrode has several advantages over the concentric needle electrodes; it will record activity from a larger area, is less painful for the patient and less expensive for the purchaser (De Luca 1997). Campbell et al (1991) utilised a monopolar technique to investigate spontaneous potentials in complete tetraplegic patients in the acute (less than four weeks) and chronic (greater than one year post injury). Campbell does not discuss his reason for

choosing the monopolar technique but the patients in the study had complete sensory loss and therefore would not perceive pain at a conscious level. However, the presence of a noxious stimulus, discomfort from the needle would influence the neuromuscular activity, as discussed previously.

In rehabilitation, kinesiology and ergonomic studies, clinicians and researchers want to look at more global activity of the muscle and therefore surface electrodes are the medium of choice. The surface EMG signal is a superimposition of many EMG signals on top of each other and therefore is a reflection of many motor unit action potentials (De Luca 1997). Surface electrodes have the advantage of being non-invasive with no risk of infection and therefore more appropriate for repeated investigation. In a review of the anatomical and technical considerations of surface electromyography, Fedjallah & Wertsch (1998) summarised the disadvantages of the technique, demonstrating that the surface signal is more susceptible to noise than the signal recorded by the needle electrode. They conclude that action potentials vary in duration, amplitude and frequency and are greatly affected by the volume conduction of the soft tissues through which the signal must pass before being picked up by the recording medium. The time variation in surface EMG is due to the difference in the distance between the electrode and the muscle fibre and the length of the axon. Cross talk signals propagate through the muscle and fatty tissue and become more of a problem as the amount of muscle activity increases. Surface electrodes lack selectivity and therefore may interpret activity from other muscles as activity from the muscle under investigation. This can be validated by comparing activity recorded by

intramuscular electrodes. In order to minimise this problem, Fedjallah & Wertsch (1998) recommend reducing the surface area of the recording electrode and decreasing the inter-electrode distance in order to reduce the range of electrode pickup.

Tissue artefact may also be minimised through adequate skin preparation to reduce impedance and appropriate electrode selection and placement. Adequate fixation of the electrode is also essential, to ensure continuous contact with the skin surface as with the pre-gelled self adhesive electrodes. Discrete electrode placement between the motor point and the tendon will optimise the signal and the operator should ensure that the electrode configuration is less than one third of the length of the muscle to prevent cross talk (electrical activity in other muscles). The size of the detection surface and the inter-electrode distance will also affect the signal: a larger surface area and greater inter-electrode distance detecting a greater number of motor units. Caution should be exercised with the inter-electrode distance, which if too great, will result in bipolar electrodes acting as 2 monopolar electrodes (De Luca 1997).

System noise is a signal that has no relation to the muscle activity and may be affected by skin impedance, the depth of fat and muscle tissue; the size, shape and location of the electrode itself or the recording equipment. If the environment is very noisy, a bipolar recording technique will assist in reducing signal artefact. Noise is a common mode signal which will be present at both electrodes. If the signal is then passed through a high impedance differential amplifier, a further reduction in signal artefact will occur. The electrodes

themselves also act as a filter to the signal and therefore different electrode substances produce different degrees of filtration (Turker 1993). This is important to consider when examining the repeatability of the EMG recording.

Studies of EMG activity in spastic muscle during movement have often been performed at different test speeds. This often presents a problem for recording system because of movement artefact. This again should be addressed through adequate skin preparation and electrode fixation and minimising the distance between the electrodes and recording system (De Luca 1997). Few authors discuss their approach to such methodological problems when assessing spastic muscle but Lamontagne et al (1998) addressed the problem of movement artefact when moving the lower leg of spinal cord injured patients by raising the high pass filtration to 20Hz. The increasing demand in the clinical environment for EMG recording during active muscle work led De Luca et al (1997) to design an active bipolar electrode (DELSYS.USA) with local preamplification. The electrode required minimal skin preparation, maintained a constant inter-electrode distance for standardisation and was reported to be robust to movement artefact. This was not, however, the experience of the author in this study, which will be discussed in Chapter 2.

Biomechanical testing examining the relationship between joint angle and threshold level of torque or EMG activity increase (Katz & Rymer 1989), torque generated and the velocity dependence of EMG activity during active and passive movement of the limb (Knutsson 1980), have been considered as measures of spasticity in a variety of patient groups.

The Pendulum Test was originally devised by Wartenberg (1951) to assess spasticity in parkinsonism. The test examines the visco-elastic properties of the quadriceps and hamstrings in a freely swinging lower limb. The subject is positioned in supine lying with the upper leg supported and the lower leg suspended freely over the edge of the support surface. The foot is raised bringing the knee to a fully extended position. The leg is then released and allowed to swing freely until movement has ceased. The angle of swing is measured using an electric goniometer. An index of spasticity was calculated as the ratio between the angle of movement in the first swing and all the movement before the leg comes to rest.

The reliability the test has been studied by many authors in a variety of neurological conditions. Bajd & Bowman (1982) observed a significant decrease in the spasticity index (a reduction in spasticity) in a group of spinal cord injury patients when the swings were repeated every 30 seconds. Vodovnik, Bowman and Bajd (1984) also concluded that the goniograms were more consistent in hemiplegic patients than in patients with spinal cord injury. They did not however state the number of patients examined in the study or provide important clinical details of the cord injured patients. Demonstration of good reproducibility endorses the use of this as a measure whilst recognising its limitations, testing only two muscle groups of the lower limb, the hamstrings and quadriceps.

Bohannon (1987) utilised an isokinetic dynamometer to determine between trial reliability in a group of thirty patients with intracranial lesions (twenty two patients with cerebrovascular accidents and eight with traumatic brain injury, although the locus of damage was not stated). He demonstrated a low variability between trials and recommended the test for extended use within the specific patient group. The findings of this study may be related to the number of repetitions (four) for each test which was less than in other studies and the fact that the dynamometer maintained a constant velocity during the movement (300°/second). The dynamometer also differs from the electrogoniometer, in that there is a damping effect caused by the deceleration of the dynamometer at the end point in both directions. This is not the case for the electrogoniometer which is essentially free swinging.

The studies utilising the pendulum test have not used standardised positioning of the patient, either high sitting or supine lying. This may affect the outcome of the measure in neurologically impaired patients. Placing the patient in a supine position is known to increase extensor tone (Bobath 1978); positioning the subject in high sitting will put stretch on hamstrings when knee is taken up into full extension which may in turn elicit a stretch reflex.

Several studies have combined the Pendulum test with other methods of measurement. Bajd & Vodovnik (1984) examining the combination of the Pendulum test and electromyography in a group of patients and reported that the EMG did not augment the data recorded from the original procedure. The findings of the study support those of Bajd et al (1984) which demonstrated a difference between a cord injured and a hemiplegic population. This may,

however, have been due to the level of spasticity in each group - the rating of the cord injured subjects ranged from mild to severe and the hemiplegic patients were rated as moderate. Bajd failed to state the number of patients reviewed in the study and also did not differentiate between patients with complete and incomplete paralysis which may also influence the outcome of the study, as previously discussed.

Jamshidi & Smith (1996) compared the electrogoniometer with a system of video analysis in only 3 spinal cord injured with varying degrees of spasticity and one neurologically intact subject. They concluded that both methods were highly reliable with intraclass correlation coefficients of greater than 0.99. The study determined the appropriate positioning of the reflective markers for the motion analysis but the number of assessors and reliability of assessors and equipment were not reported. Jamshidi subsequently concluded that the expense of the video digitisation system made the electrogoniometer a more popular choice for use in the clinical environment.

More recently, the increased presence of dynamometers in the physiotherapy clinic has enabled the clinician to record the moment generated about the joint when passive motion of the limb is performed (Campbell et al 1990). The dynamometer is a motor controlled exerciser which enables the clinician to standardise test procedures, controlling the range and velocity of movement. The current software and hardware offer the clinician simple on-line analysis or the facility for more detailed analysis off line. The most negative characteristic of the commercially available dynamometer is the cost.

Lamontagne et al (1998) examined the inter-trial reliability of a cheaper hand held dynamometer (Penny & Giles) with an isokinetic dynamometer, by comparing the torques measured about the ankle joint during passive stretching in a small group (nine) of subjects with spinal cord injury. The study was well controlled and established the reliability of the equipment but not of the operator, who aimed to match the two test speeds of the isokinetic dynamometer (5° & 180° per second). A higher repeatability was observed between the hand held device at the lower velocity (Intraclass correlation = 0.93) than at the higher velocity (ICC = 0.84). This may well have been due to operator error. Surface EMG recordings confirmed the validity of the hand held dynamometer as a measure of reflex activity. The study concluded that the hand held dynamometer was potentially useful in assessing both tonic and phasic muscle activity.

Dynamometry has been used in a variety of patient groups to examine muscle activity during passive and active movement (Knutsson 1980, Katrak et al 1992). The dynamometer can be adapted to test both upper and lower limbs and is most frequently used to examine the knee, ankle and elbow joint. Each of these joints works primarily in one plane and therefore fixation of the patient, when aiming to maintain reliability is easier. Katz and Rymer (1989) advocated measurement of torque at specific joint angles using this method, having concluded that joint stiffness was not a significant variable. Knutsson (1985) favoured both passive and active testing of the quadriceps and hamstrings at different speeds, ranging from 30° per second to 180° per second

with dual channel surface EMG recording. He examined a range of pathologies and concluded that EMG activity during eccentric muscle contraction does not decrease in the spastic patients as it does in normal subjects. He did not however differentiate between the spinal cord injured and hemiplegic patients in the study. In a small study, Perell (1996) compared a group of spastic spinal cord injured patients with a group of flaccid spinal cord injured and matched neurologically intact subject group. He concluded that movement should occur at 120° per second to detect a difference between the subject groups. The size of the subject groups were small and unequal and he did not differentiate between the complete and incomplete cord injured subjects in the spastic group. The procedure was however well controlled and only the data from the middle five repetitions was used for analysis

Early work at Royal National Orthopaedic Hospital (Barr et al 1989) explored the use of a dynamometer to record torque generated during passive movement of the lower limb at pre-set velocities. This was considered to give a composite assessment of all structures contributing to the resistance to passive movement ie. tonic and phasic muscle activity and other soft tissue changes (eg. fibrosis and shortening). The technique was not, however, able to distinguish between the level of activity in individual muscle groups. Clinical observation of patients during the conduction of the tests indicated that increased tonic muscle activity was not limited to the muscles being passively stretched ie. quadriceps and hamstrings, but also occurred in other muscle groups of the limb. Development of this method of quantification with the augmentation of

polyelectromyography, as anticipated in this study, will enable the investigators to examine the relationship between resistance to passive movement and electromyographic activity (phasic muscle activity - muscle spasm). It has been demonstrated that an increase in resistance to passive movement can be encountered without a corresponding increase in myoelectrical activity (Barr unpublished). The ability to differentiate between these two symptoms in the evaluation may assist in planning of treatment protocols for upper motor neurone dysfunction.

In more recent studies, researchers have combined and compared various methods of measuring muscle tone, often combining a subjective and an objective measure. Sköld et al (1998) examined the correlation between the Modified Ashworth Scale and simultaneous mean, peak and duration of EMG activity in a small group (fifteen) of spinal cord injured patients. She demonstrated a significant positive correlation between EMG activity and the Ashworth scale, with a stronger correlation for the flexion movements. These tests were however only repeated once and the flexion movement was always performed first.

Katrak et al (1992) combined two subjective rating scales, the Motor Assessment Scale (Carr et al 1985) and the Barthel Index (Mahoney 1965) with isokinetic dynamometry to assess the effect of Dantrium on spasticity following cerebrovascular accident. They reported that where possible the clinical assessments were performed by the same doctor but made no reference

to the number of physiotherapists performing the Motor Assessment Scale and quote only the original paper (Carr et al 1985) for the inter-tester and test-retest reliability. The study found that there was no change in the functional ability or reduction in tone in the spastic limb but a significant reduction in torque developed in the unaffected limb, indicating that the change was due to the Dantrium and not to insensitivity of the measures.

Katz et al (1992) compared six measurement tools; the Fugl-Meyer Functional Assessment, the Modified Ashworth Scale, H reflex studies, the Pendulum test, a torque meter and electromyograph in a group of ten hemiplegic patients. The subjective measures were performed on the patients prior to the four objective measures which were conducted in a random order. The time intervals between assessments were not documented. Katz concluded that there was strong negative correlation between the two clinical measures (Fugl-Meyer and Motor Assessment Scale) and concurred with the findings of Sköld that the stronger correlation was with movement in flexion (r = 0.946).

The academic exercise of comparing measurement techniques has provided interesting but not unexpected results. The author recognises the need for an objective measurement technique to quantify the effects of different treatments for spasticity in spinal cord injury patients and appreciates the advantage of measures that may be easily performed in the clinical environment. Both the literature and clinical experience indicate that both objective measurement of the symptoms of the upper motor neurone syndrome and the patient's

perception of the problem should be included in any assessment to provide a reliable and representative measure of this multi-faceted phenomenon which presents such problems to this patient group. The purpose of this study was therefore to devise a comprehensive and reliable protocol for the measurement of abnormal muscle tone in spinal cord injury patients which is compatible with the clinical environment.

Chapter 2: Development of Method

2.1. Introduction

The purpose of this study was to devise an holistic assessment programme for the evaluation of abnormal muscle tone in patients with spinal cord injury which is compatible with use in the clinical environment. This assessment package should therefore include examination of the basic muscle activity and patterns of abnormal muscle activity (strength, frequency and inter-muscular activity), limb mobility which reflects both muscle and soft tissue status, and most importantly, the patient perception of the problem. The methods for assessment of each of these parameters were reviewed in the previous chapter but the justification for the selection of the subjects and specific assessment protocols examined in the study is discussed below.

Subjects

The purpose of the study was to devise an assessment protocol for use with spinal cord injured patients. In order to establish the validity and reliability of the tests as assessments of muscle function, patients with complete motor and sensory loss were selected for the study to control for the effects of voluntary muscle activity or somatosensory or proprioceptive input.

In order to establish a baseline control measurement for the patients, it was necessary to perform the same assessment programme in a group of neurologically intact subjects. Anthropometric factors have been reported to effect the incidence and nature of spasticity (Stover 1995, Iansek 1984) and

therefore the normal subjects were matched to patients for sex, age, height and weight.

Instruments

The Ashworth Scale (Ashworth 1964) is commonly used by clinicians for the measurement of spasticity in many patient groups (Haas 1994). The Modified Ashworth Scale (Bohannon & Smith 1987) gives greater descriptive guidelines for the clinician, in order to improve reliability. Bohannon & Smith also changed the grading from 0 - 5 to 0 - 4, adding an extra grade of 1+ to maintain the number of available scores. In a review of measurement tools for neurological rehabilitation, Wade (1992) reports the classifications of the Modified Ashworth Scale as described by Bohannon, but maintains the 0 - 5 rating. This was the format chosen for evaluation of the Modified Ashworth Scale in the pilot study, to facilitate data analysis.

The inclusion of the patient perspective is an essential component to a complete assessment. Muscle tone is known to vary at different times of the day, due to specific activities, psychological stress or the presence of other noxious stimuli such as pressure areas or infection (Edgar 1992). Edgar, in his review of post-traumatic spinal spasticity, concurs with the observations made by Guttmann (1973) that there is a correlation between these factors and the level of abnormal muscle tone. Edgar's opinion is also based on anecdotal, observational evidence from his own experiences within one of the model spinal injury centres in the United States but he fails to report the number of cases reviewed or discuss the nature of the relationship and proposed causative

physiological mechanisms. The small number and the dates of the papers referenced by Edgar (Jansen 1962, Rushworth 1964 & Guttmann 1973) also indicate that no recent substantive research has been undertaken to objectively demonstrate the relationship between these factors and the level of excess muscle tone. Monitoring of muscle activity may therefore be required over a period of time using patient diaries. The purpose of this assessment package was to establish a 'snapshot view' of the patient at a particular time and to compare the findings of each facet of the assessment at that time. This negates the use of tools such as spasm diaries and focusses the question to be answered by the patient on examination. i.e. rating of their muscle tone at that particular time.

Patients do not complain of spasticity but of three main symptoms of abnormal muscle tone; the strength and frequency of the involuntary muscle spasms and the effect that the spasms have on their ability to carry out their daily activities. Each of these variables were therefore included in the patient rating component of the study. Visual analogue scales have been used successfully in the quantification of pain and are quick and simple to complete (Wall 1984). A numerical rating scale has both these qualities but the immediate availability of a numerical score enables ready comparison with other facets of the assessment package. It was therefore accepted that both the visual analogue scale and a numerical rating scale should be assessed to determine the most reliable measure for inclusion in the main study.

Measurement of the subject's disability and handicap, using recognised and validated measures in routine use on the Spinal Injuries Unit at the Royal National Orthopaedic Hospital, were not considered for this study. The Functional Independence Measure (Granger 1986), used to assess disability, was considered too insensitive to detect subtle changes in muscle tone in the paraplegic patient group, as a substantial change in tone would be required before a change in functional outcome is detected. The Craig Handicap Assessment Reporting Tool [CHART] (Whiteneck et al 1991), a measure of handicap following spinal cord injury, was also considered to be too insensitive and not to have content validity to address the question in this study as the measure examines the social interaction, occupational activity, hours of carer assistance required and financial status of the subjects.

The Pendulum test has been shown to be a reliable method of assessment of muscle tone in the quadriceps and hamstring muscles which is quick and simple to perform within the clinical situation (Bajd 1982) whilst still providing objective measurement. The technique reflects both muscle activity and also any changes in surrounding soft tissues which bear valid comparison with the therapist and patient ratings. Bespoke software rapidly provides a numerical index of spasticity which again enables simple comparison with the other facets of the assessment programme. The simplicity of conducting this objective assessment merited it worthy of inclusion in the study.

The increased availability of dynamometers in physiotherapy departments provides the facility for objective measurement and standardisation of test

parameters (controlled range of movement and velocity of passive movement) in the clinical environment. Previous work by the author (Barr et al 1989), using dynamometry to measure muscle tone in the lower limbs of paraplegics, indicated that the measurement of resistance to passive movement in isolation provides insufficient information regarding muscle activity of the lower limbs. Augmentation of this technique with electromyography, through simple adaptation of the dynamometer hardware, enables exploration of the relationship of muscle activity and resistance to passive movement through quantification of activity in specific muscle groups.

Several studies have limited EMG recording to the muscles being passively stretched (Knutsson 1985). Previous observations by the author (Barr 1989), noted that muscle activity was also present in the muscles below the knee. It was therefore thought necessary to increase the EMG recording to four muscle groups. The selection of the muscles to be assessed with electromyography was based upon the observed patterns of activity during previous assessments with the dynamometer (Barr et al 1989) and their anatomical location. Cocontraction and reflex withdrawal patterns of muscle activity in the ankle dorsiflexors and plantar flexors had been noted during passive flexion and extension of the knee in a group of paraplegic patients. Vastus Medialis and Biceps Femoris were selected as the knee extensor and flexor as they are separated by some distance such that cross talk of the EMG signal between the recording electrodes was less likely (De Luca 1997). The medial belly of Gastrocnemius and Tibialis Anterior were selected to represent muscle activity in the lower leg for this reason also.

EMG activity may be examined using needle or surface recording electrodes. The recording medium chosen for this study was surface electrodes which provide collection of activity from a larger area of the muscle. The purpose of this assessment was to examine gross muscle activity in several muscles in the leg at one time as opposed to activity in individual motor units, for which needle electrodes would be more appropriate (Aminoff 1980). Use of surface electrodes is non-invasive and requires no additional training for the operator, and therefore available to all clinicians.

Many different surface recording electrodes are available on the commercial market for diagnostic and clinical assessment of electromyographic activity. The practical considerations for each type are discussed in Chapter 1.3. Rigid silver-silver chloride EEG recording electrodes and self adhesive ECG recording electrodes are readily available to interface with standard EMG recording equipment at low cost. The more expensive active bipolar recording electrodes required adaptation of the headstage of the electromyograph but the proposed benefits of the active electrode for EMG recording during limb movement were considered significant to merit examination in the reliability studies.

H reflex testing was also considered for inclusion in the study. Whilst the importance of this method is recognised, the aim of the study was to define an assessment package for simple implementation in the clinical environment. The lack of correlation of findings from this method of testing with clinical assessments (Katz 1992) did not support its inclusion in the study.

In order to define the individual protocols for the measurement of abnormal muscle tone which were to be applied in the main study, it was first necessary to examine:-

- Intra-tester reliability for a therapist rating scale
- Reliability of three patient rating scales
- Intra-tester reliability for patient positioning for the Pendulum test and dynamometry protocol.
- Calibration & reliability of an electrogoniometer (Pendulum test)
- Calibration & reliability of a dynamometer
- Calibration & reliability of a 4 channel electromyograph
- Reliability of three surface EMG recording electrodes and skin preparation techniques

2.2 Method

2.2.1. Subjects

A group of spinal cord injured and neurologically intact subjects were selected for the reliability studies using the criteria defined for the main study. These subjects were not included in the main study. The patient group was selected from the Spinal Injuries database at the Royal National Orthopaedic Hospital Trust using the following selection criteria:-

Inclusion Criteria

- 1. Age 18 65 years
- 2. Traumatic spinal cord injury resulting upper motor neurone paralysis
- 3. Minimum of 1 year post spinal injury

- 4. A bilateral passive range of knee movement of 100 degrees present
- 5. Tolerance to supine lying
- 6. Skin tolerance to permit supine lying for period of 90 minutes on firm support surface

Exclusion criteria

- 1. Known musculo-skeletal pathology of the lower limb or lumbar spine
- 2. Pressure sores of the lower limb
- 3. History of recent deep vein thrombosis
- 4. History of recurrent urinary tract infections
- 5. History of autonomic dysreflexia
- 6. Presence of infection

The neurologically intact subjects were recruited to the study using the following criteria:-

Inclusion Criteria

- 1. Age 18 65 years
- 2. A bilateral passive range of knee movement of 100 degrees present
- 3. Tolerance to supine lying

Exclusion Criteria

1. Known musculo-skeletal pathology of the lower limb or lumbar spine

2.2.2 Instruments

Subjective Measures

Therapist Rating Scale

The Modified Ashworth Scale, as shown in Table 2.1 was assessed for intrarater reliability.

Table 2.1: Modified Ashworth Scale

Grade	Description
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch & release or by minimal resistance at the end of range of motion when affected part(s) is moved in flexion or extension.
2	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of movement
3	More marked increase in muscle tone through most of the range of movement, but affected parts easily moved
4	Considerable increase in muscle tone, passive movement difficult
5	Affected part(s) rigid in flexion or extension

Patient Rating Scales

Three scales, a visual analogue scale (Figure 2.1), a marked visual analogue scale (Figure 2.2) and a 6 point numerical rating scale (Table 2.2) were examined for reliability as a patient rating scale.

Figure 2.1: Visual Analogue Scale



Figure 2.2: Marked Visual Analogue Scale



Table 2.2: Numerical Rating Scale

Grade	Strength	Frequency	Functional
			Impairment
0	None	Never	No increase in spasm
1	Very weak	Rarely present	Does not interfere with
			ADL*
2	Weak	Sometimes present	Interferes with fine
			skills
3	Strong	Often present	Impairs ADL*
4	Very strong	Present most of the time	Need assistance with
			ADL*
5	Rigid	Always present	Unable to perform
			ADL*

* ADL Activities of Daily Living

Objective Measures

The Pendulum Test

A battery powered electrogoniometer was examined for reliability in measurement of knee joint angle during passive swing.

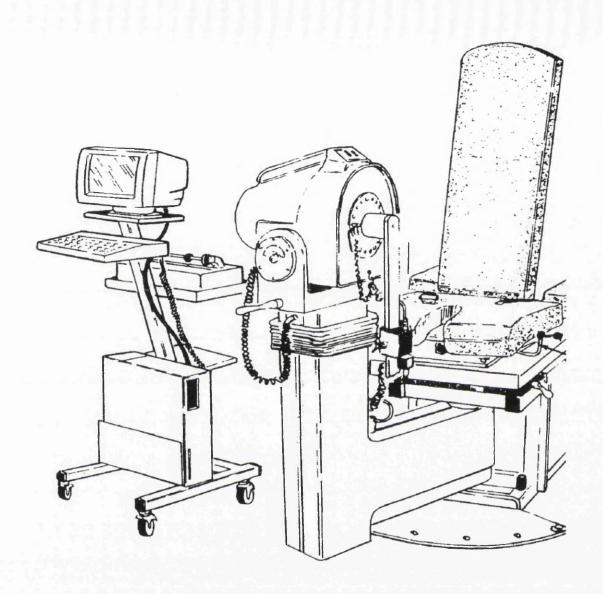
A manual goniometer and spirit level were used to calibrate the electrogoniometer for angle.

Dynamometry

The reliability of a Kin Com 125E dynamometer to produce passive motion of the lower limb at pre-set velocities and to record the force encountered during movement and interval rest periods was assessed. The Kin Com 125E dynamometer is a computer controlled exerciser which may be used for isometric, isotonic (constant force) and isokinetic (constant velocity) exercise. The dynamometer is powered by a pulsed DC servo 1.5 horse power motor. The device consists of an exercise bench with a central tower which is adjustable for height and angle. The exercise arm attaches to the central tower and can rotate through 360 degrees. Housed within the exercise arm is the load cell through which force may be applied in one plane, as shown in Figure 2.3.

The 386 processor personal computer runs a Dos operating system which controls angle, velocity and force parameters. The software has the capacity to store and analyse data, computing torque and work in relation to angle, velocity and force. For the purpose of this study, the Kin Com operating system was used to control the dynamometer only. The analogue signals were amplified within the Kin Com and then sent directly to a high performance data acquisition board (PC226 - Amplicon Liveline Ltd) in a stand alone 466-66MHz processor personal computer (Dell Computer Corporation) for off-line processing, storage and analysis.

Figure 2.3: Kin Com Dynamometer



Polyelectromyograph & Electrodes

A Neurolog system (Digitimer) was examined for reliability to record and process electromyographic activity. The Neurolog system comprised a four channel amplifier (NL824 Headstage - Digitimer, UK), an electrical isolator (NL820 - Digitimer, UK) a filter (NL 135 - Digitimer, UK) which included an optional 50Hz Notch filter and four individual integrators (NL 703 – Digitimer,

UK) which enable recording of the direct EMG signal or an integrated signal. Four bespoke (adapted from the NL100K headstage to provide a gain of 100) miniature low noise, high input impedance differential amplifiers with differential output (NL100EK Headstage - Digitimer, UK) were developed to interface each pair of pre-gelled silver-silver chloride paediatric ECG electrodes (Skintact, UniMed Electrode Supplies) to the Neurolog system. The technical specifications for each component of the polyelectromyograph are detailed in Appendix 2.1.

A buffer interface (input 0 - 5 volts), high performance data acquisition board (PC226 - Amplicon Liveline Ltd) and a 466-66MHz processor personal computer (Dell Computer Corporation) were used to acquire and process the signals received by the Neurolog system.

Bespoke software was written (Kingfisher Software Ltd) to enable real time monitoring of EMG activity during test set up.

3 types of recording electrode in routine clinical use were examined for reliability for recording EMG activity during rapid motion of the lower limb; rigid 40mm silver-silver chloride EEG cup electrodes, MDI - X10 active surface recording electrodes (Neuromuscular Research Foundation, Massachusetts, USA) and pre-gelled silver-silver chloride paediatric ECG electrodes (Skintact, UniMed Electrode Supplies).

2.2.3 Procedure

All test procedures had been approved by the Joint Research & Ethics Committee of the Royal National Orthopaedic Hospital Trust. All subjects were required to read the project information sheet and to give written consent (See Appendix 2.2 & 2.3) prior to participating in the reliability studies.

Subjective Measures

Therapist Rating - Modified Ashworth Scale

The test procedure was explained in full to the subjects prior to commencing. The five subjects were then positioned in supine lying on standard double physiotherapy treatment plinths (Huntleigh Akron) with their head supported on one pillow. Subjects were numbered 1 - 5. Subjects were randomly selected for testing by drawing numbers from a bag. If the same subject was selected on two consecutive occasions, the number was redrawn.

Subjects were asked to close their eyes and relax. The therapist grasped the right leg by the heel and placed her remaining hand under the lower aspect of the right thigh. The Therapist moved the leg at a constant speed through full range of hip and knee flexion and then returned the leg to the extended position on the bed. The therapist rated the resistance from 0 - 5 which was then recorded by an independent observer. This procedure was repeated with the left leg. Each subject was tested three times.

Patient Rating Scale

Subjects were asked to rate the strength and frequency of their spasms and the effect that this had on their ability to perform activities of daily living for that particular day using a Visual Analogue Scale, a marked visual analogue scale and the numerical scale. Each score was recorded on a separate sheet (see Appendix 2.4). This rating was repeated three times with an interval of fifteen minutes between each test. The subject was not given an indication of their previous scores. The subjects were also asked to state their preferred scale.

Objective Measures

Pendulum Test

The axis of rotation of the electrogoniometer was aligned with the axis of rotation of a long-arm goniometer. The arms of the goniometer were attached to the femoral and tibial component of the electrogoniometer. The femoral component of the electrogoniometer was placed on a flat surface in the horizontal position. This was verified with a spirit level. The tibial component of the goniometer was also moved to the horizontal position, verified with the spirit level and a reference angle taken. The tibial component of goniometer was then moved through 140 degrees flexion, pausing at 5 degree intervals to reference the angle. The electrogoniometer was calibrated again at 90 degrees flexion with the spirit level to ensure a true vertical. The goniometer was then restored back to the horizontal position, pausing at 5 degree intervals. This was repeated three times. Signals from the electrogoniometer were passed through a high performance data acquisition board on the PC for storage and offline analysis.

Dynamometry

The manufacturer's calibration procedure was followed to calibrate the dynamometer for angle, velocity and force. Each of these parameters were examined for reliability and repeatability over time. The reliability test period equated to the duration of the test procedure in the main study.

Calibration

To calibrate the Kin Com 125E for angle, the exercise arm was moved to the horizontal position. This was verified by a liquid spirit level placed on the upper edge of the exercise arm. The Kin Com was then calibrated to zero. The arm was then moved downwards to a vertical position, verified by the spirit level and the Kin Com calibrated to 90 degrees. The arm was then returned to the horizontal position and the calibration re-checked to zero.

To calibrate the Kin Com for velocity, the dynamometer's tachometer was set to zero and the speed set to zero. The tachometer was then set to 60 degrees per second and the motor switched on. The servo velocity was then calibrated to the same speed.

To calibrate the Kin Com for force, the exercise arm was locked in the horizontal position. A known 10 kilogramme weight was suspended from the load cell and the Kin Com was then calibrated to 10 kilogrammes.

Reliability

A known 10 kilogramme weight was attached to the load cell of the dynamometer and the force recorded at five minute intervals over a ninety minute period.

The dynamometer was then programmed to move through a range of 90 degrees from the horizontal position, downwards to the vertical position and then to move back up to the horizontal position. The arm stopped at 5 degree intervals for period of 4 seconds before moving through the next 5 degrees. The force and angle were recorded at each stop. This was repeated 5 times.

The reliability of the velocity of the Kin Com was assessed at the four test speeds to be used in the study - 25, 50, 100 and 200 degrees per second. The exercise arm of the dynamometer was moved from the horizontal position, down through 90 degrees to the vertical position and then back up to the horizontal position. This was repeated 5 times at each speed. The velocity analogue signal was taken directly from the Kin Com to the high speed data acquisition board in the stand alone 466 - 66MHz PC for storage and off-line processing.

Intra-Tester Reliability for Patient Positioning

The Pendulum Test and dynamometry protocol required consistent positioning of the patient and setting of joint range. Further to the demonstration of the reliability of the Kin Com to measure angle, the intra-tester reliability for patient positioning was assessed using the Kin Com.

The subjects were placed on the dynamometer in supine lying with their heads supported on one pillow and a firm pillow supporting the lumbar spine. A firm wedge was placed under the thigh of the right leg. A thigh strap was applied to prevent movement of the upper leg during the test. The central pivot of the dynamometer was aligned with the axis of rotation of the knee joint and the subject's lower leg was then strapped to the exercise arm of the dynamometer with the lower edge of the shin pad 10 centimeters proximal to the lower border of lateral condyle of the ankle joint.

The tester then moved the subject's knee joint into extension. The Kin Com angle reading was then recorded by the independent observer. The tester was blind to the Kin Com readings. The tester then returned the knee joint into flexion. This procedure was conducted 3 times. The whole procedure was then repeated with subject's left leg.

Electromyography

Eight tests were conducted to assess the calibration and reliability of the components in the Neurolog polyelectromyograph, using a voltmeter. The settings for the filters, preamplifier, isolator and integrators during the tests are shown in Table 2.3.

Table 2.3: Neurolog Settings:

Component	Setting
High Pass cut off	10 Hz
Low Pass cut off	1000 Hz
Preamp gain	1000
Isolator Gain	1
Integrator Time Constant	100 ms
50Hz notch filter	Out

Tests 1-4 recorded the direct output from the equipment and test 5-8 recorded the output when a standardised electrical output from a signal generator was passed through the Neurolog system. Full details of tests are shown in Table 2.4

Table 2.4: Neurolog Input/Output Measurement

	Input/Output Measurement
Test 1	All inputs grounded, measuring direct output from isolator
2	All inputs grounded, measuring direct output from filters
3	All inputs grounded, measuring direct output from integrators
4	All inputs grounded, measuring integrated output from integrators
5	2mV p-p 100Hz sine (one channel, others grounded) direct output from isolator
6	2mV p-p 100Hz sine (one channel, others grounded) direct output from filters
7	2mV p-p 100Hz sine (one channel, others grounded) direct output from integrators
8	2mV p-p 100Hz sine (one channel, others grounded) integrated output from integrators

Skin Preparation

In order to minimise electrical artefact in the EMG signal it is essential to minimise skin impedance through adequate skin preparation. Three protocols for skin preparation were examined to establish which method achieved the greatest reduction in skin impedance.

Two self adhesive pre-gelled electrodes were attached to the unprepared skin on the antero-lateral aspect of the lower leg. Skin impedance was recorded, using a Teca ZM2 impedance meter by an independent observer. The tester was blind to the recordings. The electrodes were removed, the skin wiped with a tissue moistened with sterile water to remove any residual gel from the electrode site and then allowed to dry. The area was then swabbed with alcohol (methylated spirits) and allowed to dry. Two electrodes were then placed on the area, as previously and skin impedance recorded again. The electrodes were removed and the skin cleansed as before. The area was then shaved of any body hair and wiped with a damp tissue, electrodes applied and skin impedance recorded. The electrodes were again removed and the skin cleansed. Finally the skin was abraded with an abrasive solution (Omniprep, Weaver & Co) and then wiped gently with a moist tissue to remove any debris. The skin was then allowed to dry in the air, prior to reapplication of new electrodes and recording of skin impedance.

Electrodes

Three experiments were conducted with each type of electrode to examine the quality of EMG signal recording from four muscles in the lower limb at rest and during passive leg movement, as shown in Table 2.5. Subjective analysis of the signal quality was performed on the real time display of the EMG activity during each experiment.

Table 2.5: Electrode Tests

Experiment 1	Subject at rest. No limb movement				
Experiment 2	Passive movement of knee from extension through 90°				
	flexion at a constant velocity of 25° per second.				
	5 repetitions.				
Experiment 3	Passive movement of knee from extension through 90°				
	flexion at a constant velocity of 200° per second.				
	5 repetitions.				

The rigid silver-silver chloride electrodes, filled with electrode contact gel and attached with Mefix and Micropore, were assessed first, the MDI - X10 active electrode, attached with Mefix and Micropore second and the self adhesive pre-gelled electrodes and local miniature amplifiers third.

A standardised protocol for skin preparation was used for all tests. The skin was wiped with methylated spirit, shaved of body hair, abraded with Omniprep and wiped with a tissue moistened with sterile water. Electrodes were stuck on to skin over the distal third of the muscle belly of Vastus Medialis, the distal third of the long head of Biceps Femoris, the proximal third of Tibialis Anterior and the proximal third of the medial head of Gastrocnemius.

During the set up for the experiment, the subject remained in high sitting on the dynamometer. When all electrodes were fixed in place, the subject was placed in supine lying with the lumbar spine supported with one pillow. A firm wedge

placed under the test leg to reduce tension on the hip flexor muscles and relieve pressure on Biceps Femoris electrode to prevent artefact in the EMG signal.

The axis of rotation of the knee joint was then aligned with the axis of rotation of the Kin Com lever arm. A thigh strap was applied to prevent displacement of the upper leg during passive movement of the knee. The lower end of the lever arm was attached to the subject with the lower edge of the pad 10 centimeters proximal to the lower border of the lateral condyle of the ankle joint. The foot was then strapped with the ankle joint in the plantar grade position to prevent movement of the foot and subsequent stretching of the Tibialis Anterior and Gastrocnemius muscles. The lower leg of the subject was then moved through 90° range of movement from flexion to extension to ensure correct alignment had been achieved. The EMG signal was observed prior to testing, to ensure a stable baseline and no artefact signal was present.

The subject was then asked to lie still and relax, whilst EMG was observed during experiments 1, 2 and 3 (See Table 2.5).

The subjects were advised to remain in the same position throughout the test but they were free to stop the procedure at any time with the panic button for the Kin Com in the event of any discomfort. The spinal injury subjects were instructed not to try and overcome any muscle spasm that may occur during the test procedure.

Further to the reliability studies on the electrodes, it was necessary to determine that discrete EMG activity could be demonstrated in each of the muscle groups to be tested, without the occurrence of cross talk. The five neurologically intact subjects only were suitable for this test.

Two self adhesive pre-gelled electrodes were stuck on to skin over the distal third of Vastus Medialis, the distal third of the long head of Biceps Femoris, the proximal third of Tibialis Anterior and the proximal third of the medial head of Gastrocnemius. Subjects were asked to contract each muscle individually for a period of five seconds. Subjective analysis was performed on the real time display of the EMG activity during each experiment.

Electrical Safety

To ensure safety of the patient, two primary safety procedures were put in place on every occasion. The power switch for the Neurolog system was switched on before connections were made with the subjects, as per the manufacturer's instructions. Failure to do this may result in some minimal ground flow (a few microamps) through the subject while the electrical supply settles. The Neurolog also contained an electrical isolator (NL820) to provide both signal and power supply isolation from the power ground supply. The NL820 complied with the BS5724 & IEC601-1 patient leakage specifications.

2.3 Results

2.3.1 Subjects

Fifteen spinal cord injured (SCI) and five neurologically intact subjects were selected for the reliability studies. The mean age of the SCI subject group was 38.7 years (range 22 – 60), the mean time post injury was 7.5 years (range 3 - 13) and the modal level of injury was T4 (range T2 - T11 The neurologically intact group comprised two female and three male subjects with a mean age was 45.4 years (range 36 - 65). Full details of all the subjects are shown in Appendix 2.5.

2.3.2 Subjective Measures

Therapist Rating

Five spinal cord injured subjects were selected for the intra-rater reliability testing of the Modified Ashworth scale. All had sustained a complete thoracic cord injury resulting upper motor neurone paralysis. The modal level of injury was T8 and the mean time post injury was 7.8 years. Individual patient scores for right and left leg are shown in Tables 2.6.

Table 2.6: Ashworth Scores - Right & Left Leg

	Patient					
Right Leg	1	2	3	4	5	
Test 1	3	2	4	3	3	
Test 2	3	2	3	3	2	
Test 3	3	2	4	3	3	
Left Leg						
Test 1	2	2	3	2	2	
Test 2	2	2	3	3	2	
Test 3	2	2	3	3	2	

The null hypothesis stated that there was no difference between the repeated ratings with the Modified Ashworth Scale. No significant difference was demonstrated, using a one way analysis of variance and intra-class correlation coefficient (p = 0.557, ICC = 0.71 for testing of the right leg, p = 0.783, ICC = 0.75 for testing of the left leg). The intra-rater reliability was therefore found acceptable for inclusion in the study (p > 0.1).

Patient Rating Scales

The three patient rating scores were examined for reliability using a one way analysis of variance and an intra-class correlation coefficient. No significant difference was demonstrated between the repeated measures with each rating scale (p > 0.1). The p and r values are shown in Tables 2.7 and 2.8.

Table 2.7: P Values

SCALE	SPASM STRENGTH	SPASM FREQUENCY	IMPAIRMENT OF FUNCTION
Visual Analogue	0.883	0.86	0.809
Marked Visual Analogue	0.603	0.55	0.277
Numerical Rating	0.801	0.693	0.851

Table 2.8: R Values

SCALE	SPASM STRENGTH	SPASM FREQUENCY	IMPAIRMENT OF FUNCTION
Visual Analogue	0.9 *	0.85 *	0.55
Marked Visual Analogue	0.83	0.75	0.81
Numerical Rating	0.82	0.71	0.92 *

All scales showed a highly significant degree of repeatability for measurement of strength (Intra-class correlation r > 0.8), a significant degree of repeatability for measurement of frequency (Intra-class correlation r > 0.6) and the marked visual analogue and numerical rating scale showed a highly significant degree of repeatability for measurement of impairment of function strength (Intra-class correlation r > 0.8).

2.3.3. Objective Measures

Pendulum Test

The reliability of the electrogoniometer to measure angle was assessed using a one way analysis of variance. The null hypothesis stated that there was no significant difference between the repeated measurements of set angles on each occasion. P = 0.997 therefore there was no evidence to reject the null hypothesis and the goniometer was shown to be highly reliable.

Dynamometry

A one way analysis of variance was performed on the repeated force, velocity and angle measurements from the Kin Com. The null hypotheses stated that there was no significant difference between the repeated force measurements, the repeated velocity measurements and the repeated angle measurements. No significant difference was demonstrated in force measurements (p = 0.909), velocity measurements (p = 0.998) and angle measurements (p = 0.925) therefore the Kin Com was accepted as a reliable measure of force, velocity and angle.

Intra-tester Reliability for Subject Positioning

Two subjects were selected for this study, one with no neurological deficit and the second with a complete spinal cord injury at the level of T4.

The repeated measures of knee extension for both subjects are shown in Table 2.9

Table 2.9: Intra-tester Measurement of Knee Extension

	Subj	ect 1	Subj	ect 2
	Right Leg	Left Leg	Right Leg	Left Leg
Series 1	162	177	152	165
	161	176	150	163
	162	177	148	164
Series 2	162	177	153	166
	162	178	153	165
	161	176	150	165
Series 3	162	177	150	164
	161	176	148	165
· -	162	177	150	164

A one way analysis of variance was performed on the repeated angle measurements for the two subjects. The null hypothesis stated that there was no significant difference between the measures. No significant difference was demonstrated between the angle measurement for the normal subject (p = 1) or the spinal cord injured subject (p = 0.21). The intra-tester reliability for patient positioning was therefore accepted as highly reliable (p > 0.1).

Electromyography

The expected and actual results of the calibration data for the Neurolog polyelectromyograph are shown in Table 2.10.

Table 2.10: Electromyograph Calibration

	Channel 1		Char	nel 2	Channel 3		Channel 4	
Test	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual
1	0	0 mv DC	0	8 mv DC	0	4 mv DC	0	4 mv DC
		± 20mv	1	± 10mv		± 10mv		± 10mv
		noise		noise		noise		noise
2	0	-8mv DC	0	0 mv DC	0	-8mv DC	0	13mv DC
		± 3mv		± 3mv		± 3mv		± 3mv
		noise		noise		noise		noise
3	0	-8mv DC	0	0 mv DC	0	-8mv DC	0	-14mv DC
		± 2mv		± 2mv		± 2mv		± 2mv
		noise		noise		noise		noise
4	0	16mv DC	0	13mv DC	0	20mv DC	0	25 mv DC
		± 2mv		0mv		0mv		Omv noise
		noise		noise		noise		
5	2	2v	2	2v	2	2v	2	2v
6	2	2v	2	2v	2	2v	2	2v
7	2	2v	2	2v	2	2v	2	2v
8	~ 2	0.6v	~ 2	0.6v	~2	0.6v	~ 2	0.6v

An unacceptable offset was determined in the integrator components of the electromyograph when operating with the integrator function switched on. All other components were found to be within acceptable limits (\pm 20mv).

Skin Preparation

The five neurologically intact subjects were selected to assess the skin preparation protocols. The skin impedance after each test is shown in Table 2.11.

Table 2.11: Skin Impedance with Different Preparation Techniques

	Skin Preparation				
Subject	None	Meths	Shave	Omniprep	
1	20Kohms	20 Kohms	18 Kohms	14 Kohms	
2	20 Kohms	20 Kohms	20 Kohms	12 Kohms	
3	20 Kohms	20 Kohms	20 Kohms	12 Kohms	
4	20 Kohms	20 Kohms	18 Kohms	8 Kohms	
5	20 Kohms	20 Kohms	16 Kohms	8 Kohms	

The null hypothesis stated that there was no significant difference in skin impedance after each of the skin preparation protocols. A highly significant difference was demonstrated between the different protocols using a one way analysis of variance (p<0.001). The greatest reduction in skin impedance was achieved after abrasion with the Omniprep solution.

Electrodes

Five subjects were selected for this test, three spinal cord injured and two neurologically intact.

All of the electrodes produced acceptable 'noise free' signals when the subject was at rest and no movement of the limb was occurring. A clear EMG signal was achieved during isometric muscle contractions in the normal subject

group. In the event of joint movement, the signals produced by both the rigid silver-silver chloride and the MDI - X10 active electrodes were grossly distorted by movement artefact, such that small amplitude EMG signals would not be detected. The pre-gelled silver-silver chloride paediatric ECG electrodes provided a satisfactory, low noise signal which enabled the detection of small amplitude EMG signals.

A summary of the advantages and disadvantages of each electrode type are shown in Table 2.12.

Table 2.12: Electrode Comparison

Electrode	Advantages	Disadvantages
Rigid 40mm silver-silver	Re-usable	Poor electrode contact &
chloride		fixation
		Unscreened cable
		Movement artefact ++
MDI - X10 active	Local amplification	Rigid recording surface
	Screened cable	Poor electrode contact &
	Re-usable	fixation
		DC offset of 10 – 15 µvolts
		Movement artefact ++
Pre-gelled self adhesive	Good electrode contact	Unscreened connector cable
	with flexibility	
	No evidence of	
	movement artefact	

2.4 Discussion

Subjective Measures

Analysis of the repeatability of the patient rating scales presents a paradox. The numerical rating scale has a higher degree of consistency but has a lower reliability coefficient. This is a result of the numerical rating scale being restricted to grading with whole numbers whereas the visual analogue and marked analogue scales were measured in fractions of whole numbers, thus any discrepancies in the numerical rating scale would appear larger.

All patient rating scales showed an acceptable level of repeatability for the grading of strength and frequency of spasm and the numerical rating scale showed the highest consistency. 87% of the subjects preferred the numerical rating scale and reported that it was the easiest to relate their spasms to. The numerical rating scale was therefore selected as the patient rating scale for the main study.

Objective Measures

Electromyograph

The calibration data for the Neurolog polyelectromyograph showed acceptable offsets for the direct output from the isolator, filters and integrators but the integrated output from the integrators was significantly higher (see Table 2.9). It was therefore decided not integrate the signal through the Neurolog system but run the signal direct from the integrators, through the data acquisition card in the PC to process and analyse the signal off line.

In order to minimise motion artefact further, the high pass filter was reset to 30Hz.

Electrodes

The rigid 40mm silver-silver chloride EEG electrodes and the MDI - X10 active surface recording electrodes were tested at an earlier stage in the study. They were found to be entirely unsuitable for use in the main study due to gross movement artefact during passive movement of the leg such that small amplitude EMG signals would not be detected.

The long unscreened cable connector from the rigid silver-silver chloride electrode to the headstage amplifier was sensitive to external noise and caused capacitive coupling to patient, a commonly recognised problem (De Luca 1997).

The poor signal quality achieved by the MDI - X10 active electrodes was disappointing. The concept of local amplification to prevent movement artefact and electrical interference with signal (De Luca 1997) seemed a solution to the problem of movement artefact at the higher velocities. Despite experimentation with fixation of the electrode, it was not possible to improve the signal quality. It was also not possible to utilise any couplant medium with the active electrodes as the inter-electrode space was small and the coupling medium would spread between the electrodes causing disruption of the signal.

Capacitive coupling was also a problem with the unscreened connector from the pre–gelled self-adhesive electrodes. In order to minimise this problem, four miniature bespoke high impedance head stages were designed to provide local amplification of the EMG signals. The leads to connect the electrodes to the amplifiers were unscreened but were only two inches in length. These were then connected to the main Headstage by screened cables to prevent any interference with the signal post amplification. This system proved to be by far the most successful in producing high quality signals.

2.5 Conclusion

Five assessment protocols for the measurement of abnormal muscle tone following spinal cord injury were assessed for reliability.

Therapist Rating Scale

The high intra-tester reliability (p > 0.1) supported use of the Modified Ashworth Scale as the therapist rating scale in the main study.

Patient Rating Scale

The numerical rating scale was shown to have a high level of repeatability (intra-class correlation r > 0.7) and was selected as the patient rating scale for the main study.

Subject Positioning

High intra-tester reliability was demonstrated for positioning of the subjects for the Pendulum test and the dynamometry protocol.

Pendulum Test

A high level of reliability (p = 0.997) was demonstrated for the electrogoniometer to measure angle.

Dynamometry

The Kin Com 125E was demonstrated to be highly reliable in the measurement of force (p = 0.909), velocity (p = 0.998) and angle (p = 0.925).

Electromyography

The Neurolog polyelectromyograph was shown to be reliable in the measurement of EMG activity (±20mv).

The protocol selected for skin preparation to minimise skin impedance was a combination all techniques examined: swabbing the skin with methylated spirit, shaving the area of body hair, abrading the skin with glass solution and wiping the skin with distilled water.

The self adhesive pre-gelled paediatric ECG electrodes with local amplification demonstrated the greatest ability to record EMG activity during rapid passive movement of the lower leg with minimum signal artefact and were therefore selected for the recording medium for the main study.

Chapter 3: Method

Five techniques for the measurement of muscle tone were applied in a group of normal and spinal cord injured subjects. The relationships between the findings of each assessment were examined within and between the subject groups and the suitability of the entire assessment package considered for use in the clinical environment.

3.1. Subjects:

A convenience sample of ten spinal cord injured subjects was sought for the main study. A further ten neurologically intact subjects would then be identified to match the selected cord injured group for age, height and weight.

Patient Group

Patients were selected from the Spinal Injuries database at the Royal National Orthopaedic Hospital Trust using the following selection criteria:-

Inclusion Criteria

- 1) Age 18 70 years
- 2) Traumatic spinal cord injury below the level of C5 resulting in complete upper motor neurone paralysis
- 3) Minimum of 1 year post spinal injury
- 4) A bilateral passive range of knee movement of 100 degrees present
- 5) Tolerance to supine lying
- 6) Skin tolerance to permit supine lying for period of 90 minutes on firm support surface

Exclusion criteria

- 1) Known musculo-skeletal pathology of the lower limb or lumbar spine
- 2) Pressure sores of the lower limb
- 3) History of recent deep vein thrombosis
- 4) History of recurrent urinary tract infections
- 5) History of autonomic dysreflexia
- 6) Presence of infection
- 7) Undergoing active treatment for any medical problem

Normal Group

Subjects were recruited to the study using the following criteria:-

Inclusion Criteria

- 1) Age 18 70 years
- 2) A bilateral passive range of knee movement of 100 degrees present
- 3) Tolerance to supine lying

Exclusion Criteria

1) Known musculo-skeletal pathology of the lower limb or lumbar spine

3.2 Instruments

Subjective Measures

The Modified Ashworth Scale (Wade 1992) was used to record the Therapist rating of lower limb tone.

A five point numerical scale was used to record the SCI subject rating of their own lower limb tone with respect to strength and frequency of spasms and the impact on their ability to conduct activities of daily living as described in Table

Objective Measures

Pendulum Test

A battery powered electrogoniometer was used to record knee angle change during passive swing.

Analogue signals from the electrogoniometer were stored and analysed using a high performance data acquisition board (PC226 - Amplicon Liveline Ltd).

EMG

A Neurolog TM System was assembled to record electromyographic activity from 4 muscle utilising 40mm self adhesive silver-silver chloride paediatric electrocardiograph recording electrodes (Skintact).

Four miniature low noise, high input impedance differential amplifiers with differential output (NL100EK Headstage - Digitimer, UK) were used, one for each pair of recording electrodes.

All four headstages were connected to a four channel amplifier (NL824 Headstage - Digitimer, UK) which was then connected to the electrical isolator (NL820 - Digitimer, UK). The four signals were passed from the isolator through one filter (NL 135 - Digitimer, UK) which included an optional 50Hz

Notch filter and then on to four individual integrators which were set to record the direct EMG signal.

The technical specifications for each component of the polyelectromyograph are detailed in Appendix 2.1.

A high performance data acquisition board (PC226 - Amplicon Liveline Ltd) in a 466 66MHz personal computer (Dell) was used for processing and storing the EMG signals. Bespoke software (Kingfisher Software Ltd) was used to display the EMG activity in real time during the set up for dynamometry assessment.

Dynamometry

A Kin Com 125E was used to produce passive motion of the lower limb at preset velocities through a fixed range of movement in all subjects and to register the force encountered during movement and interval rest periods. The analogue signals were amplified in the Kin Com and then relayed to a high performance data acquisition board (PC226 - Amplicon Liveline Ltd) in a 466-66MHz PC (Dell) for processing and recording.

MathCad software (Mathsoft Inc) was used to analyse the analogue data from the electromyograph and the dynamometer.

SPSS was used to perform statistical analysis on subjective and objective measurement data.

3.3. Procedure

All subjects were interviewed prior to acceptance on to the study and required to read the patient information sheet outlining the purpose of the study and procedures to be undertaken (Appendix 2.2). The subjects were then required to sign the consent form approved by the Joint Research and Ethical Committee of the Royal National Orthopaedic Hospital Trust (Appendix 2.3). Subjects were advised that they were able to withdraw from the study at any time without prejudice to them. All subjects underwent a clinical examination to ensure that all inclusion criteria were fulfilled and no contraindications to proceeding were present and to establish the sensory and motor neurological level in the spinal cord injured group.

All five assessment methods were applied to the patient subject group but only the objective measures (Pendulum Test, dynamometry and electromyography) and one subjective grading (Modified Ashworth scale) were applied in the normal group, Abnormal muscle tone was not present in the normal subject by definition through the selection criteria therefore the subjects rating of this variable is invalid.

The assessments were repeated on three separate days. All assessments were conducted in the same room which was controlled for temperature. Tests were conducted at the same time of day for each subject to account for diurnal variation in patterns of spasticity in the patient group and a note of previous physical activity made. Tests were conducted in the same order on each occasion, as shown in Table 3.1.

Table 3.1: Procedural Order

Test Order	Test Procedure
1	Patient Rating *
2	Modified Ashworth Scale
3	Pendulum Test
4	Electromyography & Dynamometry

^{*} Not applied in normal subject group

3.3.1 Subjective Measures

For each test session, the patient group were required to transfer from their wheel chair onto a standard double physiotherapy treatment plinth (Huntleigh Akron) and to change into a pair of shorts. They were then required to lie in supine with their head supported on one pillow for five minutes. During this time they were asked to rate the strength and frequency of their spasms at that time and the effect that this had on their ability to perform activities of daily living for that particular day. A standardised form was given to each subject on occasion (Appendix 2.4). The subject was not reminded of their scores from previous tests.

The normal subjects were also required to lie in supine with their head supported on one pillow for five minutes, prior to the Ashworth grading procedure. The test was explained to all the subjects on each attendance who were then asked to close their eyes and relax. The therapist grasped the right leg by the heel and placed her remaining hand under the lower aspect of the

right thigh. The therapist moved the leg through range to achieve 100° hip and knee flexion and then returned the leg to the extended position on the bed. The therapist then rated the resistance to movement using the Modified Ashworth Scale and recorded it in the subject's notes. On subsequent occasions, the therapist did not view the subject's score from the previous assessment.

The spinal injury subjects then transferred back into their wheelchair to move to the Kin Com for the remainder of the tests. During the set up procedure for Pendulum test and the dynamometry, the subject was allowed to sit with both hip and knee joints resting at 90°.

3.3.2 Objective Measures

Pendulum Test

The subject was positioned on the Kin Com in supine lying with a pillow supporting the lumbar spine and both legs hanging freely. The electrogoniometer rig was placed under the subject's right thigh. The tibial component of the electrogoniometer was then fastened to the subject's lower leg 10 centimeters proximal to the tip of the lateral malleolus. The researcher extended the knee and flexed it to ensure that the axis of rotation of the potentiometer was aligned with the axis of rotation of knee (to prevent any torsion occurring on the apparatus during the test) and to ensure that the leg could fall freely without contacting the dynamometer at any point. The researcher then raised the right lower leg to the horizontal (knee in extension), asked the subject to relax and waited to for any muscle activity to cease. The

leg was then released and allowed to swing freely. Analogue signals from the electrogoniometer relating to angle and velocity were sent directly to the PC for recording and analysis. This procedure was repeated three times. The rig was then transferred to the left leg and the procedure repeated exactly.

A relaxation index was calculated for each test, ie. three indices for each leg on each test occasion, using the equation derived by Bajd & Vodovnic (1984).

where starting angle is full extension, the first acute angle is the flexion angle in the first swing and the final angle when the leg ceases to swing.

Dynamometry and Electromyography

The subjects remained on the Kin Com but were restored to the sitting position during the set up for the dynamometry testing.

Prior to attachment of the electrodes, the subject's skin was prepared to minimise impedance. The area was cleaned with methylated spirit to remove any oil, shaved to remove any body hair, abraded with glass solution (Omniprep) to remove any dry skin and finally wiped with a tissue moistened with sterile water. When the skin was dry, one pair of paediatric ECG recording electrodes was placed over the four muscles to be tested in the positions shown in Table 3.2.

Table 3.2: Electrode Positions

Muscle	Electrode Position		
	Recording	Reference	
Vastus Medialis	Distal third of muscle belly	Head of fibula	
Biceps Femoris	Distal third of belly of lateral head	Tibial tuberosity	
Tibialis Anterior	Proximal third of muscle belly	Medial tibial condyle	
Gastrocnemius	Proximal third of belly of medial head	Lateral tibial condyle	

Cross referenced surface marking of the electrode positions was recorded for each subject to ensure accurate placement on subsequent tests. Inter-electrode distance was standardised for all subjects to 10 centimeters.

Electrodes were attached in a fixed order, staring from Vastus Medialis, Tibialis Anterior, Biceps Femoris and finally Gastrocnemius. One reference electrode was placed over a superficial bony prominence for each pair of recording electrodes. When all electrodes had been placed, the skin impedance was checked to ensure adequate contact and recorded in the subjects notes. The electrodes were then connected to the NL100EK Headstages which were attached firmly to the skin using Mefix adhesive strapping. The NL100EK's were then attached to the recording system via the NL824 Headstage which was placed on the patient. All connector cables were taped to the patient to

prevent movement or torsion during the test which could lead to motion artefact.

The subject was then placed in a supine position with a pillow supporting the lumbar spine and a firm wedge placed under the right thigh to prevent pressure on the Biceps recording electrode.

The axis of rotation of the knee joint was then aligned with the axis of rotation of the Kin Com lever arm. The lower end of the lever arm was attached to the subject with the lower edge of the pad 10 centimeters proximal to the lower border of the lateral condyle of the ankle joint. The foot was then strapped with the ankle joint in the plantar grade position to prevent movement of the foot and subsequent stretching of the Tibialis Anterior and Gastrocnemius muscles. The lower leg of the subject was then moved through 90 * range of movement from flexion to extension to ensure correct alignment had been achieved.

The EMG signal was observed at rest and during the movement to check alignment of the knee joint to ensure that a stable baseline and no artefact signal were present.

The subjects were advised to remain in the same position throughout the test but they were free to stop the procedure at any time with the panic button for the Kin Com in the event of any discomfort. The spinal injury subjects were instructed not to try and overcome any muscle spasm that may occur during the test procedure.

Six tests were performed on each leg. In Test 1, EMG recordings were made of muscle activity with the subject at rest with the knee positioned in full extension. In the second test, the subject's leg was then moved from full extension through to 90° knee flexion at a speed of 25° per second, held at 90° for 4 seconds and then returned to extension at a speed of 25° per second where it was held for a further 4 seconds. This manoeuvre was repeated five times. The third fourth and fifth test followed the same procedure but the limb was moved at 50, 100 and 200° per second respectively. The sixth test was a repeat of Test 1.

A bespoke programme was created using Mathcad 7 (see Appendix 3.1) to compile the data from the four EMG channels, torque and velocity signals. Each test comprised 5 complete movements, each of which were divided into four phases; pause; movement into flexion; pause: movement into extension. The EMG signals for the four muscles were rectified and averaged within each phase of the passive movement.

Data Analysis

Descriptive statistics were applied to determine the subject demographics.

Data from the therapist (Modified Ashworth Scale) and patient self report assessment (Numerical rating scale) and the electrogoniometer were ordinal level and therefore non-parametric statistical analyses were applied.

Parametric statistical analyses were considered to examine the ratio level data from the electromyograph and the dynamometer.

Chapter 4: Results

4.1. Subjects

Twenty subjects fulfilling the selection criteria were recruited to the study, ten with spinal cord injury (SCI) and ten neurologically intact subjects, matched for sex, age, height and weight.

Each subject group contained 2 females and eight males. The mean age of the SCI group was 42 years (range 24 - 69) and the mean age of the normal group was 39 years (range 29 - 60). The mean height and weight for the SCI group were 1.79 metres (range 1.63 - 1.9m) and 75.6 kilogrammes (range 66.1 - 100.8kgs) respectively. The mean height and weight for the normal group were 1.73 (range 1.63 - 1.83) and 80.5 kilogrammes (range 63 - 107.1kgs). The mean time post injury in the SCI group was 11 years (range 6 - 15 years) and the modal levels of injury were T5 and T6 (range T3 to T12). Full details of all subjects are shown in Appendix 4.1. and 4.2.

The null hypotheses stated that there was no difference between the variances of the two groups for age, height and weight. Levene's test for equality of variances indicated that there was no evidence to reject the null hypothesis (p > 0.05) and therefore equal variances were assumed for age, weight and height. Each variable was tested in turn, using a two sample Student T test. No significant differences were demonstrated between the age, height and weight of the two groups for age, height and weight respectively. The mean difference between the age of the groups was 3.6 years (95% confidence interval -6.9, 14.1, p = 0.481). The mean difference between the height of the two groups

was 5.5 cms (95% confidence interval -2.91, +13.91, p = 0.15). The mean difference in weight of the two groups was 4.85 kgs (95% confidence interval -16.57, +6.87, p = 0.39).

4.2. Subjective Measures

4.2.1. Therapist Rating

The Therapist rating for all the neurologically intact subjects on every occasion was 0 on the Modified Ashworth scale i.e. no resistance. The raw scores for the spinal cord injury subjects are shown in Appendix 4.3. The difference between the Ashworth scores of the SCI subjects and the neurologically intact subjects for both right and left leg on every occasion was examined using a Mann-Whitney 'U' test. A highly significant difference was found for each leg (p < 0.0001).

The relationship between the Therapist rating (Ashworth score) of the right and left leg of the spinal cord injured subjects on each test occasion was examined using the Spearman's rho correlation coefficient. A significant correlation was seen on the first occasion (r = 0.761, p<0.005) and a highly significant correlation was demonstrated on the second (r = 0.825, p<0.001) and third occasion (r = 0.829, p<0.001).

The Therapist rating (Modified Ashworth Score) for the right and left legs on each of the three test occasions were also examined separately, using a one way ANOVA. No significant difference was demonstrated between test occasions for the right (p = 0.471) or left leg (p = 0.429).

4.2.2. Patient Rating

The patient ratings for spasm strength, frequency and impairment of function on each of the three test occasions were examined for change using a one way ANOVA. No significant change was demonstrated in strength (p = 0.664) and frequency of spasms (p = 0.856) or the impact on functional ability (p = 0.444). Raw scores are shown in Appendix 4.4.

The relationships between the patient numerical rating of spasm strength, frequency and impairment of function on each test occasion were examined using the Spearman's rho correlation coefficient. A significant correlation was demonstrated between the patient rating of strength and frequency of spasms on the first occasion only (p < 0.05). No correlation was demonstrated between the strength and frequency of spasm and impairment of function. No correlation was demonstrated between any of the variables on the second and third occasion, as seen in Table 4.1.

Table 4.1: Correlation Coefficients (r) of Patient Ratings

	Strength	Frequency	Function
Test 1			
Strength	-	0.743 *	-0.261
Frequency	0.743 *	-	0.065
Function	-0.261	0.065	-
Test 2			
Strength	-	0.497	0.215
Frequency	0.497	-	0.315
Function	0.215	0.315	-
Test 3			
Strength	-	0.508	0.13
Frequency	0.508	-	0.466
Function	0.13	0.466	-

* Significant Correlation p < 0.05

The relationship between the Therapist rating (Modified Ashworth score) of each leg and the patient numerical rating of spasm strength, frequency and impairment of function on each test occasion was examined using the Spearman's rho correlation coefficient. No significant correlation was demonstrated on any of the three test occasion (r < 0.65), as seen in Table 4.2.

Table 4.2: Correlation Coefficients (r) for Ashworth Score With Patient ratings

	Right Ashworth	Left Ashworth
	Score	Score
Session 1		
Strength	0.379	0.007
Frequency	0.29	0.109
Function	0.041	0.324
Session 2		
Strength	0.148	-0.086
Frequency	-0.183	-0.245
Function	0.311	0.602
Session 3		
Strength	0.195	0.003
Frequency	-0.157	-0.323
Function	-0.318	-0.049

4.3. Objective Measures

4.3.1. Pendulum Test

Three Relaxation Indices were derived for each subject on each test occasion.

The scores for the SCI and normal group are shown in Appendix 4.5.

The difference between the Relaxation Index within each test for each subject group was examined using a one way analysis of variance for both left and right leg. The null hypotheses stated that there was no significant difference between the Relaxation Index within each test occasion. No significant difference was demonstrated (p > 0.1) with one exception, as shown in Table 4.3.

Table 4.3: Pendulum Test Intra-test Repeatability – p values

	Right Leg	Left Leg
SCI subjects		·
Test 1	0.75	0.88
Test 2	0.99	0.05*
Test 3	0.14	0.16
Normals		
Test 1	0.87	0.31
Test 2	0.18	0.37
Test 3	0.39	0.88

^{*} significant difference p = 0.05

The differences between the Relaxation Index of each test session for each leg in both subject groups was examined using a two way ANOVA with replication. The null hypotheses stated that there was no significant difference between the Relaxation Index of each test session. No significant difference was demonstrated between the test occasions in either subject group (SCI subjects p = 0.37, normals p = 0.54).

The difference between the Relaxation Indices for the right and left legs in each subject group was examined using a Mann-Whitney 'U' test. The null hypotheses stated that there was no significant difference between right and left leg in either subject group. A highly significant difference (p<0.001) was found between left and right legs in both subject groups.

The difference between the Relaxation Index for each leg of the spinal cord injury subjects and the subjects with no neurological deficit was examined in turn using a Mann-Whitney U test. The null hypotheses stated that there was no significant difference between the Relaxation Index of the spinal cord injury subjects and the subjects with no neurological deficit. A highly significant difference (p<0.001) was found between the subject groups for both left and right legs.

The relationship between the Relaxation Indices and the Modified Ashworth score in the spinal cord injured subjects was examined using a Spearman's rho correlation coefficient on each test occasion. A negative relationship was noted but no significant correlation was demonstrated on the first two test sessions (r<-0.5) but a weak correlation (r = -0.573) was noted in both the left and right leg on the third test occasion. The correlation coefficients are shown in Table 4.4.

Table 4.4: Correlation Coefficients Between Modified Ashworth Score & Relaxation Index (r)

	Test 1	Test 2	Test 3
Right leg	-0.488	-0.222	-0.573
Left Leg	-0.105	-0.097	-0.48

The relationship between the Relaxation Indices of each leg and the patient rating scores for strength and frequency of spasm and impairment of function were examined in the spinal cord injured group, using Spearman's rho correlation coefficient. No correlation was demonstrated on each of the three occasions with strength and frequency of spasm but a positive correlation ($r = \frac{1}{2}$)

0.621) between the right leg Relaxation Index and the patient rating of impairment of function was found on the second test occasion. A further correlation (r = 0.689) was found between the left leg Relaxation Index and the patient rating of impairment of function on the third test occasion. The correlation coefficients for each test are shown in Table 4.5.

Table 4.5: Correlation Coefficients Between Patient Rating Scores & Relaxation Indices

Strength	Frequency	Function
0.112	0.57	0.112
0.462	0.316	0.621*
0.509	0.374	0.039
0.177	0.049	0.21
0.278	0.054	0.178
0.115	0.527	0.689*
	0.112 0.462 0.509 0.177 0.278	0.112 0.57 0.462 0.316 0.509 0.374 0.177 0.049 0.278 0.054

* Correlation

Electromyography & Dynamometry

The purpose of the study was to examine the repeatability of the recording of electromyographic activity and resistance during passive movement of the lower limbs in a group of spinal cord injured and neurologically intact subjects. Any alteration in skin impedance during the test period may influence the comparison of EMG data. The skin impedance pre and post test was therefore examined for change. The raw scores are shown in Appendix 4.6. and 4.7.

The difference in skin impedance within and between groups was first examined for a normal distribution using a one sample Kolmogorov-Smirnov test. The null hypothesis stated that the data did not differ from a normal population. Evidence was found to reject the null hypothesis in twelve of the twenty four tests (p<0.05). A significant difference from the normal distribution was therefore demonstrated, indicating that non-parametric statistics were to be used.

The difference in skin impedance within and between groups was therefore examined using a Mann-Whitney 'U' test. The null hypothesis stated that there was no significant difference in skin impedance pre and post test on each test occasion. No significant difference was demonstrated (p > 0.01) at all four muscle recording sites in both subject groups, with the exception of the right Tibialis Anterior (p = 0.028) and the left Tibilalis Anterior (p = 0.018) on the first test occasion. The p values are shown in Tables 4.6 and 4.7.

Table 4.6: Change in Skin Impedance - P values

Muscle	Vast.Med.	Biceps Fem.	Tib.Ant.	Gastroc.
Test 1	0.889	0.534	0.028*	0.067
Test 2	0.28	0.33	0.503	0.914
Test 3	0.031	0.503	0.177	0.03

^{*} Significant p < 0.05

Table 4.7: Change in Skin Impedance - P values

Left Leg

Right Leg

Muscle	Vast.Med.	Biceps Fem.	Tib.Ant.	Gastroc.
Test 1	0.147	0.088	0.018*	0.147
Test 2	0.317	0.542	0.051	0.051
Test 3	0.229	0.317	0.503	0.957

^{*} Significant p < 0.05

The change in pre and post test skin impedance was also examined within the two subject groups. No significant difference was demonstrated (p> 0.05).

4.3.2. Electromyography

The EMG data were analysed using a bespoke programme written using MathCad 7 software (see Appendix 3.1). The EMG signal was rectified and averaged, as recommended by De Luca (1997). To normalise the data to each test, the difference from baseline to peak of the EMG signal was calculated.

The EMG records for each muscle were considered individually and within subject groups. For the purpose of analysis, the EMG data from each test were divided as follows:- Six tests were performed on each leg at five velocities.

Test 1	0°/second
Test 2	25°/second
Test3	50°/second
Test 4	100°/second
Test 5	200°/second
Test 6	0°/second

Each test contained five repetitions of movement of the knee from extension to flexion and from flexion to extension. Each full cycle was divided into four phases:-

Phase 1	Pause
Phase 2	Movement - Extension to flexion
Phase 3	Pause
Phase 4	Movement - Flexion to extension

Each test series was performed on three separate occasions.

The difference in average EMG activity for each muscle group in each of the subject groups within each of the three test occasions was examined in turn using a two way ANOVA with replication. The null hypothesis stated that there was no significant difference between the EMG activity in each muscle within each of the test occasions. No significant difference was found between all muscles tested in the normal group (p > 0.05). A significant difference

(p < 0.05) was found in Vastus Medialis recordings from the spinal cord injured group. The p values are shown in Table 4.8.

Table 4.8: Intra Test Repeatability-Average EMG Activity – P Values

Muscle Group	SCI Subjects	Normals
Vastus Medialis	0.04*	0.06
Biceps Femoris	0.43	0.26
Tibialis Anterior	0.26	0.69
Gastrocnemius	0.11	0.55

* Significant p < 0.05

The difference in average EMG activity for each muscle group in each of the subject groups on each of the three test occasions was examined in turn using a two way ANOVA with replication. The null hypothesis stated that there was no significant difference between the EMG activity in each muscle on each of the test occasions. No significant difference was found between all muscles tested in the normal group (p > 0.05). A significant difference (p < 0.05) was found in Vastus Medialis and Biceps Femoris recordings from the spinal cord injured group. The p values are shown in Table 4.9.

Table 4.9: Inter Test Repeatability-Average EMG Activity – P Values

SCI Subjects	Normals
0.015*	0.18
0.046*	0.06
0.47	0.59
0.47	0.6
	0.015* 0.046* 0.47

^{*} Significant p < 0.05

The relationship between the average EMG activity in each muscle during the upward and downward movement phases was examined using a two way ANOVA without replication. The null hypothesis stated that there was no significant difference between the average EMG activity in the four muscles. A highly significant difference was seen between the level of activity in all of the muscles (p<0.001) in both directions of movement.

The relationship between velocity and EMG activity during the upward and downward movement phases was examined in each muscle, for each leg, within each test occasion, within each subject group in turn using a two way ANOVA without replication. The null hypotheses stated that there was no difference in EMG activity at the different speeds and that there was no significant difference between repetitions. A highly significant difference in the EMG activity of all muscles between speeds was found in both groups (p<0.001). A significant difference between repetitions was found in the Vastus Medialis EMG recordings during the downward direction of movement in both the spinal cord injured group (p = 0.0006) and the neurologically intact

subjects (p = 0.0002). No significant differences were found between repetitions in the other muscles tested in either subject group (p>0.05) in either direction.

The correlation between the levels of EMG activity during the first and last test (i.e. with the leg held at rest) were examined for each leg in both subject groups. The results are shown in Table 4.10.

Table 4.10: Correlation Coefficients between EMG signal from Test 1 & Test 6

0.01**	
0.01**	
0.91**	0.55
0.49	0.45
0.71*	0.98**
0.72*	0.85**
0.68*	0.91**
0.95**	0.21
0.69*	0.21
0.93**	0.12
	0.71* 0.72* 0.68* 0.95** 0.69*

^{*} Correlation

^{* *} Strong Correlation

The relationship between the EMG and torque data for each muscle, in each phase of the test - pause with no movement, passive movement from knee extension to flexion, pause with no movement and passive movement from flexion to extension - at every velocity is plotted for all the subjects on the graphs shown in Appendices 4.8 - 4.22. No correlations were demonstrated within either group but trends were observed. A summary is described below.

Summary

The scatter plots clearly distinguish between the two subject groups and the difference between the EMG activity in each muscle during each phase of the movement, as described above.

The plots also indicate the spread of the data, representing the small number of spinal cord injured subjects who encountered involuntary spasms at rest and throughout the movement periods.

Phase 1 – At rest - No Movement

An increase in torque without a corresponding increase in EMG activity was observed in the Vastus Medialis, Biceps Femoris and Gastrocnemius muscles in both subject groups.

Phase 2 – Passive Movement from Knee Extension to Flexion

An increase in torque was seen with a corresponding increase in Vastus Medialis EMG activity in the patient group only at the higher velocities (100 & 200°/second).

An increase in EMG activity was noted in Tibialis Anterior at all test speeds.

Phase 3 - At rest - No Movement

Minimal EMG activity was noted in the Biceps Femoris at all test speeds except 200°/second.

An increase in EMG activity was noted in Tibialis Anterior at all test speeds.

Phase 4 - Passive Movement from Knee Flexion to Extension

Minimal EMG activity was noted in the Vastus Medialis at all test speeds except 200°/second.

An increase in EMG activity was noted in Tibialis Anterior at all test speeds.

4.3.3. Dynamometry

The relationship between velocity and torque during the upward and downward movement phases was examined in each muscle, for each leg, within each test occasion, within each subject group in turn using a two way ANOVA without replication. The null hypothesis stated that there was no difference in level of torque at the different speeds and that there was no significant difference between repetitions. A highly significant difference between speeds was demonstrated in both subject groups in all muscles (p < 0.001). No significant difference was found between the repetitions at each speed during the upward and downward movement phase for the right leg of both the normal subjects (p = 0.58, p = 0.81 respectively) and spinal cord injured subjects (p = 0.14, p=0.31). No significant difference was found between the repetitions at each

speed during the downward movement of the left leg in the normal subjects (p = 0.75) and in the spinal cord injured subjects (p = 0.1). A significant difference was, however, found between the repetitions at each speed during the upward movement phase for the left leg of the normal subjects (p = 0.016) and highly significant difference was found in the spinal cord injured subjects (p = 0.002).

The relationship between torque and the Relaxation Indices of each leg was examined using Spearman's rho correlation coefficient. No correlation was demonstrated. R values for both legs are shown in Table 4.11.

 Table 4.11: Correlation Coefficients Between Torque & Relaxation Indices

	Test 1	Test 2	Test 3
Right Leg	0.231	0.263	0.012
Left Leg	0.135	0.268	0.316

The relationship between the torque and Modified Ashworth score for each leg was examined in the spinal cord injured group, using Spearman's rho correlation coefficient. A correlation was demonstrated between the Ashworth scores of the right leg on the first (r = 0.676) and third test (r = 0.642) occasion and a strong correlation was seen on the second test (0.719). A correlation was also seen in the left leg on the second test occasion. R values are shown in Table 4.12.

Table 4.12: Correlation Coefficients Between Torque and Modified Ashworth Score

	Test 1	Test 2	Test 3
Right Leg	0.676**	0.719**	0.642**
Left Leg	0.242	0.654*	0.095

* Correlation

** Strong correlation

The relationship between the torque generated by each leg and patient rating scores for strength and frequency of spasm and impairment of function on each test occasion was examined in the spinal cord injured group, using Spearman's rho correlation coefficient. No correlation between torque and spasm strength was seen in either leg on each of the test occasions. The r values are shown in Table 4.13.

Table 4.13: Correlation Coefficients Between Torque and Patient Rating of Spasm Strength

	Test 1	Test 2	Test 3
Right Leg	0.374	0.395	0.175
Left Leg	0.186	0.271	0.121

No correlation between torque and spasm frequency was seen in either leg on each of the test occasions. The r values are shown in Table 4.14.

Table 4.14: Correlation Coefficients Between Torque and Patient Rating of Spasm Frequency

	Test 1	Test 2	Test 3
Right Leg	0.118	0.022	0.191
Left Leg	0.477	0.178	0.266

A weak correlation between torque and impairment of function was seen in the right leg on the first and third test occasion and in the left leg on the second test occasion. The r values are shown in Table 4.15.

Table 4.15: Correlation Coefficients Between Torque and Patient Rating of Impairment of Function

	Test 1	Test 2	Test 3
Right Leg	0.572	0.279	0.529
Left Leg	-0.222	0.538	0.371

Chapter 5: Discussion

The purpose of this study was to devise and evaluate a comprehensive assessment programme for the measurement of abnormal muscle tone and to consider it's application in the clinical environment. This chapter will discuss the methods employed in the study, review the results in relation to other related work and provide comment on the compatibility of the assessment with the clinical environment.

5.1. Method

Subjects

Many studies have examined the presence and characteristics of abnormal muscle tone in a variety of patient populations, often combining patients with different pathologies in one study (Knutsson 1985, Katrak 1992, Seib 1994). Although the subject numbers in this study were small, it was considered a homogenous group. The neurologically intact control group were closely matched anthropometrically and all the spinal injury subjects had thoracic cord lesions without any other complications. The inclusion of tetraplegic subjects in the study was rejected as the difference in incidence, severity and patterns of spasticity have been noted to be different between thoracic and cervical injuries (Stover 1992). As discussed in Chapter 1, the incidence of spasticity is also more marked in patients with incomplete injuries and patterns of spasticity more varied, hence the exclusion of this group from the study.

The spinal cord injury subjects were recruited to the study using strict selection criteria with respect to the level and severity of their impairment. All subjects

had sustained a thoracic injury to the cord resulting in complete motor and sensory loss. This was established through a clinical examination only. Only by further assessment of sensory and motor evoked potentials could this diagnosis be confirmed. This would provide additional invaluable information which could be examined in light of the electromyography and dynamometry assessment data.

Instruments

The problems with the recording medium and data analysis for the electromyography were not anticipated at the start of the project. The concept of local amplification in the active electrode (Delsys) and the simplicity of application made it very attractive for use in the clinical environment. The electromyograph was adapted to accommodate the four active electrodes directly into the headstage, reducing the set up time of the assessment to approximately five minutes. This was in stark contrast to the 20 minutes taken to set up the EMG recording system using the self adhesive electrodes which were eventually used in the study. Despite the time taken to set up for the test, the quality of the data obtained from the latter equipment was far superior to that of the active electrodes. Development of the miniature amplifiers, using the concept of local amplification from the active electrode, to prevent signal disruption through movement and prevention of capacitive coupling, served to minimse the substantial artefact from the rapid velocity, large amplitude movements during the dynamometry testing.

The importance of reducing skin impedance was noted. The skin preparation technique selected was not however popular with subjects, and some complained of skin irritation for 24 hours after the test. This did not present a major problem for the patients but irritation of the skin is known to act as a noxious stimulus (Guttmann 1973) which in turn may affect the level of abnormal muscle activity and should be taken into consideration when performing repeated testing.

The data collection and analysis programmes were written to meet the specific requirements of the project. Three different programmes were used in the course of the study. The first displayed the EMG and torque recordings in real-time to establish a smooth baseline. A second programme was then implemented to record the EMG and torque and velocity signals. The data were then transferred to the third programme for off-line processing and analysis. This was a long and tedious task and offered several opportunities for data to be corrupted in transit. The combination and redevelopment of the programmes to enable speedy online analysis within one programme and one PC would greatly enhance the acceptability of the technique for the clinical environment.

Procedure

The researcher advocated the use of bilateral testing in the protocol, in contrast to some previous studies (Lamontagne 1998). The significant difference demonstrated between legs in both subjects groups with each of the tests indicates that the assessment protocol should include bilateral limb testing.

Review of the Pendulum test data shows that the Relaxation indices of the left leg were significantly higher in both groups i.e the left leg was more relaxed. This may be due to the fact that the right leg was always tested first and therefore a learning effect may have occurred. This could certainly be the case for the neurologically intact subjects who have voluntary control over the muscles of the lower limb but conscious learning is obviously not possible for the complete spinal cord injured subjects. A repeat of this procedure, randomising the order in which the legs are tested would examine this phenomenon further.

The purpose of this study was not to perform in-depth analysis on the configuration of the EMG signal, as the nature of the neurophysiological pathology was not the object of the research question. Quantification of EMG with surface recording electrodes does present methodological problems, as discussed in Chapter 1.3. Campbell et al (1990) utilised surface EMG to examine spastic activity during passive motion in a group of spinal cord injured patients. Unable to perform quantification, her outcome measure was the presence or lack of EMG activity. LaMontagne (1998) used simple rigid electrodes to compare EMG activity during rapid velocity movements with an isokinetic dynamometer and hand held dynamometer. The electrodes were able to detect large amplitude signals but direct comparison between the level and nature of the EMG activity between the two measurement tools was not distinguishable.

The dynamometry testing was performed at four speeds. This was not in keeping with other work, where two or three test speeds were used (Campbell 1990, Bohannon 1987, Knutsson 1985). Previous authors have selected a low velocity (30°/second) and a high velocity (180 - 240°/second) to examine both the tonic and phasic capacity of the muscle. Knutsson (1985) noted that the velocity threshold in voluntary movements was variable and therefore he advocated testing at normal joint velocities. This would exclude the very high speeds that Bohannon (1987) was testing at (360°/second). A reduction in the number of test speeds would reduce the time taken to complete the assessment package. However, the data showed a difference at each of the test speeds such that the researcher would advocate the continuation of the protocol as described ie. four test speeds.

The relationship between the levels of EMG activity and velocity of movement have been noted by several authors. Various speeds of movement have been applied through the use of dynamometers in different patient groups. Sköld et al (1998) also noted a relationship with the direction of movement in addition to the speed, noting more EMG activity in movement to extension than flexion. This was not the experience of this study, wherein increased EMG activity was noted predominantly during movement into flexion. The findings in this study show quite clearly that there is a relationship between velocity and both EMG activity and resistance to passive movement, but an increase in either of these variables may occur without a corresponding increase being demonstrated in the other.

The number of repetitions of movement within each test also varies between studies. Perrell (1996) repeated movements 10-12 times but then selected the middle 5 for data analysis. This may reject valuable information regarding the initial reaction of muscle to movement which may change. Campbell et al (1990) also disregarded the first and last part of their recordings but presented no evidence for doing so. The results from this study showed no significant difference between repetitions in the neurologically intact group but a significant difference was observed between repetitions in the cord injured group, such that the author will continue to analyse all of the data recorded.

The test position for the dynamometer has varied also. Perrel (1996) tested in high sitting but rightly noted that with knee in extension, a substantial stretch was applied to the hamstrings. This was the justification for performing the dynamometry testing and Pendulum testing in this study with the subject positioned in supine lying, with the hip in slight flexion to relieve tension on the hamstrings that may elicit reflex activity.

Bohannon (1985) used a dynamometer to perform the Pendulum Test at a fixed velocity in thirty patients with hemiplegia. He noted the deceleration effect as compared to the free swinging goniometer which may affect the muscle response at the change of direction. The Kin Com used in this study had the facility to 'soften' the turns by means of deceleration but this facility was deactivated for the testing.

5.2. Results

Subjective Measures

The level of intra-rater reliability demonstrated for the Modified Ashworth Scale was in keeping with the work of others (Bohannon & Smith 1987). It is interesting to note however that several of the papers evaluating other measures of spasticity against the Ashworth Scale (Katz et al 1992, Priebe 1996, Skold 1998) have failed to examine the issue of reliability, which perhaps is a reflection of the common acceptance of the Ashworth Scale within the clinical environment.

The limitations of the Modified Ashworth scale were recognised in the study in that the researcher detected a difference in resistance to passive movement between the subject's right and left leg, but the difference was not sufficient to rate a higher score on the Ashworth scale. The therapist rating of inter-leg difference also concurred with the subject. This was, however, an anecdotal observation during the study and was not evaluated in a formal manner. This may be given further consideration for inclusion in the data set at a later stage.

The lack of correlation between the assessment findings was surprising. Katz et al (1992) had demonstrated a correlation between the Modified Ashworth and the Fugel-Meyer motor assessment score. Katz also demonstrated a correlation between the level of EMG activity and Ashworth score. The Ashworth scoring and the electromyography were not performed simultaneously, but the Ashworth score was performed at predetermined times during the day. It is not reported how the timings were planned and what factors were taken into

account. This may have a bearing on the level of muscle tone. The assessments in this study were conducted at a set time of day, but also took in to consideration, the patient's bladder and bowel regime and menstrual cycle to ensure that comparability was maximised.

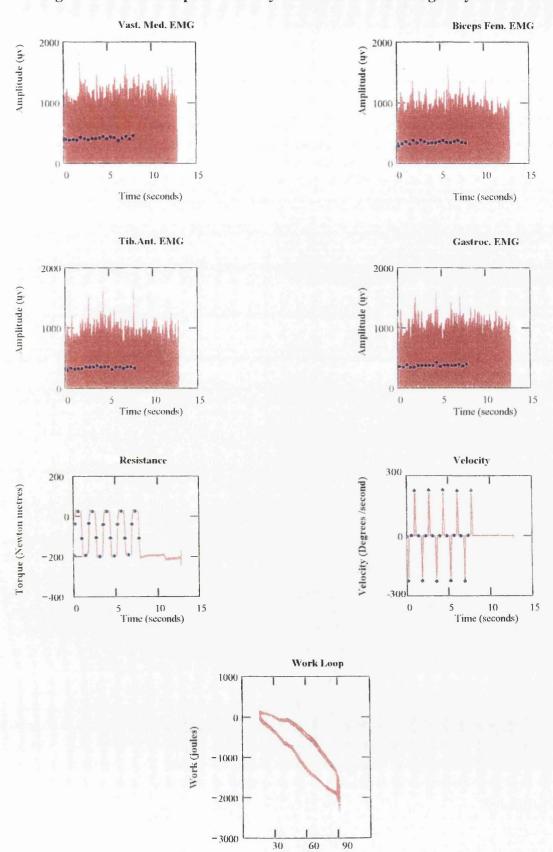
The lack of correlation between the individual patient rating scores does not reduce the importance of this information. It was interesting to note the difference in patient perceptions regarding the level of their tone which did not correlate with the therapist rating. Priebe (1996) also observed a lack of correlation between the patient rating of spasm frequency and clinical measures. The lack of correlation with functional activity and strength and frequency of spasm is not surprising in this study, as all of the spinal cord injury subjects in the study were particularly capable in activities of daily living. It would therefore take a significant degree of spasticity to cause functional compromise with this group. A point of note, that when considering the patient rating, it is also important to register that this was a combined assessment of both limbs whereas all the other measures tested each leg individually.

The only variable to correlate with the patient perception was the Pendulum Test. This adds support for implementation of the Pendulum test within the clinical examination, to compliment the subjective information gained from the patient and the therapist ratings. The defined relationship does not signify that one test should substitute for the other.

Figures 5.1, 5.2 and 5.3 show the rectified, averaged EMG signal for the four individual muscles, the velocity trace which indicates the periods of movement and rest (as described in Section 3.3.2) and the torque generated during the movement and rest periods.

The dynamometer proved to be highly reliable in the measurement of normal subjects, as indicated by the consistent work loop in Figure 5.1. It also produced repeatable measures for cord injured subjects.

Figure 5.1: Data Output - Velocity 200°/second - Neurologically Intact Patient



Angle (degrees)

Analysis of individual records identified two subjects who demonstrated burst of EMG activity through out the duration of the test, regardless of position or movement. Figure 5.2 demonstrates that bursts of phasic activity in all four muscles occur when the subject is at rest. Figure 5.3 shows that the level of activity in Tibialis Anterior is reduced by repeated movement but is still significant. Further investigation of this subject with more precise neurophysiological testing may be merited in this case. The subject presented here was the most extreme example but the levels of EMG activity were higher than expected in the SCI subject group. Two theories are proposed as the cause of this activity. Firstly, that the foot was not strapped adequately and sufficient movement of the ankle joint applied stretch to the muscle, eliciting the activity. The second hypothesis was that the strapping served as a proprioceptive afferent stimulus. Further investigation of this finding is clearly merited whereby the protocol is repeated with alternative fixation of the ankle is provided.

The correlation between the Modified Ashworth Score and the dynamometry results is again supported in the literature (Katz 1992). This relationship is to be expected in that the two measurement techniques purport to measure the same variable, the resistance to passive movement. The dynamometer however provides an increased degree of sensitivity as it is not confined to the five grades of the Ashworth Scale.

Figure 5.2: Data Output – Velocity - 0°/second - SCI Subject

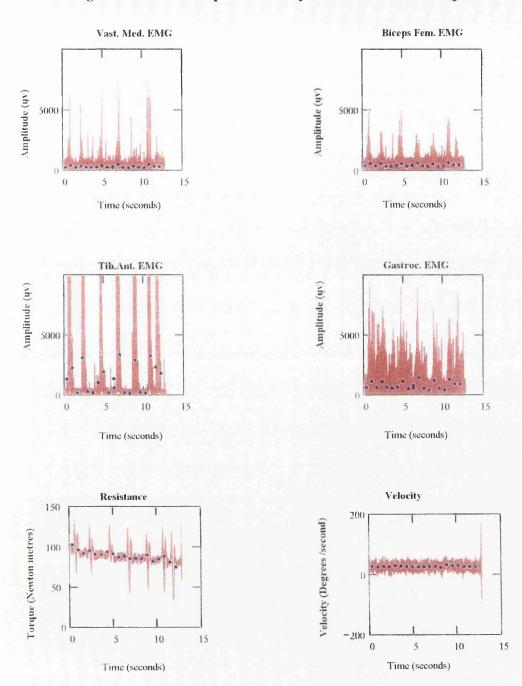
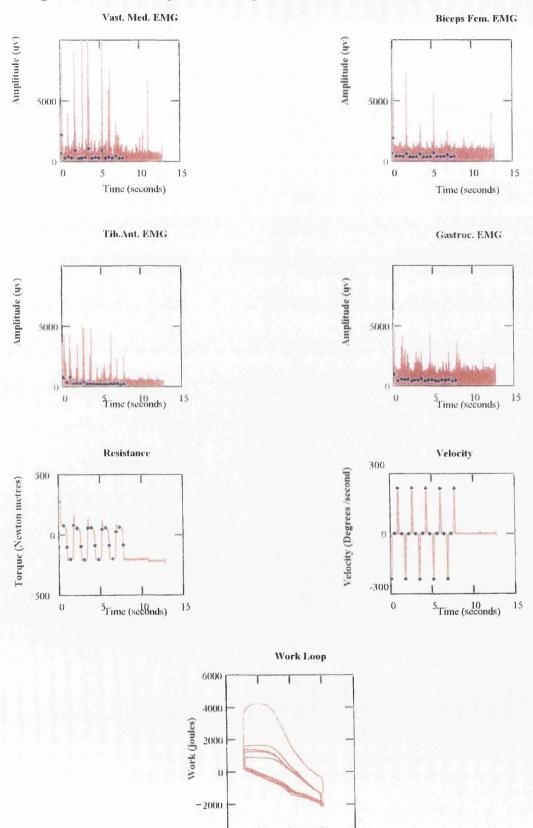


Figure 5.3: Data Output - Velocity 200°/second - SCI Subject



-4000

Angle (degrees)

It is important to form a view regarding the compatibility of all the measures utilised in the study with the clinical situation, both on an individual basis and as an assessment package.

The Modified Ashworth Scale is widely recognised by clinicians from all disciplines (Haas 1994) as a tool for the assessment of abnormal muscle tone. High intra-tester reliability has been demonstrated in this study and high intertester reliability seems likely (Bohannon & Smith 1987) but to date there is insufficient evidence in the literature to support this assumption. Evaluation of intra and inter-tester reliability presents few problems in the clinical environment and could be implemented as part of an induction programme for new clinical staff as appropriate. The assessment requires no equipment and is performed quickly, and therefore has no financial implications for the clinical service.

The patient rating provides an interesting source of information, which although may not correlated with the findings of the other assessments, as in this study, but is still a valid parameter to measure. The advent of clinical governance within healthcare provision in England (NHS Executive 1998) emphasises the importance of user involvement and patient satisfaction with the treatment received. If the treatment of abnormal muscle tone and subsequent problems associated with it is to be deemed as successful, it is essential that the patient perceives the benefit of the intervention in a way that is quantifiable to him. As with the therapist rating, the recording of patient perceptions regarding the their spasm strength, frequency and effect on

functional ability requires no equipment and minimal time, thus providing a low cost valuable source of information.

The Pendulum Test has attained considerable popularity in the clinical environment due to its objectivity and simplicity (Bajd 1982, Bohannon 1985). The test is quick and simple to perform with the minimum amount of equipment. To date, the analysis programmes are not commercially available but simple programming to compute Bajd's formula requires little investment from any hospital information technology or medical physics department.

The polyelectromyograph is a moderately expensive piece of equipment which may be put to practical use for in-depth investigation of patients with various neuromuscular pathologies. The use of pre-gelled self adhesive electrodes improved the quality of signal recording substantially, combined with the miniature amplifiers produced a robust but not portable EMG system that would be of great value to a clinical department. However, the electrodes selected for this study were not reusable and therefore carry a cost implication. The time taken to set up and conduct the test in conjunction with the dynamometry would however pose a problem for the clinician in most physiotherapy departments where the time allocation per patient is less than one hour. The process required for the analysis of the data in its present developmental state would also not be acceptable for use by the busy clinician.

The dynamometer provides an extremely reliable measurement tool which requires little operator training and will also interface with other equipment, should it be required. The dynamometer also provides the facility for controlled, repeatable active and passive testing of muscle function which can be used with many patient groups. A dynamometer is therefore a useful instrument to include in the clinical environment but the substantive cost may be prohibitive to smaller physiotherapy departments.

Each individual test has its own merits & shortfalls. The purpose of this study was to create an assessment package to measure several properties of abnormal muscle tone and augment the findings of the other assessments incorporated in the package. This should, in turn, identify the most appropriate treatment modality for the problem, assist in setting treatment goals and provide substantive evaluation of outcome. To omit any one component of the package would severely impact on the complimentary balance reflected in the assessment protocol.

The primary criticisms of the package of assessments are the need for training for skilled assessors to ensure reliable measurement in all five assessment procedures, the considerable time required to undertake the five components of the assessment package and the cost of the purchase, maintenance and support of the equipment. These factors would support the use of the assessment protocol in specialist units rather than more generic units where the presence of spinal injury patients is uncommon.

The management and treatment of abnormal muscle tone is undertaken as routine in specialist rehabilitation and spinal injury centres, where a staff skill

base has been established and will be routinely updated by regular staff training and development. As a consequence, all new techniques and treatments should be considered, to ensure that all clinical practice undertaken is evidence based. The volume of patients presenting at specialist units with problematic muscle tone, requiring careful assessment to determine appropriate assessment, as proposed by the findings of this study, would ensure staff commitment and sufficient repeated practice to maintain their skills in reliable assessment. This would not however be practicable in a general hospital, where the staff training programmes would focus on their large volume and more usual patient groups. Although the principles of reliable measurement to demonstrate treatment outcome should be mirrored across all practice, the choice of one simple, reliable and valid measure would no doubt be the preference for the nonspecialist units. Although the skills required to operate the dynamometer would be transferable for the assessment and treatment of other patient groups, this equipment is not often present in physiotherapy departments, as will be discussed later in this section.

The increasing demands on the Health Service to treat a greater number of patients, whilst still reducing waiting times, applies further pressure on staff to maximise the use of their clinical time. Only one assessor participated in this study and the time required to complete the full assessment package reduced considerably during the course of the study, as her skills developed. However, the average duration of each test session at completion of this study was still ninety minutes and required the assessor to be present throughout the procedure. This time included preparation of the patient and completion of the

five individual assessment protocols but did not entail the substantial time required for the offline data analysis. This would not be acceptable within a busy physiotherapy department in a small clinical centre or even a larger general district hospital where patient volume prevents long periods of individual face to face assessment and treatment. It would seem far more appropriate for patients with problems with abnormal muscle tone to be referred for assessment and treatment at a specialist centre, where sufficient patient numbers could make the case for a viable, regular clinic service supported by the appropriately skilled staff, to optimise the use of time.

In order to adopt the full assessment programme, a health care facility would need to make considerable investment in the equipment itself and the time required to train staff to operate the equipment reliably. The equipment for the assessment programme (dynamometer, electromyograph, electrogoniometer, acquisition and analysis software) and necessary technical support would not be available routinely in a small hospital physiotherapy department where the number of spinal injuries patients or patients with problematic abnormal muscle tone is likely to be negligible. The cost of the dynamometer (in excess of £35,000) would be prohibitive for most physiotherapy departments, despite its potential role in the assessment and treatment of other patient groups. The role of a polyelectromyograph and electrogoniometer would be minimal with the patient case mix in most general physiotherapy departments and therefore purchase of such equipment would not prove cost effective.

Increasingly, there is an international demand to treat the spinal cord injured individual in specialist centres (Stover 1995, SIA 1998, Wang 1999), where the appropriate skills and resources can be concentrated to ensure appropriate diagnosis, management and long term follow-up for optimum quality of life for this patient group. In 1998, the Spinal Injuries Association in the United Kingdom drew up a charter (SIA 1998) to campaign for all individuals with spinal cord injury to be treated a specialist spinal injuries centre in the U.K. Their campaign was further supported by a large survey of their membership (Smith 1999) which demonstrated improved impairment, disablity and handicap outcomes for patients managed in a specialist centre.

The SIA further advocate the life long management and treatment of all medical conditions experienced by the spinally injured within the specialist spinal injuries centres, regardless of the relationship between the medical problem and the cord injury. This view is not upheld by all the Spinal Injuries centres in the U.K (Middleton 1999 - personal communication) as these centres do not always have the necessary expertise readily available to deal with the vast range of medical problems that may be experienced by this patient group, which may be totally unrelated to the cord injury. It is entirely appropriate, however, for the management of patients with problematic abnormal muscle tone following cord injury to be continued by the specialist spinal injuries centres where the specialist skills, time and equipment would be more available for comprehensive assessment (as proposed by the findings of this study) and therapy required to manage this complex problem.

The increasing demand on bed availability within the specialist centres dictates the need for planned re-admissions with clear patient-focussed goals. The purpose of the comprehensive assessment protocol developed in this study was to ensure appropriate holistic evaluation to guide specific treatment interventions. The investment required in equipment and minimal training of expert staff for introduction of the proposed assessment protocol within such specialised units should therefore prove beneficial to both the cost and quality of the service provided.

5.3 Limitations of the Study

The tester reported a lack of sensitivity in the Modified Ashworth Scale. Detectable differences between the subject's legs were noticed by the therapist during rating but these were not represented in the scoring as the differences were not sufficient to merit a higher score on the scale.

The only tests performed simultaneously during the assessment programme were the EMG and the dynamometry. In retrospect, it may have been advisable to perform the patient rating before, after and during the other tests. The inclusion of simultaneous EMG recordings during the Pendulum test (which was rejected as unnecessary by Bajd, 1985) and Ashworth scoring would add to the strength of the comparison of data.

In the event of broadening the assessment programme to less able patients, the question of functional assessment should be reviewed again. The use of a functional assessment was excluded from the present study due to the

functional competence of the subjects, such as it was felt that a functional index would lack sensitivity to record change in this patient group.

The choice of methods to be included in an assessment programme for the evaluation of abnormal muscle tone following spinal cord injury must be appropriate to answering the question posed. The purpose of this study was to define a broad spectrum of measures to examine the various component of the upper motor neurone syndrome, to provide useful and practical information on which to base any treatment plan. Each of the methods used in the study could be developed further to answer more specific questions but this was outside the remit for this study.

5.4 Suggestions For Further Study

Inter-tester reliability

This study was undertaken by a single operator. If this assessment programme is to be implemented into routine clinical use, it is essential that the inter-rater reliability of the assessment protocol is examined. The process was designed to be as simple as possible, utilising equipment that could be easily imported into the clinical environment. None the less, inter-tester reliability cannot be assumed.

Patient Group

This assessment was evaluated in a small, yet homogenous group of patients. A natural progression from this study would be to implement the assessment protocol in a larger group of paraplegics and to extend the group to patients with cervical and incomplete cord injuries.

Treatment Modalities

The aim of the study was to devise and examine the assessment protocol in a group of patients that was likely to demonstrate minimal change from test to test. In order to examine the sensitivity of the protocol to change, it would be valuable to use the assessment of patients pre and post a known treatment to reduce spasticity, for example pre and post a bolus dose of intrathecal baclofen.

EMG Studies

The purpose of this study was not to examine the physiology underlying the characteristics of abnormal muscle tone but simply to measure the state that

these mechanisms place the patient in. Observation of the subject during testing with the dynamometer indicated that muscle activity was also occuring in the contralateral leg. Quantification of this activity using the method described in this study would provide additional clinical information. Further in-depth analysis of the electromyographic activity in this patient group would be of great interest but would require a different recording technique (intramuscular electrodes) and would therefor not be easily taken up in the clinic.

Chapter 6: Conclusion

The purpose of this study was to devise and evaluate a comprehensive assessment programme for the measurement of abnormal muscle tone and to consider it's application in the clinical environment.

Five measurement techniques were examined in a group of spinal cord injured subjects and a matched group of neurologically intact subjects: Therapist rating, patient rating, electrogoniometry, surface electromyography and dynamometry.

The findings of the study concluded that all of the measurement techniques were reliable and that the four measurement techniques implemented in both subject groups were able to differentiate between the spinal cord injured and the neurologically intact subjects.

The Therapist rating scale (Modified Ashworth Scale) showed a high intrarater reliability for the right leg (Intraclass correlation = 0.71) and left leg (Intraclass correlation =0.75). The scale detected a significant difference between the spinal cord injured and the neurologically intact subjects (p<0.001) and a significant difference between left and right leg (p<0.0001). No significant difference was demonstrated in either group over each of the three test occasions (p>0.1)

The numerical patient rating scale was shown to be reliable for the patient rating of spasm strength (Intraclass correlation = 0.82) and frequency

(Intraclass correlation = 0.71) and impairment of function (Intraclass correlation = 0.92). The patient rating scale demonstrated no significant change in the strength (p = 0.664) and frequency of spasms (p = 0.856) or the impact on functional ability (p = 0.444) between test occasions.

A significant correlation was demonstrated between the strength and frequency of spasms (p<0.05). No correlation was demonstrated between the strength or frequency of spasms with impairment of function.

No significant correlation was demonstrated between the therapist and patient rating.

Electrogoniometry demonstrated no significant intra-test and inter-test difference within each subject group (p<0.1). A highly significant difference was demonstrated between the Relaxation Indices of the spinal cord injured and the neurologically intact subjects (p<0.001) and a highly significant difference was demonstrated between right and left leg in each subject group (p<0.001).

No correlation was demonstrated between the Relaxation Indices and the therapist rating (r<0.6) or with the patient rating of spasm strength and frequency. A correlation was demonstrated between the Relaxation Indices and patient rating of impairment of function (r>0.62).

The polyelectromyograph showed no significant intra-test and inter-test difference in the levels of EMG activity in the Vastus Medialis, Biceps Femoris, Tibialis Anterior and Gastrocnemius in the neurologically intact subjects. No significant intra-test difference was seen in the spinal cord injured subjects but a significant inter-test difference was demonstrated (p<0.05). A significant difference in the degree of EMG activity between the two subject groups was demonstrated (p<0.01).

A significant difference was demonstrated between the degree of EMG activity in individual muscles (p<0.001) and between right and left leg in both subject groups. A significant difference was also observed between the degree of EMG activity at the four different speeds (p<0.001).

Dynamometry demonstrated no significant intra-test difference (p>0.1) in the torque generated during passive movement in both subject groups. No significant inter-test difference was observed in the neurologically intact subjects but a significant inter-test difference was observed in the spinal cord injured group (p<0.001). A significant difference was demonstrated between the torque generated in each leg in each subject group (p< 0.01) and a significant difference was demonstrated between the two subject groups (p<0.001).

No correlation was demonstrated between the torque generated during passive movement and the Relaxation Indices in both subject groups (r<0.5).

A correlation was demonstrated between the therapist rating scale and the torque generated in the spinal cord injured group on each test occasion (r>0.64). No correlation was demonstrated between the patient rating of spasm strength and frequency and impairment of function (r<0.6).

All the measures examined in the study proved to be reliable and practical for implementation within the clinical environment. The lack of correlation between the findings of the components of the assessment protocol supports the hypothesis that it is necessary to include all the measures investigated if the clinician wishes to undertake a comprehensive evaluation of the individual person with a spinal cord injury presenting with abnormal muscle tone.

References

Alfieri V 1982 Electrical treatment of spasticity.

Scand.J.Rehab.Med.14:177 - 182

American College of Surgeons Advanced Trauma Life Support.

Pub. American College of Surgeons. Chicago. IL60611-3211

Aminoff MJ 1980 Electrodiagnosis in clinical neurology.

Pub. Churchill Livingstone. New York.

Ashworth B 1964 Preliminary trial of carisoprodol in multiple sclerosis.

Practioner.192:540 - 542

Bachy-y-Rita P, Illis LS 1993 Spinal shock: possible role of receptor plasticity and non synaptic transmission.

Paraplegia 31: 82 - 87

Bajd T, Gregoric M, Vodovnic L, Benko H 1985 Electrical stimulation in treating spasticity resulting from spinal cord injury.

Arch.Phys.Med.Rehabil.66:515 - 517

Bajd T, Bowman RG 1982 Testing & modelling of spasticity.

J.Biomed.Eng.4:90 - 96

Barolat G, Maiman D 1987 Spasms in spinal cord injury. A study of 72 patients.

J.Am.Paraplegia Soc. 10:25 – 38

Barr FMD, Moffat B, Bayley JIL, Middleton FRI 1989 Evaluation of the effects of functional electrical stimulation on muscle power and spasticity in spinal cord injury patients.

Clin.Rehab.3:17 - 22

Beck AT, Ward Ch, Mendolson M, Mock J, Erborough J 1961 An inventory for measuring depression.

Arch.Gen.Psychol.4:351 - 363

Bedbrook GM, Sedgely GI 1980 The management of spinal injuries - past and present.

Int. Rehabil. Med. 2: 45 - 61

Benjamen J 1976 The Northwick Park ADL index.

Br.J.Occ.Ther. 39:301 – 306

Benton LA, Baker LL, Bowman BR, Waters RL 1981 Functional Electrical Stimulation - A practical guide.

Copyright. Rancho Los Amigos Rehabilitation Engineering Centre.

Berry M, Carlile J, Hunter A 1996 Peripheral nerve explants grafted into the vitreous body of an eye promote the regeneration of retinal ganglion cell axons severed in the optic nerve.

J. Neurocytol. 25: 147 - 170

Blumer CE, Quine S 1996 Surveillance of traumatic spinal cord injury in Australia: the identification of information needs.

Spinal Cord 34;11:639 – 643

Bobath B 1978 Abnormal postural reflex activity caused by brain lesions.

Pub. William Heinemann Medical Books Ltd. London

Bohannon RW 1993 Tilt table standing for reducing spasticity after spinal cord injury.

Arch.Phys.Med.Rehabil. 74:1121 - 1122

Bohannon RW, Smith MB 1987 Inter rater reliability of a modified Ashworth Scale of muscle spasticity.

Phys. Ther.67: 206 - 207

Bohannon RW 1987 Variability and reliability of the pendulum test for spasticity performed using a Cybex II isokinetic dynamometer.

Phys.Ther. 67:659 - 661

Bracken MB, Shephard MJ, Collins WF, Holford TR, Young W, Basakin

1990 A randomized controlled trial of methylprednisolone or naxolone in the

treatment of spinal cord injury: results of the second National Acute Spinal

Cord Injury Study.

N.Engl. J Med 322: 1405 - 1411

Brar SP, Smith MB, Nelson LM, Franklin GM, Cobble ND

Evaluation of treatment protocols on minimal to moderate spasticity in multiple

sclerosis.

Arch.Phys.Med.Rehabil.72:186 - 189

Brooks ME, Ohry A 1992 Conservative versus surgical treatment of the

cervical and thoracolumbar spine in spinal trauma.

Paraplegia 30: 46 - 49

Buckelew SP, Frank RG, Elliott TR, Chaney J, Hewett J 1991 Adjustment

to spinal cord injury: Stage theory revisited.

Paraplegia 29;2:125 - 130

Burke D, Hagbarth KE, Lofstedt L, Wallin BG 1976 The response of

human muscle spindle endings to vibration of non-contracting muscles.

J.Physiol.(London).261:673 - 693

201

Burke D, Lance JW 1973 Studies of the reflex effects of primary and

secondary spindle endings in spasticity.

Ed. Desmedt JE. New developments in electromyography and clinical

neurophysiology. Vol.3.475 - 495. Pub.Karger. Basel.

Campbell JW, Herbison GJ, Chen YT, Jaweed MM, Gussner CJ 1991

Spontaneous electromyographic potentials in chronic spinal cord injured

patients: relation to spasticity and length of nerve.

Arch.Phys.Med.Rehabil.72:23 - 27

Campbell J, Waters RL, Meadows PM, Nakai R, Carter CL, Grek AE,

Miller L, Gillim RL 1990 Spasticity: Correlate of FES motor performance in

spinal injury.

In.Proc.Advances in External Control of Human Extremities. Ed. Popovic

DB.Pub.Nauka.Belgrade

Carr JH, Shepard RB, Nordholm L, Lynne D 1998 Investigation of a new

motor assessment scale for stroke patients.

Phys.Ther.65:175 - 180

Chapman CE, Wiesendanger M 1982 Physiological and anatomical basis of

spasticity: A review.

Phys.Ther.Canada.34:125 - 136

202

Chen HY, Chiu WT, Chen SS, Lee LS, Hung CI, Hung CL, Wang YC, Hung CC, Lin LS 1997 A nation-wide epidemiological study of spinal cord injuries in Taiwan from July 1992 to June 1996.

Neurol.Res.19;6: 617 - 622

Cioni B, Meglio M, Pentimalli L, Visocchi M 1995 spinal cord stimulation in the treatment of paraplegic pain.

J.Neurosurg.82:35 - 39

Close R 1972 Dynamic properties of mammalian skeletal muscle.

Physiol.Review. 52:129 - 197

David S, Aguayo AJ 1981 Axonal elongation into peripheral nervous system bridges

after central nervous system injury in rats.

Science 241:931 - 933

de la Torre JC, Goldsmith HS 1990 Collogen-omental graft in experimental spinal cord transection.

Acta Neurochirg (Wien) 102:152 - 163

De Luca CJ 1997 The use of surface EMG in biomechanics.

J.Appl.Biomechan.13;2:192 -200

DELSYS. Neuromuscular Research Foundation. Boston University. MA 02181

Delwaide PJ 1984 Contribution of human reflex studies to the understanding and management of the pyramidal syndrome.

In. Ed:Shahani BT. Electromyography in CNS disorders: Central EMG. Pub.Butterworth .Boston. 77 -111.

Delwaide PJ Young RR 1985 Clinical Neurophysiology in spasticity Pub. Elsevier Amsterdam.

Ditunno JF, Young W, Donovan WH, Creasey G 1994 The international standards booklet for neurological and functional classification of spinal cord injury.

Paraplegia 32: 10 - 80

Department of Health 1998 A First Class Service. Quality in the New NHS. Health Services Circular 113;1998

Edgar RE 1992 Post-traumatic spinal spasticity.

In. Handbook of Clinical Neurology. Spinal Cord Trauma. Ed. Vinken PJ, Bruyn GW, Klawans HL, Frankel HL. 367 - 373

Exner G, Meinecke FW 1997 Trends in the treatment of spinal cord lesions seen within a period of 20 years in German centres.

Spinal Cord 35;7:415 -419

Fawcett JW 1998 Spinal cord repair: from experimental models to human application.

Spinal Cord 36:811 - 817

Frankel HL, Coll JR, Charlifue SW, Whiteneck GG, Gardner BP, Jamous MA, Krishnan KR, Nuseibeh I, Savic G, Sett P 1998 Long term survival in spinal cord injury: a fifty year investigation.

Spinal Cord 36;4:266 - 274

Fugel-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S 1975 The post-stroke hemiplegic patient: a method for evaluation of physical performance.

Scand.J.Rehabil.Med7:13 - 31

Gildenberg PL, Campos RJ, Dimitrijevic MR 1985 Characteristics of the tonic stretch reflex in spastic spinal cord and head injured patients.

Appl.Neurophysiol.48:106 - 110

Goldberg DP 1972 A scaled version of the General Health Questionnaire Psychol.Med. 9:139 – 45

Goldberg MJ 1991 Measuring outcomes in cerebral palsy.

J.Pediatr.Orthop.11:682 - 685

Granger CV, Hamilton BB, Sherwin FS 1986 Guide for the use of the uniform data set for medical rehabilitation.

Uniform Data System for Medical Rehabilitation Project Office, Buffalo General Hospital, New York 14203, USA.

Grays Anatomy 1980 Eds. Williams PL, Warwick R. 36th edition.

Pub. Churchill Livingstone

Guttmann L 1973 Spinal cord injuries: Comprehensive management and research.

pub. Blackwell. Oxford

Haas BM 1994 Measuring spasticity: a survey of current practice among health care professionals.

B.J.Ther. Rehabil.1;2:90 -95

Harvey RF, Jellinek HM 1981 Functional performance assessment: a program approach.

Arch.Phys.Med.Rehabil.62:456 - 460

Henneman E, Somjen G, Carpenter DO 1965 Functional significance of cell size in spinal motoneurones.

J.Neurophysiol. 28: 560 - 580

Iansek R 1984 the effects of reflex path length on clonus frequency in spastic muscles.

J.Neurol.Neurosurg.Psych.47:1122 - 1124

Jamshidi M, Smith AW 1996 Clinical measurement of spasticity using the Pendulum test: comparison of electrogoniometric and videotape analyses.

Arch.Phys.Med.Rehabil.77:1129 - 1132

Jansen JKS 1962 Spasticity: Functional aspects.

Acta Neurol.Scand.38;3:41 – 51

Jones DA, Round JM 1992 Skeletal muscle in health and disease.

Pub.Manchester University Press

Kakulas BA, Taylor JR 1992 Pathology of injuries of the vertebral coloumn and spinal cord.

In. Handbook of Clinical Neurology. Spinal Cord Trauma. Ed. Vinken PJ, Bruyn GW, Klawans HL, Frankel HL. 367 - 373

Katrak PH, Cole AMD, Poulos CJ, McCauley JCK 1992 Objective

assessment of spasticity, strength and function with early exhibition of

dantrolene sodium after cerebrovascular accident: a randomised double-blind

study.

Arch.Phys.Med.Rehabil.73:4 – 9

Katz RT, Rovai GP, Brait C, Rymer WZ 1992 Objective quantification of

spastic hypertonia: Correlation with clinical findings.

Arch.Phys.Med.Rehabil.73:339 - 347

Katz RT, Rymer WZ 1989 Spastic hypertonia.

Arch.Phys.Med.Rehabil.70:144 - 155

Keirstead HS 1995 Axonal regeneration and physiological activity following

transection and immunological disruption of myelin within the hatching chick

spinal cord.

J.Neurosci. 15: 69663 - 6974

Kiwerski JE 1993 Neurological outcomes from conservative or surgical

treatment of cervical spinal cord injured patients.

Paraplegia 31: 192 - 198

208

Knutsson E 1980 Restraint of spastic muscles in different types of movement.

In Spasticity. Disordered Motor Control. Eds.Feldman RG, Young RR, Koella WP. Year Book Publishers. Chicago

Knutsson E 1985 Quantification of spasticty

In.Eds. Struppler A, Weindl A. Electromyography and evoked potentials. Pub. Springer.Berlin.

Krawetz P, Nance P 1996 Gait analysis of spinal cord injured subjects: effects of injury level and spasticity.

Arch.Phys.Med.Rehabil.77:635 - 638

Krause JS, Sternberg M, Lottes S, Maides J 1997 Mortality after spinal cord injury: an 11 year prospective study.

Arch.Phys.Med.Rehabil.78; 8:815 -821

Krause JS 1998 Dimensions of subjective well-being after spinal cord injury: an analysis by gender and race/ethnicity.

Arch.Phys.Med.Rehabil.79: 900 - 909

Lakie M, Walsh EG, Wright GW 1984 Passive wrist movements – thixotropy – measurement of memory time,

J.Physiol.346:6 -12

Lamontagne A, Malouin F, Richards CL, Dumas F 1998 Evaluation of reflex and non reflex induced muscle resistance to stretch in adults with spinal cord injury using hand-held dynamometry and isokinetic dynamometry.

Phys.Ther.78;9:964 - 978

Lance JW 1980 Symposium synopsis. Spasticity: Disordered motor control. Eds.Feldman RG, Young RR, Koella WP. Year Book Publishers. Chicago

Lehmann JF, Price R, deLateur BJ, Hinderer S, Traynor C 1989

Spasticity: Quantitative measurements as a basis for assessing effectiveness of therapeutic intervention.

Arch.Phys.Med.Rehabil.70:6 - 15

Lenman JAR, Ritchie AE 1970 Clinical electromyography.

Pub. Medical & Scientific Publishing Company. Bath

Li Y, Field PM, Raisman G 1997 Repair of adult cortico-spinal tract by transplants of olfactory ensheathing cells.

Science 277:2000 - 2002

Lieber RL 1986 Skeletal muscle adaptability. 1: Review of basic properties.

Dev.Med.& Child Neurol.28:390 - 397

Mahoney F, Barthel D 1965 Functional evaluation: The Barthel index.

Md.State Med.J.14:61 - 65

Mathsoft Inc. 101 Main Street. Cambridge. MA02142. USA

Mayer NH 1997 Clinico-physiologic concepts of spasticity and motor

dysfunction in adults with an upper motoneuron lesion.

Muscle & Nerve Suppl.6:1 - 13

Midha M, Schmitt JK 1998 Epidural spinal cord stimulation for the control

of spasticity in spinal cord injury patients lacks long-term efficacy and is not

cost effective.

Spinal Cord 36:190 - 192

Mitchell GAG, Mayor D 1977 The essentials of neuroanatomy.

Pub.Churchill Livingstone. Edinburgh.

National Health Service Executive 1997

The new NHS. Modern & dependable

Pub. The Stationary Office Ltd

Noseworthy JH, Vandrvoort MK, Wong CJ, Ebers GC, & Canadian

Cooperative MS Study Group 1990 Interrater variability with the Expanded

Disability Status Scale (EDSS) and Functional Systems (FS) in a multiple

sclerosis clinical trial.

Neurol. 40: 971 - 975

211

Odéen I, Knutsson E 1981 Evaluation of the effects of muscle stretch and weight load in patients with spastic paraplegia.

Scand.J.Rehabil.Med.13:117 - 121

Penn RD, Savoy SM, Corcos D, Latash M, Gottlieb G 1989 Intrathecal baclofen for severe spinal spasticity.

N.Eng.J.Med.320:1517 - 1554

Perrell K, Scremin A, Scremin O, Kunkel C 1996 Quantifying muscle tone in spinal cord injury patients using isokinetic dynomometric techniques.

Paraplegia 34:46 - 53

Peterson T, Klemar B 1988 electrical stimulation as a treatment of lower limb spasticity.

J.Neuro.Rehab.2:103 - 108

Pierrot-Deseilligny E, Mazieres L 1985 Spinal mechanisms underlying spasticity.

In. Clinical Neurophysiology in spasticity Ed.Delwaide PJ Young RR. Pub. Elsevier Amsterdam.

Pierson SH, Katz DI, Tarsy D 1996 Botulinum toxin A in the treatment of spasticity: Functional implications and patient selection.

Arch.Phys.Med.Rehabil. 77:717 - 721

Price R, Lehmann JF, Boswell-Bessette S, Burleigh A, de Lateur BJ 1993

Influence of cryotherapy on spasticity at the human ankle.

Arch.Phys.Med.Rehabil.74:300 - 304

Priebe MM, Sherwood AM, Thornby JI, Kharas N, Markowski J 1996

Clinical assessment of spasticity in spinal cord injury: a multidimensional

problem.

Arch.Phys.Med.Rehabil.77:713 - 716

Priebe MM, Sherwood AM, Graves DE, Mueller M, Olson WH 1997

Effectiveness of gabapentin in controlling spasticity: a quantitative study.

Spinal Cord 35:171 - 175

Reger SI, Negami S, Reyes ET, Navarro R 1990 Wound healing and

perfusion of pressure ulcers in direct current stimulated denervated tissues.

In.Proc.Advances in External Control of Human Extremities. Ed. Popovic

DB.Pub.Nauka.Belgrade

Robinson CJ, Kett NA, Bolam JM 1988 Spasticity in spinal cord injured

patients: 2.Initial measures and long term effects of surface electrical

stimulation.

Arch.Phys.Med.Rehabil.69:862 - 868

Rosenfalck A, Andreassen 1980 Impaired regulation of force and firing pattern of single motor units in patients with spasticity.

J.Neurol.Neurosurg. Psych.43:907 - 916

Rothwell JC, Traub JJ, Day BL, Obeso JA, Thomas PK, Marsden CD 1982 Manual motor performance in a deafferented man.

Brain.105:515 - 542

Rushworth G 1964 The nature and management of spasticity.

Proc.R.Soc.Med.57:715 – 719

Schwab ME, Kapfhammer JP, Bandtlow CE 1993 Inhibitors of neurite growth.

Annu. - Rev. Neurosci. 16:565 - 595

Silberstein M, Brown D, Tress BM, Hennessey O 1992 Suggested MRI criteria for surgical decompression in acute spinal cord injury. Preliminary observations.

Paraplegia 30:704 - 710

Sehgal N, McGuire JR 1998 Beyond Ashworth: Electrophysiologic quantification of spasticity.

Phys.Med.Rehabil.Clin.North AM.9;949 -979

Seib TP, Price R, Reyes MR, Lehmann JF 1994 The quantitative

measurement of spasticty: Effect of cutaneous stimulation

Arch.Phys.Med.Rehabil.75:746 - 750

Shindo N, Jones R 1987 Reciprocal patterned electrical stimulation of the lower limbs in severe spasticity.

Physiotherapy 73;10:579 - 582

Silver JR Stewart D 1994 The porevention of spianl injuries in rugby football

Paraplegia July;32(7):442-53

Sköld C, Harms-Ringdahl K, Hultling C, Levi R, Seiger A 1998 Simultaneous Ashworth measurements and electromyographic recordings in tetraplegic patients.

Arch.Phys.Med.Rehabil.79:959 - 965

Smith MJ 1999 Making the difference: Efficacy of specialist versus nonspecialist management of Spinal Cord Injury

Pub. Spinal Injuries Association. 76 St. James's Lane. London N10 3DF

Spinal Injuries Association 1998

A Charter for Support

Spinal Injuries Association. 76 St. James's Lane. London N10 3DF

Spivak JM, Weiss MA, Cotler JM, Call M 1994 Cervical injuries in patients 65 and older.

Spine 15;19(20):2302 - 2306

Stover SL, DeLisa JA, Whiteneck GG 1995 Spinal Cord Injury. Clinical outcomes from the model systems. Aspen Publishers, Gaithersburg, Maryland, USA

Sullivan M, Karsson J, Ware JE 1995 The Swedish SF-36 health survey - 1. Evaluation of data quality, scaling assumptions, reliability and construct validity across general population in Sweden.

Soc.Sci. Med. 41:1349 - 1358

Spinal Cord 35;11:720 -724

Suyama T, Nihei R, Kimura T, Yano H, Tobimatsu Y, Hatsuyama Y, Nakamura R 1997 Rehabilitation of spinal cord injury in the national rehabilitation center for the disabled of Japan: profile of a spinal service.

Teddy PJ 1995

In.Clinical Neurology. International Practice & Research.

Neuroprostheses.4;1:95 – 114. Eds.Brindley GS, Rushton DN. Pub. Baillière

Tindall. London.

Tosi L, Righetti C, Terrini G, Zanette G 1993 Atypical syndromes caudal to the injury site in patients following spinal cord injury. A clinical neurophysiological and MRI study.

Paraplegia 31: 751 - 756

Turker KS 1993 Electromyography: Some methodological problems and issues.

Phys.Ther.73;10:57 - 68

Vallbo AB, Hagbarth KE, Torebjork HE, Wallin BG 1979

Somatosensory, proprioceptive and sympathetic activity in human peripheral nerves.

Physiol.Rev.59:919 - 957

Wade DT 1992 Measurement in Neurological Rehabilitation.

Pub. Oxford Medical Publications

Wall PD, Melzak R 1984 Textbook of pain.

Pub. Churchill Livingstone. Edinburgh.

Walton J 1981. Disorders of voluntary muscle. Pub. Churchill Livingstone. Edinburgh.

Wang D 1999 Mobilisation of patients having spinal surgery following acute spinal cord injury.

Pub. Proc. 16th Annual Guttmann Multi-disciplinary Meeting. Southport.

Wartenberg R 1951 Pendulousness of the legs as a diagnostic test.

Neurol.1:18 -24

Waters RL, Adkins R, Yakura J, Vigil D 1994 Prediction of ambulatory performance based on motor scores derived from standards of the American Spinal Injury Association.

Arch.Phys.Med.Rehabil.75;7:756 - 760

Westgren N, Levi R 1998 Quality of life and traumatic spinal cord injury.

Arch.Phys.Med.Rehabil. 79:1433 - 1439

Whiting S, Lincoln N 1980 An ADL assessment for stroke patients.

Br.J.Occ.Ther. 43:44 – 46

Williams B 1990 Post-traumatic syringomyelia, an update.

Paraplegia 28: 296 - 313

World heath Organisation 1980 International classification of Impairments, disabilities and handicaps.

W.H.O. Geneva.

Xu XM, Guenard V, Kleitman N, Bunge MB 1995 Axonal regeneration into Schwann cell-seeded guidance channels grafted into transected adult rat spinal cord.

J.Comp.Neurol. 351: 145 - 160

Yeo JD, Walsh J, Rutkowski S, Soden R, Craven M, Middleton J 1998

Mortality following spinal cord injury.

Spinal Cord 36;5:329 - 336

Young RR, Wiegner 1987 Spasticity.

Clin. Orthop. Rel. Res. 219:50-62

Zigmond AS, Snaith 1983 The Hospital Anxiety and Depression Scale.

Acta. Psych. Scand. 67:361 - 70

Appendices

Appendix 1.1: ASIA Scale for Measurement of Impairment – Key Sensory & Motor Testing Points

Neurological	Surface Marking Point	Muscle (Motor)	
Level	(Sensory)		
C2	Occipital protuberance	_	
C3	Supraclavicular fossa	-	
C4	Top of acromioclavicular joint	-	
C5	Lateral side of the antecubital	Elbow flexors (Biceps,	
	fossa	Brachialis	
C6	Thumb	-	
C7	Middle finger	-	
C8	Little finger	-	
T1	Medial side of the antecubital	•	
	fossa		
T2	Apex of axilla	-	
T3	Third intercostal space (IS)	-	
T4	Fourth IS	-	
T5	Fifth IS	-	
T6	Sixth IS (Xiphisternum)	-	
T7	Seventh IS	-	
T8	Eight IS		
Т9	Ninth IS -		
T10	Tenth IS (Umbilicus) -		
T11	Eleventh IS -		
T12	Inguinal ligament (Mid point)	nt) -	
L1	Half distance between T12 & L2	-	
L2	Mid anterior thigh Hip flexors (Iliopsoas)		
L3	Medial femoral condyle	Knee extensors (Quadriceps)	
L4	Medial malleolus Ankle dorsiflexors (Tibial		
	anterior)		
L5	Dorsum of foot -3^{rd} metatarsal Long toe extensors (Extension Long toe)		
phalangeal joint hallucis lor		hallucis longus	
S1	Lateral heel Ankle plantar flexors		
		(Gastrocnemius, Soleus)	
S2	Popliteal fossa	-	
S3	Ischial tuberosity	-	
S4-5	Perianal area	-	

Appendix 2.1: Technical Specification of Neurolog EMG System

NL100EK Head Stage

Input Impedance	108 ohmss.//10pF	
Input Leakage	<5pA	
Input sockets	3 x 2mm with compliance	
Gain	X 10	
Frequency response	> 50kHZ	
Common Mode rejection Ratio	40 dB	
Output Impedance	< 0.01Ω	
Differential O/P offset	500μV	
Output socket	4-way Lemo plug	
Lead length	1 metre	
Supply Current	< 2.5mA	
Dimensions	20x30x15	

NL824 Pre Amplifier

Input Impedance	100 Ω each input	
Input Leakage		
Input sockets	x 2mm sockets	
Gain	X 100, X 1000, X 10,000	
Frequency response	3Hz 10Hz 30Hz	
Common Mode Rejection Ratio	> -80 dB	
Output Impedance		
Differential O/P offset		
Output socket	9 way connecting plug	
Lead length		
Supply Current		

Appendix 2.1: Continued

NL820 Isolator

Isolation		
Voltage	+2500 volts DC	
Capacitance	35pf	
Resistance	10k Mohms	
Ground signal attenuation	2 x signal frequency Hz/10 ⁶	
Gain	x 1, x2, x5	
Input		
Amplitude	± 1 volt linear range:± 10 volts absolute	
Impedance 10 kohms		
Bias Current	< 50 nA	
Noise	< 4 mV at 150 kHz	
Offset	< 10 mV	
Output		
Amplitude	±5 volts maximum dependent on	
	gainsetting	
Frequency response	DC to 15kHz (3dB point)	
Isolated Power	±13 volts nominal unregulated	
	current maximum ± 40 mA	
	Output impedance 35 ohms nominal	
Input Power	± 15 volts regulated at 150 mA for	
	maximumisolated power specified	

NL125 Filter

Input voltage range	±	
Input impedance	56Kohms	
Low frequency cutoff range	DC, 0.5 Hz \rightarrow 5kHz continuouslty adjustable	
High frequency cutoff range	Wide band (>50kHz) 7 5Hz to 50kHz continuously adjustable	
Gain within passband	+ 1.0	
Attenuation beyond cutoff	40dB/decade	
Notch attenuation	> 50dB	
Notch width at - 3dB points	20Hz	
Output voltage range	± 10 volts	
Output Impedance	600 ohms	

Appendix 2.2: Project Information Sheet

Project Information Sheet

The aim of this study is to to devise a reliable method of quantification of spasticity and spasmodic muscle contractions and to examine the relationship between these phenomena. The effects of passive movement on the muscles of the leg will be observed with the subjects at rest and following interventions to change the resting state of the muscle.

Tests will be repeated on three occasions to examine reliability. The test environment will remain constant and tests will be conducted at the same time of day

The tests will include:-

1. Pendulum Test

An electrogoniometer will be attached to the leg of the subject to record the angle and speed of movement of the subjects lower leg when allowed to swing freely. The subjects will be positioned in lying with their lower leg (below the knee) hanging unsupported over the plinth. The therapist will move the leg to the extended position and when the subject is relaxed, release the leg and allow it to swing freely.

2. Dynamometry

The Kin Com dynamometer is an hydraulically powered, computer controlled exercise machine enabling active and passive exercise of the limbs. For the purpose of this study, passive movement of the legs, through a fixed range at constant predetermined speeds, will be performed using the dynamometer. The subjects will be positioned in lying on the dynamometer, with a strap around the pelvis and thigh for stabilisation. The subject's leg will be fixed to the arm of the dynamometer by a strap just above the ankle joint. The leg will be flexed from a straightened position to 90 degrees and then returned to the straightened position.

3. Electromyography

Electrical activity in 4 muscle groups in the leg will be recorded during passive movement of the limb using surface recording electrodes. Preparation of the skin prior to the application of the electrodes will be necessary to ensure adequate EMG recording. This will entail swabbing of the skin with alcohol, removal of leg hair by shaving and abrading of the skin using an abrasive solution.

4. Subjective rating of spasticity

The spinally injured subjects will rate their spasticity on each occasion using an established reliable 0 - 5 rating scale.

The therapist will rate the spasticity of the spinally injured subjects on each occasion using an established reliable 0 - 5 rating scale.



ORTHOPÆDIC HOSPITAL TRUST

in association with the Institute of Orthopædics

Brockley Hill, STANMORE Middlesex HA7 4LP

Your Ref.
Appendix 2.3: Consent Form

Telephone 0181-954 2300

Our Ref.

Fax 0181-....

Assessment of Abnormal Muscle Tone Following Spinal Cord Injury
I freely and voluntarily consent to participate in the research project being undertaken by Miss Fiona Barr of the FES Research Unit, R.N.O.H.T. under the supervision of Dr Frederick Middleton of the Royal National Orthopaedic Hospital, to determine a protocol for the assessment of abnormal muscle tone following spinal cord injury.
The outline of the research project and all the risks involved have been explained to me in full by and I have read the Project Information Sheet.
I understand that the aim of the project is to investigate the electrical activity of nerve and muscle tissue in normal and paralysed subjects at rest, during passive movement of the limbs. This will involve:-
Examination of joint range in the lower limb Manual measurement of resistance to passive movement Recording of electromyographic activity during passive movement of the lower limbs, by means of a dynamometer.
It will be necessary for me to attend the FES Research Unit at Stanmore on three occasions.
I have discussed the possible hazards of the research project with members of the research team, and understand them. These include:-
Skin problems - The electrodes for recording EMG are pre-gelled and an allergic skin reaction, although unlikely, may occur. Muscle, tendon and joint damage - I realise that there is a very small risk of this but that it could cause temporary incapacity.
I understand that I may withdraw from the project at any time and that this will not affect any further treatment for myself at the Royal National Orthopaedic Hospital.
Continued:-

225

have been assured that all data obtained during the research project will be kept anonymous, and that all personal data held on computer is registered under the Data Protection Act 1984.
have had no payment offered to me for my participation in this research project.

If I have any questions or queries, I can contact either:

Miss Fiona Barr Dr Frederick Middleton		081-954-2300 ext. 590 081-954-2300 ext. 596	
I have read and	l understood this consent form	n.	
Signed:			
Witnessed:			
Date:			

Measurement of Muscle Tone

Name:	 Test Number:
Date of Test:	Time of Test:
Strength	
Frequency	 ·····
Impairment of function	

Grade	Strength	Frequency	Functional Impairment
0	None	Never	No increase in spasm
1	Very weak	Rarely present	Does not interfere with ADL
2	Weak	Sometimes present	Interferes with fine skills
3	Strong	Often present	Impairs ADL
4	Very strong	Present most of the time	Need assistance with ADL
5	Rigid	Always present	Unable to perform ADL

Appendix 2.5: Pilot Study Subjects

Spinal Cord Injured

Subject	Sex	Age	Level	Time post injury (Years)
1	F	40	T8	12
2	F	45	T4	6
3	M	22	T9	4
4	M	24	Т6	6
5	M	29	Т8	10
6	M	31	T2	13
7	M	34	T4	4
8	M	34	T4	11
9	M	35	T6	3
10	M	36	Т9	4
11	M	42	T5	6
12	M	45	T11	10
13	M	47	T3	10
14	M	56	T4	6
15	M	60	Т8	7

Neurologically Intact

Subject	Sex	Age	
1	Female	37	
2	Female	36	
3	Male	40	
4.	Male	49	
5	Male	65	

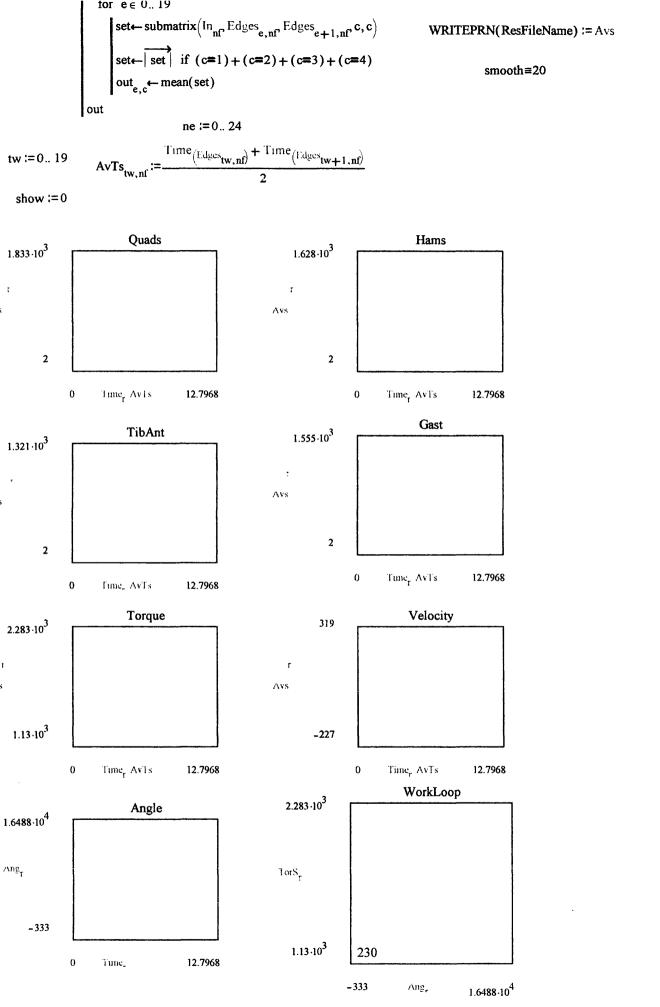
Appendix 3.1: MathCad Analysis Programme

Subject := "mism" FileNumber := 43 nf := 0..11FileName, num2str(FileNumber + nf)) FileName := concat("\\Roger\c\Fiona B\", Subject) ResFileName := concat(FileName₀, ".res") FileName_{nf}:=concat(FileName_{nf}, ".dat") ResFileName := concat("C:\Fiona9M\", Subject) ResFileName := concat(ResFileName, num2str(FileNumber)) ResFileName := concat(ResFileName, ".res") ResFileName = "C:\Fiona9M\mism43.res" In_{nf} := READPRN (FileName_{ni}) $r := 0.. \text{ rows}(\ln_{0}) - 1 \qquad \text{rr} := 1.. \text{ rows}(\ln_{0}) - 1 \qquad \text{Offset}_{\mathbf{nf}} := -27$ $\text{Time}_{r} := r \cdot 0.0024 \qquad \text{Quads}_{r, \mathbf{nf}} := \left| \left(\ln_{\mathbf{nf}} \right)_{r, 1} \right| \qquad \text{Hams}_{r, \mathbf{nf}} := \left| \left(\ln_{\mathbf{nf}} \right)_{r, 2} \right| \qquad \text{TibAnt}_{\tau, \mathbf{nf}} := \left| \left(\ln_{\mathbf{nf}} \right)_{r, 3} \right| \qquad \text{Gast}_{\tau, \mathbf{nf}} := \left| \left(\ln_{\mathbf{nf}} \right)_{r, 4} \right|$ $\operatorname{Ang}_{r,\operatorname{nf}} := \operatorname{Ang}_{rr,\operatorname{nf}} + \operatorname{Vel}_{rr-1,\operatorname{nf}} + \operatorname{Offset}_{\operatorname{nf}}$ $TorS^{\langle nf \rangle} := \begin{vmatrix} out \leftarrow Tor^{\langle nf \rangle} \\ for \ r \in smooth... \ rows(out) - 1 - smooth \\ out \leftarrow \frac{1}{2 \cdot smooth + 1} \cdot \sum_{k=r-smooth}^{r+smooth} Tor_{k,nf} \end{vmatrix} = \begin{vmatrix} out \leftarrow Vel^{\langle nf \rangle} \\ for \ r \in smooth... \ rows(out) - 1 - smooth \\ out \leftarrow \frac{1}{2 \cdot smooth + 1} \cdot \sum_{k=r-smooth}^{r+smooth} Vel_{k,nf} \end{vmatrix}$ $n \leftarrow 0$ $T1 \leftarrow \frac{\max(\text{VelS}^{<\text{nf}>})}{2}$ $T2 \leftarrow \frac{\min(\text{VelS}^{<\text{nf}>})}{2}$ $\text{for } r \in 1 ... \text{rows}(\text{Vel}) - 3$ $\text{if } \left[\left(\text{Vel}_{r-1, \text{nf}} < \text{T1} \right) \cdot \left(\text{Vel}_{r, \text{nf}} > \text{T1} \right) \right] + \left[\left(\text{Vel}_{r-1, \text{nf}} < \text{T1} \right) \cdot \left(\text{Vel}_{r, \text{nf}} > \text{T2} \right) \right]$ $n \leftarrow n + 1$ $\text{out}_n \leftarrow r$ ne := 0... rows(Edges) - 1

Edges $_{\text{ne},5} := \text{Edges}_{\text{ne},1}$ Edges $_{\text{ne},1} := \text{Edges}_{\text{ne},1}$

Edges_{ne,11} := Edges_{ne,1}

 $Edges_{ne.0} := Edges_{ne.1}$



NS.

Ang_T

```
"fiba"
              "jash"
              "chyu"
              "chfr"
                           Dir := "C:\Fiona9M\"
              "mism"
              "stca"
                                   ext := ".res"
              "ante"
                             Session := 7, 25...43
              "anch"
              "dath"
              "miwo"
FileRoot :=
              "jabe"
                           f := 0.. rows(FileRoot) -1
              "papa"
                                                      := concat(Dir, concat(FileRoot_f, concat(num2str(Session), ext)))
              "wibe"
              "adth"
              "shco"
                           f := 0.. rows(FileName) - 1
              "maba"
                             Data<sub>f</sub> := READPRN (FileName<sub>f</sub>)
              "kera"
              "toch"
              "anwa"
              "mabl"
```

WRITEPRN("FBDBase.asc") := Data

$$\begin{aligned} & \text{leg} := 0.. \ 1 \quad \text{vel} := 0.. 5 \quad \text{rep} := 0.. 4 \quad \text{phs} := 0.. 3 \quad \text{sig} := 0.. 6 \quad \text{Norm} := 0.. 29 \quad \text{Pats} := 30.. 59 \\ & \text{c(1,v,r,p,s)} := 1.840 + \text{v} \cdot 140 + \text{r} \cdot 28 + \text{p} \cdot 7 + \text{s} \\ & \text{Out}_{f,c(\text{leg,vel,rep,phs,sig})} := \left[\left(\text{Data}_f \right)_{\text{leg} \cdot 6 + \text{vel}} \right]_{\text{rep} \cdot 4 + \text{phs,sig}} \end{aligned}$$

$$VN_{Norm \cdot 5 + rep} := \left[\left(Data_{Norm} \right)_{0.6+3} \right]_{rep.4+1,1}$$
 mean(VN) = 461.462 stdev(VN) = 153.961

$$VP_{Norm.5+rep} := \left[\left(Data_{Pats} \right)_{0.6+3} \right]_{rep.4+1,1}$$
 mean(VP) = 532.62 stdev(VP) = 69.402

Set(NorP,L,V,R,P,S) :=
$$\begin{bmatrix} \text{for } r \in 0..29 \\ \text{out}_{r} \leftarrow \left[\left(\text{Data}_{\text{NorP} \cdot 30 + r} \right)_{\text{L-}6 + \text{V}} \right]_{\text{R-}4 + \text{P,S}} \\ \text{out} \end{bmatrix}$$

NorP := 1 Leg := 0 Vel := 1 Rep := 0 Phs := 1 Sig := 3

 $SDF1 := SetA(NorP, Leg, Vel, Rep, Phs, Sig) \qquad mean(SDF1) = 603.42 \qquad stdev(SDF1) = 758.363 \qquad \frac{stdev(SDF2)}{stdev(SDF1)} = 239.8$

NorP := 0 Leg := 0 Vel := 1 Rep := 0 Phs := 0 Sig := 3

SDF2 := Set(NorP, Leg, Vel, Rep, Phs, Sig) mean(SDF2) = 373.507 stdev(SDF2) = 49.348

show :=27

 $SDF1_{show} = SDF2_{show} = 391.6$ corr(SDF1, SDF2) =

O:=0..2 V:=0..6

$$SDF_{O,V} := \begin{cases} SetA(NorP, Leg, V, Rep, Phs, Sig) & \text{if } O=0 \\ SetB(NorP, Leg, V, Rep, Phs, Sig) & \text{if } O=1 \\ SetC(NorP, Leg, V, Rep, Phs, Sig) & \text{if } O=2 \end{cases}$$

$$SDF = \begin{cases} \{10,1\} &$$

 $Means_{(O,V)} := mean(SDF_{O,V})$

 $\mathsf{Means}_{3,\,\mathbf{V}} \coloneqq \mathsf{mean} \big(\mathsf{stack} \big(\mathsf{SDF}_{0,\,\mathbf{V}}, \mathsf{stack} \big(\mathsf{SDF}_{0,\,\mathbf{V}}, \mathsf{SDF}_{0,\,\mathbf{V}} \big) \big) \big)$

$$\mathbf{Means} = \begin{bmatrix} 367.48 & 357.4 & 344.49 & 371.68 & 360.71 & 368.44 & 1.328 \cdot 10^3 \\ 348.48 & 369.46 & 356.42 & 366.23 & 361.01 & 365.71 & 476.18 \\ 370.47 & 393.66 & 384.3 & 396.46 & 393.89 & 388.39 & 384.38 \\ 367.48 & 357.4 & 344.49 & 371.68 & 360.71 & 368.44 & 1.328 \cdot 10^3 \end{bmatrix}$$

Appendix 4.1: Subject Details

Neurologically Intact Subjects

Subject	Sex	Age	Height	Weight
1	F	37	163	63
2	F	43	163	63.9
3	M	30	170	70.2
4	M	29	183	93.6
5	M	32	178	80.1
6	M	31	178	72
7	M	35	175	75.6
8	M	45	178	84.6
9	M	43	170	94.5
10	M	60	173	107.1
Mean		38.5	173.1	80.46
Std Dev		9.55	6.64	14.439
Std.Error Mean		3.02	2.1	4.566

SCI Subjects

Subject	Sex	Age	Height	Weight
1	F	36	165	66.1
2	F	45	163	67.5
3	M	24	183	72.5
4	M	32	188	80.1
5	M	35	190	77.9
6	M	39	183	67.5
7	M	40	173	71.1
8	M	47	183	78.8
9	M	54	173	73.8
10	M	69	185	100.8
Mean		42.1	178.6	75.61
Std Dev		12.6	9.48	10.14
Std.Error Mean		3.98	3	3.207

Appendix 4.2: SCI Subjects - Level of Injury

Subject	Subject	Level of Injury	Time Post Injury (Years)
1	JВ	T3	11
2	PP	T5	12
3	WB	T5	8
4	AT	T4	15
5	SC	T5	12
6	MB	T12	7
7	KR	T6	11
8	TC	T6	6
9	AW	T10	14
10	MB	T6	11
Mean		-	10.7
Mode		T5/T6	-

Appendix 4.3: Subjective Rating Scores

SCI Subject Ashworth Sores

	Ashwor	th Score - R	light leg	Ashwo	rth Score - 1	Left leg
Subject	Test 1	Test 2	Test 3	Test 1	Test 2	Test 3
1	2	2	2	2	2	2
2	2	2	2	2	2	2
3	2	3	2	2	2	3
4	3	2	1	3	2	1
5	4	4	4	4	4	4
6	3	2	2	3	2	2
7	2	3	2	2	3	2
8	4	3	3	4	3	3
9	4	4	4	4	4	4
10	4	3	3	2	2	2

Patient Rating Scores

Strength

Subject	Test 1	Test 2	Test 3
1	3	3	3
2	2	1	1
3	1	2	2
4	3	0	1
5	2	0	1
6	2	2	2
7	2	2	2
8	2	2	1
9	3	3	3
10	5	5	5

Appendix 4.3: Continued

Frequency

Subject	Test 1	Test 2	Test 3
1	3	3	3
2	2	5	5
3	1	2	2
4	3	1	2
5	2	0	0
6	2	1	2
7	3	3	2
8	3	1	1
9	3	3	3
10	3	3	3

Impairment of Function

Subject	Test 1	Test 2	Test 3
1	1	2	2
2	1	1	2
3	1	0	2
4	0	0	2
5	0	1	0
6	2	0	2
7	1	1	1
8	3	1	1
9	3	3	3
10	0	0	0

Appendix 4.4: Relaxation Indices - SCI Subjects

Right Leg

					Pat	ient				
Test	1	2	3	4	5	6	7	8	9	10
1	0.5	0.39	0.77	0.53	0.75	0.32	0.32	0.58	0.18	0.59
	0.5	0.24	0.77	0.56	0.76	0.38	0.45	0.42	0.25	0.61
	0.58	0.29	0.75	0.44	0.76	0.31	0.49	0.56	0.18	0.77
2	0.6	0.4	0.73	0.5	0.74	1.21	0.09	0.29	0.22	0.82
	0.65	0.47	0.73	0.58	0.73	0.5	0.47	0.44	0.27	0.79
	0.58	0.19	0.77	0.53	0.72	0.51	0.47	0.57	0.55	0.79
3	0.99	0.7	0.77	0.52	0.63	0.86	0.33	0.47	0.41	0.76
	0.76	0.63	0.74	0.51	0.61	0.93	0.46	0.67	0.34	0.77
	0.78	0.53	0.79	0.58	0.74	0.29	0.18	0.13	0.46	0.74

Left Leg

					Pat	ient				
Test	1	2	3	4	5	6	7	8	9	10
1	1.03	0.59	0.56	0.62	0.96	0.45	0.66	0.79	0.59	0.56
	0.7	0.42	0.63	0.59	0.98	0.54	1.18	0.6	0.68	0.71
	0.95	0.5	0.66	0.57	1.06	0.39	0.56	0.72	0.65	0.67
2	1.22	0.75	0.94	0.75	0.85	1.02	0.95	0.78	0.64	0.62
	0.41	0.78	0.86	0.69	0.84	0.98	0.35	0.6	0.54	0.6
	0.51	0.77	0.89	0.56	0.85	1.03	0.56	0.85	0.61	0.63
3	1.05	0.6	0.68	0.72	0.91	0.31	0.6	0.58	0.64	0.24
	1.05	0.57	0.67	0.59	0.92	0.2	1.19	0.85	0.68	0.79
	1.05	0.6	0.86	0.59	0.9	0.21	0.91	0.72	0.71	0.73

Appendix 4.5: Relaxation Indices - Neurologically Intact Subjects

Right Leg

					Nor	mal				
Test	1	2	3	4	5	6	7	8	9	10
1	0.88	0.83	0.9	0.81	0.79	0.93	0.82	0.85	0.8	0.86
	0.86	0.9	0.84	0.83	0.76	0.95	0.87	0.83	0.83	0.85
	0.87	0.91	0.74	0.82	0.76	0.95	0.98	0.86	0.83	0.85
2	0.89	0.89	0.87	0.92	0.82	0.86	0.84	0.86	0.87	0.86
	0.87	0.87	0.86	0.94	0.85	0.93	0.91	0.85	0.75	0.87
	0.86	0.91	0.92	0.92	0.85	0.94	0.93	0.88	0.81	0.9
3	0.91	0.88	0.9	0.95	0.79	0.9	0.94	0.88	0.85	0.88
	0.9	0.91	0.83	0.95	0.91	0.91	0.94	0.88	0.86	0.85
	0.78	0.53	0.79	0.58	0.74	0.89	0.95	0.84	0.87	0.87

Left Leg

					Sub	ject				
Test	1	2	3	4	5	6	7	8	9	10
1	1.05	1.03	0.92	1.02	0.95	1.11	0.96	1.01	0.81	1.06
	1.08	1.03	0.92	1.02	0.92	1.11	1.06	1.03	0.97	1.02
	1.06	1.03	0.93	1.02	0.9	1.08	1.07	1.03	0.88	1.01
2	0.95	0.97	0.93	1.05	0.91	1.05	0.96	1	0.87	1.05
	0.91	0.93	0.94	1.07	1.02	0.99	1.07	0.97	0.93	1.07
	0.94	0.99	0.9	1.08	1.01	1.08	1.01	1.01	0.93	1.02
3	1.01	1.13	0.98	1.07	0.93	1.08	1.12	0.98	0.94	1.06
	1	1.14	0.98	1.08	1.06	1.09	0.95	0.98	0.98	1.07
	1	1.16	1.01	1.05	1.06	1.11	1.04	0.98	0.92	1.06

Appendix 4.6: Difference in Electrode Impedance - SCI Subjects

nt VM BF TA GA VM BF TA GA VM BF +4 +12 0	Right I ea		Tect	24 J			Test 2	# 2			Toct	st 3	
+4 +12 0	Patient	ΜΛ	1		GA	VM		. I	GA	VM	1		GA
0 0		+	+12	0	0	0	0	-2	0	0	-2	0	0
0 +2 0 -10 0 -10 +2 </th <th>2</th> <th>0</th>	2	0	0	0	0	0	0	0	0	0	0	0	0
0 0 0 +2 0	3	0	+2	0	0	-10	0	-10	+2	+2	0	0	0
0 0 0 -2 -4 0 -4 0	4	0	0	0	0	+2	0	0	0	0	0	0	0
0 -2 0 0 0 0 0 0 0 -2 -8 -2 0 0 0 0 0 +2 1 0 0 0 0 -4 0 0 0 0 0 0 -2 -2 0 0 0 0 0 0 0 0 0 -4 -10 -2 0 0 0 0 -2 0 0	S	0	0	0	0	-2	-4	0	7 -	0	0	0	0
-2 -8 -2 0 0 0 0 0 +2 +2 0 0 0 0 -4 0 </th <th>9</th> <th>0</th> <th>-2</th> <th>0</th> <th>0</th> <th>0</th> <th>0</th> <th>0</th> <th>0</th> <th>0</th> <th>0</th> <th>-2</th> <th>0</th>	9	0	-2	0	0	0	0	0	0	0	0	-2	0
0 0 0 0 -4 0 0 0 0 -2 -2 0 0 0 0 0 0 0 -4 -10 -2 0 0 0 0 -2 0	7	-2	8-	-2	0	0	0	0	0	+2	0	0	0
-2 -2 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	8	0	0	0	0	-4	0	0	0	0	0	+2	0
-4 -10 -2 0 0 0 0 -2 0 0 0 0 0 -3 0 0 0 0 0 0 0 0 0 0 0 0 0	6	-2	-2	0	0	0	0	0	0	0	0	0	0
	10	4-	-10	-2	0	0	0	0	-2	0	0	0	0

	В	0	0	0	0	4+	0	0	0	ò	0
Test 3	TA	-2	0	9-	0	0	0	0	0	0	0
Tes	BF	0	0	0	0	0	0	0	0	0	0
	ΜΛ	0	0	-2	+16	9-	0	-2	-2	0	0
	GA	0	0	0	0	0	0	0	-2	0	0
t 2	TA	0	0	0	0	8-	0	0	-2	0	0
Test 2	BF	0	0	0	-2	-2	0	0	0	0	0
	VM	0	0	0	0	0	0	0	-4	0	0
	GA	0	0	0	0	0	9-	-2	0	0	0
t 1	$\mathbf{T}\mathbf{A}$	0	0	+8	0	0	0	-2	-4	0	0
Test 1	BF	0	0	0	0	0	9+	0	0	0	0
	VM	0	0	0	0	0	0	0	0	0	0
Left Leg	Patient	1	2	3	4	2	9	7	8	6	10

VM - Vastus Medialis

BF - Biceps Femoris

GA - Gastrocnemius

TA - Tibialis Anterior

Appendix 4.7: Difference in Electrode Impedance - Neurologically Intact Subjects

ct VM BF TA GA VM BF TA CA 0 0 +2 -2 0 <t< th=""><th>Dight I og</th><th></th><th>Tot</th><th>1</th><th></th><th></th><th>Tas</th><th>+ 2</th><th></th><th></th><th>Toct 3</th><th>7,7</th><th></th></t<>	Dight I og		Tot	1			Tas	+ 2			Toct 3	7,7	
0 0 +2 -2 0	Subject	VM	1	1	GA	VM	1		GA	MA	BF	TA	GA
0 0	1	0	0	+2	-2	0	0	0	0	0	0	0	-2
-10 0 0 0 0 0 0 0 0 0 0 0 -2 0	2	0	0	0	0	0	0	0	0	0	0	0	0
0 0 0 -2 0 0 0 0 0 0 0 +2 0	3	-10	0	0	0	0	0	0	0	-2	-4	0	-2
0 0 +2 0 0 0 0 0 0 0 0 +2 -2 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	4	0	0	0	-2	0	0	0	0	-2	-2	0	0
0 0 +2 -2 0	S	0	0	+2	0	0	0	0	0	-2	0	0	0
0 0 0 0 0 0 0 0 -4 -2 0 0 0 0 -14 -2 -2 0 0 0 0 0 0 0 0	9	0	0	+2	-2	0	0	0	0	7-	0	-2	- 4
-4 -2 0 0 0 -14 -2 -2 0 0 0 0 0 0 0 0	7	0	0	0	0	0	0	0	0	-2	0	0	0
	8	4-	-2	0	0	0	-14	-2	-2	-2	0	7-	-2
	6	0	0	0	0	0	0	0	0	0	0	0	0
	10	0	0	0	0	0	0	0	0	0	0	-2	0

	GA	0	0	0	0	+4	0	0	0	0	0
st 3	TA	-2	0	9-	0	0	0	0	0	0	0
Test 3	BF	0	0	0	0	0	0	0	0	0	0
	VM	0	0	-2	+16	9-	0	-2	-2	0	0
	GA	0	0	0	0	0	0	0	-2	0	0
it 2	TA	0	0	0	0	8-	0	0	-2	0	0
Test 2	BF	0	0	0	-2	-2	0	0	0	0	0
	VM	0	0	0	0	0	0	0	-4	0	0
	GA	-2	0	0	0	0	+2	0	0	0	0
st 1	TA	0	0	0	0	0	0	0	0	0	0
Test 1	BF	0	0	0	0	0	+2	0	0	9+	0
	MA	7 -	0	0	0	0	0	0	0	8-	0
Left Leg	Subject	1	2	3	4	w	9	7	∞	6	10

VM - Vastus Medialis

BF - Biceps Femoris

TA - Tibialis Anterior

GA - Gastrocnemius

Legend for Figures 4.8-4.23

Figures 4.8 – 4.23 illustrate the relationship between the torque generated (y axis) and the EMG activity (x axis) in the each of the four individual muscles (Quadriceps - Figures 4.8, 4.12, 4.16 & 4.20: Biceps Femoris – Figures 4.9, 4.13, 4.17 & 4.21: Tibialis Anterior – Figures 4.10, 4.14, 4.18 & 4.22: Gastrocnemius – Figures 4.11, 4.15, 4.19 & 4.23) and during an individual phase of the dynamometer passive movement tests.

Each test comprised a sequence of 4 phases, repeated 5 times:-

Phase 1 - No movement (Figures 4.8 – 4.11)

Phase 2 – Movement from Extension to Flexion (Figures 4.12 – 4.15)

Phase 3 - No movement (Figures 4.16 - 4.19)

Phase 4 – Movement from Flexion to Extension (Figures 4.20 – 4.23)

Each test was conducted under 4 separate velocity conditions (A, B, C & D) and was repeated on 3 different days.

Condition	Passive Movement Velocity
A	25°/second
В	50° / second
С	100° / second
D	200° / second

Each figure contains 240 data points from the four velocity conditions on all of the three test occasions for both subject groups (twenty subjects in total).

Each point of the scatter plot represents the total amount of EMG activity and torque generated during the five repetitions of one individual phase on a single test occasion. Differentiation of the data points for the spinal cord injured and neurologically intact subjects and four test velocities conditions (25, 50, 100 & 200 degrees per second) are shown in the key for each figure.

Despite bunching of the data points on the left side of the plots, a significant difference between the two subject groups is demonstrated by the outlying points of the cord injured subjects along the x axis.

Symbol Blue - Neuro Intact Subjects Magenta - SCI Subjects Velocity ° / second 100 25 200 50 Test Condition A B C O Relationship Between Vastus Medialis EMG Activity & Torque Generated During Phase 1 0000 8000 7000 0009 5000 4090 0 (No Movement) 3000 COOC. × 1000 Appendix 4.8: 0 Torque (Nm) 5 30 25 35 20 15 10 242

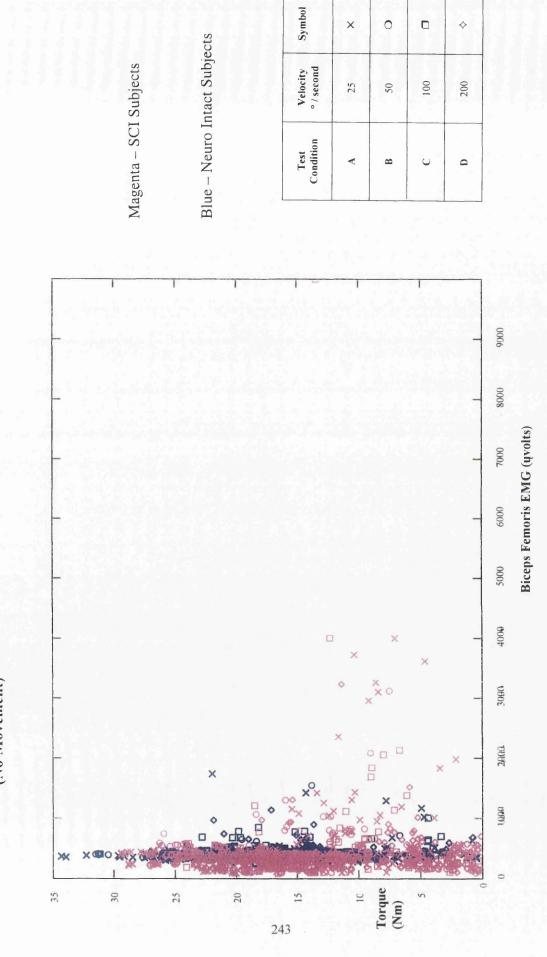
0

×

<

Vastus Medialis EMG (uvolts)

Relationship Between Biceps Femoris EMG Activity & Torque Generated During Phase 1 (No Movement) Appendix 4.9:

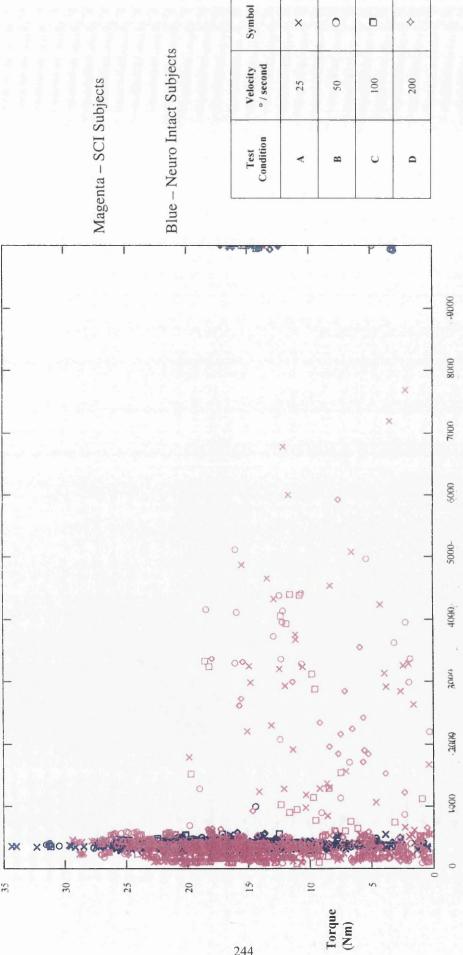


X

0

 \Diamond

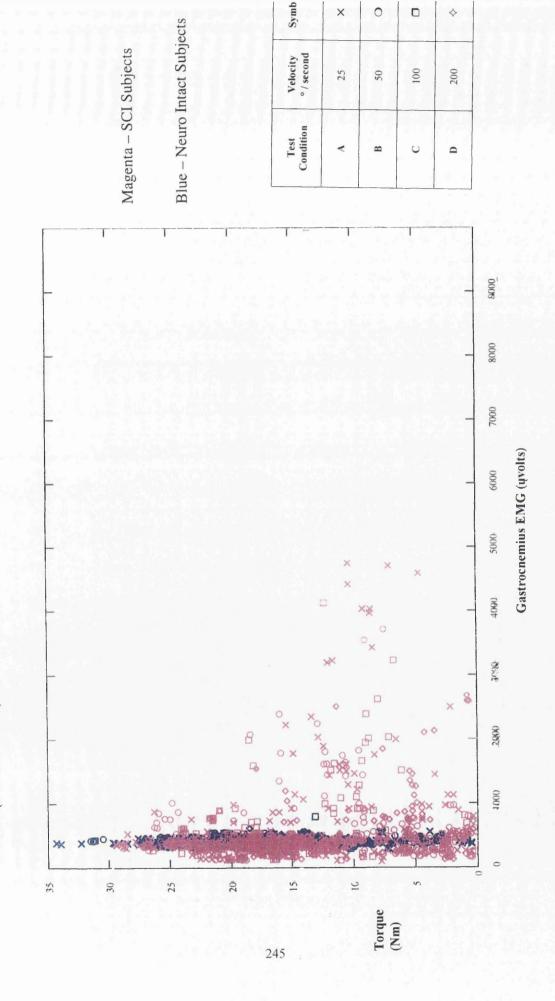
Relationship Between Tibialis Anterior EMG Activity & Torque Generated During Phase 1 (No Movement) XX Appendix 4.10: 30 35

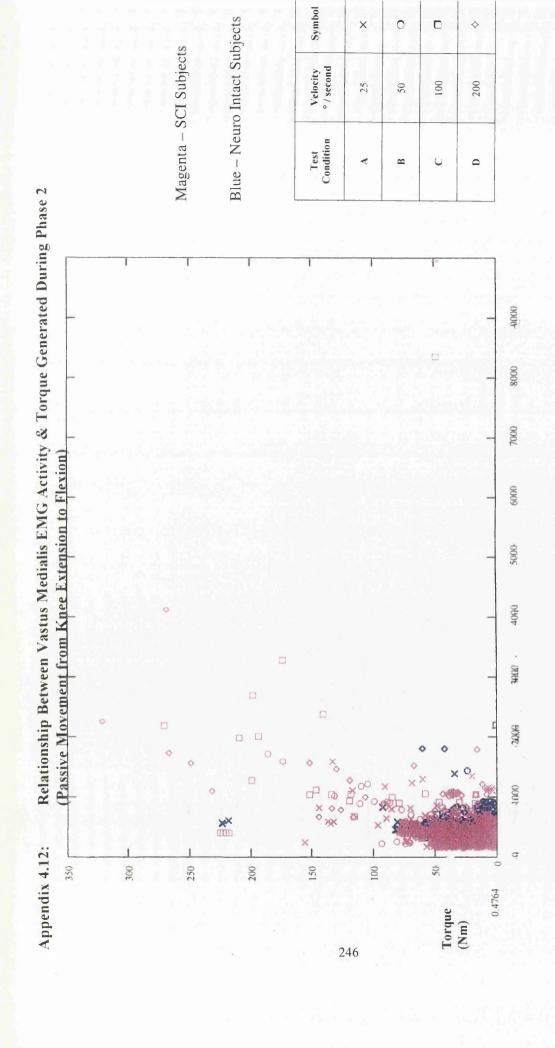


244

Tibialis Anterior EMG (uvolts)

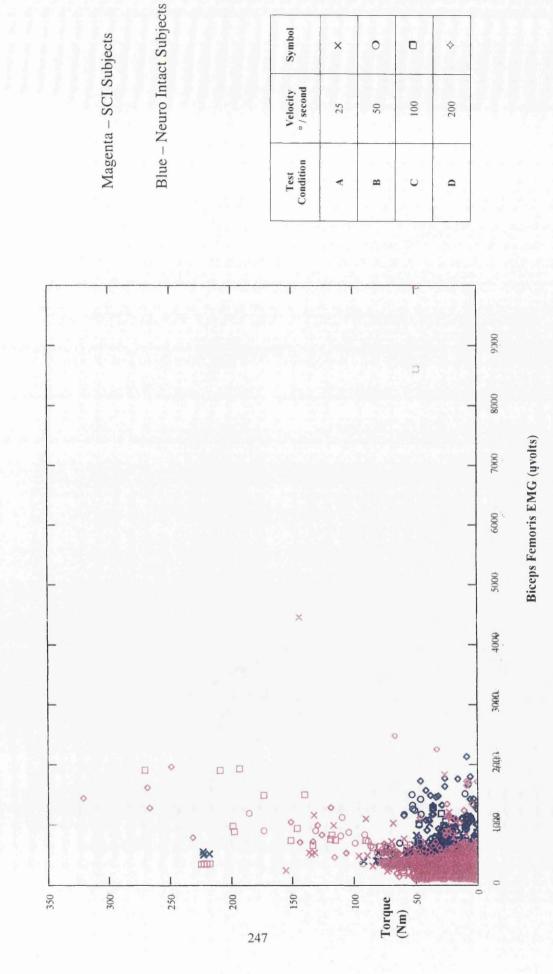
Relationship Between Gastrocnemius EMG Activity & Torque Generated During Phase 1 (No Movement) Appendix 4.11:





Vastus MedialisEMG (uvolts)

Relationship Between Biceps Femoris EMG Activity & Torque Generated During Phase 2 (Passive Movement from Knee Extension to Flexion) Appendix 4.13:



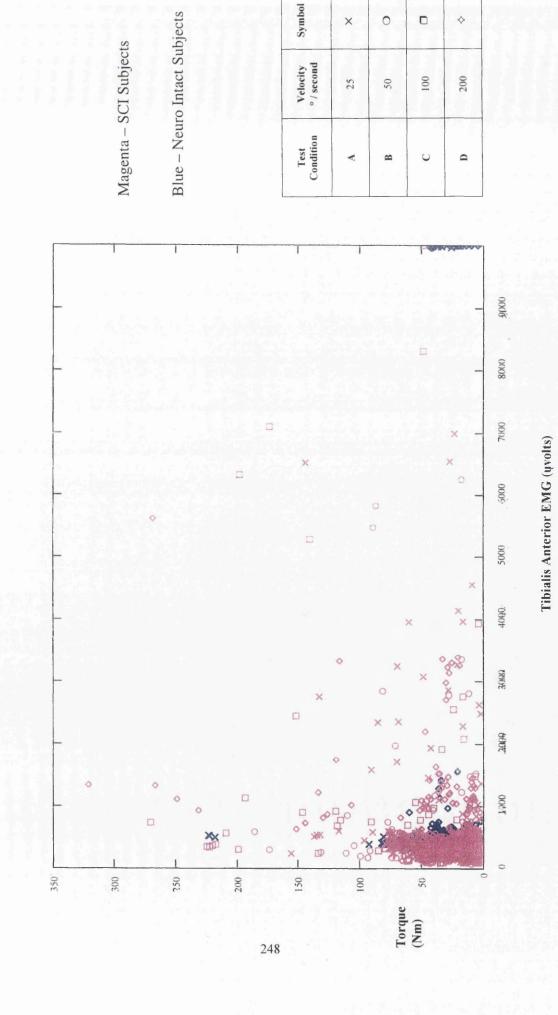
Symbol

0

<

×

Relationship Between Tibialis Anterior EMG Activity & Torque Generated During Phase 2 (Passive Movement from Knee Extension to Flexion) Appendix 4.14:



Symbo Blue - Neuro Intact Subjects Magenta - SCI Subjects Velocity ° / second 100 200 50 25 Test Condition C O B 0000% 8000 2000 (Passive Movement from Knee Extension to Flexion) 0009 5000 4000-. Ta8899 0 1000 8 J 300 250 100 200 150 50 350 Torque (Nm) 249

Relationship Between Gastrocnemius EMG Activity & Torque Generated During Phase 2

Appendix 4.15:

0

×

 \Diamond

Gastrocnemius EMG (uvolts)

Blue - Neuro Intact Subjects Magenta - SCI Subjects Velocity ° / second 100 200 50 25 Test Condition K 8 C Q Relationship Between Vastus Medialis EMG Activity & Torque Generated During Phase 3 (No Movement) 0000 8000 7000 0009 5000_TabP1 4090 3000 2009 1000 **%** × Appendix 4.16: θ 250 200 150 20. 100 300 Torque (Nm) 250

Symbol

0

×

 \Diamond

Vastus MedialisEMG (uvolts)

Blue - Neuro Intact Subjects Magenta - SCI Subjects Velocity °/second 100 200 25 50 Test Condition B C Q Relationship Between Biceps Femoris EMG Activity & Torque Generated During Phase 3 (No Movement) 0 XX × Appendix 4.17: 250 150 200 100 20 300 Torque (Nm) 251

Symbol

0

×

<>

0006

8000

7000

. 0009

5000

4000

3000

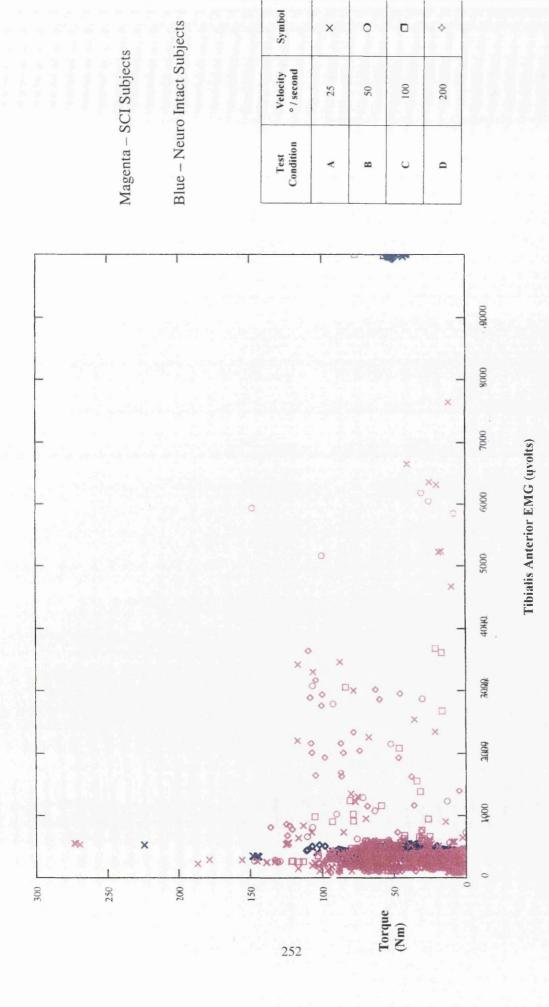
2000

1000

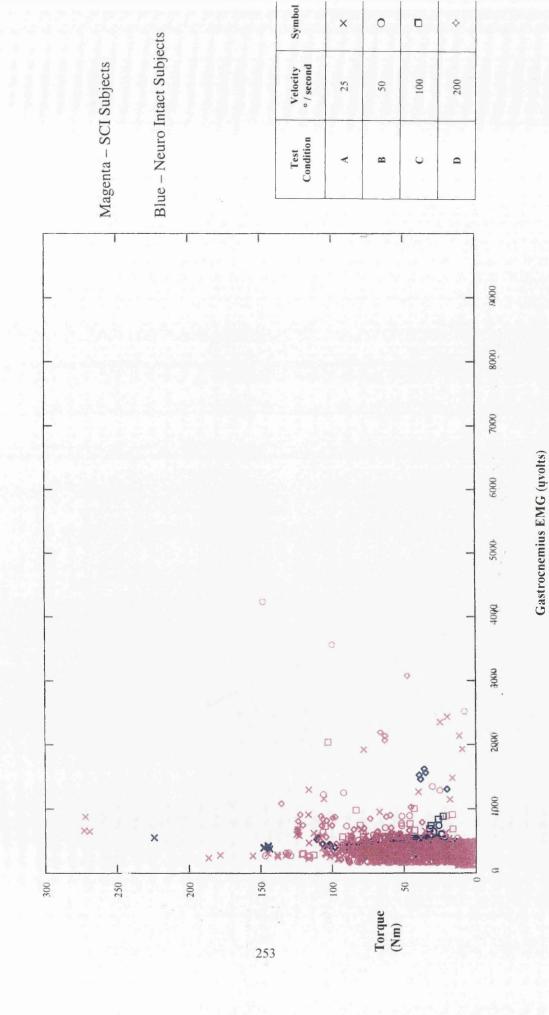
0

Biceps FemorisEMG (qvolts)

Relationship Between Tibialis Anterior EMG Activity & Torque Generated During Phase 3 (No Movement) Appendix 4.18:



Relationship Between Gastrocnemius EMG Activity & Torque Generated During Phase 3 (No Movement) Appendix 4.19:



Relationship Between Vastus Medialis EMG Activity & Torque Generated During Phase 4 Appendix 4.20:



250

200

150

Blue - Neuro Intact Subjects Magenta - SCI Subjects Velocity ° / second 100 25 50 200 Test Condition A C B O

Symbol

×

0

<

Vastus Medialis EMG (uvolts)

0000

8000

7000

0009

5000

400C

BOOK

2000

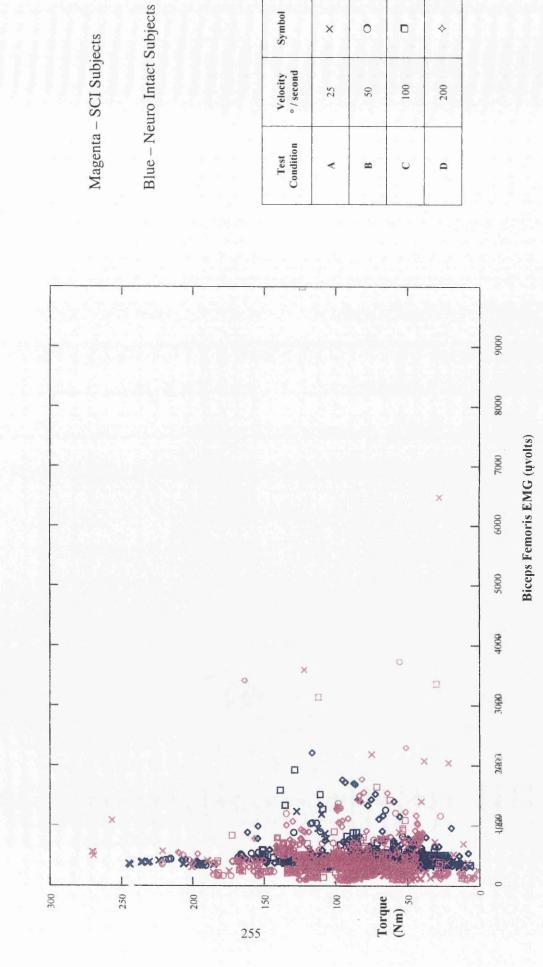
1000

Torque (Nm) 50.

100

254

Relationship Between Biceps Femoris EMG Activity & Torque Generated During Phase 4 (Passive Movement from Knee Flexion to Extension) Appendix 4.21:



Symbol

Velocity ° / second

×

25

A

0

20

B

100

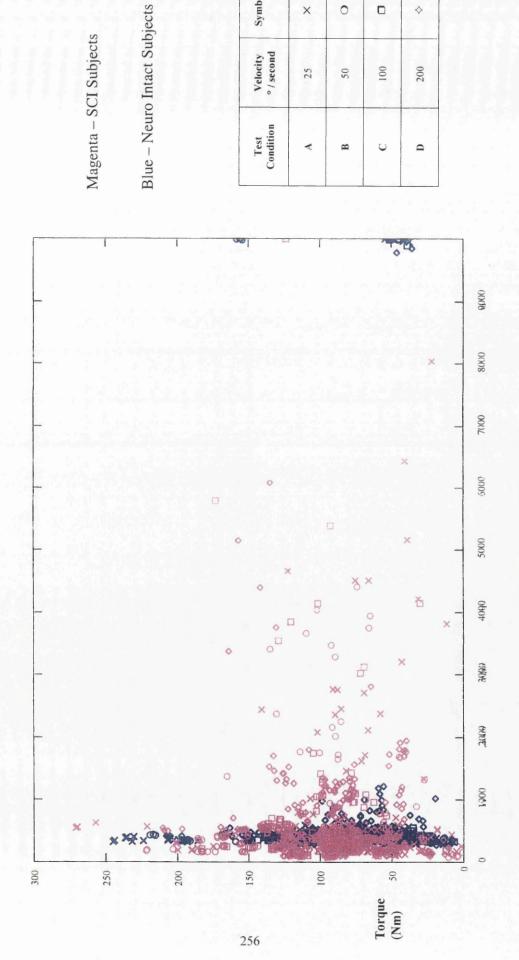
C

<

200

Q

Relationship Between Tibialis Anterior EMG Activity & Torque Generated During Phase 4 (Passive Movement from Knee Flexion to Extension) Appendix 4.22:



Symbol

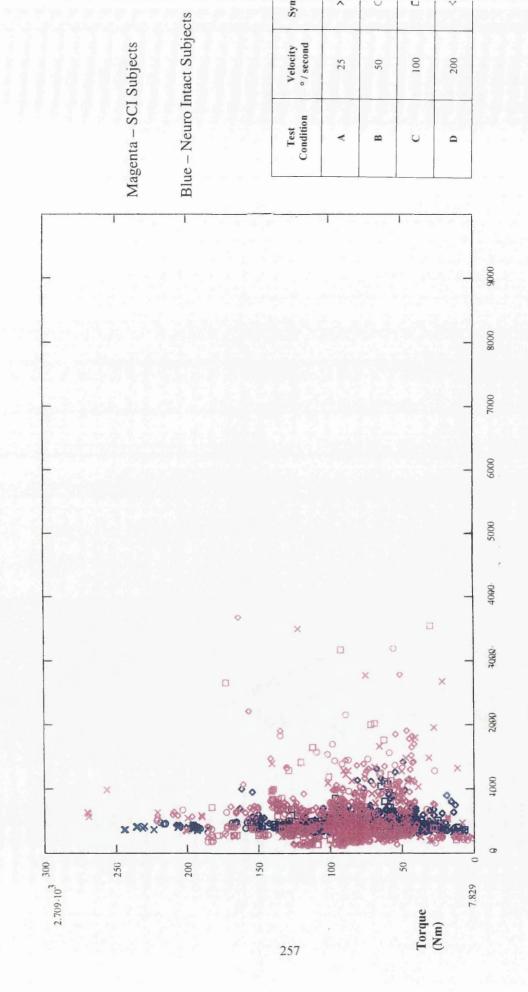
0

<

X

Tibialis Anterior EMG (qvolts)

Relationship Between Gastrocnemius EMG Activity & Torque Generated During Phase 4 (Passive Movement from Knee Flexion to Extension) Appendix 4.23:



Gastrocnemius EMG (uvolts)