The effect of tone-reducing ankle-foot orthoses for the treatment of lower limb spasticity

Submitted by

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BPO (Hons), La Trobe University, Australia.

A thesis submitted in total fulfillment

of the requirements for the degree of

Doctor of Philosophy

National Centre for Prosthetics and Orthotics

Division of Allied Health

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Australia

June, 2010

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List of Abbreviations

ABI	Acquired brain injury
ADLs	Activities of daily living
AFO	Ankle-foot orthosis
ANOVA	Analysis of variance
ASIS	Anterior superior iliac spine
BTX-A	Botulinum toxin-A
CI	Confidence interval
CNS	Central nervous system
CP	Cerebral Palsy
DFO	Dynamic foot orthosis
EMG	Electromyography
EVA	Ethylene vinyl acetate
GRF	Ground reaction force
GTO	Golgi tendon organ
H-reflex	Hoffmann reflex
Hmax:Mmax ratio	Ratio of the maximum Hoffmann reflex amplitude to the maximum muscle response amplitude
ICC	Intraclass correlation coefficient
ICF	International Classification of Functioning, Disability and Health
ITB	Inhibitory toe bar
M-response	Muscle response as a result of direct electrical stimulation of the alpha motoneuron
MAS	Modified Ashworth Scale
MN	Motoneuron
MS	Multiple sclerosis
OCG	Orthokinetic compression garment
PEDI	Paediatric Evaluation of Disability Index
PNS	Peripheral nervous system
Post-op shoe	Post-operative shoe
$R_{95\%}$	95% range of change
ROM	Range of motion

SCI	Spinal Cord Injury
SD	Standard deviation
SPASM	Support Programme for Assembly of database for Spasticity Measurement
SPSS	Statistical Package for the Social Sciences
TRAFO	Tone-reducing ankle-foot orthosis
TRO	Tone-reducing orthosis
UMN	Upper motor neurone

Dedication

To my mum,
Who endured heartache and poverty,
To give me every possible opportunity in life.
I did this for you.

這是謹獻給我敬愛的媽媽,
為要給我生命中的　一個可能成功的機會,
忍受心痛和貧困。

Statement of Authorship

Except where reference is made in the text of the thesis, this thesis contains no material published elsewhere or extracted in whole or in part from a thesis submitted for the award of any other degree of diploma.

No person's work has been used without due acknowledgment in the main text of the thesis.

The thesis has not been submitted for the award of any degree or diploma in any other tertiary institution.

All research procedures involving human subjects reported in the thesis were approved by the Health Sciences Faculty Human Ethics Committee of La Trobe University.

Signed:

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Summary

Tone-reducing ankle-foot orthoses (TRAFOs) are prescribed for the specific purpose of reducing spasticity in the lower limbs to improve function. However, their effects on spasticity and function are unclear. Therefore, the aim of this thesis was to examine the tone-reducing (neurophysiological) effect of TRAFOs in subjects with lower limb spasticity.

The first study conducted in this thesis aimed to determine the reliability of the Hoffmann reflex (H-reflex), which was the main measurement tool used in this thesis. The H-reflex is a measure of motoneuron (MN) excitability, and this study demonstrated that it was a reliable measure in the standing position. The H-reflex was subsequently used to examine the neurophysiological effect of TRAFOs on soleus MN excitability in able-bodied subjects, and subjects with spasticity while standing.

The results of these studies demonstrated no significant effects of TRAFOs on MN excitability suggesting that they were ineffective in reducing spasticity neurophysiologically. While these results were true at the group level, some subjects demonstrated significant effects to the orthosis conditions when the results were analysed individually. This highlighted the fact that the way in which spasticity responds to treatments can be unique for each individual.

Despite the main finding that TRAFOs had no significant effect on MN excitability, it was necessary to evaluate their effect on functional aspects of spasticity since the aim of TRAFO use is to improve spastic gait. The final study involved assessing the effect of TRAFOs using three-dimensional gait analysis and electromyography in subjects while walking. This study found no significant effect of TRAFOs on temporo-spatial gait parameters, joint kinematics or soleus muscle activity when compared with standard AFO designs. This thesis concluded that TRAFOs have no significant neurophysiological effect on soleus MN excitability, temporo-spatial and kinematic gait parameters or soleus muscle activity in subjects with spasticity.

Acknowledgements

This thesis was a collective team effort and would never have made it to the printer without the help of many people. First and foremost, thanks to my supervisors A/Prof. Tim Bach, A/Prof. Doug Rogers and A/Prof. Julie Bernhardt who invested so much of themselves professionally and personally.

Thanks to all of my family (most especially my mum) and Greg, for encouraging me, supporting me and loving me. A big thanks to all of my friends for not ditching me throughout my candidature. I must have been the biggest bore at times. To those who helped with proofreading, that chocolate I promised is on its way! A special mention to all of the staff at Melbourne Orthotics for actually understanding what my thesis was about and giving me time off work when I needed it. Thanks also to everyone at South Camberwell Uniting Church for their constant prayers and support, and thanks to my biggest supporter Jesus Christ.

Thanks for those who were needed along the way to give a professional helping hand: Dr. Alex Ward, Dr. Geoff Cumming, all of the staff at the National Centre for Prosthetics and Orthotics (La Trobe University), the Musculoskeletal Research Centre (La Trobe University) and the National Stroke Research Institute (Austin Health).

A special thanks to all of the stroke survivors I have met along the way. I admire you all for your perseverance and determination in life. Thanks to the Stroke Association of Victoria and the Victorian stroke support groups who helped with subject recruitment.

Finally, I would like to acknowledge the financial support of the Faculty of Health Sciences and the National Centre for Prosthetics and Orthotics at La Trobe University.

Publications, Conference Presentations and Awards

Publications

1. Ibuki, A. and Bernhardt, J. (2008) What is spasticity? The discussion continues. *International Journal of Therapy and Rehabilitation* 14(9):391-394.
2. Beischer, A. D., Clarke, A., de Steiger, R.N., Donnan, L., Ibuki, A. and Unglik, R. (2008) The practical application of multimedia technology to facilitate the education and treatment of patients with plantar fasciitis: a pilot study. *Foot and Ankle Specialist* 1(1):30-38.
3. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2010) The effect of tone-reducing orthotic devices on soleus muscle reflex excitability while standing in patients with spasticity following stroke. *Prosthetics and Orthotics International* 34(1):46-57.
4. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2010) 2009 ISPO Australia Best Research Paper Award: An investigation of the neurophysiologic effect of tone-reducing AFOs on reflex excitability in subjects with spasticity following stroke while standing. *Prosthetics and Orthotics International* 34(2):154-165.
5. Ibuki, A., Cornoiu, A., Clarke, A., Unglik, R. and Beischer, A. D. (2010) The effect of orthotic treatment for midfoot osteoarthritis assessed using specifically designed patient evaluation questionnaires. *Prosthetics and Orthotics International.* Currently under review.

Presentations

1. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2005) The effect of tone-reducing ankle-foot orthosis modifications on the reflex excitability of able-bodied subjects. International Society for Prosthetics and Orthotics ASM, Sydney, Australia.
2. Ibuki, A. (2006) The orthotic management of spasticity. PhD upgrade seminar. La Trobe University, Victoria, Australia.

3. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2006) The effect of TRAFO modifications on the H-reflexes of able-bodied subjects. APA Neurology special group, New Research Forum, Victoria, Australia.
4. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2007) The effect of TRAFO modifications on the reflex excitability of able-bodied subjects. International Society for Prosthetics and Orthotics 12th World Congress, Vancouver, Canada.
5. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2008) The effect of tone-reducing devices on soleus reflex excitability in patients with spasticity following stroke. International Society for Prosthetics and Orthotics ASM, Adelaide, Australia.
6. Ibuki, A., Beischer, A. D., Clarke, A. and Unglik, R. (2008) The use of multimedia technology to facilitate the education and treatment of patients with plantar fasciitis. International Society for Prosthetics and Orthotics ASM, Adelaide, Australia.
7. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2009) The effect of tone-reducing devices on soleus reflex excitability in patients with spasticity following stroke. Musculoskeletal Research Centre seminar, Victoria, Australia.
8. Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2009) An investigation of the neurophysiologic effect of tone-reducing AFOs on the reflex excitability of subjects with spasticity following stroke. International Society for Prosthetics and Orthotics ASM, Queensland, Australia.

Awards

1. 2005 David Myers university medal (La Trobe University).
2. 2007 E. H. Armstrong prize for services to lower limb amputees (National Centre for Prosthetics and Orthotics, La Trobe University).
3. 2008 George Carter new investigator's award (International Society for Prosthetics and Orthotics, Australia).
4. 2009 Best research paper award (International Society for Prosthetics and Orthotics, Australia).

Chapter One

Introduction and thesis overview

1.1 The mystery behind tone-reducing orthoses

Tone-reducing orthoses (TROs) may be considered a black hole in the world of orthotics. Orthotists are aware of them, but generally do not know much about them except that they are splints used for the specific purpose of reducing spasticity in the upper or lower limbs (Ford, Grotz and Shamp, 1986). There are many definitions for spasticity. In this thesis, it is defined as a pathological increase in muscle tone that results following an upper motor neurone (UMN) lesion, and presents as intermittent or sustained involuntary muscle activation that is characteristically velocity dependent (due to reflex elements), but is also influenced by non-reflex (mechanical) elements (Pandyan, Gregoric, Barnes *et al.*, 2005). Muscle tone can generally be defined as the constant tension between the origin and insertion of a muscle (Stratton, 1981).

Much speculation has been made regarding the effect of TROs, but there is a significant gap in our current knowledge regarding exactly what they do. TROs make only a brief appearance in key orthotic texts and are merely mentioned in passing in orthotics education. This is surprising since tone-reducing concepts have existed for decades with literature dating back to the 1950s. Tone-reducing concepts made their way into orthotic management in the form of inhibitory (tone-reducing) casts which later evolved into TROs with literature first published in the early 1980s (Zachazewski, Eberle and Jefferies, 1982). Despite the fact that TROs have been around for decades, the quality of TRO literature in academic journals is low and experimental studies are scarce. Literature on TROs is predominantly comprised of low-level evidence papers of anecdotal and observational single case studies (Dieli, Ayyappa and Hornbeak, 1997). As a result, our knowledge of TROs is poor (Lin, Sabharwal and Bibbo, 2000).

The majority of literature on TROs make very general speculations regarding what they do and what their potential benefits may be. As their name suggests, TROs supposedly reduce spasticity (Ford *et al.*, 1986) and are used when muscle tone becomes pathologically increased in conditions involving UMN lesions. There is currently no

solid evidence to support or refute the claims that previous authors have made regarding the tone-reducing capabilities of TROs. Research has failed to keep up with the continued use of TROs in the clinical world leaving a big question mark over the evidence base for their use (Teplicky, Russell and Law, 2003). In her article on dynamic orthoses (a form of TROs), Nancy Hylton (1990a, p198) identified this problem clearly by stating that "clinical practice techniques are often years ahead of an exact understanding and a quantifiable documentation of their effects".

People who have sustained an UMN lesion and subsequently experience spasticity face a multitude of associated problems. Spasticity greatly impairs one's ability to perform useful works with the motor system (Gans and Glenn, 1990), and when spasticity affects muscles in the lower limbs, a functional, efficient and safe gait is significantly hindered (Abe, Michimata, Sugawara, Sugaya and Izumi, 2009). This thesis will focus on the effects of spasticity in the lower limbs, which is quite different from the effects of spasticity in the upper limbs.

Ankle-foot orthoses (AFOs) are often prescribed to support, correct and control spastic lower limbs by offering biomechanical support and control (Abe *et al.*, 2009). There is substantial evidence in the literature to demonstrate that standard AFOs are effective in improving various aspects of gait, posture and function in people with spasticity (Abe *et al.*, 2009; Franceschini, Massucci, Ferrari, Agosti and Paroli, 2003; Gok, Kucukdeveci, Altinkaynak, Yavuzer and Ergin, 2003; Hesse, Luecke, Jahnke and Mauritz, 1996c; Hesse, Werner, Matthias, Stephen and Berteanu, 1999; Lehmann, Condon, Price and deLateur, 1987; Mojica, Nakamura, Kobayashi *et al.*, 1988; Tyson and Thornton, 2001; Tyson, Thornton and Downes, 1998; Wang, Lin, Lee and Yang, 2007). Tone-reducing ankle-foot orthoses (TRAFOs) are purported to provide further benefits for people with spasticity because along with the biomechanical support that AFOs provide, TRAFOs are said to have additional neurophysiological functions to directly reduce the spasticity in lower limb muscles (Ford *et al.*, 1986; Lohman and Goldstein, 1993; Nash, Roller and Parker, 2008; Teplicky *et al.*, 2003). It is believed that reducing spasticity in lower limb muscles subsequently leads to an improvement in the control and movement of those limbs. However, there is insufficient evidence regarding the neurophysiological effects of TRAFOs to directly inhibit muscle spasticity.

Perhaps the primary reason for this significant gap in knowledge is due to the complexity of the phenomenon of spasticity. Authors of TRAFO literature generally come from clinical backgrounds, and while they are able to document what they can see an orthosis is doing to a patient's posture, gait and balance, more often than not, they will be unable to explain what the orthosis is doing to the patient's spasticity neurophysiologically. As a result, the majority of literature on TRAFOs is comprised of observational studies that fall short of going that step further to explain why the investigator observed what they did. In order to thoroughly evaluate the effects of TRAFOs, the fields of neurophysiology and orthotics need to come together.

There has been a small handful of experimental studies conducted on TRAFOs published in the literature (Crenshaw, Herzog, Castagno *et al.*, 2000; Hassani, Roh, Ferdjallah *et al.*, 2004; Iwata, Kondo, Sato *et al.*, 2003; Lam, Leong, Li, Hu and Lu, 2005; Mills, 1984; Nash *et al.*, 2008; Naslund, Jesinkey, Sundelin, Wendt and Hirschfeld, 2005; Radtka, Skinner, Dixon and Johanson, 1997; Romkes and Brunner, 2002). The results of these studies are mixed, with some finding no significant differences when the TRAFOs were tested with other AFO designs and others finding improvements with the TRAFOs. The main problem with these studies is that they are unable to make firm conclusions regarding the neurophysiological effect of TRAFOs on spasticity. This is predominantly due to methodological shortcomings and the choice of measurement tools used by the investigators.

To adequately assess the neurophysiological effect of TRAFOs, the biomechanical effect must first be controlled, and the measurement tool used must exclusively measure at least one aspect of spasticity. Previous studies have failed to adequately control the biomechanical effect of TRAFOs and therefore, cannot make conclusions about the neurophysiological effect of the TRAFOs as their results were contaminated by confounding variables (Lin *et al.*, 2000). Some studies have investigated differences in muscle activity (Lam *et al.*, 2005; Mills, 1984; Nash *et al.*, 2008; Radtka *et al.*, 1997) and temporo-spatial gait parameters (Crenshaw *et al.*, 2000; Iwata *et al.*, 2003; Lam *et al.*, 2005; Nash *et al.*, 2008; Radtka *et al.*, 1997; Romkes and Brunner, 2002) between TRAFOs and other AFO designs, however, due to the test conditions and measurement procedures, they can not draw conclusions regarding the tone-reducing effects of the TRAFOs. It is not possible for the investigators of these studies to determine the

neurophysiological effect of the TRAFOs on spasticity as any changes in outcome measures could have been due to the biomechanical effect of the orthoses that were not controlled.

1.2 Thesis aims and hypotheses

Due to the shortcomings of previous TRAFO research, it remains unknown whether TRAFOs can reduce spasticity in the lower limbs. Therefore, the main aim of this thesis was to determine the effect of TRAFOs on lower limb spasticity. As spasticity is comprised of various reflex and mechanical elements, a number of outcome measures were used to assess the effect of TRAFOs on MN excitability, gait and muscle activity. The specific aims of this thesis were:

1. To develop a method by which the neurophysiological effect of TRAFOs could be evaluated separately from their biomechanical effect;
2. To investigate the neurophysiological effect of TRAFOs on the motoneuron (MN) excitability of the soleus muscles in subjects with spasticity following stroke while standing; and
3. To investigate the effect of TRAFOs on temporo-spatial gait parameters, joint kinematics and soleus muscle activity in subjects with spasticity following stroke while walking.

Based on these aims, the following hypotheses were derived:

1. TRAFO use results in a reduction of MN excitability of the soleus muscle in subjects with spasticity while standing;
2. TRAFO use results in improved temporo-spatial gait parameters and joint kinematics in subjects with spasticity; and
3. TRAFO use results in improved soleus muscle function in subjects with spasticity.

1.3 Thesis overview

The next chapter of this thesis, Chapter Two, begins with a detailed review of spasticity. Particular attention is paid to the aetiology and pathophysiology of spasticity to demonstrate the continued need for research into this complex phenomenon. Spasticity is a condition that continues to puzzle experts in the field and although knowledge is constantly progressing, there is still a lot to learn. For instance, it has previously been thought that muscle spasticity was predominantly the result of reflex based changes in the muscle (Singer, Dunne and Allison, 2001a). However, it is now known that changes in non-reflex (mechanical) elements (contractile and connective tissues) are also responsible for the phenomenon of spasticity in muscles. This recent differentiation between the reflex and mechanical elements responsible for muscle spasticity is presented in the review which challenges many aspects of our current knowledge of spasticity, from how spasticity is defined to how it is adequately quantified and treated.

A sound knowledge of the neurophysiology behind spasticity is required to understand how TRAFOs may be effective in altering lower limb spasticity, so the review discusses the basic neurophysiology underlying spasticity. The treatment of spasticity is probably just as complex as the condition itself. There are several methods for treating spasticity which include pharmacological methods, surgical methods and conservative methods. The review only details the conservative methods of treatment, in particular orthotic treatment. An historical account for how TRAFOs came about and how they have evolved over time is presented. Neurophysiological mechanisms which underlie the principles of TRAFOs are explored, followed by a critical review of the literature on TRAFOs to provide justification and purpose for this thesis. Reviewing past research on TRAFOs highlights the shortcomings of previous research and also the need for an appropriate measurement tool to be used when assessing the effect of TRAFOs on spasticity.

Measurement tools for the evaluation of spasticity are then discussed in three main sections; clinical methods, biomechanical methods and neurophysiological methods. Particular attention is placed on the H-reflex which was the main measurement tool used in this thesis. A thorough review of the H-reflex is presented detailing the neurophysiology behind the measurement tool. The advantages and limitations of the H-

reflex are also presented. Clear justification for the H-reflex being the most appropriate measurement tool for the assessment of TRAFOs in this thesis is provided. This chapter is then concluded by summarising the gaps in knowledge, the main research questions and the aims of this thesis.

Chapter Three presents the first study conducted in this thesis which aimed to determine the reliability of the H-reflex as adopted for use in this thesis. A detailed review of the methodology of the H-reflex is presented with particular attention placed on its reliability as established by previous investigators. As the H-reflex was the primary measurement tool used in this thesis, its reliability was crucial to support the subsequent investigations. This study was conducted on able-bodied subjects and assessed the inter-session reliability of the H-reflex measured from the soleus muscle with subjects in two positions: prone and standing. The results of this study demonstrated that the H-reflex had high inter-session reliability in both positions - a finding that is consistent with those of previous investigations.

With the reliability of the H-reflex established, it was then used as the main measurement tool in the subsequent three studies. Chapter Four details a study that examined the effect of TRAFOs on soleus MN excitability in able-bodied subjects while standing. This study was conducted on able-bodied subjects in the same way that other investigators have assessed the effects of spasticity interventions on able-bodied subjects prior to testing pathological subjects (Kukulka, Fellows, Oehlertz and Vanderwilt, 1985; Robichaud and Brunt, 1994). By first testing the TRAFOs on able-bodied subjects, the response of the healthy neuromuscular system to the TRAFOs was determined. This study was also helpful to smooth out the methodological procedures and built the framework for the subsequent studies in this thesis.

The results of this study demonstrated that the TRAFOs had no significant effect on soleus MN excitability in able-bodied subjects while standing. It was possible that the design of the TRAFOs (specifically the tone-reducing features) were insufficient to stimulate the necessary inhibitory afferent fibres to alter MN excitability, resulting in the non-significant results. As TRAFOs are simply AFOs with the addition of tone-reducing features, the effect of TRAFOs is largely dependent upon the design and construction of

the tone-reducing features. Therefore, prior to repeating the study on pathological subjects, the design of the tone-reducing features was examined.

The aim of the study presented in Chapter Five was to examine the effect of three individual tone-reducing features on soleus MN excitability in subjects with spasticity following stroke while standing. To establish an homogenous subject group, survivors of stroke exhibiting spasticity in their affected lower limb were recruited for the study. Three orthotic devices were used to assess the tone-reducing features which were:

1. A dynamic foot orthosis (DFO) to assess the effect of an inhibitory footplate;
2. An orthokinetic compression garment (OCG) to assess the effect of cutaneous stimulation through materials in contact with the skin, and circumferential pressure around the leg; and
3. A range of motion (ROM) walker to assess the effect of continuous stretch of the plantarflexor muscles.

The orthotic devices were assessed individually to determine their effects on soleus MN excitability, and variations of the devices were assessed to determine how the devices could be most effectively utilised to alter MN excitability. The results of the study found that overall, the devices were ineffective in altering soleus MN excitability in subjects with spasticity while standing. However, when assessed individually, two subjects responded with statistically significant decreases in their MN excitability to the DFO and one of those two subjects also responded to the OCG with a class 1 level of compression. These results supported the claims of previous investigators who have stated that the way in which individuals with spasticity respond to treatments can be unpredictable and unique.

To continue testing the aims and hypotheses of this thesis, the tone-reducing features were then incorporated into AFO designs to determine the effect of TRAFOs in subjects with spasticity. The studies conducted in Chapters Two to Five were necessary preparation for the final study presented in Chapters Six and Seven.

In the final study, TRAFOs were investigated alongside identical standard AFOs without any tone-reducing features, to determine the neurophysiological effect of the TRAFOs on MN excitability, temporo-spatial gait parameters, joint kinematics and soleus muscle

activity. In part one of this study which is presented in Chapter Six, the H-reflex was used to evaluate the neurophysiological effect of the TRAFOs on soleus MN excitability in subjects with spasticity while standing. Unlike other investigations that have been conducted on TRAFOs, this study was able to distinguish and isolate more clearly the neurophysiological effect of the TRAFOs from their biomechanical effect by comparing two AFO designs that were identical, except for the tone-reducing features of the TRAFOs.

The results of part one of this study demonstrated that overall the TRAFOs had no significant effect on the soleus MN excitability of subjects with spasticity. These results were consistent with the results of the studies detailed in Chapters Four and Five. However, when the results were analysed individually, four subjects had statistically significant increases in their MN excitability to one or more of the orthosis conditions, suggesting that the orthosis conditions were increasing their level of spasticity. These individual results were in contrast to those demonstrated in the previous study (Chapter Five) where MN excitability decreased in response to the orthosis conditions. Again, these findings highlighted the individual way in which subjects with spasticity may respond to treatment.

Part two of this study is presented in Chapter Seven. The aims of this study were to assess the effect of TRAFOs on temporo-spatial gait parameters, joint kinematics and soleus muscle activity in subjects with spasticity. Unlike the previous studies in this thesis that focused on measuring an aspect of the reflex element of spasticity (MN excitability), this study focused on measuring functional aspects of spasticity that involve the mechanical elements. Again, the TRAFOs were tested alongside standard AFOs without tone-reducing features to determine the neurophysiological effect of the TRAFOs. The results of this study found no significant differences in temporo-spatial gait parameters, joint kinematics or soleus muscle activity with the TRAFOs compared with the standard AFOs. However, when observing individual results, the electromyographic (EMG) data suggested that for some subjects, the TRAFOs were beneficial in reducing soleus muscle activity during walking (compared with the standard AFOs).

Chapter Eight of this thesis forms an overall discussion of the main findings of the studies presented in Chapters Three to Seven, and explores their implications in the clinical field of orthotics. The main limitations of the studies are acknowledged and suggestions for future studies are presented. Finally, the conclusions of the thesis are presented. Overall, this thesis concluded that TRAFOs are ineffective in altering soleus MN excitability, gait parameters and soleus muscle activity in subjects with spasticity following stroke. While there may be significant effects observed for some individuals, the effects are not always beneficial. Furthermore, it appears almost impossible to be able to predict these outcomes as subjects seemed to respond to treatments on an individual basis. The results of the studies in this thesis highlight the complexity of treating spasticity and emphasise the need to monitor orthotic treatments carefully to ensure that any effects are beneficial for the individual.

Chapter Two

Review of the literature

This chapter presents a detailed review of muscle spasticity which is the pathological focus of this thesis. Spasticity affects the lives of many people with UMN lesions in numerous ways, from causing difficulties with motor control, to severely limiting any form of functional movement. Spasticity is a serious condition that requires continued research to improve the lives of the people it affects. To grasp a fundamental understanding of spasticity, it is necessary to begin by exploring the basics of normal muscle tone, and then move on to what happens when normal muscle tone is disturbed. Treatments for spasticity will be reviewed as well as the methods for quantifying spasticity to evaluate the effects of treatment.

2.1 Muscle tone

The definition for muscle tone depends upon the state of the muscle, which can either be relaxed (passive) or active. When a muscle is in a state of voluntary relaxation, muscle tone can be clinically defined as the slight constant tension between the origin and insertion of a muscle, resulting in a sensation of resistance to passive displacement when a limb is moved through a range of motion (ROM) (Carr, Shepherd and Ada, 1995; Katz and Rymer, 1989; Pisano, Miscio, Colombo and Pinelli, 1996). This definition can only be applied to muscle tone when a muscle is relaxed, not when a muscle is active. Therefore, when a muscle is active, muscle tone can be defined as the balanced tension in the muscle that maintains its readiness to respond to stimulation (Davidoff, 1992). These two characteristics of muscle tone consist of the simultaneously combined influences of three distinct components (Katz and Rymer, 1989; Morris, 2002b; Pisano *et al.*, 1996):

1. The physical inertia of the extremity producing reaction forces proportional to acceleration;
2. Mechanical/visco-elastic characteristics of muscular and connective tissues, tendons and joints; and
3. Spinal reflex activity.

For muscles to function normally, the peripheral nervous system (PNS) and the central nervous system (CNS) must work together so that the state of the muscle is always known. This allows active muscle tone to be adequately maintained and constantly adjusted to the requirements of posture and movement (Gelber and Jozefczyk, 1999). This regulation of active muscle tone occurs through an intricate network of somatic reflexes that are mediated by the spinal cord and thus known as spinal reflexes. Normal active muscle tone depends on a balance between the excitatory and inhibitory influences on these spinal reflexes (Losseff and Thompson, 1995). These influences are facilitated or inhibited by the supraspinal systems (cortex, basal ganglia, cerebellum and brainstem) which constantly modulate activity in the PNS (Lennon, 1996).

A spinal reflex is a reflex with a pathway through the spinal cord usually not requiring supraspinal input or higher processing (Felten and Felten, 1982). As long as sensory input and lower MN output are intact, spinal reflexes can function even in the absence of supraspinal connections (Felten and Felten, 1982). The simplest example of a spinal reflex is the monosynaptic reflex which involves one afferent and one efferent neuron which have one synapse in the spinal cord (Felten and Felten, 1982). The two basic kinds of spinal reflexes are superficial (cutaneous) reflexes and deep reflexes. The deep reflexes include the muscle stretch reflex and the Golgi tendon organ (GTO) reflex.

Superficial reflexes are polysynaptic and include withdrawal reflexes, exteroceptive reflexes or flexor reflexes (Felten and Felten, 1982). They cause motor responses to cutaneous stimulations of sensory exteroceptors in the superficial tissues on the exterior of the body (Felten and Felten, 1982). Exteroceptors are responsive to heat, cold and touch. If the stimulus is intense enough to cause pain or potential injury to the body, the withdrawal reflex can be initiated in which many muscles contract in a coordinated fashion. The stronger the stimulus, the more interneurons recruited and the more muscles involved in the reflex, which can involve all four limbs and the trunk (Felten and Felten, 1982).

Muscle stretch reflexes occur in response to the stimulation of sensory receptors known as muscle spindles, which detect slight changes in muscle length (Carew and Ghez, 1985; Stratton, 1981). Muscle spindles are small stretch-sensitive encapsulated units that lie

parallel to muscle fibres within muscle bellies (Stratton, 1981). Muscle spindles respond to changes in the length or the amount of stretch of muscles, as well as changes in the velocity of the muscle stretch (Gelber and Jozefczyk, 1999; Meythaler, 2001). When muscle spindles are activated, primary spindle endings with type Ia afferent fibres and secondary spindle endings with type II afferent fibres transmit impulses to the spinal cord (Davidoff, 1992). These afferent fibres synapse with alpha MNs and rapidly excite the extrafusal muscle fibres of the stretched muscle resisting further stretch (Felten and Felten, 1982). It appears that type Ia fibres are concerned with relaying information concerning changes in muscle length and contraction velocity, while type II fibres are only concerned with changes in muscle length (Carew and Ghez, 1985; Stratton, 1981). The Ia afferent fibres also synapse with inhibitory interneurons that inhibit alpha MNs controlling the antagonist muscles so that they relax and do not interfere with the desired movement (Stratton, 1981).

GTOs are located within the tendons of skeletal muscles and are proprioceptors responsible for muscle relaxation and lengthening in response to an increase in muscle tension whether due to active muscle contraction or not (Carew and Ghez, 1985; Felten and Felten, 1982). GTOs inform the nervous system of the tension exerted by the muscle on its tendinous insertion to the bone (Carew and Ghez, 1985). Once activated, impulses are transmitted via Ib afferent fibres which exert an inhibitory influence on the alpha MNs of the same muscle, relaxing it and preventing the tendon from being further stretched (Felten and Felten, 1982). The Ib afferent fibres also synapse with interneurons that activate the antagonist muscles (known as reciprocal activation), and transmit information to the cerebellum.

These muscle stretch reflexes can be described as tonic or phasic. Tonic reflexes respond to changes in the length of muscles and cause a prolonged asynchronous discharge of MNs to produce a sustained muscle contraction for the alteration or maintenance of posture (Morris, 2002b). Phasic reflexes respond to quick stretching of muscles and cause a synchronous discharge of MNs (Morris, 2002b). Phasic stretch reflexes are usually assessed clinically by a tendon tap whereas tonic stretch reflexes are assessed by the passive stretching of muscles (Mayer, Esquenazi and Keenan, 2007).

Together, the muscle spindles, GTOs, cutaneous efferents and higher brain centres modify muscle responses to produce and regulate active muscle tone (Ivanhoe and Reistetter, 2004). (For a more detailed description of these processes, see Kandel and Schwartz, 1985.) Following an UMN lesion in the motor cortex or the associated descending motor tracts, changes are observed firstly in spinal reflex activity (as the connections between the CNS and PNS are compromised) and secondly in soft tissue characteristics (Katz and Rymer, 1989; Morris, 2002b). These changes lead to either decreased muscle tone (hypotonia) or increased muscle tone (hypertonia). Hypotonia will not be discussed in this thesis. Hypertonia is a general term used to describe any abnormal increase in muscle tone (Anderson, Keith, Norvak and Elliot, 2002). If the increase in muscle tone is velocity dependent (assessed by moving limbs passively through their range at different speeds), then there is a neural contribution to the hypertonia which is indicative of spasticity (Morris, 2002b).

2.2 Spasticity

2.2.1 Definition

Spasticity is a complex phenomenon that continues to attract much attention in the clinical and research arenas. The word spasticity itself originates from the Greek word *spastikos* meaning to tug or to draw (Farnan and Saulino, 2002; Losseff and Thompson, 1995). The fact that there is still no universally accepted definition for spasticity highlights the many problems that still exist regarding its exact aetiology, quantification and treatment (Ibuki and Bernhardt, 2008)[1].

In the 1950s, the concept of spasticity as a velocity-dependent increase in stretch reflexes was proposed, which at the time was quite innovative (Morris, 2002b). In 1980, the concept was written into the widely utilised definition of spasticity as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (Lance, 1980, p485). Since this

[1] A copy of the published manuscript that further explores the issue of defining spasticity is included as Appendix A.

definition of spasticity was proposed, understanding of the phenomenon has grown and this definition now seems too narrow focused. Spasticity is not as straight forward as a velocity-dependent increase in stretch reflex activity as it was once thought. Spasticity is a collection of clinical manifestations of both reflex and non-reflex (mechanical) related symptoms of the UMN syndrome, and Lance's definition does not wholly encompass this (Burridge, Wood, Hermens *et al.*, 2005; Wood, Burridge, Van Wijck *et al.*, 2005).

Recently, a consortium known as SPASM (Support Programme for Assembly of database for Spasticity Measurement), which is funded by the European Commission and made up of an international group of experts in the field, proposed a new definition for spasticity. They defined it as "disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles" (Burridge *et al.*, 2005; Pandyan *et al.*, 2005, p5). This new definition purposely excluded the term "velocity-dependent" to steer away from the perception that spasticity is entirely neural with no mechanical component.

It appears that the definition proposed by the SPASM group was based on a paper by Tardieu in 1954, and in an attempt to cover all aspects of spasticity, the definition has become too broad in comparison with Lance's definition. Therefore, spasticity has been defined for the purposes of this thesis as a pathological increase in muscle tone that occurs following an UMN lesion, and presents as intermittent or sustained involuntary muscle activation that is characteristically velocity dependent (due to its reflex elements), but is also comprised of non-reflex (mechanical) elements (such as changes in muscle and soft tissue characteristics). It is believed that this definition covers all of the aspects of spasticity that exist to our knowledge today and can be applied in both a research and clinical context. However, it is also recognised that as our knowledge of spasticity increases, the definition for it may need to be adjusted accordingly.

2.2.2 Epidemiology and symptoms

Conditions that may lead to spasticity include trauma, congenital inherited metabolic defects, acquired metabolic defects, stroke, spinal cord injury (SCI) and demyelinating

disease (Hudgson, 1976). To convey the prevalence of spasticity, it has been reported to occur in approximately:

- 70% of people following SCI (Adams, Martin and Hicks, 2007; Biering-Sorensen, Nielsen and Klinge, 2006; Meythaler, 2001);
- 60% of people following stroke (Wallesch, Maes, Lecomte and Bartels, 1997; Watkins, Leathley, Gregson *et al.*, 2002);
- 78% of people with multiple sclerosis (MS) (Naghdi, Ansari, Mansouri *et al.*, 2008); and
- 80% of people with cerebral palsy (CP) (Carlson *et al.,* 1997; Scherger, 1995).

Burridge *et al.* (2005) reported that approximately 75% of people with severe acquired brain injury (ABI), 20% of people with stroke and 60% with MS require treatment for spasticity itself, as well as its associated problems.

Symptoms following spasticity depend on factors such as the severity and location of the lesion, the age of the person affected and the presence of associated disorders of other pathways (Gautier-Smith, 1976; Ward, 2003). As a result, the symptoms of spasticity are highly variable between individuals and the diagnosis and treatment of spasticity cannot be generalised. The main global consequence of spasticity is the inability to produce selective, functional, controlled movements of body segments which are essential for normal postural reactions and skilled movements (Gans and Glenn, 1990; Goff, 1976). Spasticity is also usually accompanied by muscle weakness which mostly affects the extensor muscles in the upper limbs, and the flexor muscles in the lower limbs (Hudgson, 1976).

Not only does spasticity affect motor control but it can also contribute to poor balance, falls, poor bowel and bladder function, pain, spasms, clonus and fatigue (Hsieh, Wolfe, Miller, Curt and Team, 2008; Hudgson, 1976). It increases metabolic demands (Farnan and Saulino, 2002; Meythaler, 2001) and the risk of joint contracture development (Selles, Li, Lin *et al.*, 2005). Spasticity can also negatively affect body image, self esteem, social participation, emotional and mental health, sleep and sexual function, among many other things (Adams *et al.*, 2007; Lechner, Kakebeeke, Hegemann and Baumberger, 2007; Priebe, 2006). Furthermore, spasticity negatively affects activities of daily living (ADLs) and adds to the cost of medications and attendant care (Hsieh *et al.*,

2008). Sufferers of spasticity will further testify that it can be aggravated by pain, poor positioning, infections, constipation, urinary retention, skin irritation or pressure sores, fatigue, stress, drugs and even weather (Moore, 1998). In surveys related to perceptions of problems associated with SCI, patients consistently ranked spasticity among their top four life concerns (Mahoney, Engebretson, Cook *et al.*, 2007).

2.2.3 Pathophysiology

The mechanisms precipitating spasticity are still not completely understood and remain controversial (Galiana, Fung and Kearney, 2005; Morris, 2002b; Satkunam, 2003). However, it is quite clear that spasticity develops as a result of lesions causing malfunctioning transmissions in the spinal networks of the descending reticulospinal and vestibulospinal pathways (Biering-Sorensen *et al.*, 2006; Brown, 1994; Hudgson, 1976). Lesions in the motor pathways result in a loss of control over spinal reflexes which are normally tightly regulated by supraspinal centres. This loss of control leads to either too much facilitation (abnormal enhancement of activity in facilitatory systems) or too little inhibition of MNs (an abnormal decrease in the activity of inhibitory systems) (Meythaler, 2001; Sheean, 2002; Singer, Dunne and Allison, 2001b; Wyke, 1976). In other words, there is an imbalance in the system between the inhibitory and excitatory inputs into the MNs that originate from changes in the way the segmental spinal cord circuitry handles information from proprioceptive, exteroceptive, and suprasegmental descending inputs (Meythaler, 2001). Input signals at the level of the spinal cord are "mishandled" and result in signals gaining access to alpha MNs causing their depolarisation, where normally those signals would be subject to supraspinal inhibitory control (Mayer *et al.*, 2007).

It is generally acknowledged that spasticity is of neurological origin (Wyke, 1976). The initial neurophysiological disturbance then leads to a secondary biomechanical disturbance in the limbs and body segments affected. Therefore, spasticity has two elements, a reflex (neural) element and a non-reflex (mechanical) element. The contribution of both of these elements makes up the signs of spasticity which are clinically manifested. These two elements will now be discussed in detail.

2.2.3.1 The reflex element of spasticity

Spasticity occurs in response to an UMN lesion and generally requires time to develop after the initial injury (Carr *et al.*, 1995; Chapman and Wiesendanger, 1982; O'Dwyer, Ada and Neilson, 1996b). This period could be days or even months post-injury during which there is a transition in the muscles from being flaccid with hypoactive or absent reflexes, to developing hyperactive reflexes (Little and Halar, 1985; Ward, 2003). This suggests that plastic changes in synaptic connections may contribute to the development of spasticity such as (Carr *et al.*, 1995; Chapman and Wiesendanger, 1982):

1. The formation of new synaptic connections through axonal sprouting;
2. Increased chemical sensitivity (an increase or abnormal sensitivity of pre- or post-synaptic elements to the afferent input); and
3. Changes in the normal patterns of synaptic use (where previously inactive synapses become active).

These suggestions all refer to the reflex or neural element of spasticity. Hypotheses that together, or in some combination explain the reflex element of spasticity include (Harburn, Hill, Vandervoort *et al.*, 1992; Ivanhoe and Reistetter, 2004):

1. Alteration in the motorneuronal electrical properties, thereby lowering their threshold for discharge;
2. Fusimotor hyperactivity;
3. Hyperactive MNs; and
4. A facilitation of the synaptic excitability of MNs during muscle stretch that may include interneuronal facilitation via muscle and or flexor reflex afferents from loss of supraspinal influences (inhibitory and/or excitatory).

Changes in the reflex element of spasticity can be broken down into the following components - increased tonic stretch reflexes, increased phasic stretch reflexes, increased cutaneous reflexes and increased pathologic radiation of reflexes between spinal segments (Lechner, Frotzler and Eser, 2006; Skold, 2000). Although spasticity is characterised by a velocity dependent increase in reflex activity, tonic rather than phasic stretch reflexes are generally recognised to be of greater significance in the overall phenomenon of spasticity (O'Dwyer and Ada, 1996a). A study conducted on the biceps brachii muscles of hemiplegic and normal subjects, found differences in

electromyographic (EMG) activity in response to passive displacement of the elbow joint between the two subject groups (Thilmann, Fellows and Garms, 1991a). The investigators attributed the increased EMG activity of the hemiplegic subjects to increased stretch reflex activation due to a pathological increase in stretch reflex gain. The EMG activity was highly correlated with displacement velocity, and the duration of the EMG activity was highly correlated with the duration of the applied displacement.

It has previously been suggested that when the CNS is damaged in adults, primitive tonic reflexes that are normally latent return to dominate motor activity and may contribute to patterns of spasticity (Duncan, 1960; Duncan and Mott, 1983; Lohman and Goldstein, 1993). Although there is no evidence that spasticity in adults is caused by the return of primitive reflexes, this theory will be discussed here as some tone-reducing principles in orthotics were developed based on this theory. Primitive reflexes are readily elicited in normal infants during the first year of life by gentle pressure, stroking or vibratory stimuli. The disappearance of primitive reflexes as clinical entities during the early years of childhood is thought to be due to suppression accomplished by the maturing cerebral cortex, however, they may also function throughout life as the peripheral inputs for automatic postural recovery systems (Duncan and Mott, 1983; Pratt, 2000). When primitive reflexes persist throughout childhood, the suppressive ability of the cortex is said to be impaired, as may be observed in children with CP (Pratt, 2000).

These primitive tonic reflexes are deployed in such a pattern that they are capable of reacting to displacement of the centre of gravity in the upright body to maintain upright posture. In addition to the local corrective muscle contraction as a result of cutaneous stimulation, it is thought that each reflex recruits participation of proximal musculature in the thigh, hip and trunk (Duncan and Mott, 1983). These reflexes are important in the regulation of upright posture and also in controlled, coordinated movements such as walking.

Following an UMN lesion, it is said that the supraspinal suppression of these primitive tonic reflexes is compromised and they can return as tonic movements often extremely difficult to control voluntarily (Duncan and Mott, 1983). The reflexes and their associated movements can become problematic, especially if they are left unopposed and

uncontrolled. Constant stimulation of the reflexes is said to cause lower limbs to adopt the positions of the reflex movements that are being stimulated (Duncan, 1960). This is believed to lead to deformities, contractures and muscle imbalances (Lima, 1990).

2.2.3.2 The mechanical element of spasticity

Spasticity is frequently explained from a neurological standpoint, and less emphasis is placed on the mechanical changes that occur within spastic muscles. Over the last few decades, researchers have determined that non-reflex or mechanical elements (otherwise referred to as muscle thixotropic factors) also contribute to spasticity alongside the reflex elements mentioned above (Carr *et al.*, 1995; Singer *et al.*, 2001b). This new insight into spasticity has received considerable attention, generated new research and called for a revision of past knowledge (Malouin, Bonneau, Pichard and Corriveau, 1997).

A key study on the mechanical element of spasticity was conducted by Sinkjaer and Magnussen (1994) who measured the passive stiffness (response from passive soft tissues), intrinsic stiffness (response from the properties of fibres contracting prior to a stretch) and reflex stiffness (response from the stretch reflex-mediated contraction of muscle fibres) of the ankle plantarflexor muscles during isotonic contractions in nine hemiplegic subjects and eight healthy control subjects. The investigators found that the passive stiffness of the spastic legs was significantly increased by 94% compared with the contralateral legs, and by 278% compared with the control subjects. In addition, the passive stiffness of the contralateral legs in the hemiplegic subjects was increased by 95% compared with the healthy subjects. Traditionally, the increased resistance encountered during passive lengthening of a spastic muscle was thought to result principally from inappropriate muscle activation associated with velocity dependent hyperexcitable tonic stretch reflexes. However, the increased passive stiffness observed in this study was more likely to be a result of mechanical elements since the investigators also found that the reflex stiffness of both legs of the hemiplegic subjects were within the normal ranges of reflex stiffness found in the control subjects. There were no significant differences for intrinsic stiffness between the spastic leg, contralateral leg and the leg of the control subjects.

The theory behind the contribution of the mechanical elements of spasticity is also based on the results of studies conducted on spastic muscles that have found increases in the mechanical response to passive muscle stretch without a parallel increase in EMG activity of the stretched muscle (Damiano, Quinlivan, Owen *et al.*, 2002; Malouin *et al.*, 1997). More studies are now being performed to try to separate the reflex and mechanical elements of spasticity by examining the mechanical features of muscles before and after altering aspects of the reflex elements (Chung, Van Rey, Bai *et al.*, 2008). There have even been a number of studies conducted suggesting that mechanical elements have a more profound and consistent effect on spasticity than reflex elements (Chung *et al.*, 2008; Singer *et al.*, 2001b). However, this may be dependent upon the time elapsed since the onset of spasticity, as it has been suggested that the mechanical elements play more of a role as spasticity becomes more chronic (Thilmann *et al.*, 1991a).

Some electrophysiological studies have shown abnormally high levels of EMG activity in the tibialis anterior muscle during the swing phase of gait despite a lack of EMG activity in the antagonist triceps surae muscles (Brown, 1994; Dietz, Quintern and Berger, 1981; Thilmann, Fellows and Ross, 1991b). These studies demonstrated no evidence of co-activation of the tibialis anterior muscles and triceps surae muscles during the swing phase. This suggested that mechanical changes in muscle fibre properties (and possibly connective tissues) of the triceps surae muscles were responsible for the increased resistance to dorsiflexion movement rather than over-activity of the plantarflexor muscles (Dietz *et al.*, 1981).

The mechanically mediated stiffness discussed in these studies is comprised of responses from passive tissues and muscle fibres. The stiffness of passive tissues could be attributed to alterations in connective tissues, tendons, ligaments or joint capsule compliance (Chung *et al.*, 2008). The stiffness of muscle fibres could be attributed to physiological, morphometric or histochemical changes in the mechanical properties of active motor units affected by the contractile properties of the engaged cross-bridges (Brown, 1994; Singer *et al.*, 2001b; Voerman, Gregoric and Hermens, 2005). It has been shown that the resting sarcomere length of muscle cells is shorter in spastic muscles with increased modulus of elasticity, suggesting remodeling of structural muscle components (Galiana *et al.*, 2005; Mirbagheri, Alibiglou, Thajchayapong and Rymer, 2008). The

occurrence of changes in fibre size and fibre type distribution and potentially fibre length secondary to spasticity may also contribute to the mechanically mediated stiffness (Mirbagheri *et al.*, 2008).

Mechanically mediated stiffness could also be caused by limited use of spastic muscles causing fibrotic changes in the collagen tissues of tendons, joint capsules or muscle fibres over time (Nielsen and Sinkjaer, 1996; Singer *et al.*, 2001b). In chronic spasticity, this can lead to a permanent shortening of muscles and connective tissues, resulting in reduced active and passive joint mobility, which is defined as contracture (Boyd and Ada, 2001; Mehrholz, Wagner, Meibner *et al.*, 2005). A vicious cycle might arise whereby spasticity leads to muscle contracture, which in turn potentiates spasticity (O'Dwyer *et al.*, 1996b).

2.2.4 Summary

This review has only painted a brief picture of spasticity but has highlighted the complexity of this phenomenon. It is still unknown how much responsibility the reflex or mechanical elements have in contributing to the overall phenomenon of spasticity because of the difficulty in separating the two elements to assess their effects individually (Galiana *et al.*, 2005). Further research is clearly required to determine the exact influence of the mechanical elements in the development of spasticity, especially as spasticity becomes more chronic over time (Malouin *et al.*, 1997). The good news is however, that as more information is being unveiled about this phenomenon, the picture of spasticity is becoming clearer and this should lead to more targeted and effective treatment strategies.

2.3 Treatment of spasticity

The treatment of spasticity is a complex issue. With an aging population, advances in medicine and an increasing awareness of neurological disorders like stroke, the number of people developing spasticity is undoubtedly increasing due to higher survival rates following neurological attacks. Spasticity can be hugely disabling and its effect on ADLs and quality of life can be significant. The need for targeted treatment strategies for people with spasticity is an important issue requiring much attention and development. Unfortunately, since spasticity is not life-threatening, it may not be perceived as having high priority in the field of medical research.

The complexity of the mechanisms behind spasticity have made any attempt to treat and assess treatment very difficult (Brown, 1993). So far, there is no single method which alone can solve the problem of spasticity (Odeen, 1981a) and perhaps there never will be since spasticity is a combination of different elements. Despite the difficulties behind treating spasticity it is still a necessary and important aspect of clinical care which needs to be carefully considered, administered and monitored.

The main aims of treatment for spasticity are (Losseff and Thompson, 1995):

1. Functional improvement;
2. Prevention of complications or the development of further spasticity; and
3. Relief of pain and discomfort.

To achieve these aims, a combination of treatment strategies generally needs to be adopted to address both the reflex and mechanical elements of spasticity.

Treatments for spasticity can generally be separated into two categories - invasive methods and conservative methods. Invasive methods include the use of antispasticity drugs and surgical interventions. Drugs can be classified as those which act centrally either as depressors of cortical and supraspinal activity or on internuncial neurons within the spinal cord, those which act as neuromuscular blocking agents and those which act on the muscles themselves (Gautier-Smith, 1976). Such drugs include baclofen, dantrolene, diazepam, tizanidine and Botulinum toxin-A (BTX-A) (Barnes, 2001b).

Surgical interventions can be categorised according to procedures that interfere with the neuronal pathways and those that correct musculoskeletal deformity (Bhakta, 2000). These include peripheral nerve blocks, posterior or anterior root rhizotomies (Levine, Kabat, Knott and Voss, 1954), DREZ-otomies (dorsal root entry zone), myelotomies (Gelber and Jozefczyk, 1999), cordotomies (Lima, 1990), peripheral neurectomy (Levine *et al.*, 1954; Lima, 1990), tenotomy/tendon transfers, tendon lengthening (Lima, 1990) and joint arthrodeses.

The disadvantages of these invasive methods are that they can be associated with serious risks and detrimental side effects. Surgical interventions especially neurological interventions may not be reversible and drug treatments may only have temporary effects with the possibility of negative side effects on muscle strength and cognitive function (Bhakta, 2000). Therefore, conservative methods for reducing spasticity are usually preferred in the first instance (Lechner *et al.*, 2007). In cases where conservative methods are deemed ineffective, pharmacological and surgical interventions may be necessary and beneficial. Conservative methods of treatment may include physiotherapy, inhibitory casting, heat and cold therapies, electrical stimulation and orthotics (Barnes, 1998). The last of these is the focus of this thesis.

2.4 Orthoses

An orthosis is defined by the International Standards Organisation (1989) as an externally applied device used to modify the structural or functional characteristics of the neuromuscular and skeletal systems. Orthoses are used as reinforcements for parts of the body when function and mechanics are not optimal (Akashi, 2004). Orthoses in general aim to prevent and correct deformity, maintain positioning, provide support and control, correct undesirable movements and improve function (Akashi, 2004; Cusick, 1988; Yamanaka, Ishii and Suzuki, 2004b).

All orthotic devices directly affect the biomechanics of target body segments to achieve these aims via the application of carefully targeted external forces to body segments in order to encourage or prevent certain movements. In some cases, movements can be completely restricted when the aim is to achieve immobilisation.

2.4.1 Tone-reducing orthoses

There is a particular group of orthoses that were developed with the aim to more effectively treat spasticity through targeted neurophysiological mechanisms. These orthoses are termed tone-reducing orthoses (TROs) and have been referred to as neurological orthoses/splints, inhibitive orthoses/splints and dynamic orthoses/splints. There are numerous designs of TROs that have been described in the literature, but the theory and principles behind how they work are the same. This thesis will refer to them collectively as TROs and will not use the different names that have been given to specific designs since there is no consistency in the literature. For example, Dieli, Ayyappa and Hornbeak (1997), refer to the AFO in their study as a dynamic AFO, whereas Siegel and Bernardoni (1997), refer to an almost identical AFO as a supramalleolar tone-balancing orthosis.

Since the term "tone-reducing orthosis" will be used throughout this thesis, clarification of the terminology is required to avoid confusion as the terms "tone" and "spasticity" do not mean the same thing. While these orthoses are called "tone-reducing", their aim is actually to reduce spasticity (see section 2.2.1 for a definition of spasticity), not just muscle tone (see section 1.1 for a definition of muscle tone). These orthoses would probably be more accurately defined as "spasticity-reducing orthoses" however the term "tone-reducing orthoses" is commonly used throughout the literature and is known among clinicians when referring to these distinct orthotic devices.

The concept and development of TROs especially for the treatment of spasticity in the lower limbs appears to have stemmed from the theory and practice of inhibitory casting (otherwise known as serial casting) (Lin *et al.*, 2000; Zachazewski *et al.*, 1982). Inhibitory casting is a neurophysiological treatment tool that has been widely used for several decades as an adjunct to therapeutic interventions for the treatment of spasticity (Carlson, 1984). Inhibitory casts for the lower limbs are typically made of plaster-of-Paris and are applied below the knee with the foot and ankle in a neutral position, i.e. with the subtalar joint neither inverted or everted and the ankle joint in 90 degrees (Carlson, 1984; Sussman and Cusick, 1979). Inhibitory casts involve specific tone-reducing features to inhibit or facilitate the CNS to alter motor output (Carlson, 1984). These features include inhibitory toe bars (ITBs), the application of pressure to tendons,

orthokinetics, metatarsal domes, circumferential pressure around muscle bellies, internal heels, hyperextension footplates, compression into joints through weight bearing, prolonged stretch and positioning the ankle in a neutral position (Blashy and Fuchs, 1959; Carlson, 1984; Lohman and Goldstein, 1993; Sussman and Cusick, 1979). (Further details of these tone-reducing features are provided in subsequent chapters.) Inhibitory casting is often used in conjunction with physical therapy to treat spasticity by improving patterns of movement and function (Watt, Sims, Harckham *et al.*, 1986).

The theory behind inhibitory casting suggests that the casts can reduce spasticity by (Carlson, 1984; Smelt, 1989):

1. The autogenic inhibitory response to prolonged stretch of the Ib afferent fibres of GTOs;
2. Inhibiting primitive reflexes in the foot through the use of contoured footplates to achieve a more controlled alignment of the foot and to stimulate or inhibit specific reflexogenous areas on the sole of the foot;
3. Stabilising the base of support for weight-bearing;
4. Positioning the extremity in a reflex-inhibiting pattern of neutral ankle position with extension or hyperextension of the toes; and
5. Applying total contact to all surfaces of the foot and lower leg to alter cutaneous stimulation.

The disadvantages of inhibitive casts are that they are heavy, cumbersome, friable, permeable and not adjustable. To overcome these disadvantages the neurophysiological principles of inhibitory casting were isolated and developed into specific tone-reducing features, which were then incorporated into orthoses. The earliest literature on TROs emerged in the early 1980s, and literature has suggested that they are just as effective as inhibitory casts for the treatment of lower limb spasticity (Harris and Riffle, 1986).

TROs for the foot and ankle are referred to as tone-reducing ankle-foot orthoses (TRAFOs) and are frequently prescribed for the treatment of patients with spasticity in the lower limbs, where the spasticity has a detrimental effect on the functioning of the motor system (Weber, 1990).

The specific aims of TRAFOs are to (Charlton and Ferguson, 2001):

1. Reduce or inhibit an abnormal spastic reaction by proper positioning of the limb;
2. Prevent abnormal movement patterns;
3. Promote normal alignment and normal movement;
4. Prevent contractures and maintain joint ranges; and
5. Target motor learning;

Where it is achievable, orthotic interventions aim to realign the limb segments as near as possible to normal positioning in the hope that normal posture will result and muscle groups will become appropriately active (Charlton and Ferguson, 2001).

Any splint or orthotic device can be modified to include tone-reducing features in an attempt to achieve these aims. This includes orthoses for the upper limbs, however, this thesis will mainly focus on orthoses for the lower limbs as the functions of the upper limbs and lower limbs are very different. Although it may be possible that all standard orthosis designs have some tone-reducing benefits (for example, by improving limb positioning and applying stretch to muscles), TROs use a more deliberate approach to specifically reduce or inhibit spasticity neurophysiologically (Nash *et al.*, 2008). When used appropriately, it is said that the TROs perfect and enhance effective orthotic control for patients with spasticity (Rogers and Vanderbilt, 1990).

2.4.2 Theories regarding how tone-reducing orthoses work

Patients with spasticity generally have a combination of neurophysiological (muscle control) and biomechanical problems as the body is not merely a passive recipient of mechanical inputs, but a reactive entity (Hylton, 1990a). Thus, orthoses act within two environments; the purely mechanical one and the body's own physiological one (Pratt, 2000). TRAFOs are purported to function in two ways: to improve the biomechanics of a limb and improve the neurophysiological control of a limb (Teplicky *et al.*, 2003).

The biomechanical effect of TRAFOs primarily acts to apply external forces to limbs to alter or control their positioning and movement. This is done to mechanically stabilise and control the motion of joints, thus decreasing the reaction of muscle spindles to stretch (Gelber and Jozefczyk, 1999). TRAFOs also maintain muscles at certain lengths to

provide prolonged stretch on muscles and tendons (Corn, Imms, Timewell *et al.*, 2003; Lin *et al.*, 2000) which can help to discourage contracture formation and the development of fixed deformity. Maintaining the foot and ankle structures in a correct alignment is also said to facilitate improved function (Cusick, 1988) while supporting the body in a position that promotes more typical muscle balance (Lin *et al.*, 2000; Teplicky, 2002). It is believed that each spastic foot has an optimum position relative to the leg which will produce the most significant tone-reducing effects (Ricks and Eilert, 1993; Sankey, Anderson and Young, 1989). TRAFOs also function to redistribute pressures, especially on the plantar surface of the foot which can play an important role in the treatment of spasticity by redistributing the pressures which are said to stimulate primitive tonic reflex responses.

The neurophysiological effect of TRAFOs is much more complex. Knowledge of spinal reflex pathways (particularly those that innervate spastic lower limb muscles) is required to understand the theories behind how TRAFOs are said to function. As was previously discussed, spasticity occurs when the inhibitive influence and control from higher brain centres on spinal reflex activity is compromised. Within the spinal reflex arc, the MN serves as the final conduit, carrying a host of different synaptic and modulating influences including:

1. Pre-synaptic inhibition initiated by descending fibre input;
2. Inhibitory post-synaptic potentials from interneuronal connections from antagonistic muscles; and
3. Excitatory post-synaptic potentials from type Ia and type II afferent fibres.

Hence, there is potential to influence the neural mechanisms underlying spasticity by changing the level of MN excitability (Morris, 2002a). This can be done by stimulating various cutaneous receptors in the skin, GTOs and reflexogenous areas on the sole of the foot. TRAFOs are also said to work by decreasing the effects of detrimental stimuli such as pressure on the metatarsal heads which triggers the positive support reaction (the extensor muscle activation in response to loading the foot) (Charlton and Ferguson, 2001).

It is said that neurophysiologically, TRAFOs act to:

1. Alter spasticity via sensory feedback (Lohman and Goldstein, 1993; Pratt, 2000);

2. Prevent the stimulation of reflexes induced by tactile stimulation especially reflexes of the foot (Lin *et al.*, 2000);
3. Influence proprioceptors through joint compression provided by weight-bearing in the proper alignment (Lin *et al.*, 2000);
4. Alter muscle length to improve recruitment and sequencing of muscle activity (Lin *et al.*, 2000; Teplicky, 2002);
5. Apply continuous pressure at the point of a muscle insertion specifically the Achilles tendon to stimulate GTOs (Nash *et al.*, 2008); and
6. Influence cutaneous receptors via the close contact of materials with different textures (Blashy and Fuchs, 1959; Lohman and Goldstein, 1993).

TRAFOs are believed to produce these neurophysiological effects to enhance function by improving voluntary control over spastic muscles. While TRAFOs may only have a direct effect on foot and ankle joint mechanics and physiology, this distal control is believed to exert a positive effect on more proximal joints and muscles throughout the body as well (Abel, Juhl, Vaughan and Damiano, 1998).

2.4.3 Critical review of the orthotic literature

A search of the literature was conducted using PubMed, MEDLINE, CINAHL and a hand search of lists of references in identified papers to find further papers. Search terms included “orthosis”, “orthoses”, “ankle”, “foot”, “splint”, “brace”, “cast”, “tone-reducing”, “tone-inhibiting”, “inhibitory”, “dynamic”, “spasticity”, “spastic” and “hypertonia”. Any study or report that mentioned an orthotic device used for the purpose of treating spasticity neurophysiologically was collected and reviewed.

The body of literature regarding TROs is unfortunately very limited and largely comprised of low level evidence papers such as observational studies and expert opinions (Dieli *et al.*, 1997). Therefore, it was not possible to conduct a systematic review of the literature. Of the literature available, research has been conducted on individual tone-reducing features and devices, various designs of TRAFOs, as well as TROs for the upper limbs. Studies have been conducted on several subject groups including both

children and adults with spasticity resulting from various neurological disorders, but predominantly CP and stroke.

Experimental studies that have been conducted on TRAFOs generally report favourable results following their use, and most authors of observational studies write positively of their experiences with TRAFOs. The possible benefits of TRAFOs that have been described in the literature include:

- Improved gait function (Bronkhorst and Lamb, 1987; Crenshaw *et al.*, 2000; Diamond and Ottenbacher, 1990; Dieli *et al.*, 1997; Ford *et al.*, 1986; Iwata *et al.*, 2003; Nash *et al.*, 2008);
- Carry-over effects of positive gait improvements when the TRAFOs were removed (Bronkhorst and Lamb, 1987);
- Improved ankle joint position (Mills, 1984; Taylor and Harris, 1986);
- Improved ankle joint ROM (Bronkhorst and Lamb, 1987);
- Inhibition of the tonic reflexes in the foot (Bronkhorst and Lamb, 1987; Iwata *et al.*, 2003);
- Inhibition of the positive support reaction (Taylor and Harris, 1986; Zachazewski *et al.*, 1982);
- Improved posture and symmetry (Harris and Riffle, 1986; Taylor and Harris, 1986; Zachazewski *et al.*, 1982);
- Improved balance (Harris and Riffle, 1986);
- Decreased hypertonicity and spasticity (Iwata *et al.*, 2003; Shamp, 1990);
- Improved upper limb position (Taylor and Harris, 1986); and
- Positive subjective reports by patients (Iwata *et al.*, 2003).

This forms quite an impressive list, however, with such claims comes much skepticism. Interestingly, the few controlled experimental studies that have been conducted on TRAFOs were unable to provide solid evidence to support the many perceived advantages of TRAFOs that have been documented by descriptive studies (Crenshaw *et al.*, 2000; Lam *et al.*, 2005; Naslund *et al.*, 2005; Romkes and Brunner, 2002). As a result, it remains unclear how effective TRAFOs are in reducing spasticity and which TRAFO designs and features may be the most effective.

There are a number of limitations evident when examining previous research that has been conducted on TRAFOs. These are detailed in Table 2.1 but will be described in further detail here. The majority of research consists of single subject case studies which limits the generalisability of the results. Researchers who have used such study designs state that the symptoms of spasticity are so varied between individuals that it is difficult to obtain a homogenous subject group (Smelt, 1989), and therefore, single subject case studies are the most appropriate study design.

Uncontrolled orthosis mechanics introduced a number of limitations into studies. Many studies compared TRAFOs with different orthosis designs such as dynamic AFOs, supramalleolar orthoses, leaf spring AFOs, solid AFOs and hinged AFOs. Differences in the effects of permitted ROM at the ankle joint, length of lever arms and location of trimlines means that changes in outcome measures could have been due to these mechanical differences rather than due to any neurophysiological effects. There were also studies that compared prefabricated orthoses with custom-made orthoses (Diamond and Ottenbacher, 1990; Dieli *et al.*, 1997) which may have resulted in differences in the fit and comfort of the orthoses. Some investigators identified these limitations in their studies and suggested that further research be conducted to control the orthosis mechanics in order to adequately isolate differences resulting from tone-reducing features (Dieli *et al.*, 1997).

The effect of the tone-reducing features of the TRAFOs was difficult to determine in some studies due to methodological issues. Several studies used a barefoot baseline condition and then tested the TRAFOs with shoes on without distinguishing between the effects of the shoes and the effects of the TRAFOs. It is known that the use of shoes alone may positively affect gait characteristics when compared with barefoot conditions (Rogers De Saca, Catlin and Segal, 1994). Some studies also lacked adequate control groups or control conditions so that it cannot be determined with confidence that the results observed were solely due to the tone-reducing effects of the orthoses and not due to potential series effects (eg. learning). This applies especially to those studies where subjects continued to receive concurrent therapies (such as physiotherapy) during the testing period. There were also some studies tested TRAFOs alone and provided no other orthosis for comparison. Of the studies that compared TRAFOs with similar AFO

designs, several found no significant differences between the orthoses suggesting that the addition of tone-reducing features to the orthosis designs provided no further advantages (Crenshaw *et al.*, 2000; Hassani *et al.*, 2004; Radtka, Skinner and Johanson, 2005).

Table 2.1 List of published experimental studies on TRAFOs in chronological order. The main limitations of each study are identified.

Authors and Year	**Orthosis design tested**	**Cause of spasticity of the subject group tested**	**Single subject design**	**Barefoot baseline condition while orthoses tested with shoes**	**Subjects received other therapies during the testing period**	**No other orthosis used for comparison**	**Where two orthoses were compared, orthosis mechanics were not controlled**	**Did not measure any aspect of spasticity directly**
Zachazewski *et al.*, (1982)	TRAFO	ABI	✓		✓	✓		✓
Mills, (1984)	Bivalved inhibitory splint*	ABI and stroke				✓		✓
Harris and Riffle, (1986)	TRAFO and SMO*	CP	✓	✓	✓	✓		✓
Taylor and Harris, (1986)	TRAFO	CP	✓		✓	✓		✓
Bronkhorst and Lamb, (1987)	TRAFO	CP and ABI	✓			✓		✓
Diamond and Ottenbacher, (1990)	DAFO* and LSAFO*	Stroke	✓	✓			✓	✓

Table 2.1 Continued.

Authors and Year	Orthosis design tested	Cause of spasticity of the subject group tested	Single subject design	Barefoot baseline condition while orthoses tested with shoes	Subjects received other therapies during the testing period	No other orthosis used for comparison	Where two orthoses were compared, orthosis mechanics were not controlled	Did not measure any aspect of spasticity directly
Embrey *et al.*, (1990)	Inhibitive SMO*	CP	✓		✓	✓		✓
Mueller *et al.*, (1992)	DAFO*	Stroke	✓	✓		✓		✓
Dieli *et al.*, (1997)	DAFO* and LSAFO*	Stroke		✓			✓	✓
Radtka *et al.*, (1997)	DAFO* and SAFO*	CP		✓			✓	✓
Crenshaw *et al.*, (2000)	TRAFO, HAFO* and SMO*	CP						✓
Romkes and Brunner, (2002)	DAFO* and HAFO with PF stop*	CP		✓			✓	✓
Iwata *et al.*, (2003)	AFO with ITB*	Stroke						

Table 2.1 Continued.

Authors and Year	Orthosis design tested	Cause of spasticity of the subject group tested	Single subject design	Barefoot baseline condition while orthoses tested with shoes	Subjects received other therapies during the testing period	No other orthosis used for comparison	Where two orthoses were compared, orthosis mechanics were not controlled	Did not measure any aspect of spasticity directly
Hassani *et al*., (2004)	DAFO* and HAFO*	CP		✓			✓	✓
Lam *et al.*, (2005)	DAFO* and SAFO*	CP		✓			✓	✓
Naslund *et al*., (2005)	DAFO*	CP				✓		✓
Nash *et al.*, (2008)	TRAFO	SCI	✓		✓			

TRAFO= Tone-reducing ankle-foot orthosis; ABI= Acquired brain injury; SMO= Supramalleolar orthosis; CP= Cerebral palsy; DAFO= Dynamic ankle-foot orthosis; LSAFO= Leaf-spring ankle-foot orthosis; SAFO= Solid ankle-foot orthosis; HAFO= Hinged ankle-foot orthosis; PF= Plantarflexion; ITB= Inhibitory toe bar; SCI= Spinal cord injury.

*The original terminology for the orthosis designs used by the authors of these studies has been reported here. The various designs differ mostly in their trimlines and ankle joint motion restriction.

Perhaps the most notable limitation of most TRAFO studies was in the outcome measures used to determine the effects of TRAFOs on spasticity. Most studies measured functional outcomes such as gait, posture and balance without measuring any aspect of spasticity itself. Consequently, those studies were unable to clearly support or refute their hypotheses regarding an effect on spasticity because their outcome measures did not measure spasticity. The investigators of those studies failed to adequately quantify the neurophysiological effect of TRAFOs, and therefore, could not provide evidence that TRAFOs were effective in reducing spasticity.

The few studies that assessed EMG as a dependent variable were unable to demonstrate statistically that TRAFOs had a significant effect on muscle activity (Lam *et al.*, 2005; Mills, 1984; Nash *et al.*, 2008; Smith, 1995). One study measured EMG during passive rest (Mills, 1984), another measured EMG during passive controlled movement of the ankle joint (Smith, 1995), and two studies measured EMG during walking (Lam *et al.*, 2005; Nash *et al.*, 2008). Radtka *et al.* (1997) also assessed EMG, but only to monitor the timing of lower limb muscle activation in children with CP while they walked. They found that neither solid AFOs nor TRAFOs significantly affected muscle timing when compared with barefoot conditions. The use of EMG for the evaluation of treatments for spasticity needs to be carefully considered. EMG can be useful to determine changes in the pattern of muscle activations during functional activities such as walking, but EMG measures from muscles during passive rest are unlikely to demonstrate any changes in spasticity. This is because the evaluation of spasticity by EMG in a relaxed muscle requires activation of the reflex element of spasticity which is dependent upon movement (Biering-Sorensen *et al.*, 2006).

A number of the studies mentioned above focused on assessing the effects of dynamic AFOs. Dynamic AFOs are TRAFOs that generally have lower proximal trimlines to allow for sagittal plane ankle movements. They also incorporate tone-reducing footplate features to provide an optimal weight-bearing surface for the foot (Lam *et al.*, 2005; Mueller *et al.*, 1992). The studies that assessed dynamic AFOs without comparison with another AFO design reported some improvements in measured outcomes over no-brace conditions (Mueller *et al.*, 1992; Naslund *et al.*, 2005). However, the studies that did compare dynamic AFOs with another AFO design found no significant differences between the orthoses (Hassani *et al.*, 2004), or found the dynamic AFOs to be less

effective (Lam *et al.*, 2005; Romkes and Brunner, 2002). The authors of these studies attributed the poorer results from the dynamic AFOs to their reduced lever arms (as a result of their lower proximal trimlines). The authors suggested that the reduced biomechanical effect could not be compensated by the tone-reducing features of the dynamic AFOs, suggesting that biomechanical effects of orthoses are more important than any potential neurophysiological effects.

Only two studies have been conducted on the effect of TRAFOs that adequately controlled the biomechanical components of the orthoses to determine their tone-reducing effects (Crenshaw *et al.*, 2000; Iwata *et al.*, 2003). Crenshaw *et al*. (2000) examined the effect of TRAFOs in children with CP and failed to demonstrate any significant functional improvements in gait with TRAFOs compared with standard AFOs. Iwata *et al*. (2003) examined the effect of TRAFOs (AFOs with the addition of ITBs) in hemiplegic adults and found that the TRAFOs were only effective in subjects who exhibited an active toe grasp reflex. In subjects who did not exhibit the reflex, there were no significant differences in gait performance.

In light of this, the potential benefits of TRAFOs observed by some clinicians and investigators need to be interpreted cautiously. A subject with spasticity in the lower limbs which has a detrimental effect on their gait is likely to improve with the use of an AFO, whether it has tone-reducing features or not, due to the biomechanical effect of the orthosis. The only way to assess the tone-reducing features of TRAFOs is to compare them with standard AFOs and control the biomechanical effects of both orthoses, as was done by Crenshaw *et al*. (2000) and Iwata *et al.* (2003). The limitation of these two studies was that both used gait parameters as their primary outcome measure and neither measured spasticity directly.

2.4.4 Summary

Tone-reducing concepts in orthotics are still promoted today by clinicians and centres worldwide, without substantial evidence to support any additional benefits over the standard orthosis designs into which they are incorporated (Teplicky *et al.*, 2003). A review of the literature demonstrates that TRAFOs have been reported to provide a considerable number of benefits for people with spasticity. If the claims of their

beneficial effects are true, then they can be utilised as an effective, non-invasive treatment to improve the lives of people with spasticity. However, if their claims are not true, then the time, effort and costs that go into making and fitting TRAFOs are unjustified.

Unfortunately, little objective data exists to demonstrate unequivocally beneficial effects of TRAFOs for people with spasticity (Lin *et al.*, 2000), and there is presently more support for the biomechanical rationale than there is for the neurophysiological rationale (Teplicky, 2002). This is because it has already been demonstrated that AFOs (whether tone-reducing or not) can be effective in altering the biomechanics of the lower limbs.

Lack of support in the literature for the neurophysiological effect of TRAFOs is mainly due to the fact that the majority of investigations have not measured spasticity directly. This stems in part from the difficulties in reliably measuring spasticity, especially in the clinical setting (Taylor and Harris, 1986). But this is not to say that there is no reliable way of measuring the neurophysiological effect of TRAFOs on spasticity. There are various methods available to quantify spasticity, some of which have not been previously utilised in TRAFO research. These methods will be explored in the next section.

2.5 The measurement of spasticity

The measurement of spasticity is a challenging problem due to the fact that there is no universally accepted definition for spasticity (Priebe, 2006). The development of a standard measure of spasticity is also hindered by the lack of understanding of the underlying mechanisms of spasticity (Haas and Crow, 1995), therefore, clinicians rarely measure it objectively beyond using a simple subjective scale (Ansari, Naghdi, Moammeri and Jalaie, 2006). But just because something is difficult to do, does not mean that it can be avoided. An objective measurement of spasticity is important because it (Goff, 1976; Haas, 1994):

1. Contributes to establishing a diagnosis;
2. Contributes to forecasting the outcome of disease or injury;
3. Aids treatment planning;
4. Provides a record of the state of the patient at any one time to indicate deterioration or improvement to evaluate the effect of treatment; and
5. Facilitates research.

There are numerous experimental methods for quantifying spasticity. Some methods are quite simple while others are much more complex and involve the use of sophisticated equipment. Methods for measuring spasticity can focus on quantifying aspects of the reflex elements (like stretch reflex activity) or the mechanical elements (like muscle and joint stiffness) (Damiano *et al.*, 2002).

Due to the multidimensional nature of spasticity with its reflex and mechanical elements, it is recommended that spasticity be objectively measured with a combined neurophysiological and biomechanical approach (Hsieh *et al.*, 2008; van der Salm, Veltink, Hermens, Ijzerman and Nene, 2005; Voerman *et al.*, 2005; Wood *et al.*, 2005). This means that in order to obtain a reliable and comprehensive measure of spasticity, both reflex and mechanical elements need to be quantified (Wood *et al.*, 2005). No single measurement tool will be able to assess both elements of spasticity completely and measuring only a single component of spasticity is likely to under-represent its magnitude and severity (Priebe, 2006).

Measurement tools for quantifying spasticity can be broken up into three groups. In order of complexity they are (Lechner *et al.*, 2006):

1. Clinical methods;
2. Biomechanical methods; and
3. Neurophysiological methods.

Some of these are commonly used in spasticity research but rarely used in the clinical setting. It has been shown that the perceived requirements of a clinical spasticity measurement tool are very different from a research tool due to the differences in what researchers and clinicians feel is important in a spasticity measurement tool (Burridge *et al.*, 2005). Researchers place high importance on distinguishing the various aspects and components of spasticity, while clinicians place more importance on a muscle's response to movement and its effect on function.

Each spasticity measurement method has different advantages and disadvantages which need to be recognised when selecting an appropriate measurement tool. This depends on numerous factors such as the aim of the measurement, the ability of the patient, the available instrumentation and the knowledge of the assessor. The main disadvantage of clinical and biomechanical methods is that they may not adequately distinguish between the reflex and mechanical elements of spasticity (Burridge *et al.*, 2005). The main disadvantages of neurophysiological methods are that they require expertise and specialised equipment, and that they may lack functional relevance. A brief overview of various measurement methods to assess treatment outcomes on spasticity will now be documented with particular focus on the H-reflex.

2.5.1 Clinical methods

Rating scales are the most common clinical method for measuring spasticity and would appear to be the simplest method for assessing spasticity (Haas and Crow, 1995). Numerous scales have been presented in the literature which all seem to measure different aspects of spasticity (Priebe, Sherwood, Thornby, Kharas and Markowski, 1996). Scales can measure components of the UMN syndrome associated with spasticity or assess the effect of spasticity on ADLs and participation, all of which may change in response to treatment (Burridge *et al.*, 2005). It has been shown that different clinical

scales correlate poorly with each other, supporting the idea that they all measure different aspects of spasticity (Hsieh *et al.*, 2008). The measurement of different aspects of spasticity may be important to define the most disturbing component of the condition in each patient (Burridge *et al.*, 2005).

Clinicians mainly identify spasticity by evaluating the level of resistance of a limb to passive movements (Haugh, Pandyan and Johnson, 2006). The resistance is due to neural and biomechanical factors, however most clinical measures are unable to identify the exact cause of the increased resistance (O'Dwyer and Ada, 1996a; Yelnik, Albert, Bonan and Laffont, 1999). The success of evaluating spasticity by these rating scales also lies greatly on the ability and impartiality of the assessor as these scales can be prone to poor inter-rater and inter-session reliability. The number of spasticity rating scales that have been proposed is quite large, and Table 2.2 lists a few that have been used in the literature.

Table 2.2 Spasticity rating scales.

Name of scale	What does it measure?	Reference
Ashworth Scale	Resistance to passive movement	Ashworth (1964)
Modified Ashworth Scale (MAS)	Resistance to passive movement	Bohannon and Smith (1987)
Modified Modified Ashworth Scale	Resistance to passive movement	Ansari *et al.* (2006); Naghdi *et al.* (2008)
Tardieu Scale	Resistance to passive movement and dynamic catch	Haugh *et al.* (2006)
Modified Tardieu Scale	Resistance to passive movement and dynamic catch	Boyd and Ada (2001); Morris (2002b)
The Spasticity Test	Resistance to passive movement and dynamic catch	Van Den Noort *et al.* (2009)
Spasm Severity Scale	Spasm severity	Priebe *et al.* (1996)
Spasm Frequency Scale	Spasm frequency	Penn (1988)
Tendon jerk grading scores	Phasic tendon reflex	De Jong (1984); Vattanasilp and Ada (1999)
Tone Assessment Scale	Tonal abnormality	Gregson *et al.* (1999)
5-point National Institute of Neurological Disorders and Stroke (NINDS) myotatic reflex scale	Tendon reflex	Hallett (1993); Litvan *et al.* (1996)
Mayo clinic scale	Tendon reflex	Manschot *et al*. (1998)
Spinal Cord Assessment Tool for Spastic reflexes (SCATS)	Clonus and spasms	Benz *et al.* (2005)
Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET)	Impact of spasticity on ADLs	Adams *et al.* (2007)
Oswestry scale of grading	Spasticity	Goff (1976)

The most widely used scale for the clinical assessment of spasticity is currently the Modified Ashworth Scale (MAS) (Bohannon and Smith, 1987; Morris, 2002b; Skold, 2000; Watkins *et al.*, 2002). The MAS is based on the original Ashworth Scale (Table 2.3) which was first described in 1964 in a pharmacological trial investigating the effects of muscle relaxants on spasticity (Ashworth, 1964; Morris, 2002b). The Ashworth Scale is a five-point scale that grades the amount of tone felt as a limb is passively moved through a ROM by an examiner (Damiano *et al.*, 2002). This scale (along with several other spasticity scales) includes a category for a "catch" which can be defined as a transient increase in the force that opposes the passive extension (Pandyan, Price, Rodgers, Barnes and Johnson, 2001). The Ashworth Scale is easy to use and does not require any special equipment, however, it is highly subjective and inter-rater reliability is low (Katz and Rymer, 1989). The scale also suffers from a cluster effect where the majority of scores tend to be grouped in the middle of the scale. Furthermore, the Ashworth Scale may lack precision as it is unable to detect small degrees of change (Priebe, 2006).

To overcome the problems with the Ashworth Scale, it was modified by Bohannon and Smith (1987) to spread out the grades in the middle portion (Table 2.4). The authors added an extra category, making the scale a six-point scale to render it more discriminative, and they also redefined the criteria for each of the scores. Although the Ashworth Scale was designed to assess spasticity, most authors agree that the scale is more accurately a measure of muscle tone rather than spasticity as it does not take into consideration the velocity of the joint movement (Ansari *et al.*, 2006; Morris, 2002b; Vattanasilp, Ada and Crosbie, 2000). As a result, the scale has been the focus of much criticism in the literature and numerous authors have challenged its validity and reliability (Allison, Abraham and Petersen, 1996; Johnson, 2002; Kumar, Pandyan and Sharma, 2006; Morris, 2002b; Pandyan, Price, Rodgers, Barnes and Johnson, 1999). Despite the scale's limited objectivity, it is still very commonly used and is a standard to which other measurement methods are compared (Damiano *et al.*, 2002; Katz, Rovai, Brait and Rymer, 1992). Some authors have continued to modify the scale in an attempt to improve its validity and reliability and there is now a modified MAS (Table 2.5) (Ansari *et al.*, 2006; Naghdi *et al.*, 2008).

Table 2.3 The original Ashworth Scale (Ashworth, 1964, p541).

Score	Description
0	No increase in tone.
1	Slight increase in tone giving a "catch" when the limb is moved in flexion or extension.
2	More marked increase in tone, but limb easily flexed.
3	Considerable increase in tone, passive movements difficult.
4	Limb rigid in flexion or extension.

Table 2.4 The Modified Ashworth Scale (Bohannon and Smith, 1987, p207).

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

Table 2.5 The Modified MAS (Ansari *et al.*, 2006; Naghdi *et al.*, 2008).

Score	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension.
2	Marked increase in muscle tone, manifested by a catch in the middle range and resistance throughout the remainder of the range of motion, but affected part(s) easily moved.
3	Considerable increase in muscle tone, passive movement difficult.
4	Affected part(s) rigid in flexion or extension.

The Tardieu Scale is another commonly used scale for the clinical measurement of spasticity. It was first described in French by Tardieu, Shentoub and Delarue (1954) who proposed a method of measuring spasticity that took into consideration the velocity of the joint movement. This method was later constructed into a quantifiable scale by Held and Pierrot-Deseilligny (1969) (Table 2.6). The original Tardieu Scale has since been updated with the addition of detailed instructions regarding the procedures for carrying out the test, and clearer definitions for the criteria of each score (Boyd and Graham, 1999b; Morris, 2002b). Some authors refer to the updated Tardieu Scale as the Modified Tardieu Scale but this terminology is not consistent in the literature. For a full description of the updated Tardieu Scale see the papers by Boyd and Ada (2001) and Morris (2002b).

The Tardieu Scale is conducted by passively moving a relaxed limb through its ROM at three velocities:

V1- As slow as possible;

V2- Speed of the limb segment falling under gravity; and

V3- As fast as possible.

The quality of the muscle reaction to stretch is then rated at a specified velocity which is determined according to the muscle of interest. Two parameters are recorded for each muscle: X and Y. The X parameter measures the quality of the muscle reaction on a five-point scale (Table 2.7), and the Y parameter measures the angle at which the muscle

reaction occurs (also known as the "catch") (Morris, 2002b). The Y parameter can be further defined by measuring R1 (the angle at which the catch occurs at V3) and R2 (the angle at the full passive ROM at V1) (Boyd and Graham, 1999b). If there is a small difference between R1 and R2 then there is predominantly a fixed muscle contracture present, whereas if there is a large difference between the two measures then there is a large reflexive component present. The scale contains clear instructions for the positioning of the patient during testing, the prescribed velocity for each muscle stretch and the scoring of the quality of the muscle reaction (Boyd and Ada, 2001; Morris, 2002b).

The Tardieu Scale is similar to the MAS, however, it is generally considered to be better at quantifying spasticity as it takes into consideration the velocity of the joint movement. This allows the clinician to determine whether the cause of an abnormal increase in resistance is due to neural or mechanical factors (Boyd and Ada, 2001; Haugh *et al.*, 2006). In a study conducted by Patrick and Ada (2006), the Tardieu Scale was found to be more accurate than the Ashworth Scale at identifying the presence of spasticity in the elbow flexors and ankle plantarflexors of hemiplegic subjects following stroke. This was determined by comparing the percentage exact agreement between the Tardieu Scale and the Ashworth Scale with two laboratory measures of spasticity (stretch-induced EMG activity and maximum passive joint excursion) in 16 hemiplegic stroke affected subjects. The reliability and validity of the Tardieu Scale still remains under question due to a lack of research, however, initial results have found the Tardieu Scale to be more reliable and valid that the Ashworth Scale and the MAS (Boyd, Barwood, Bailleau and Graham, 1998; Mehrholz *et al.*, 2005).

Table 2.6 The Tardieu Scale.
Grading as developed by Held and Pierrot-Deseilligny in 1969 (Haugh *et al.*, 2006).

Score	Description
0	No stretching.
1	Only visible contraction.
2	Simple jump felt during mobilization but that passes just as suddenly as it came.
3	Lasting contraction or a few clonic tremors that go away after a few seconds.
4	Contraction or clonus that does not cease, even after a few seconds.

Table 2.7 The Modified Tardieu Scale (Boyd and Graham, 1999b).

Score	Description
0	No resistance throughout the course of the passive movement.
1	Slight resistance throughout the course of the passive movement, with no clear catch at a precise angle.
2	Clear catch at a precise angle, interrupting the passive movement, followed by release.
3	Fatigable clonus (<10 seconds when maintaining pressure) occurring at a precise angle.
4	Infatigable clonus (>10 seconds when main maintaining pressure) occurring at a precise angle.

While subjective clinical scales for the quantification of spasticity are useful, they also carry a number of limitations. These limitations can either be due to the design of the scales or the ways in which the scales are applied. The problem with the design of most scales is that they have low sensitivity to detect slight changes, and they may present a cluster effect in which most subjects tend to be grouped in the intermediate degrees of the scale (Pizzi, Carlucci, Falsini, Verdesca and Grippo, 2005b).

Problems with the practical application of the scales relate to issues with both the patient and the assessor. Regarding the assessor, it is essential that the assessor has received adequate training before commencing the testing procedures, and that well described, standardised procedures are available and adhered to, to avoid rater errors and poor repeatability (Biering-Sorensen *et al.*, 2006). Regarding the patient, the ability to relax during passive movements as well as the patient's "state" are believed to affect muscle tone measurements (Pomeroy, Dean, Sykes *et al.*, 2000). The "state" of the patient refers to factors such as the level of anxiety, bladder filling, positioning and fatigue, since all of these can affect the level of spasticity in a limb (Harburn *et al.*, 1992). The circumstances under which the individuals are tested like the time of day, type of activity performed before the test, ambient temperature, emotional status, general health and the use of drugs may also contribute to variability in the scores (Biering-Sorensen *et al.*, 2006; Hsieh *et al.*, 2008). These patient factors need to be considered regardless of which measurement tool or method is used. It must also be considered that there may be times when measurement procedures will be disrupted and thorough testing will be compromised by the patient's limited abilities, endurance and comfort (Tyson *et al.*, 1998).

Overall, the value of clinical subjective measurements appears to be in their ability to provide a simple method of establishing clinical impressions, and therefore, they may be useful tools in the clinical setting (Haas, 1994). However, they cannot distinguish between the reflex and mechanical elements of spasticity and for that reason, should be used in conjunction with other measurement methods. Further effort is required to determine the reliability and validity for many of the spasticity measurement scales that have been proposed in the literature.

2.5.2 Biomechanical and electrophysiological methods

Unlike subjective clinical measurement methods, objective methods provide a more quantifiable and reliable measure that should not be affected by the person taking the measure (Haas, 1994). Biomechanical methods for measuring spasticity observe the behaviour of muscles, joints and limb segments in response to controlled mechanical manipulations of the angle of deflection of a joint and the rate at which a limb is moved (Burridge *et al.*, 2005; Katz and Rymer, 1989). They can also be used to measure the mechanical aspects of spasticity such as joint stiffness, joint ROM and joint torque. The method of moving a joint or limb segment can be classified according to manual displacement, controlled displacement, controlled torque and gravitational methods (such as the pendulum test) (Wood *et al.*, 2005). For a thorough review of these biomechanical methods for measuring spasticity see Wood *et al.* (2005).

Some biomechanical methods can only be performed on certain joints or limb segments and may require specialised equipment and training. Devices used to measure spasticity biomechanically can be based on three different principles: gravity controlled, position controlled and force/torque controlled (Salazar-Torres, Pandyan, Price *et al.*, 2004). These devices can include potentiometers, trachometers, torque meters and isokinetic dynamometers.

Electrophysiological methods provide the most reliable method of determining the stretch reflex threshold, therefore, they may be of value for evaluating spasticity in combination with biomechanical and neurophysiological techniques (Biering-Sorensen *et al.*, 2006). Several authors (Boyd and Ada, 2001; Katz and Rymer, 1989) have recommended using EMG to measure the stretch reflex threshold (the angle at which EMG begins to increase during passive stretching of a muscle) to ascertain the neural component of spasticity. While EMG is useful to determine whether a neural component to hypertonia is present, it cannot be solely used to measure changes in spasticity to assess the effects of an intervention. This is because changes in spasticity may not be reflected in EMG activity due to the reflex and mechanical elements that make up the phenomenon of spasticity (Carey and Burghardt, 1993). Furthermore, the measurement of EMG during activity (such as walking) cannot distinguish between voluntary muscle responses and involuntary reflex responses. The only way to separate voluntary

responses from involuntary responses is in a laboratory with controlled stimulus evoked EMG.

2.5.3 Gait

The majority of studies on the evaluation of TRAFOs have used some form of gait analysis (whether observational or objective) as their primary measurement tool. This may be considered a functional method of measuring spasticity, however, it has been suggested that gait analysis should not be regarded as a measure of spasticity as no conclusive association between gait and spasticity has been made (Johnson, 2002; Lin and Brown, 1992). Furthermore, gait measures cannot distinguish the contribution of factors such as spasticity, muscle weakness, muscle imbalance and reduced joint ROM to the gait impairment (Yelnick *et al.,* 1999).

Some may argue that gait outcomes are more useful than outcomes that specifically measure spasticity since gait is a functional measure and may have greater clinical and functional significance. However, it is important especially in the evaluation of TRAFOs to determine the effect of the tone-reducing features of the orthoses to ascertain:

1. Whether or not they are necessary; and
2. How they can be improved and better utilised if they really do work.

Gait analysis by itself cannot determine this.

While both clinical and biomechanical methods have their place in research, a more valid and direct method of measuring spasticity is required for the evaluation of TRAFOs. Of the methods discussed so far, none are able to separate the biomechanical and neurophysiological effects of TRAFOs adequately to demonstrate the tone-reducing effects on spasticity. But such methods are available and will be discussed next.

2.5.4 Neurophysiological methods

Neurophysiological methods for the assessment of spasticity measure the electrical responses of the motor control system to a variety of stimuli and conditions (Burridge *et al.*, 2005). Neurophysiological methods can be used to solely measure the neural aspect

of spasticity when the mechanical aspect is adequately controlled. Since reflexes are heightened in spasticity, attempts to quantify the excitability of MNs should be a prime target for investigating the reflex element of spasticity (Haas and Crow, 1995). A commonly used neurophysiological method for measuring spasticity is the Hoffmann reflex (H-reflex).

The H-reflex was first shown by Piper in 1912 and described in more detail by Hoffmann in 1918 (Voerman *et al.*, 2005). It is used in neurophysiological investigations in humans to estimate a number of parameters that cannot be measured directly (Brinkworth, Tuncer, Tucker, Jaberzadeh and Turker, 2007). The H-reflex is an electrically induced reflex analogous to the mechanically induced spinal stretch reflex. It measures the excitability of alpha MNs located within a target MN pool (Hwang, Lin and Ho, 2002b; Palmieri, Hoffman and Ingersoll, 2002). However, unlike the spinal stretch reflex, the H-reflex bypasses the mechanical sense organs, mainly the muscle spindles (Chalmers and Knutzen, 2000). The H-reflex can provide useful information regarding changes in the level of inhibition or excitation of a MN pool (Voerman *et al.,* 2005)

The H-reflex can be used as an indication of the level of spasticity or an alternative method for measuring muscle tone (Kukulka, Haberichter, Mueksch and Rohrberg, 1987; Pizzi *et al.*, 2005b). It has a clear experimental role but also a clinical role to aid in the diagnosis of neurological pathologies and musculoskeletal injuries (Palmieri *et al.*, 2002; Williams, Sullivan, Seaborne and Morelli, 1992). It can also be used to monitor the progress of treatments (Crayton and King, 1981). The H-reflex has been used extensively in neurophysiology research to investigate the efficacy of various therapies such as massage (Morelli, Seaborne and Sullivan, 1990a), tendon pressure (Kukulka *et al.*, 1987), cooling (Bell and Lehmann, 1987) and muscle stretch (Hwang, 2002a).

The H-reflex can be recorded in most voluntary muscles that have a main supply nerve that can be accessed by percutaneous electrical stimulation (Misiaszek, 2003). Tests have shown that the soleus provides the greatest amplitude of response when compared with other muscles, and recordings from the soleus have been shown to be valid and reliable (Maryniak, Yaworski and Hayes, 1991; Sabbahi and Khalil, 1990; Strakowski, Redd, Johnson and Pease, 2001). As such, the soleus muscle was the muscle chosen to be the focus of testing in this thesis.

The H-reflex itself is an EMG response seen when artificial electrical stimulation of a peripheral mixed nerve activates Ia afferent fibres resulting in excitation of spinal MNs (Figure 2.1) (Hilgevoord, Koelman, Bour and Ongerboer de Visser, 1994). To record the H-reflex of the soleus muscle the tibial nerve (mixed peripheral nerve) is percutaneously stimulated in the popliteal fossa with an electronic stimulator (Little and Halar, 1985). This causes stimulation of the large diameter Ia sensory afferent fibres and sends action potentials to the spinal cord where essentially monosynaptic connections cause homonymous alpha MNs to reach threshold. This in turn results in the depolarisation of the extrafusal muscle fibres in the soleus muscle and their subsequent contraction (Robichaud and Brunt, 1994; Vujnovich and Dawson, 1994). The depolarisation is detected by surface EMG electrodes, amplified by an EMG amplifier and recorded on a recording device. The response of the soleus to the stimulation of the tibial nerve is called the H-reflex and it has a latency of approximately 35 milliseconds following the stimulation (Figure 2.1) (Schiepatti, 1987). Variations in the size of the H-reflex provide a reliable indication of the level of excitatory or inhibitory influences acting on the alpha MNs (Taborikova, 1973). The amplitude of the H-reflex provides a reproducible, reliable and quantifiable reflection of MN excitability specifically of the alpha MNs lying within the L5-S1 reflex arc for the soleus muscle (Vujnovich and Dawson, 1994).

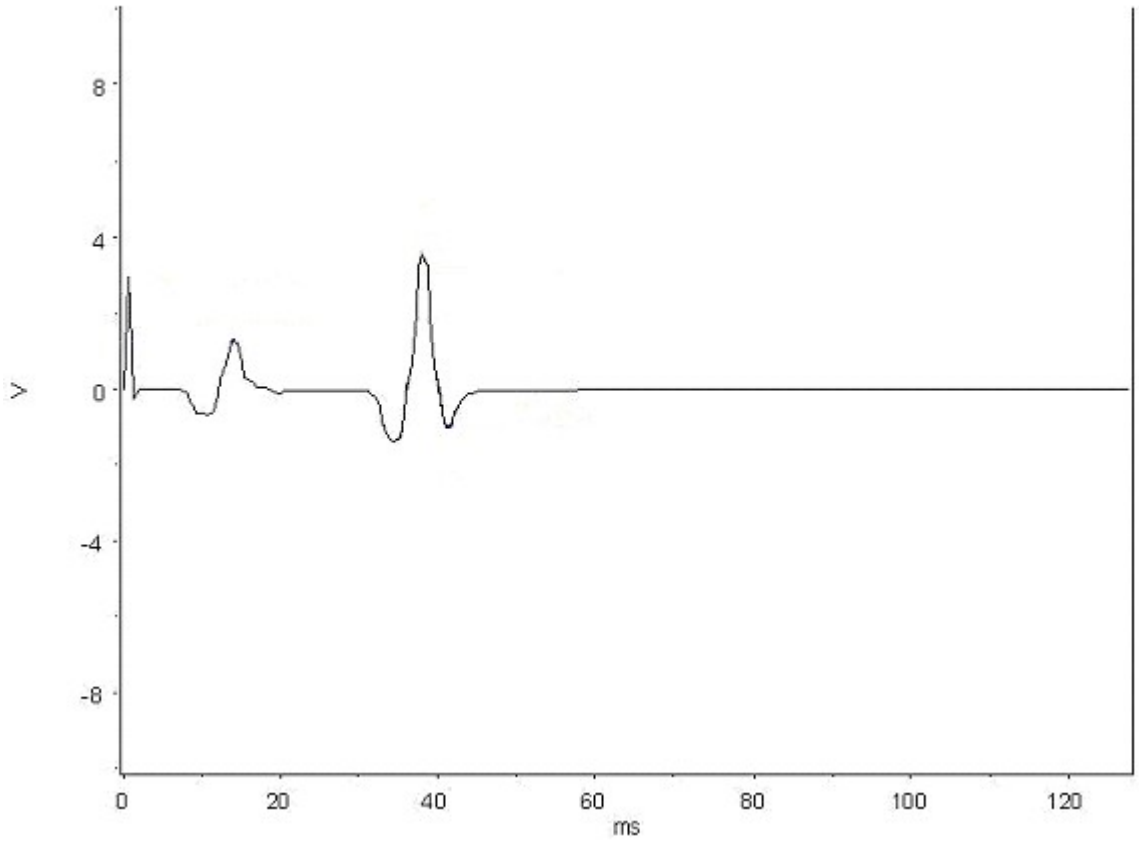

Figure 2.1 EMG recording of the H-reflex.
The stimulus artefact occurs at 0 milliseconds, the M-response then follows at approximately 5-10 milliseconds and the H-reflex occurs at approximately 35 milliseconds after the stimulus.

While the Ia afferent fibres are being stimulated by the percutaneous electrical stimulation, MNs are also stimulated within the mixed tibial nerve causing their direct depolarisation. This depolarization of the MNs results in contraction of the soleus muscle which is also recorded by the surface electrodes. As this activity does not pass through the spinal cord it is not called a reflex but simply a direct motor response or muscle response (M-response) (Palmieri, Ingersoll and Hoffman, 2004). The M-response represents the orthodromic conduction in the distal motor axons (Little and Halar, 1985) and it has a latency of approximately 5-10 milliseconds following the stimulation (Figure 2.1) (Voerman *et al.*, 2005). The size of the M-response directly reflects the intensity of the electrical stimulus. The M-response amplitude is thought to be a fairly stable value because it is simply caused by the depolarisation of the motor axons in response to an electrical stimulus and is not influenced by spinal centres (Palmieri *et al.*, 2002).

Consistency in the amplitude of the M-response provides an indication of stability in the stimulating and recording conditions (Vujnovich and Dawson, 1994). If the M-responses remain consistent across several recordings at a constant stimulus intensity while changes are observed in the H-reflex curves, it can be assumed that the changes in the H-reflex curves are due to changes in the level of MN excitability (Hugon, 1973a).

The H-reflex is most effectively used to study the excitability of a MN pool by the H-reflex and M-response recruitment curves (Figure 2.2) (Stolp-Smith, 1996). These recruitment curves illustrate the H-reflex and M-response amplitudes as functions of the level of stimulus intensity (Hilgevoord *et al.*, 1994). To construct the recruitment curves, the stimulus intensity is gradually increased while the maximum responses of the H-reflexes and M-responses are plotted at each level of stimulus intensity (Figure 2.2). The ratio of maximum H-reflex amplitude to the maximum M-response amplitude (H:M ratio) can then be obtained from the recruitment curves. The H:M ratio indicates the relation between the maximum number of motor units that can be activated through reflexes compared with the total number of motor units in the whole MN pool that can be activated by percutaneous orthodromic conduction (Voerman *et al.*, 2005). The H:M ratio is a useful measure as it minimises the influence of variability with recording or subject physiology. It is therefore preferable to simply reporting H-reflex amplitudes alone (Garrett and Caulfield, 2001; Hilgevoord *et al.*, 1994). The fraction of the soleus MN pool activated in an H-reflex is usually about 50% but can be as high as 100% of the maximal M-response (Biering-Sorensen *et al.*, 2006; Fisher, 1992). This differs between individuals and can be significantly affected by the presence of a neurological disorder. Further information regarding the procedures for recording the H-reflex and M-response recruitment curves as well as their reliability is detailed in the next chapter.

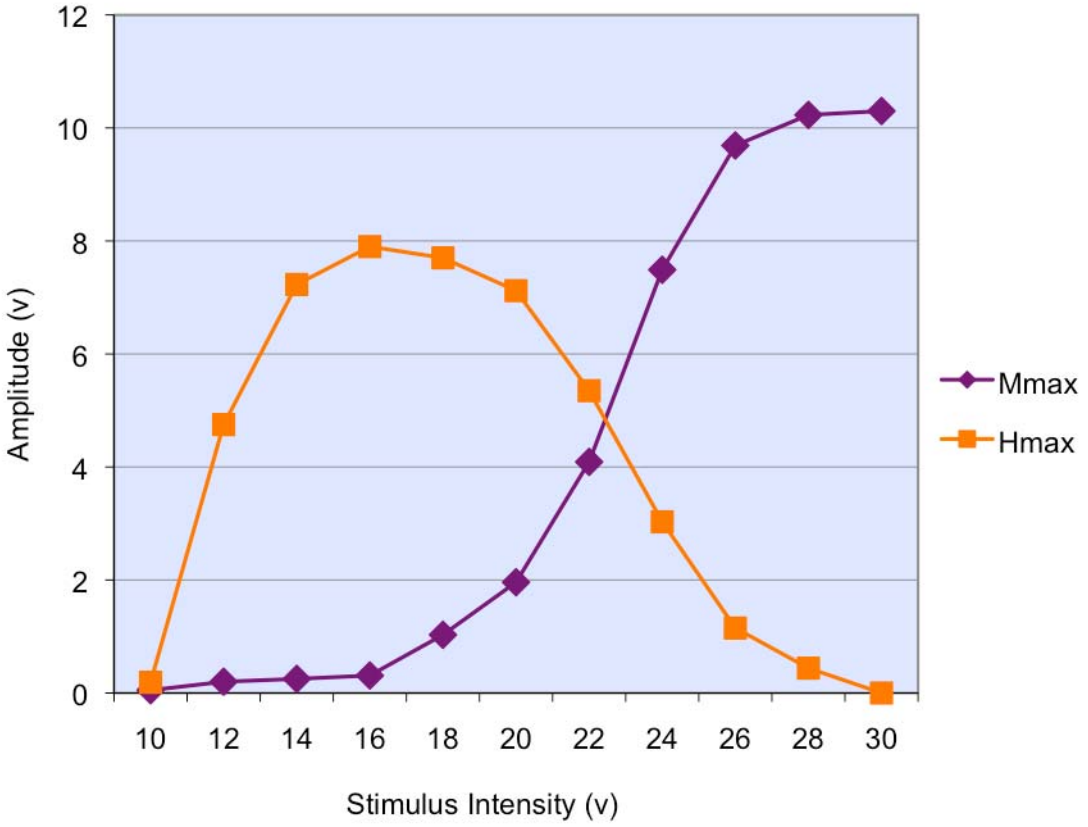

Figure 2.2 H-reflex and M-response recruitment curves.

Changes in the amplitudes of the H-reflexes between various testing conditions can be explained by at least three possible mechanisms:

1. Alteration in the level of MN excitability;
2. Variation in the amount of neurotransmitter released by the afferent terminals; or
3. Variation in the intrinsic properties of the MNs by neuromodulator substances such as serotonin (Capaday, 1997; Misiaszek, 2003).

When interpreting data, it is important to take these factors into consideration. Some authors have suggested that due to the pre-synaptic and post-synaptic effects on MN excitability, the H-reflex cannot be described as solely monosynaptic in nature and therefore, is not a true direct measure of alpha MN excitability (Brinkworth *et al.*, 2007; Capaday, 1997).

However, the H-reflex can still be useful to assess the effects of an intervention on MN excitability. The problem is that the H-reflex cannot distinguish which pre- or post-synaptic factors are responsible for a potential change in MN excitability. For the assessment of the neurophysiological effect of TRAFOs, the H-reflex is sufficient to demonstrate whether or not the tone-reducing features of the TRAFOs have any effect on MN excitability. If a change in MN excitability is found following TRAFO use, further investigations could then be performed to determine which pre- or post-synaptic factors are responsible.

The disadvantage of using the H-reflex to measure MN excitability is that it is a research tool and is impractical for use in a clinical setting (Barnes, 1998). The electrical stimulations can cause discomfort to the patient and it can be difficult to obtain consistent trial-to-trial results. This is because there may be variability in the placement of electrodes, variability in the stimulus parameters, and complications may arise if electrodes detect the activity of muscles other than the one being studied (Schiepatti, 1987). The H-reflex may also give a false estimation of the functional effects of spasticity as the measurements are usually taken in static rather than dynamic situations (Barnes, 1998). Strict methodological protocols need to be followed to ensure reliable results.

2.5.4.1 The use of neurophysiological measures of spasticity in orthotics research

The H-reflex can be used to evaluate the neurophysiological effect of TRAFOs on MN excitability as a measure of spasticity. If the biomechanical effects of a TRAFO are adequately controlled, the H-reflex can be used to determine if the TRAFOs have any effect on spasticity by changing the level of excitability of the MNs that innervate spastic muscles.

The majority of experimental research that has been conducted on TRAFOs has used functional outcome measures to determine an effect on spasticity. Very few studies have used biomechanical or electrophysiological methods to measure spasticity and none have used neurophysiological methods. However, there have been studies that have used the H-reflex to assess the effects of other orthosis designs on MN excitability.

Childers, Biswas, Pertoski and Merveille (1999) used a measure called the vibratory inhibition index of the H-reflex to assess the effect of inhibitory casting on the spastic flexor carpi radialis muscle in eight subjects following stroke. The results demonstrated that the inhibitory casts decreased MN excitability consistently over the three days that the casts were in place. On the fourth day when the casts were removed, the MN excitability increased demonstrating that the inhibitory casts were effective in decreasing MN excitability but only while they were being worn.

Similarly, Ushiba, Masakado, Komune *et al.* (2004) and Pizzi, Carlucci, Falsini, Verdesca and Grippo (2005a) both demonstrated that the use of static wrist-hand orthoses to extend the wrist joints were successful in decreasing the MN excitability of the flexor carpi radialis muscles in subjects with upper limb spasticity following stroke. Both of the studies used the H:M ratio as their primary outcome measure. Ushiba *et al*. (2004) monitored subjects over a period of 20 minutes while wearing the wrist-hand orthoses and Pizzi *et al.* (2005a) monitored subjects over a period of three months.

The only published study that has assessed MN excitability in the lower limbs following application of an orthosis was conducted by Nishikawa and Grabiner (1999b). These researchers assessed the MN excitability of the peroneal muscles in healthy subjects and found that MN excitability increased immediately by approximately 10% following the application of prefabricated ankle braces.

The results of these studies cannot be used to predict the potential neurophysiological effects of TRAFOs in spastic lower limb muscles. This is due to differences in the results found between the studies, the different muscles tested and the different subject groups studied. Nevertheless, these studies provide evidence to suggest that TRAFOs may have an affect on the MN excitability of spastic muscles.

2.5.5 Summary

Any outcome measure must be valid, reliable and sensitive to change in the clinical condition being investigated to produce meaningful results (Priebe, 2006). The clinical condition in this case is spasticity which presents a challenge to measure due to its multifactorial aetiology (Lechner *et al.*, 2006). Current measurement tools for spasticity may be seen as inadequate since there is no single tool which can provide a valid and reliable measure of the various components of spasticity (Burridge *et al.*, 2005), but perhaps a single universal measurement system for spasticity is impossible to achieve (Johnson, 2001).

There are numerous methods for measuring different aspects of spasticity mentioned in the literature. The reliability of some of these measures has been established, however, confusion still surrounds issues regarding their relevance and validity. A measurement tool can be shown to be reliable, but this is of no use if it does not measure the variable that needs to be measured. This has been the case for the MAS. Despite it being the "gold standard" clinical measure for spasticity, some authors have recently argued that it does not measure spasticity but simply measures muscle tone (Morris, 2002b; Vattanasilp and Ada, 1999).

Although all measures of spasticity have their limitations, careful selection of a combination of measures can provide an appropriate thorough assessment of spasticity (Adams *et al.*, 2007). Following careful review, in this thesis the Tardieu Scale was used to assess the presence of spasticity in subjects but not to monitor the effects of treatment as the Tardieu Scale cannot be administered on the ankle joint while a subject is wearing an AFO in a standing position. The main outcome measures chosen for use in the planned studies to assess the effects of orthotic interventions were MN excitability, temporo-spatial gait parameters, joint kinematics and soleus muscle activity. These outcomes were measured using the H:M ratio, three-dimensional motion gait analysis and EMG. The use of these outcome measures was considered to provide a comprehensive assessment of the effects of TRAFOs on spasticity.

2.6 Overall summary

Spasticity has been the focus of research in the fields of neurophysiology and physical therapy for decades. However, there is no doubt that continued research is required to advance our understanding of spasticity, especially as the number of people experiencing spasticity increases (Page, 2004). Research on spasticity is required in three main areas:

1. The pathophysiology and neurophysiology of spasticity;
2. The treatment of spasticity; and
3. The measurement of spasticity.

One method for managing spasticity is through the use of orthotics, specifically TROs. The benefits of orthoses in comparison to surgical or pharmacological treatments are that they are non-invasive, have minimal side-effects and risks, provide long-term effects, are widely available and are easily produced and maintained. TRAFOs for the lower limbs may be able to improve movement and walking allowing a sufferer of spasticity to be more mobile, independent and confident, improving their ADLs and quality of life. It is already well established that the biomechanical effect of AFOs are effective in improving function, however, it may be possible that AFOs can be further improved with the addition of tone-reducing features. There is evidence in the literature to suggest that TRAFOs can affect the level of muscle spasticity in the lower limbs, but there are a number of problems with the research that has produced this evidence. Following critique of this literature, confidence in the evidence is reduced identifying a need for further research.

With the use of appropriate measurement methods, it should be possible to determine the neurophysiological and biomechanical effects of TRAFOs on spasticity. If TRAFOs really do reduce spasticity, then they should be more frequently prescribed for people with spasticity to avoid the need for more risky treatments involving surgery or drugs. If they do not reduce spasticity, then the time, effort and costs that go into making and fitting them are unnecessary and unjustified.

2.7 Aims and hypotheses

The overall aim of this thesis was to examine the effect of TRAFOs on lower limb spasticity. Since this aim was quite broad, its focus was narrowed in order to be thorough. TRAFOs are assumed to have effects on all muscles in the leg, however, only the soleus muscle was tested in the subsequent studies. Spasticity can arise from numerous neurological pathologies but only subjects with spasticity secondary to stroke were chosen for the studies in this thesis.

To fulfil the overall aim of the thesis, more targeted aims were composed as follows:

1. To develop a method by which the neurophysiological effect of TRAFOs could be evaluated separately from their biomechanical effect;
2. To investigate the neurophysiological effect of TRAFOs on the MN excitability of the soleus muscles in subjects with spasticity following stroke while standing; and
3. To investigate the effect of TRAFOs on temporo-spatial gait parameters, joint kinematics and soleus muscle activity in subjects with spasticity following stroke while walking.

Based on these aims, the following hypotheses were derived:

1. TRAFO use results in a reduction of MN excitability of the soleus muscle in subjects with spasticity while standing;
2. TRAFO use results in improved temporo-spatial gait parameters and joint kinematics in subjects with spasticity; and
3. TRAFO use results in improved soleus muscle function in subjects with spasticity.

Chapter Three

The inter-session reliability of the soleus H-reflex

3.1 Introduction

The choice of a suitable outcome measure is a critical factor for any experimental study that aims to evaluate the effects of an intervention (Becker, Stuifbergen, Rogers and Timmerman, 2000). In order for a measure to be valid, it must first be shown to be reliable especially in the case of spasticity assessment (Hopkins, Ingersoll, Cordova and Edwards, 2000). The H-reflex was chosen as the main measurement tool used in this thesis as it was likely to be the best measure of spasticity for the evaluation of TRAFOs. However, given the specific questions of this thesis, it was first necessary to establish its reliability within this particular context.

3.1.1 Factors that may influence the H-reflex and M-response amplitudes

The H-reflex is known to be a challenging test to conduct as it is difficult to obtain consistent trial-to-trial results that are controlled for extraneous variables (Hopkins *et al.*, 2000). Since the H-reflex amplitude is susceptible to many influencing factors (Gottlieb and Agarwal, 1971) its ability to consistently assess MN excitability within and between testing sessions has been questioned (Palmieri *et al.*, 2002).

The amplitude of the soleus H-reflex is thought to be an expression of the relative excitability of soleus alpha MNs to excitatory inputs from type Ia afferent fibres of the posterior tibial nerve (Goulart, Valls-sole and Alvarez, 2000). However, this is not completely accurate as the H-reflex amplitude may be subject to a number of influencing factors. These influencing factors can be categorised into two groups (McIlroy and Brooke, 1987):

1. Factors relating to the stimulating and/or recording conditions; and
2. Factors that can cause fluctuations in the excitability of the MN pool.

Factors relating to the stimulating and/or recording conditions include the stimulus intensity, stimulus duration, stimulus frequency (Imura, Kishikawa, Wada, Iwai and Fuziwara, 1997a), skin resistance (Palmieri *et al.*, 2002), subcutaneous fat (Palmieri *et al.*, 2002) and movement of the stimulating and/or recording electrodes (McIlroy and Brooke, 1987). Factors that can affect the H-reflex amplitude by causing fluctuations in MN excitability can include the state of the subject (level of arousal, patient comfort or anxiety) (Crayton and King, 1981; Matthews, 1966), bladder distention (Little and Halar, 1985), auditory stimulations (Voerman *et al.*, 2005), head and neck position (Biering-Sorensen *et al.*, 2006) and length of the muscle being tested (Hwang, 2002a). There are many other factors that may specifically cause suppression or facilitation of MN excitability and some of these have been listed in Table 3.1

These factors can be categorised according to whether they act pre-synaptically or post-synaptically. Pre-synaptic factors are dependent upon the extrinsic properties of a MN (eg. number of synaptic terminals and spatial distribution of synaptic terminals onto MNs from a given input system), and post-synaptic factors are dependent upon the intrinsic properties of a MN (eg. total membrane area and electrotonic architecture of the MN which depends on the cell anatomy) (Funase, Imanaka and Nishihira, 1994).

Table 3.1 Factors that may affect the H-reflex amplitude.
These factors are not related to circumstances surrounding the stimulating and/or recording electrodes.

Suppression	Facilitation
• Voluntary contraction of antagonist muscles (Gottlieb and Agarwal, 1971; Knikou, 2008; Stolp-Smith, 1996). • Slow passive stretch (Voerman *et al.*, 2005). • Drugs including nicotine, pentobarbital, diazepam, aspirin and baclofen (Eke-Okoro, 1982; McLellan, 1977; Stolp-Smith, 1996). • Eyes closed (Kameyama, Hayes and Wolfe, 1989). • Sleep (Stolp-Smith, 1996). • Vibration (Fisher, 1992; Stolp-Smith, 1996; Voerman *et al.*, 2005). • Tendon tap (Stolp-Smith, 1996). • Spinal anaesthesia (Stolp-Smith, 1996).	• Mild contraction of agonist (Fisher, 1992; Gottlieb and Agarwal, 1971; Stolp-Smith, 1996). • Muscle fatigue (Eke-Okoro, 1982). • Labyrinthine vestibular stimulation (Stolp-Smith, 1996). • Jendrassik maneuver (Voerman *et al.*, 2005). • Grasping of hands (Imura, Kishikawa, Wada, Iwai and Fuziwara, 1997b). • Teeth clenching (Stolp-Smith, 1996; Voerman *et al.*, 2005). • Alcohol (Eke-Okoro, 1982). • Caffeine (Eke-Okoro, 1982).

The M-response is generally thought to be a fairly stable response that directly reflects the stimulus intensity (Palmieri *et al.*, 2004). As it is almost impossible to measure the current travelling through the tibial nerve, the M-response is used as an indication of the number of efferent fibres being stimulated. If the M-response amplitude remains constant while being stimulated at a constant intensity, it can be assumed that any inhibition or facilitation of the H-reflex is due to factors occurring in the spinal cord at synaptic junctions. However, it has been demonstrated that the M-response amplitude may also vary due to certain influencing factors (Table 3.2). Since the M-response does not involve any synaptic connections, these influencing factors must act on either the MN itself or on the neuromuscular junction.

Table 3.2 Factors that may affect the M-response amplitude.
These factors are not related to circumstances surrounding the stimulating and/or recording electrodes.

Suppression	Facilitation
• Prolonged voluntary contraction of muscle being tested (Fenton, Garner and McComas, 1991). • Increase in temperature (Denys, 1990). • Repetitive stimulation (Hicks, Fenton, Garner and McComas, 1989). • Drugs like aspirin (Eke-Okoro, 1982). • Muscle fatigue (Eke-Okoro, 1982).	• Maximal voluntary contraction of muscle being tested (Hicks *et al.*, 1989). • Decrease in temperature (Denys, 1990). • Alcohol (Eke-Okoro, 1982). • Caffeine (Eke-Okoro, 1982).

In order for the H-reflex and the M-response to accurately reflect changes in MN excitability, investigators must control the many extraneous factors known to affect these two responses. Some may be easy to control while others are very difficult to control. Failure to control these factors may result in variability not only between subjects and between trials, but also from trial-to-trial within the same subject (Brinkworth *et al.*, 2007).

Several investigations have been conducted to examine the intra-session and inter-session reliability of the soleus H-reflex and M-response under different testing conditions. Investigators have particularly focused on controlling subject positioning and electrode placement. The results of these studies vary, but the general trend suggests that the H-reflex is a reliable measure of MN excitability both within and between sessions (Palmieri *et al.*, 2002).

3.1.2 H-reflex and M-response reliability

Overall, the results of studies that have been conducted on H-reflex and M-response reliability have demonstrated that intra-session reliability is very high, whereas inter-session reliability is not clearly defined. McIlroy and Brooke (1987) assessed the

reliability of the soleus H-reflex amplitude at two set stimulus intensities and found that within session variability was much smaller (10%) than between sessions variability (30-40%). These results were based on the means and 95% confidence bands for H-reflex measures obtained from five subjects. The authors also investigated the effect of removing and reattaching the stimulating and recording electrodes in four subjects and found that H-reflex variability increased as a result of the electrode disturbance. The authors concluded that treatments should be tested within a session and effects should be compared within individuals rather than between individuals. They also concluded that poor between session variability could mostly be attributed to the removal and reattachment of electrodes (McIlroy and Brooke, 1987).

Several authors have also demonstrated that within session reliability of the H-reflex is good (Crayton and King, 1981; Morelli, Sullivan and Seaborne, 1990b; Williams *et al.*, 1992). Morelli *et al.* (1990b) tested the reliability of H-reflex and M-response amplitudes from two recording sites over the soleus muscle and found that simultaneously recording recruitment curves from the two locations was highly repeatable over a period of 15 minutes during the same session within subjects. Intra-class correlation coefficients (ICCs) for the H-reflex amplitudes for ten normal subjects were 0.99. Williams *et al.* (1992) also found good within subjects reliability of the H-reflex in a single session with coefficients ranging from 0.96 to 0.99. Within subjects' M-response reliability was also assessed and found to be equally repeatable with a correlation of 0.995.

Crayton and King (1981) demonstrated that the H-reflex recruitment curve was a highly reproducible parameter in an individual who was assessed over a six month period. (This claim was made based only on observations of three recruitment curves superimposed over one another on a single graph. No statistical analyses were conducted.) However, when studying the inter-session reliability of eight subjects, they found non-significant test-retest correlations for the maximum H-reflex amplitudes (r=0.6) and maximum M-response amplitudes (r=0.016) while the H:M ratios were associated with a statistically significantly correlation (r=0.719, p<0.05). It was not stated how much time had elapsed between the two testing sessions, but the investigators attributed the low test-retest correlations to alterations in electrode placement.

Inter-session reliability was also assessed by Palmieri *et al*. (2002) who studied H-reflexes from the soleus, peroneal and tibialis anterior muscles in ten healthy subjects. The authors found that the ICCs for the maximum H-reflex amplitudes were highly reliable for the soleus (0.99), peroneal (0.99) and tibialis anterior (0.85) muscles. Maximum M-response amplitudes and H:M ratios were also found to be highly reliable. These results contrasted those of McIlroy and Brooke (1987) and the authors attributed the differences to the testing procedures between the two studies.

All of the studies mentioned so far tested their subjects in relaxed non-weight-bearing positions. The H-reflex has also been shown to be reliable in weight-bearing positions (Ali and Sabbahi, 2001; Handcock, Williams and Sullivan, 2001; Hopkins *et al.*, 2000). Ali and Sabbahi (2001) tested the inter-session reliability of the H-reflex amplitude when assessed in three different positions (prone, free standing and standing while lifting 20% of body weight) on two separate days. They concluded that testing the H-reflex amplitude at separate sessions resulted in significantly reduced reliability values. They also demonstrated a higher reliability during the standing and loaded conditions than compared to the prone position indicating more stability and consistency, and less variability of the reflexes during standing and loading (Ali and Sabbahi, 2001).

A similar study was conducted to test the reliability of the H-reflex in supine and one-legged standing over five consecutive days (Hopkins *et al.*, 2000). Intra-session reliability was estimated using the ICC and found to be high for both the supine (0.932) and standing positions (0.853). Inter-session reliability was equally high for supine (0.938) and standing (0.803). Interestingly, the supine position was found to produce more reliable results than the standing position, a result also observed by Mynark (2005), but not by Ali and Sabbahi (2001). These differences between studies may be attributed to the different subject positions used by the studies (i.e. prone versus supine and free standing versus loaded standing and one-legged standing). The differences in reliability between the different positions demonstrates that subject positioning is an important factor in H-reflex testing (Palmieri *et al.*, 2004).

The age of the subjects has also been shown to be an important factor for reliable results. Mynark (2005) conducted a study to determine the reliability of the soleus H-reflex in

both young and elderly subjects during supine and standing positions. Higher reliability of the soleus H-reflex was obtained in ten young healthy subjects (mean age 23.2 years) compared with ten elderly healthy subjects (mean age 69.1 years) when the results from the supine and standing conditions were combined and the reliability for all of the trials were calculated. The ICCs were 0.98 and 0.93 respectively demonstrating a 5.4% decrease in the reliability of the H-reflex in older subjects compared with younger subjects. While the reliability of the H-reflex in young subjects remained quite constant between the conditions and when decreasing the number of trials from ten to four, this was not the case for the elderly subjects. Changing the subject position from supine to standing caused a 6% decrease in H-reflex reliability and decreasing the number of trials from ten to four in the standing position caused an 8% decrease in reliability (Mynark, 2005).

To reduce variability in H-reflex testing, authors have suggested that:

1. As many samples as possible (approximately ten samples) should be averaged at each stimulus intensity within a trial for reliable results (McIlroy and Brooke, 1987; Mynark, 2005);
2. Effects should be compared within a day without disturbing the electrodes (Ali and Sabbahi, 2001; McIlroy and Brooke, 1987; Voerman *et al.*, 2005); and
3. Effects should be compared within individuals rather than between individuals (McIlroy and Brooke, 1987).

3.1.3 H-reflex and M-response recruitment curves

Many studies on H-reflex reliability have constructed H-reflex and M-response recruitment curves for analysis (Figure 3.1). This is more useful than simply comparing H-reflex responses from a constant level of stimulus intensity (Brinkworth *et al.*, 2007) since recruitment curves allow the measurement of recruitment threshold (the minimum afferent stimulation which activates the most excitable MNs), recruitment gain (the slope of the curve describing MN activation as a function of stimulation intensity), and maximum amplitude values of the H-reflex and M-response curves (Hilgevoord *et al.*, 1994).

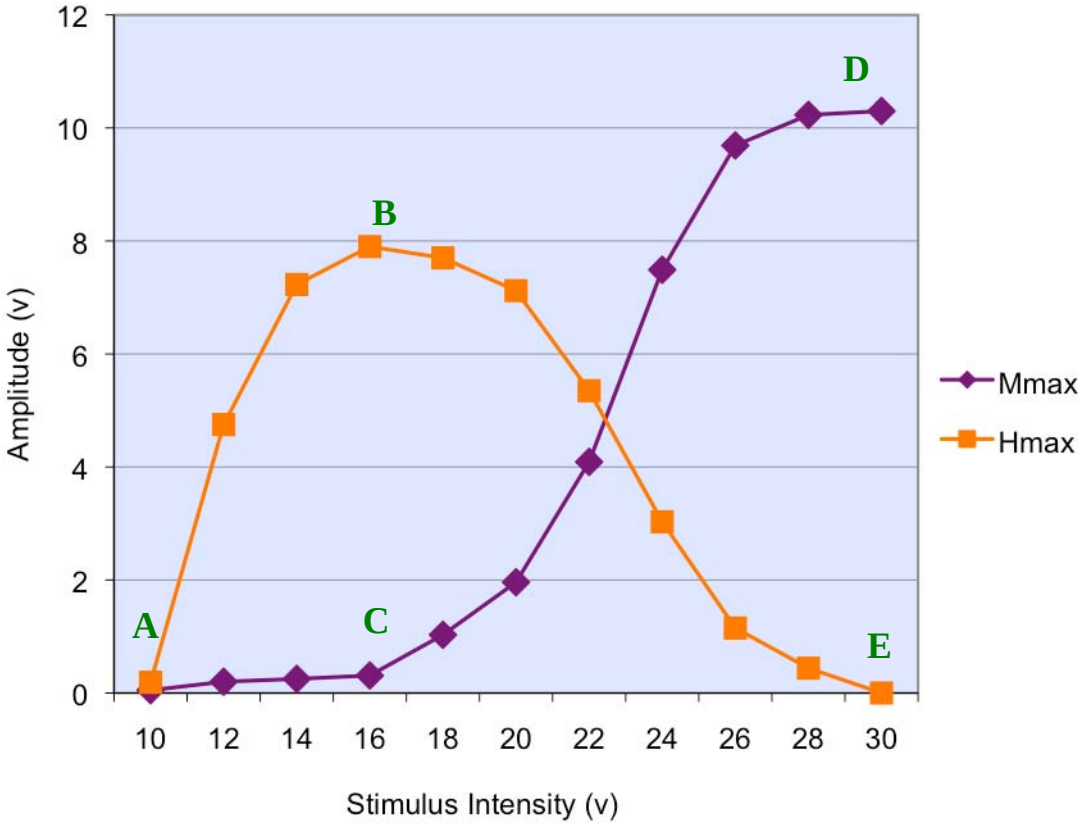

Figure 3.1 H-reflex and M-response recruitment curves.
A= H-reflex threshold; B= H-reflex maximum amplitude; C= M-response threshold; D= M-response maximum amplitude; E= H-reflex extinction.

To record H-reflex and M-response recruitment curves, electrical stimulus voltage is slowly increased from low intensities to high intensities while recording amplitudes of the EMG responses of the H-reflexes and M-responses at set increments along the way. The relative recruitment of Ia afferent fibres and alpha MNs in the tibial mixed nerve depends on the duration and intensity of the electrical stimulations which can determine the thresholds for the H-reflex and the M-response (Voerman *et al.*, 2005). This is because the strength-duration curve of the electrical stimulus differs for the recruitment of larger diameter Ia afferent fibres and smaller diameter alpha MNs, meaning that it is possible to evoke an H-reflex with stimuli below the threshold for the MNs (Pierrot-Deseilligny and Mazevet, 2000; Voerman *et al.*, 2005). Several investigators have recommended that the stimulus duration be set at 1 millisecond to preferentially recruit

the Ia afferent fibres before the alpha MNs (Hilgevoord *et al.*, 1994; Panizza, Nilsson and Hallett, 1989; Pierrot-Deseilligny and Mazevet, 2000; Stolp-Smith, 1996).

When the stimulus intensity has reached a sufficient voltage, Ia afferents fibres in the mixed nerve are depolarised causing the propagation of action potentials towards the spinal cord (Palmieri *et al.*, 2004). The Ia afferent fibres synapse with alpha MNs and cause a depolarisation in the MNs and the propagation of action potentials towards the soleus muscle. The muscle fibres in the soleus subsequently contract and this muscle activity appears as the H-reflex tracing on the EMG. The H-reflex recruits MNs in an orderly fashion from neurons with the smallest diameter axons (as they are more excitable with larger Ia excitatory post-synaptic potentials) to neurons with the largest diameter axons (which are less excitable with smaller Ia excitatory post-synaptic potentials) (Knikou, 2008). The H-reflex amplitude slowly rises with increasing stimulus voltage. Eventually, the stimulus intensity is large enough to cause the direct depolarisation of the alpha MNs in the tibial nerve resulting in the initiation of an M-response (Palmieri *et al.*, 2004).

The direct artificial stimulation of the alpha MNs recruits them in the opposite order from the H-reflex, with larger diameter axons activated first followed by the smaller diameter axons. Increasing the stimulus intensity increases the M-response amplitude, while the H-reflex reaches a maximum amplitude and then decreases until it eventually becomes extinct (Hilgevoord, Bour, Koelman and Ongerboer de Visser, 1995). The M-response also reaches a maximum amplitude but then plateaus giving it a uniform sigmoidal shape (Hilgevoord *et al.*, 1994). The H-reflex usually reaches its maximum amplitude slightly above the M-response threshold, and the M-response usually reaches its maximum amplitude at approximately the same time when the H-reflex becomes extinct (Figure 3.1).

The phenomenon of H-reflex extinction is not yet completely understood, and it appears that identifying the responsible mechanisms can only be accurately detected by invasive methods (Gottlieb and Agarwal, 1976). The extinction of the H-reflex is commonly attributed to antidromic collision (Hilgevoord *et al.*, 1995), which occurs when the antidromic action potential (volley of electrical activity travelling in the wrong direction

along the motor axon) collides with the reflexive orthodromic action potential (volley of electrical activity travelling in the correct direction along the motor axon after passing through the spinal cord) (Palmieri *et al.*, 2004; Voerman *et al.*, 2005).

The antidromic action potential is caused by the artificial stimulation of the mixed nerve, which sends action potentials travelling in both directions along the motor axon from the point of stimulation (Palmieri *et al.*, 2004). If the antidromic volley is smaller than the orthodromic volley which has travelled through the spinal cord, then the orthodromic volley is reduced but continues along the motor axon to the muscle. This explains why the H-reflex curve begins to decrease after reaching its maximum amplitude (Palmieri *et al.*, 2004). When the size of the antidromic volley is equal to or larger than the orthodromic volley, the orthodromic volley can not continue along its path and the H-reflex becomes extinct (Gottlieb and Agarwal, 1976).

3.1.4 Summary and aims

The reliability of a measurement tool such as the H-reflex, must be verified to ensure that the changes observed in dependant variables are due to inhibitory or facilitatory effects caused by an intervention, and not due to other sources of variability (Hopkins *et al.*, 2000). Although the reliability of the H-reflex has previously been explored, techniques for measuring and recording the H-reflex vary greatly among investigators, so it is important to establish the reliability of instrumentation and protocols in individual laboratories. While studies have been conducted to examine the reliability of the H-reflex in both prone and standing, no study has been conducted with the aim to determine if one position provides more reliable responses than the other.

Since the intra-session reliability of the H-reflex has been previously documented and uniformly reported to be highly reliable (McIlroy and Brooke, 1987; Morelli *et al.*, 1990b; Williams *et al.*, 1992), only the inter-session reliability was examined in this study. The aims of this study were:

1. To assess the inter-session reliability of the H-reflex, M-response and H:M ratio measured from the soleus muscle in able-bodied subjects in two positions: prone and standing; and

2. To determine if the measures were more reliable in one position than the other. It was hypothesised that the H-reflex, M-response and H:M ratio would demonstrate high inter-session reliability and that the standing position would be more reliable than the prone position.

3.2 Methods

3.2.1 Subjects

Ten able-bodied students and staff were recruited from the University for this study. The subjects consisted of five males and five females between the ages of 18 and 52 (mean age 22.6, SD=10.41). Inclusion in the study required subjects to have a good level of health and a medical history free from any neurological disorders. Subjects were also required to have a dominant leg that was free from any current injury.

3.2.2 Apparatus

All of the equipment used for this project was located in the Biomechanics laboratory at La Trobe University (Bundoora, Australia, 3086). A schematic diagram of the equipment setup is detailed in Figure 3.2.

EMG signals were measured with an EMG system that had been developed in-house (La Trobe University, Bundoora, Australia, 3086). A pre-amplifier and amplifier provided an overall gain of 1000. Signals were bandpass filtered between DC and 2 KHz and monitored on a four channel oscilloscope (Medelec Ltd. Surrey, UK, GU22 9JU). Power spectral analysis of the EMG signals indicated that almost all of the signal power was below 200 Hz. A Cardiometrix artefact eliminator (Cardiometrix, Bothell, WA, 98011) was used to ensure that the impedance between recording electrodes was below 5 kohms.

H-reflexes were initiated percutaneously using a Grass SD9B Stimulator (Grass Instruments Co. Quincy, MA, USA, 02169). A MacLab4 (AD Instruments, Bella Vista, Australia, 2153) was used to acquire the data which was displayed, processed and stored on a laboratory computer system (Apple Macintosh Quadra 650) using MacLab software (Scope version 3.3). EMG and stimulus data were sampled at 20 kHz for 100 msec. EMG activity was monitored on channel one of the MacLab and stimulus intensity was monitored on channel two.

A Tektronix ramp generator (Tektronix, Inc., Beaverton, USA, 97077) was used to generate a trigger pulse to start the data acquisition as the trigger pulse from the Grass

stimulator was too short to initiate data acquisition directly. The stimulus was passed through a 10:1 voltage reduction circuit (La Trobe University, Bundoora, Australia, 3086) as the stimulus voltage exceeded the range of MacLab. The responses were converted back to real voltages using appropriate multiplication factors.

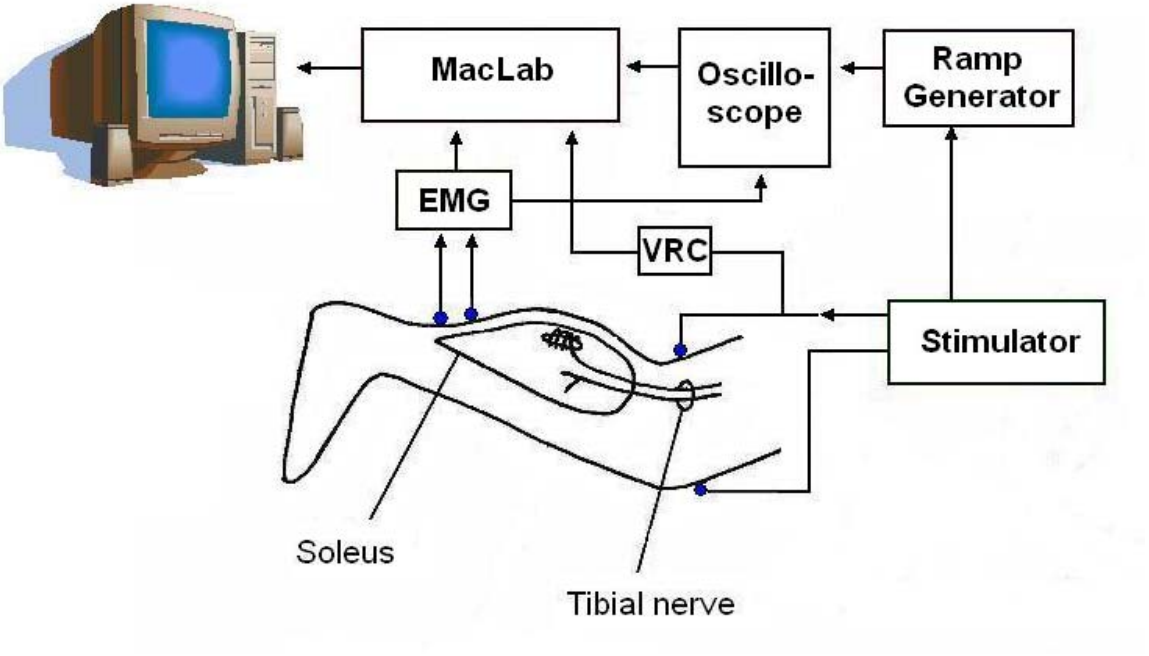

Figure 3.2 A schematic diagram of the experimental setup.
VRC= 10:1 voltage reduction circuit.

3.2.3 Procedures

Prior to the commencement of testing, experimental procedures were approved by the Health Sciences Faculty Human Ethics Committee at La Trobe University (ethics approval number FHEC04/110), and informed consent was obtained from each subject. Subjects were required to attend two testing sessions on two separate days approximately one week apart. Both of the sessions occurred at similar times in the day, and each session followed exactly the same procedures. Each session lasted approximately two hours. Subjects were asked to refrain from ingesting stimulating substances and from performing strenuous exercise for 12 hours prior to the testing session.

3.2.3.1 Subject preparation

Subjects were positioned prone on a plinth with their ankles extended past the end of the plinth. Shoes and socks were removed. The same investigator prepared and tested all of the subjects. In preparation for electrode placement, the skin over the soleus muscle and the fibular head of the dominant leg was shaved, abraded, and cleansed with isopropyl alcohol. Surface recording electrodes (Ag/AgCl 10 mm disk monitoring electrodes) were then adhered over the soleus and fibular head. Electrode placement over the soleus was determined using Hugon's method (Hugon, 1973a) according to the instructions detailed by Kukulka *et al.* (1985). The distance between the fibular head and the lateral malleolus was measured. One third of that measurement was then measured proximally from the medial malleolus and marked on the posterior coronal plane mid-line of the calf (Hugon, Delwaide, Pierrot-Deseilligny and Desmedt, 1973b; Kukulka *et al.*, 1985). This was the site for the positive surface electrode.

The negative surface electrode was placed distal to the positive electrode with a center-to-center electrode distance of 30 mm. Hugon's method is said to place the positive electrode approximately 2 cm distal to the insertion of the gastrocnemius muscle on the Achilles tendon, so that selective pick-up of the EMG responses of the soleus muscle can be obtained with minimal contamination from the gastrocnemius muscles (Hugon *et al.*, 1973b). The ground electrode was positioned over the fibular head which was chosen as a relatively silent area. A small amount of electro-conductive gel was applied on the electrodes to increase conductivity. The impedance between electrodes was tested, and

when satisfactorily low (<5 kohms), the electrodes were secured to the leg with foam tape.

The anode (positive stimulating electrode) was a 5x7 cm copper plate positioned on the anterior thigh just proximal to the patella. It was secured to the leg with an elastic Velcro strap. The cathode (negative stimulating electrode) was custom-made for the study. It consisted of a 5x7 cm lead plate wrapped around a 5x7 cm piece of polyethylene plastic with all of the edges on one side bevelled (Figure 3.3). Two elastic straps with Velcro ends were riveted to the two ends of the plastic in order for the cathode to be secured to the leg. The cathode was specially designed for the experiment based on the design used by Robinson, McComas and Belanger (1982). The cathode was placed in the popliteal fossa over the popliteal crease. A section of the lead on the back of the electrode was bent away from the plastic in order for an alligator clip to be attached. Electroconductive gel was applied on the anode and cathode for good coupling. Unipolar stimulation where the cathode is positioned over the nerve and the anode is positioned on the opposite side of the limb is recommended to selectively activate Ia afferent fibres at lower thresholds and to reduce the stimulus artefact (Palmieri *et al.*, 2004).

Figure 3.3 The custom-made cathode.

Commonly used cathodes comprise of small (approximately 1 cm diameter) spheres. The lead plate cathode was chosen over smaller designs as it is able to stimulate over a wider area. Trial tests found that it was very difficult to maintain the small cathode in a consistent position and its stimulation was very localised. This problem was not experienced with the lead plate cathode, and subjects reported that the sensation of the stimulations was more tolerable with the lead plate cathode than with the smaller design.

Correct placement of the cathode in the popliteal fossa was verified using the following criteria (Kukulka *et al.*, 1985; Voerman *et al.*, 2005; Vujnovich and Dawson, 1994):

- The H-reflex could be elicited without or with very small M-response;
- The H-reflex and M-responses displayed the same waveform through all stimulus intensities; and
- There was no change in the M-response amplitude with repeated submaximal stimulus intensities.

3.2.3.2 Testing procedures

The procedures involved two testing conditions (prone and standing) which were always tested in the same order. Prior to the commencement of testing, subjects were informed of the testing procedures and given a verbal description of the sensations they would experience from the electrical stimulations. For the prone position, subjects were instructed to relax and to maintain a constant body position with the head turned to the right throughout the testing period.

The tibial nerve of the right leg was stimulated percutaneously in the popliteal fossa with a stimulus duration of 1 millisecond and a frequency of 0.2 Hz. Stimulus frequency of 0.2 Hz has been recommended by previous investigators to prevent carry-over effects from the previous stimulation that can distort results if the stimulations occur too quickly (Voerman *et al.*, 2005). H-reflex and M-response recruitment curves were constructed by gradually increasing stimulus voltage from below threshold for the H-reflex to supramaximal for the M-response. The stimulus voltage was gradually increased rather than randomly selected, in an attempt to avoid any psychological stress that might be caused by an unexpectedly strong stimulation (Funase *et al.*, 1994). Ten stimulations

were recorded at each level of stimulus voltage before the voltage was manually increased by an increment of approximately one volt. Stimulus voltage was progressively increased in this manner until the H-reflex had become extinct and the M-response had reached its maximum amplitude. The curves of ten recordings at each level of stimulus intensity were averaged, and the peak-to-peak maximum H-reflex and M-response amplitudes were measured and used to generate the recruitment curves. Peak-to-peak amplitudes of the H-reflexes and M-responses were taken from the peak of the positive deflection from baseline to the trough of the subsequent negative deflection from baseline (Figure 3.4). In this way a variable number of 15-25 samples were obtained from each subject to construct the curves.

Subjects then assumed a position of relaxed, free standing. They were instructed to stand comfortably bearing equal weight through both feet and to keep their eyes fixed on a target placed on a wall approximately 3 metres in front of them. All subjects stood barefoot on a carpeted floor. A walking frame was placed in front of the subjects if required for balance and a chair was placed just behind them. Recruitment curves were again constructed in the same manner as previously described. When the subjects returned approximately one week later for their second testing session, exactly the same procedures were undertaken.

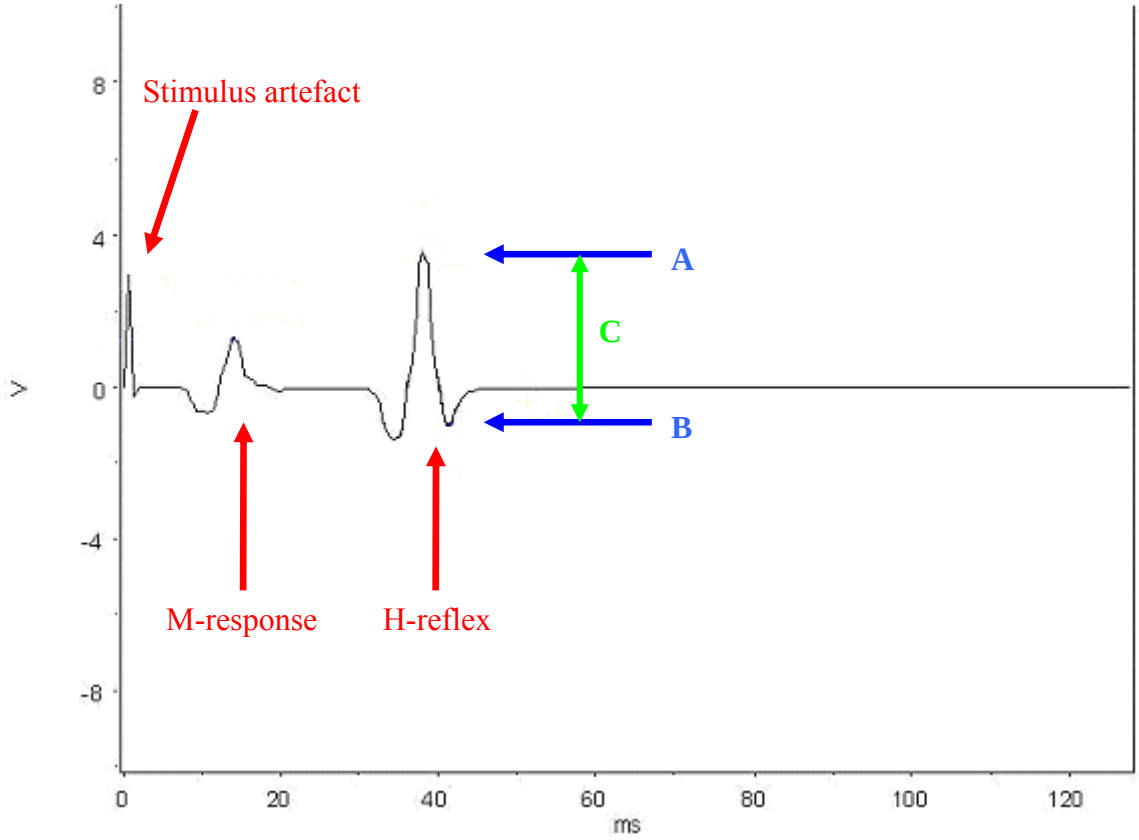

Figure 3.4 Example of measuring the peak-to-peak H-reflex amplitude.
A= The positive deflection from baseline; B= The subsequent negative deflection from baseline; C= The amplitude of the H-reflex.

3.2.4 Statistical analyses

Maximum M-response and H-reflex amplitudes were identified from recruitment curves recorded for each subject in both the prone and standing positions during each session. The maximum M-response and H-reflex amplitudes were then used to calculate H:M ratios. The inter-session reliability of the maximum M-response and H-reflex amplitudes as well as the H:M ratios were estimated using a two-way mixed effects model Intraclass Correlation Coefficient (ICC) for unordered pairs. ICCs are more appropriate to use than Pearson's correlation when the variables being correlated are unordered. ICCs and their associated 95% confidence intervals (CIs) were calculated using the Statistics Package for the Social Sciences (SPSS) Release 11.5.0 for Windows (SPSS Inc, Chicago, USA, 60606-6412).

The ICCs between the prone and standing positions were compared to determine if the dependent variables in one position were more reliable than another. A simple method of checking the 95% CIs for the ICCs was used. According to Lu and Shara (2007), an overlap between two 95% CIs indicates that there is no significant difference between two ICCs and no overlap indicates that there is a significant difference between two ICCs.

3.3 Results

The maximum M-response amplitudes were unable to be recorded in subjects 1 and 5 in the prone position, and subject 5 in the standing position as the EMG responses exceeded the range of the amplifier. Therefore, the M-response amplitudes from only eight subjects were able to be analysed for the prone position and the M-response amplitudes from nine subjects for the standing position (Table 3.3).

The maximum M-response and H-reflex amplitudes as well as the H:M ratios in both prone and standing for all subjects are presented in Table 3.3. The results of the statistical analyses demonstrated that the inter-session reliability for the maximum H-reflex amplitudes and H:M ratios were satisfactorily high both in prone (0.93 and 0.85) and standing (0.98 and 0.95) (Table 3.4). The ICCs for the maximum M-response amplitudes in prone (0.61) were not as reliable as in standing (0.96) which also reduced the reliability of the H:M ratios in prone (0.85). According to the method of Lu and Shara (2007), there was no significant difference between the reliability of the variables in prone or in standing.

Table 3.3 Maximum M-response amplitudes, maximum H-reflex amplitudes and H:M ratios for all subjects in prone and standing.

	Prone (mV)						Stand (mV)					
Subject	**M1**	**M2**	**H1**	**H2**	**H:M1**	**H:M2**	**M1**	**M2**	**H1**	**H2**	**H:M1**	**H:M2**
1	-	-	12.23	9.65	-	-	13.81	12.56	7.15	5.87	0.52	0.47
2	4.19	6.06	2.61	2.98	0.62	0.49	5.65	6.29	1.92	1.38	0.34	0.22
3	13.66	10.98	9.88	7.14	0.72	0.65	6.84	6.00	5.37	5.04	0.79	0.84
4	10.51	8.58	8.37	8.81	0.80	1.03	9.92	9.14	8.00	8.87	0.81	0.97
5	-	16.11	9.36	10.46	-	0.65	-	-	8.45	7.66	-	-
6	9.98	9.31	6.99	7.24	0.70	0.78	9.62	10.04	7.08	6.93	0.74	0.69
7	7.77	7.16	3.03	2.46	0.39	0.34	13.65	12.10	2.31	2.72	0.17	0.22
8	9.31	11.44	10.45	11.06	1.12	0.97	12.21	12.52	11.43	11.4	0.94	0.91
9	7.64	5.39	4.84	4.46	0.63	0.83	6.21	4.87	4.46	4.07	0.72	0.84
10	7.08	10.3	2.19	2.74	0.31	0.27	8.53	7.35	1.73	1.94	0.20	0.26
Mean	8.77	9.48	7.00	6.70	0.66	0.67	9.60	8.99	5.79	5.59	0.58	0.60
SD	2.80	3.27	3.62	3.32	0.25	0.26	3.10	3.00	3.21	3.21	0.28	0.31

M1= M-response session 1; M2= M-response session 2; H1= H-reflex session 1; H2= H-reflex session 2; H:M1= H:M ratio session 1; H:M2= H:M ratio session 2.

Table 3.4 Inter-session reliability of the maximum M-response amplitudes, maximum H-reflex amplitudes and H:M ratios in prone and standing.

Position	Dependent variable	N	ICC	95% CI
Prone	M-response	8	0.61	-0.09, 0.91
	H-reflex	10	0.93	0.73, 0.98
	H:M ratio	8	0.85	0.43, 0.97
Standing	M-response	9	0.96	0.84, 0.99
	H-reflex	10	0.98	0.92, 0.99
	H:M ratio	9	0.95	0.81, 0.99

3.4 Discussion

The results of this study have demonstrated that the inter-session reliability of the maximum H-reflex amplitudes and the H:M ratios were acceptable in both the prone and standing positions when assessed in able-bodied subjects. The maximum M-response amplitudes were reliable in the standing position but not as reliable in the prone position. This resulted in reduced reliability of the H:M ratios in the prone position. The standing position appeared to be more reliable for all of the variables than the prone position, although this was not statistically significant.

3.4.1 Reliability in prone and standing

The results of this study are consistent with the findings of a previous investigation that assessed inter-session reliability of the H-reflex in the prone position and two different standing positions: free standing, and standing while carrying a weighted box equivalent to 20% of body weight (Ali and Sabbahi, 2001). The authors found the standing positions to be more reliable than the prone position by observing the Pearson's correlation coefficient. (No statistical tests were conducted to compare the correlation coefficients between the positions.) The authors suggested that the higher reliability in standing compared with prone indicated more stability, consistency and less variability of the reflex during standing. During free standing, it is said that the spinal reflexes are "engaged" in closed loop activity with greater cortical control of movement and peripheral feedback signals to the CNS, as compared to the prone position when spinal reflexes are "disengaged" with less cortical control and less peripheral feedback (Ali and Sabbahi, 2001; Voerman *et al.*, 2005).

Hopkins *et al.* (2000) and Mynark (2005) found contrary results where the inter-session reliability was lower in standing and higher in supine when tested in able-bodied subjects. (Again, no statistical tests were conducted to compare the ICCs between the positions.) This discrepancy may be attributed to the different positions tested, as it appears that prone and supine positions have different effects on MN excitability. The difference between these two positions on MN excitability has not previously been investigated and warrants further research. How the subject is standing also appears to have an effect on MN excitability. Hopkins *et al.* (2000) tested subjects while standing

on a single leg and balancing with one hand on a table. It was suggested that sway by the subjects during recording in this position may have affected the measures. The distance the contralateral foot was lifted from the ground and the amount of pressure placed on the balancing hand may also have affected the results (Hopkins *et al.*, 2000). Variability in descending cortical control may have contributed to the poorer reliability in the standing position due to increased mental alertness and concentration required to maintain standing balance on one leg. This may have been especially relevant since the electrical stimulations would have caused contractions of the soleus muscle leading to perturbations in posture.

3.4.2 M-response amplitudes

The maximum H-reflex amplitudes in both positions were more reliable than the maximum M-response amplitudes. This may be due in part to the smaller sample size for the M-responses. The maximum M-response amplitudes for two subjects were unable to be recorded as the maximum recordable value of the analogue to digital converter was 10.23 volts, and those subjects exceeded this range. The EMG amplifier and digital analogue converter used in this study were designed to record the EMG of muscles during natural movements such as walking. By electrically stimulating the tibial nerve at very high intensities, the whole MN pool was activated causing activity which exceeded the design specifications of the EMG amplifier. This kind of muscle activity is normally not exerted during natural movements and may explain why the equipment used was inadequate for the recording of maximum M-response amplitudes. This can be overcome by using equipment with larger ranges or incorporating another voltage reduction converter into the equipment setup.

Another reason why the M-responses were not as reliable as the H-reflexes may be because it was more difficult to ascertain the maximum amplitude of the M-responses than the H-reflexes. The H-reflex reaches a clear maximum amplitude before it then declines with increasing stimulus voltages. The M-response amplitude increases and eventually plateaus, however, it is difficult to determine the point at which the curve plateaus without stimulating at increasingly high voltages. Difficulty with identifying the maximum M-response amplitude may also be experienced in cases where the M-response

recruitment curve does not rise smoothly. Variability in the shape of the curve could be misinterpreted as the maximum amplitude. There was a risk that the testing sessions may have been ceased too early or that stimulus voltages were not progressed high enough. However, increasing the length of the testing session and stimulating at high voltages may increase patient discomfort and fatigue which is known to affect MN excitability.

3.4.3 Implications

The findings of this study suggested that specific practices need to be employed when conducting H-reflex testing to ensure high reliability of measurements. This had important implications for the planned experiments in this thesis which used the H-reflex as the main measurement tool. Pierrot-Deseilligny and Mazevet (2000, p78) stated that "the technique of the H-reflex is simple, but a strict methodology is required to be able to validly interpret the results". It may not be possible to control each factor that is known to have an effect on MN excitability, however, good reliability can be achieved if a strict protocol is used and assessors are adequately trained (Voerman *et al.*, 2005).

Subject positioning appeared to be an important factor in the determination of reliability evidenced by the comparison of results between the current study and those of previous studies where different positions were tested. The standing position appeared to be the best position for obtaining reliable results in this study. This study has also identified the need to ensure that the equipment used to record H-reflexes and M-responses is adequate to record at all expected ranges.

3.5 Conclusion

This study has demonstrated that the H-reflex amplitudes are highly reliable when tested in prone and standing at two separate testing sessions. The M-response amplitudes were highly reliable in the standing position but less reliable in the prone position which affected the reliability of the H:M ratios. There was no statistically significant difference between the reliability of the values in prone or in standing. The study hypothesis was not completely supported by the results of this study as not all of the dependent variables were found to be reliable. This study contributed to meeting the first aim of this thesis as it established the reliability of the H-reflex which was used in measuring the neurophysiological effect of TRAFOs.

Chapter Four

The neurophysiological effect of TRAFOs in able-bodied subjects

4.1 Introduction

When assessing the effects of an intervention on MN excitability, previous investigators have conducted their investigations by assessing able-bodied subjects before assessing pathological subjects (Kukulka, Beckman, Holte and Hoppenworth, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988). This serves two purposes: firstly, it allows the procedures to be fine tuned so that they run smoothly when testing pathological subjects, and secondly, it demonstrates the response of a healthy neuromuscular system to the intervention. The results of the able-bodied subjects may then be compared with the results of the pathological subjects, which may provide further information regarding any observed effects.

4.1.1 Tone-reducing devices

A detailed review of spasticity, TRAFOs and the H-reflex was presented in Chapter Two. As was mentioned in the literature review, any orthosis can be made into a TRO by the addition of tone-reducing features to specifically target and treat spasticity (Rogers and Vanderbilt, 1990). This study focused on the evaluation of three common tone-reducing features for the lower limbs that can be added to a standard AFO design: an inhibitory toe bar (ITB), the application of pressure to the Achilles tendon and an orthokinetic compression garment (OCG).

4.1.1.1 Inhibitory toe bar

An ITB is a long narrow pad placed in the toe sulcus region. It is mainly used to inhibit the toe grasp reflex which is said to be a primitive tonic reflex of the foot that may become hyperexcitable following an UMN lesion (Duncan, 1960; Duncan and Mott, 1983). There are supposedly four tonic reflexes of the foot that can cause deformity and muscle imbalance in patients with spasticity (Duncan, 1960):

1. Toe grasp reflex;
2. Eversion reflex;
3. Inversion reflex; and
4. Dorsiflexion reflex.

Although there is no evidence to prove that primitive reflexes return following UMN lesions and contribute to spasticity, they will be discussed as many tone-reducing features were developed based on this theory. Primitive tonic foot reflexes are believed to be superficial reflexes elicited by cutaneous stimulation or pressure in specific regions on the plantar surface of the foot (Figure 4.1) (Duncan and Mott, 1983). The toe grasp reflex occurs in response to stimulation of the ball of the foot, near the head of the second and third metatarsal heads. This reflex causes flexion and adduction of the toes as well as associated contractions in the soleus, gastrocnemius and hamstring muscles (Duncan, 1960). The eversion reflex occurs in response to stimulation of the lateral border of the foot over the fifth metatarsal head. It causes eversion of the foot and associated contractions in the vastus lateralis and tensor fascia lata muscles (Duncan, 1960). The inversion reflex occurs in response to stimulation of the medial border of the foot near the head of the first metatarsal. It causes inversion of the foot and associated contractions in the internal hamstrings. Lastly, the dorsiflexion reflex occurs in response to stimulation of the plantar surface of the heel and causes the foot to dorsiflex (Duncan, 1960).

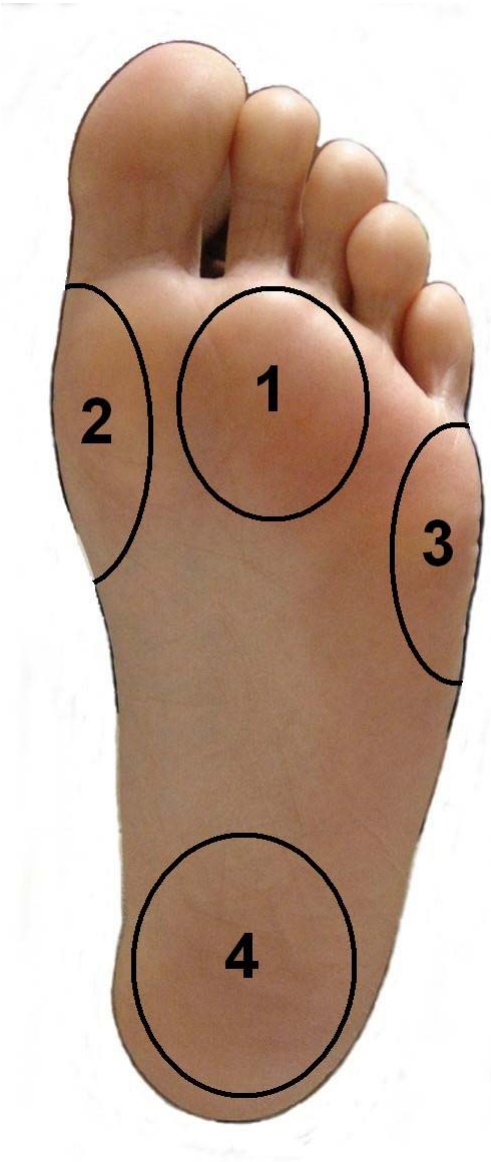

Figure 4.1 The reflexogenous areas on the plantar surface of the foot.
1= Toe grasp reflex; 2= Inversion reflex; 3= Eversion reflex; 4= Dorsiflexion reflex; (adapted from Duncan and Mott, 1983).

The ITB is thought to inhibit the toe grasp reflex by decreasing the amount of pressure on the ball of the foot which is the reflexogenous area responsible for the reflex (Bronkhorst and Lamb, 1987). It also extends the toes at the metatarso-phalangeal joints and prevents them from contracting into a flexed position. By inhibiting the toe grasp reflex, the ITB may also inhibit spasticity in the extensor muscles of the lower limbs (including the soleus muscle) which are said to exhibit associated contractions when the toe grasp reflex

is elicited (Duncan, 1960). It is also believed that extension of the toes has a tone-reducing effect on abnormal extensor responses in the lower limbs (Nash *et al.*, 2008). Studies that have evaluated the effects of ITBs have generally found favourable results. Rogers de Saca *et al.* (1994) evaluated the effects of toe spreaders on hemiplegic subjects who exhibited the toe grasp reflex. The toe spreader is similar to the ITB except that it also abducts the toes. The investigators found that the toe spreaders effectively inhibited the toe grasp reflex and improved walking parameters, however, there were no effects on muscle activity. Iwata *et al.* (2003) also found that ITBs were effective in inhibiting the toe grasp reflex and improved walking parameters in hemiplegic subjects when they were attached to standard AFOs. These improvements were only evident in subjects who exhibited the toe grasp reflex. Hemiplegic subjects who did not exhibit the reflex did not experience any significant improvements in their walking with the use of ITBs.

4.1.1.2 Tendon pressure

It has been reported that firm pressure applied at the insertion of a muscle has tone-reducing effects (Lohman and Goldstein, 1993). This theory can be applied to AFO management by applying pressure to the insertion of the Achilles tendon to inhibit spasticity in the triceps surae muscles. Tendon pressure is said to inhibit spasticity by stretching the tendon and stimulating GTOs which have inhibitory effects on the alpha MNs of the muscle whose tendon is being stretched (Kukulka *et al.*, 1985; Robinson *et al.*, 1982).

Zachazewski *et al.* (1982) found that pressure applied to both sides of the Achilles tendon in a TRAFO improved gait characteristics and eliminated the positive support reaction in a 25 year old male with spasticity secondary to ABI. The positive support reaction is the onset of spasticity in the lower limb characterised by excessive ankle plantarflexion and knee extension in response to loading of the foot, particularly the metatarsal heads (Charlton and Ferguson, 2001; Nash *et al.*, 2008). These results were only based on observation and no other AFO was used for comparison.

Smith (1995) conducted a study on four healthy subjects to determine if the application of pressure to both sides of the Achilles tendon by an AFO would alter muscle activity in

the triceps surae muscles. No effects were demonstrated in this study, however, this may have been due to the very small sample size, the measurement method used, the fact that subjects were seated and the application of pressure to either sides of the Achilles tendon rather than directly over the tendon. It has been suggested that pressure applied to either sides of the tendon is less effective than pressure applied directly over the tendon (Carlson, 1984). A study conducted to determine the effect of tendon pressure on the soleus H-reflex of ten healthy subjects found that pressure applied directly onto the tendon decreased H-reflex amplitudes significantly in all subjects (Robinson *et al.*, 1982). However, pressure applied to the side of the tendon had no effect in seven subjects and caused an increase in H-reflex amplitude in one subject (Robinson *et al.*, 1982).

Other studies that have applied pressure directly over the Achilles tendon have demonstrated decreased MN excitability in the soleus muscle as indicated by reduced H-reflex amplitudes (Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988; Robinson *et al.*, 1982). In these studies, pressure was applied using purpose built pressure applicators and all subjects were tested in the prone position. Both healthy subjects and hemiplegic subjects were tested, and MN excitability was significantly reduced with the application of tendon pressure in both subject groups. No similar studies have been conducted to test the effects of pressure applied directly over the Achilles tendon by an AFO while in a standing position.

4.1.1.3 Orthokinetics

The concept of orthokinetics was developed in 1927 by an orthopaedic surgeon named Julius Fuchs (Lohman and Goldstein, 1993). He intended to create a flexible, dynamic device to discourage joint immobilisation and avoid the discomfort that can be associated with wearing rigid, static orthoses (Farber, 1982). Orthokinetics involves the construction of flexible sleeves that incorporate elastic and inelastic segments called “active” and “inactive” fields to facilitate or inhibit muscle activity. The elastic part of the orthokinetic sleeve is placed where muscle activity is desirable, and the inelastic part is placed where muscle activity is undesirable.

Lohman and Goldstein (1993) described other materials that could have similar effects on muscle activity. They stated that cool, rigid, and smooth materials could be used as inactive field materials to inhibit muscle activity, and that warm, expansive, and textured materials could be used as active field materials to facilitate muscle activity. It is said that contraction of the muscle underlying the textured material causes the active field to stimulate the dermatome over that muscle (Farber, 1982). In this way, orthokinetics is said to utilise the exteroceptors of the skin and indirectly the proprioceptors of muscles and tendons to decrease muscle tone (Blashy and Fuchs, 1959).

The benefits of orthokinetics may also be due to the circumferential pressure that the sleeves apply around muscles. Therefore, orthokinetic sleeves have been termed "orthokinetic compression garments (OCGs)" for this thesis. It has been previously demonstrated that circumferential pressure applied around the leg decreases soleus H-reflex amplitudes in healthy subjects (Robichaud, Agostinucci and Vander Linden, 1992), as well as subjects with stroke (Robichaud *et al.*, 1992) and SCI (Robichaud and Agostinucci, 1996). Air splints were used to apply the circumferential pressure in these investigations, and the reduced H-reflex amplitudes were only observed during the period of pressure application. The investigators attributed these effects to the stimulation of cutaneous afferent nerve fibres. It is believed that group III and/or IV afferent fibres from cutaneous receptors can be responsible for reducing the spasticity of underlying muscles (Ushiba *et al.*, 2004).

The effects of OCGs on patients with spasticity have been reported as rapid pain relief, immediate mobilisation, increased muscle power and increased ROM of the joints and limb segments enclosed by the OCG (Blashy and Fuchs, 1959). These results suggest that OCGs have the ability to reduce spasticity. However, these results were taken from case studies that were documented in 1959 (Blashy and Fuchs, 1959), and the authors based their results entirely on clinical observations rather than objective testing. Since then, there has been very limited research conducted on OCGs.

4.1.2 Summary and aims

The tone-reducing features mentioned above can potentially all be used in conjunction with AFO management to decrease MN excitability in subjects with spasticity. In this study, the application of tendon pressure, ITB and OCG were selected for assessment as they represent three commonly proposed mechanisms by which tone-reducing features are said to act: by the stimulation of GTOs, stimulation of reflexogenous areas on the plantar surface of the foot, and stimulation of cutaneous receptors over target muscles. Despite the research that has been conducted on these tone-reducing features, their effect on MN excitability in conjunction with AFO use has not yet been examined.

Therefore, the purpose of this study was to assess the effect of ITBs, tendon pressure and OCGs when incorporated into AFOs on soleus MN excitability in able-bodied subjects while standing. It was hypothesised that the soleus MN excitability would decrease when the tone-reducing features were used with the AFOs.

4.2 Methods

4.2.1 Subjects

Ten able-bodied subjects were recruited from the University student population for this study. The subject characteristics are summarised in Table 4.1. Inclusion in the study required subjects to be between the ages of 18 and 80, have a good level of health and a medical history free from any neurological disorders. None of the subjects had any history of skeletal or soft tissue injury to their dominant lower limbs.

Table 4.1 Subject characteristics.

Subject	Sex	Age	Weight (Kg)	Height (cm)	Dominant Side
1	Male	21	75	179	Right
2	Male	20	71	176	Right
3	Female	21	50	159	Right
4	Male	21	73	178	Right
5	Male	20	65	170	Right
6	Female	19	52	156	Right
7	Female	19	64	160	Right
8	Female	23	52	165	Right
9	Female	21	73	165	Right
10	Male	22	77	177	Right
Total and Means	5 Female, 5 Male	20.7 ± 1.25	65.2 ± 10.39	168.5 ± 8.66	10 Right, 0 Left

4.2.2 Apparatus

The same experimental apparatus and equipment was used from the previous study (see section 3.2.2). However, there were few changes to the equipment and setup. Firstly, the computer system in the laboratory was changed from an Apple Macintosh computer to a Dell computer (Optiplex GX260). As a result, the MacLab was changed to a PowerLab/410 (AD Instruments, Bella Vista, Australia, 2153) which was used to digitise EMG and stimulator signals. Data were sampled at 20 kHz for 0.128 seconds and processed using PowerLab software (Scope version 3.3). The relatively high sampling frequency facilitated detection of peak amplitudes in H and M waves during analysis.

In the previous study, the EMG responses exceeded the range of the analogue to digital converter, therefore, the maximum M-response amplitudes for some subjects were unable to be recorded. To overcome this problem, another 10:1 voltage reduction circuit (La Trobe University, Bundoora, Australia, 3086) was used to reduce EMG signals from the muscle recordings. A schematic diagram of the experimental setup is shown in Figure 4.2.

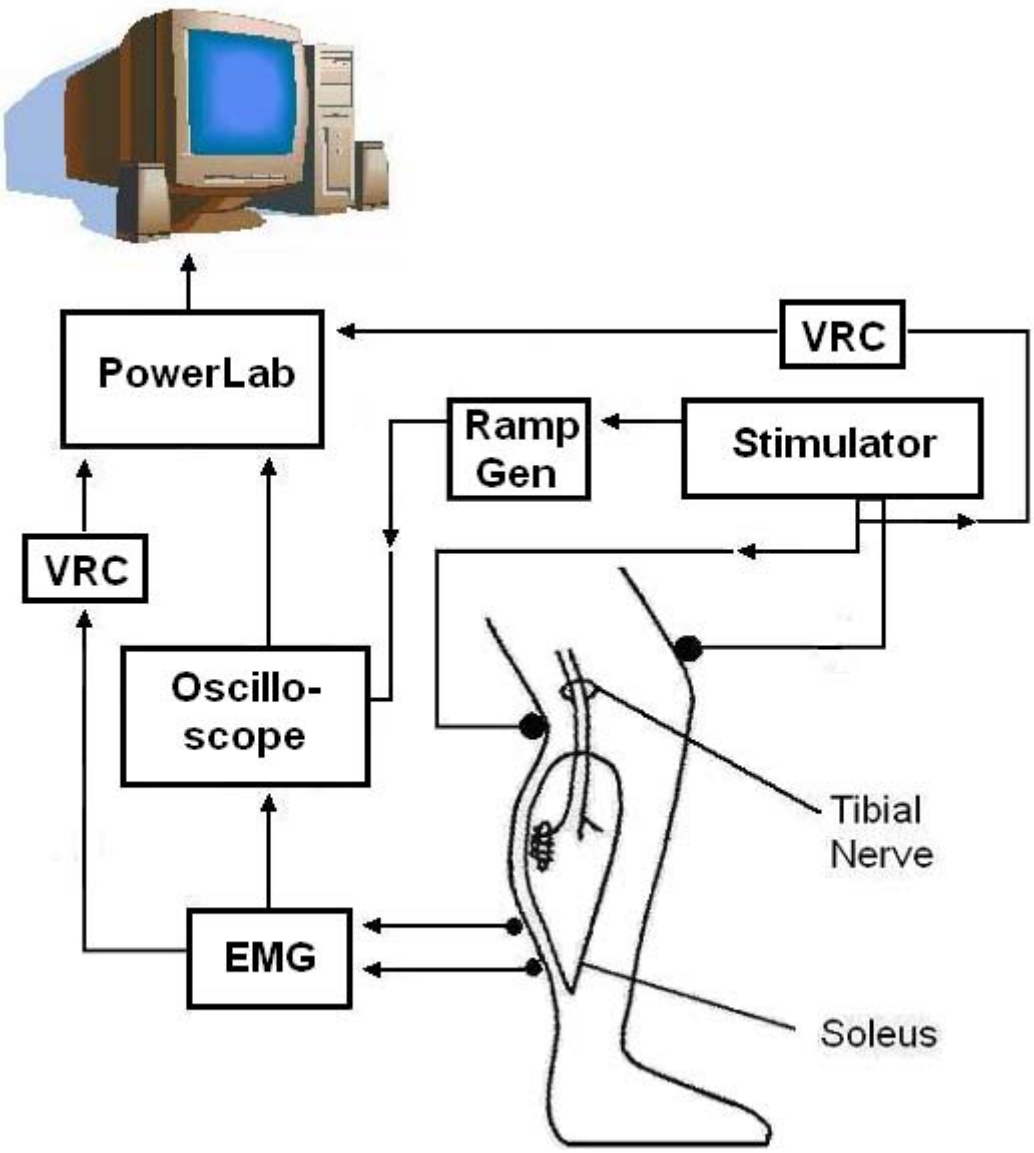

Figure 4.2 A schematic diagram of the experimental setup.
VRC= 10:1 voltage reduction circuit; Ramp gen= Ramp generator.

4.2.3 Procedures

Prior to the commencement of testing, experimental procedures were approved by the Health Sciences Faculty Human Ethics Committee at La Trobe University (ethics approval number FHEC04/110) and informed consent was obtained from each subject.

4.2.3.1 TRAFO fabrication

The dominant leg of each subject was cast to fabricate a custom-made solid AFO. The ankle was cast in a neutral position (tibia and foot aligned at 90 degrees and the subtalar joint neither inverted or everted). The trim-lines followed those for standard solid AFOs with full-length footplates (Weber, 1990). The only unusual feature of the AFOs was that they had windows (approximately 35x50 mm) cut out of the posterior shells to allow for electrode placement over the soleus muscle (Figure 4.3).

Each of the plaster casts underwent the same standard AFO modifications:

- The lateral and medial malleoli were built up by approximately 5 mm;
- The area over the Achilles tendon was built up so that it was not too curved in the sagittal plane;
- The medial longitudinal arch was slightly loaded;
- The plantar surface of the forefoot was levelled so that the cast stood stable on a bench with no inversion or eversion;
- The forefoot section was extended distally to accommodate a full-length footplate that would finish distal to the toes; and
- The anterior trim-lines were built up to facilitate donning and doffing.

The shoes that were used in this project were flat soled casual lace-up shoes, so no pitch was modified into the casts.

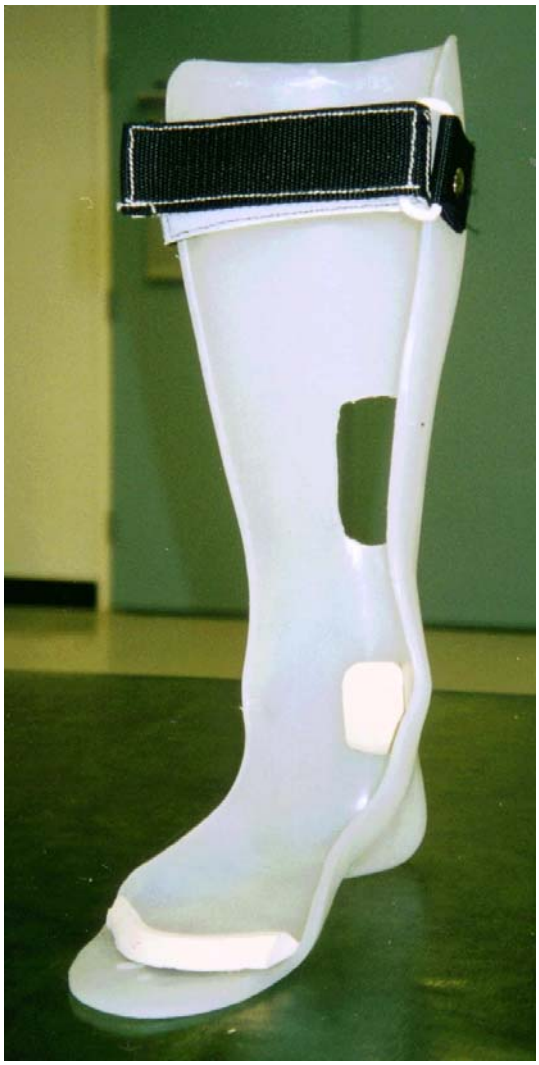

Figure 4.3 A custom-made TRAFO with the inhibitory toe bar and tendon pressure pad attached.

In preparation for moulding, each cast had two ¼ inch holes drilled into the mandrel, one at the ankle and one at the proximal third of the leg on the anterior surface to facilitate suction of the plastic onto the cast. Each cast also had a length of Dacron twisted into a rope stapled down the anterior surface of the cast to further facilitate suction.

Before moulding, the casts were placed in an oven set at 180 degrees for at least 15 minutes. Five minutes before the plastic was ready for moulding, the casts were removed from the oven, dressed with a nylon stockinette and sprayed with silicon. The casts were then moulded with 5 mm polypropylene plastic. Once cooled, the AFOs were cut off the

casts, trimmed, and all of the edges were smoothed. The posterior windows were then cut into the plastic directly over the area for the electrode placement (Figure 4.3).

Custom-made ITBs were fabricated for each subject following the fabrication process detailed by Iwata *et al.* (2003). Each subject's foot was placed on a piece of paper and traced with a pen. The distal edge of the ball of the foot and the touching point of the toes to the floor were marked with two points adjacent to each toe. When the foot was removed from the paper, the points were connected to create a template for the ITB (Figure 4.4). The templates were traced onto Pelite (a polyethylene closed cell foam), cut out, and the edges were rounded. In their study of the effects of ITBs, Iwata *et al.* (2003) used 5 mm Pelite to create the bars and found that although the bars significantly improved walking, they failed to completely inhibit the toe grasp reflex. The authors suggested that thicker toe bars would be more effective, therefore, 10 mm Pelite was used in this study.

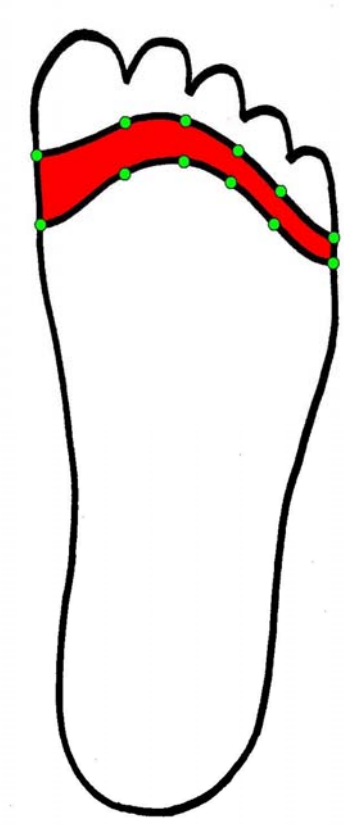

Figure 4.4 Foot tracing to produce the inhibitory toe bar template.
The green dots show the points adjacent to each toe which were connected to produce the ITB template (coloured in red).

The tendon pressure pads were fabricated from 15 mm Pelite. Rectangles (30x40 mm) were cut out and heat moulded into the Achilles relief of the AFOs at the level of the malleoli. The edges were then smoothed (Figure 4.3).

The OCGs were fabricated using vinyl and 3 mm neoprene. The shiny, smooth, inelastic vinyl was used as the inactive field over the plantarflexors, and the two-way stretch neoprene was used as the active field over the dorsiflexors (Figure 4.5). Separate templates for each of the two materials were drawn from measurements of the subject's legs. Four measurements were taken to produce the templates:

1. Distance from the fibular head to the lateral malleolus;
2. Proximal calf circumference;
3. Mid-calf circumference; and
4. Distal calf circumference (just proximal to the malleoli).

The template for the vinyl to cover the posterior half of the leg was produced by halving the circumferential measures. The template for the neoprene to cover the anterior half of the leg was produced by halving the circumferential measurements and then reducing them by 20%. The 20% reduction accommodated the stretch in the fabric and allowed for the sleeves to apply some circumferential pressure to the limb. The two materials were sewn together with the seams on the outer surface.

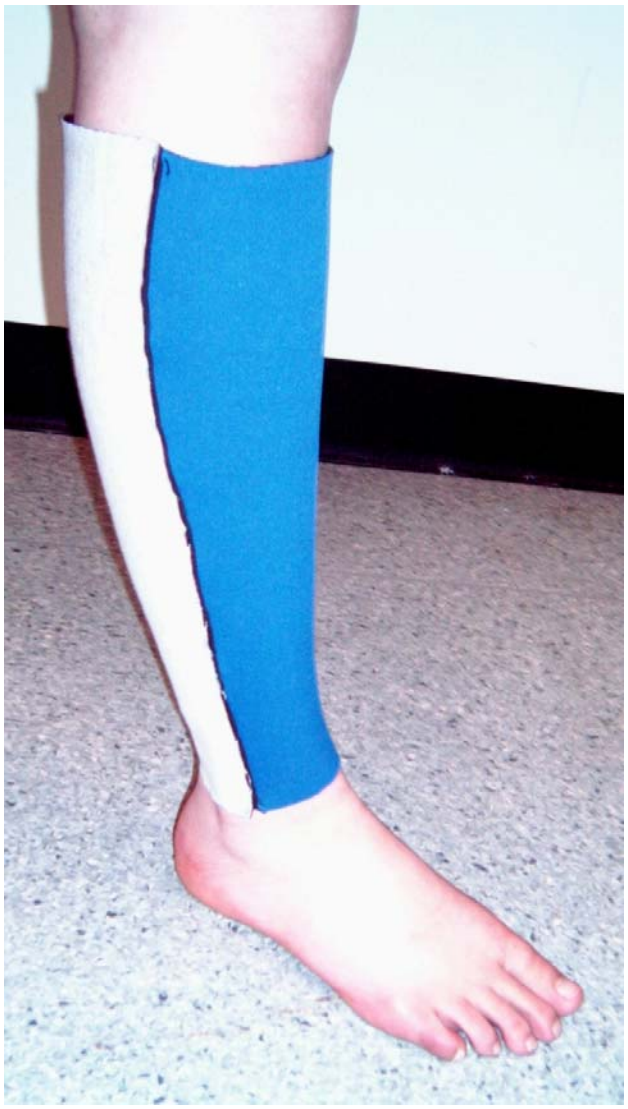

Figure 4.5 An orthokinetic compression garment.

4.2.3.2 Subject preparation

Subjects were asked to refrain from ingesting stimulating substances and from performing any strenuous exercise for the 12 hours prior to the testing session. The procedures for preparing subjects for experimental testing were the same as those detailed in the previous study (see section 3.2.3.1). All of the subjects were required to verbally confirm that the AFOs were comfortable to stand in before the testing could begin. If there were any areas of discomfort or issues with the fitting of the orthoses, these were rectified before the testing commenced.

4.2.3.3 Testing procedures

Once subjects were prepared and ready to begin the testing, they were given a verbal explanation of the testing procedure and a description of what the stimulator would feel like. Subjects were then given an accommodating period to become accustomed to the sensation of the stimulator in the standing position. A walking frame was positioned in front of the subjects and a chair was placed behind them. Subjects were instructed to bear equal weight through both legs, to use the frame only as a balance aid (if required), to remain relaxed, and to fix their gaze on a target placed on a wall approximately 3 metres in front of them.

The design of the repeated-measures intervention study followed an ABA format. An initial baseline (A_1) observation phase was used to identify the typical baseline performance characteristics of each subject's MN excitability. In the subsequent intervention (B) phases, the interventions were introduced with continuing monitoring of subject's MN excitability. In the final baseline (A_2) phase, the intervention was withdrawn while subject monitoring continued to determine the presence of any carryover effects.

Testing began once the subjects indicated that they were comfortable with the testing situation. For each of the testing conditions, H-reflex and M-response recruitment curves were constructed. There were six conditions tested:

A_1 - AFO only (baseline);
B_1 - AFO with tendon pressure;
B_2 - AFO with ITB;
B_3 - AFO with OCG;
B_4 - AFO with all three tone-reducing features; and
A_2 - AFO only (return to baseline).

To minimise the possibility of any series effects, the order of conditions B_1 to B_4 was presented either forwards or backwards for alternate subjects. This was done instead of presenting the conditions in a random order to decrease the effects of electrode movement caused by donning and doffing the OCG. Since conditions B_3 and B_4 required the OCGs to be worn, they were always presented one after the other and would occur

either at the beginning or end of the intervention conditions. The subject's own flat lace-up shoes were worn throughout the testing. The different tone-reducing devices were attached to the AFOs using double-sided adhesive tape allowing them to be removed and attached as required for each condition. The two conditions involving the tendon pressure pads required the addition of ankle straps to be worn around the AFOs to increase pressure on the Achilles tendon, and ensure that the tendon pressure pads were not pushing the foot and ankle forward in the AFOs.

The stimulus duration was 1 millisecond and the stimulus frequency was 0.2 Hz. Recruitment curves were constructed by gradually increasing stimulus voltage from below threshold for the H-reflex to supramaximal for the M-response. Further details on the experimental procedures can be found in section 3.2.3.2. In order to decrease the length of the testing session, it was decided to decrease the number of recordings at each level of stimulus intensity from ten stimulations (which was done in the previous study) to four. This was done since there were six conditions to be tested, and any more than four stimulations at each level of stimulus intensity would have caused the whole testing session to exceed two hours. It is known that muscle fatigue and mental fatigue of the subject can affect the H-reflex (Brinkworth *et al.*, 2007; Crayton and King, 1981; Eke-Okoro, 1982; Matthews, 1966), therefore, it was deemed more important to preserve the subject's state than to gather more data. Furthermore, previous investigators have demonstrated that decreasing the number of recordings from ten to four does not significantly decrease the reliability of the results (Handcock *et al.*, 2001; Hopkins *et al.*, 2000; Williams *et al.*, 1992). In the reliability study reported in Chapter Three, reliability estimates based on ten stimulations were similar to those reported by other investigators (Handcock *et al.*, 2001; Hopkins *et al.*, 2000; Williams *et al.*, 1992). It was assumed, without reanalysis of the reliability data reported in Chapter Three, that similar reliability would be obtained for four stimuli at each stimulus voltage as has been reported by those investigators.

Four stimulations were recorded at each level of stimulus voltage before the voltage was increased by an increment of approximately one volt. Stimulus voltage was progressively increased until the H-reflex had become extinct and the maximum M-response amplitude had been reached. The mean curve of the four recordings at each level of stimulus

intensity was obtained and used to construct the recruitment curves for the H-reflexes and the M-responses. In this way, data for a variable number of 15-25 stimulus voltages were obtained from each subject to construct the recruitment curves.

4.2.4 Statistical analyses

H:M ratios were calculated from the recruitment curves for each condition. A one-way repeated measures Analysis of Variance (ANOVA) was used to test for differences in mean H:M ratios between the conditions. Mauchly's test of sphericity was used to determine whether distributions were normal, and where they were not, a Greenhouse-Geiser adjustment was used (Corston and Colman, 2003). Statistical significance was set at α=0.05 and all statistical analyses were performed using SPSS Release 11.5.0 for Windows (SPSS Inc, Chicago, USA, 60606-6412).

In addition to the ANOVA, the 95% range of change ($R_{95\%}$) for the H:M ratio was calculated to determine which within subject measures were significantly different (Boyd, Fatone, Rodda *et al.*, 1999a). According to Chinn (1991), the $R_{95\%}$ can be calculated as:

$$R_{95\%} = 1.96SD(y_1 - y_2)$$

where y_1 and y_2 are the mean H:M ratios for the two baseline conditions (AFO only conditions), and SD is the standard deviation of the mean difference between the two baseline H:M ratios.

However, using 1.96 as the critical value for *z* assumes a large sample and is highly optimistic. For a more conservative estimate, the critical value for *t* with *df*=9 was used as the multiplier. Any measures that fell outside of the range were considered to be indicative of a significant response to the intervention.

4.3 Results

The ANOVA demonstrated that there were no significant differences in the mean H:M ratios between any of the conditions (F=0.360, df=1.645, p=0.663) (Figure 4.6). The $R_{95\%}$ was used to further examine the data to determine whether any substantial changes within individual subjects had been overlooked in the averaging process. The $R_{95\%}$ interval for the H:M ratio was found to be 0.225. None of the H:M ratios for any individual for any of the conditions fell outside of the $R_{95\%}$ of the mean baseline conditions (Table 4.2).

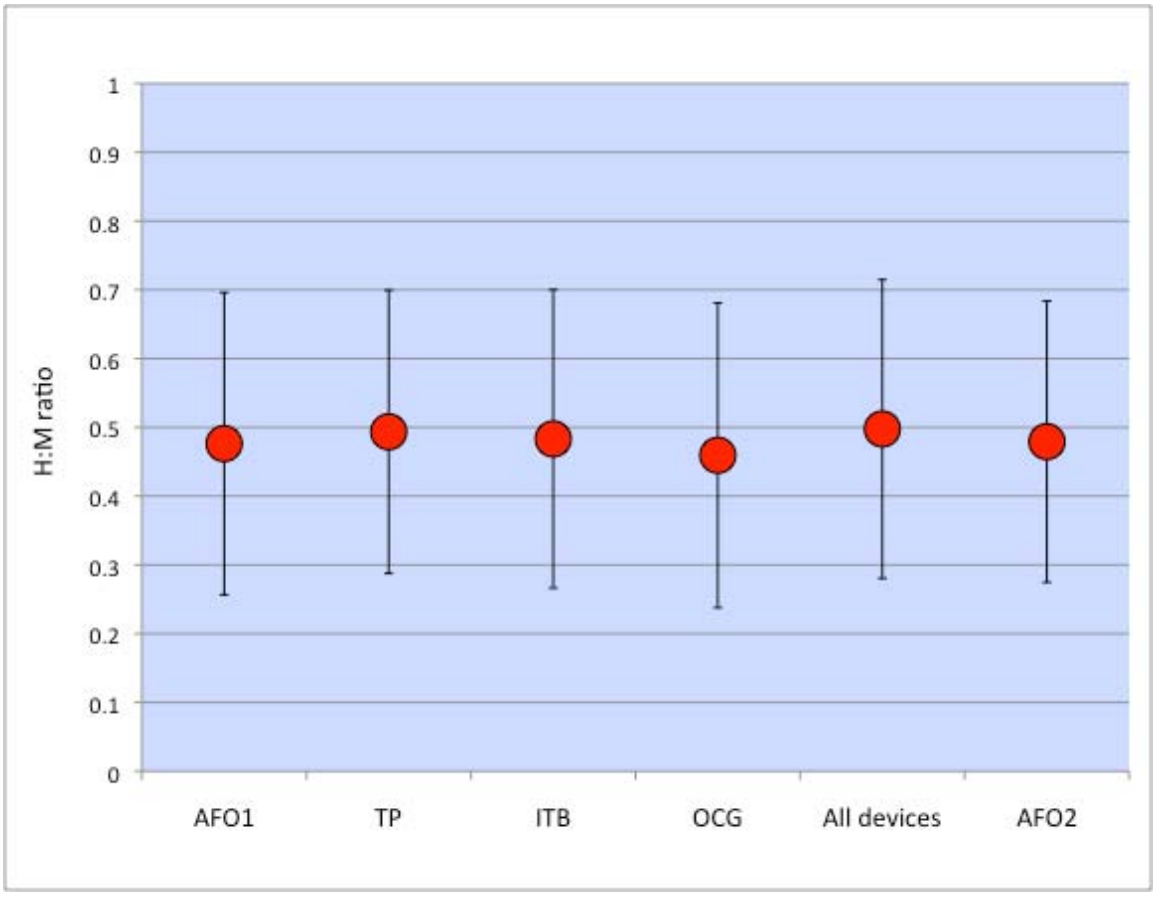

Figure 4.6 Mean (±1 SD) H:M ratios for all subjects across all conditions.
AFO1= Ankle-foot orthosis initial baseline; TP= Tendon pressure; ITB= Inhibitory toe bar; OCG= Orthokinetic compression garment; AFO2= Ankle-foot orthosis baseline return.

Table 4.2 H:M ratios and the $R_{95\%}$ for each subject.

Subject	AFO1	TP	ITB	OCG	All devices	AFO2	Mean of AFO1 and AFO2	$R_{95\%}$
1	0.58	0.51	0.45	0.36	0.46	0.38	0.48	0.26 - 0.71
2	0.39	0.39	0.40	0.42	0.50	0.42	0.41	0.18 - 0.63
3	0.13	0.20	0.20	0.14	0.17	0.14	0.14	-0.09 - 0.36
4	0.21	0.53	0.48	0.45	0.28	0.48	0.34	0.12 - 0.57
5	0.53	0.58	0.61	0.62	0.67	0.64	0.58	0.36 - 0.81
6	0.86	0.93	0.94	0.90	0.87	0.87	0.86	0.64 - 1.09
7	0.29	0.28	0.27	0.24	0.27	0.28	0.29	0.06 – 0.51
8	0.59	0.62	0.69	0.67	0.71	0.66	0.63	0.40 - 0.85
9	0.55	0.54	0.43	0.47	0.54	0.51	0.53	0.31 - 0.76
10	0.64	0.35	0.36	0.34	0.52	0.42	0.53	0.31 - 0.76

AFO1= Ankle-foot orthosis baseline 1; TP= Tendon pressure; ITB = Inhibitory toe bar; OCG = Orthokinetic compression garment; AFO2= Ankle-foot orthosis baseline 2.

4.4 Discussion

The results of this study indicated that the application of pressure to the Achilles tendon, the ITBs and the OCGs did not significantly alter soleus MN excitability in able-bodied subjects when attached to standard solid AFOs. These results did not support the study hypothesis. The lack of effect of the tone-reducing features challenges the results of previous investigations on TRAFOs that have attributed observed improvements in gait to the tone-reducing effects of the orthoses (Bronkhorst and Lamb, 1987; Carlson, Vaughan, Damiano and Abel, 1997; Dieli *et al.*, 1997; Ford *et al.*, 1986; Haberman, 1990; Harris and Riffle, 1986; Iwata *et al.*, 2003; Taylor and Harris, 1986; Zachazewski *et al.*, 1982). Although the TRAFOs of the present study were assessed in standing and not walking, this study was the first to directly measure the neurophysiological effect of TRAFOs on MN excitability in an upright standing position.

Iwata *et al.* (2003) examined the effects of ITBs on the gait of hemiplegic subjects and found that use of ITBs attached to AFOs significantly increased walking speed, stride length and cadence in subjects who exhibited the toe grasp reflex. They stated that the ITBs may have improved gait by reducing spasticity diffusely in the leg even though they did not measure spasticity directly. The results of this present study suggest that the improvements observed by Iwata *et al.* (2003) may have been due to alterations in biomechanics rather than alterations in the underlying neurophysiology.

Early literature on the original OCGs claimed that the devices may improve joint ROM, speed of limb reaction, muscle strength, co-ordination and pain primarily through cutaneous stimulation (Blashy and Fuchs, 1959; Gracies, Marosszeky, Renton *et al.*, 2000; Whelan, 1964). However, no studies have been conducted to demonstrate these effects through the direct measurement of spasticity. The OCGs fabricated for subjects in this study were designed based on the best available literature. However, the details provided in the literature lack sufficient detail and are quite outdated (Blashy and Fuchs, 1959; Farber, 1982; Whelan, 1964). It was possible that the materials used to fabricate the OCGs or the circumferential pressures applied to the limbs by the OCGs differed from those of previous investigations and were insufficient to cause an effect. The amount of circumferential pressure applied by the OCGs was not measured and this was a limitation of this study. Future investigations should measure the amount of

circumferential pressure applied by the OCGs or use prefabricated compression garments that apply standardised levels of compression.

Kukulka *et al.* (1986; 1985), Leone and Kukulka (1988), and Robinson *et al.* (1982) all reported that the application of pressure to the insertion of the Achilles tendon had inhibitory effects on soleus MN excitability in both able-bodied and hemiplegic subjects. The application of tendon pressure supposedly activates cutaneous afferents subserving touch and pressure, as well as the Ib afferent fibres from GTOs (Kukulka *et al.*, 1985). The use of a tendon pressure pad in the present study failed to replicate these effects. Previous studies (Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988; Robinson *et al.*, 1982) utilised pressure applicators which applied a direct, quantifiable force to the Achilles tendon while subjects lay in a prone position. Robinson *et al.* (1982) did not state how much pressure was applied in their study. In the studies conducted by Kukulka *et al.* (1986; 1985) 2 kg (20 N) of force was applied through a 20x15 mm transducer head (equivalent to 67 kPa). In the study conducted by Leone and Kukulka (1988) forces of 5 kg (5 N) and 10 kg (10 N) were applied over an area of 4.4 cm^2 (equivalent to 114 and 228 kPa). The tendon pressure pads that were used to apply pressure in the present study may not have applied sufficient pressure to activate the inhibitory afferent fibres.

The pressure applied by the tendon pressure pad in the orthosis was a constant pressure, which may also have resulted in the lack of effect. The studies conducted by Kukulka *et al.* (1986, 1985) found that reduction of the H-reflex amplitude only occurred at the onset of pressure application, and therefore, they recommended that pressure should be applied intermittently. While there were differences in the amount of pressure applied and the duration of pressure applied between the studies by Kukulka *et al.* (1986, 1985) and the present study, the method for pressure application used in the present study replicated an appropriate application of this concept in orthotic practice.

In order for tendon pressure to be effective in altering MN excitability in an AFO, it may be necessary to fabricate thicker tendon pressure pads from materials with higher densities, to apply greater pressure to the Achilles tendon. However, it may also be the case that it is not practical to achieve the level of pressure required through an AFO without causing discomfort to the subject and risking skin injury or the development of

pressure sores. An alternative method may be to dorsiflex the ankle joint and stretch the Achilles tendon. This would activate the GTOs and lead to the same inhibitory effect on alpha MNs as was demonstrated in the study conducted by Robinson *et al.* (1982). The effect of ankle dorsiflexion angle in TRAFOs warrants further research as an alternate method for eliciting an inhibitory effect on MNs.

A key difference between these previous studies that were conducted on H-reflex responses to tendon pressure, and the study being reported here, was in the positioning of subjects. Previous studies tested their subjects in non-weight-bearing positions, whereas this study tested subjects in the standing position. In the upright position, descending control is greater than in a prone or seated position (Ali and Sabbahi, 2001; Voerman *et al.*, 2005). This is because peripheral sensory inputs as well as descending cortical control from the CNS, exert a larger inhibition of the soleus H-reflex circuit in a standing position. Influences from supraspinal centres are anticipated to be minimal in lying or sitting positions, and spinal inhibitory neurons that are affected by muscle contraction might be less active in non-weight-bearing positions (Kawashima, Sekiguchi, Miyoshi, Nakazawa and Akai, 2003; Knikou, 2008). It may be possible that tone-reducing interventions are only effective when a subject is in a non-weight-bearing position where there is less cortical control and less peripheral feedback to MNs (Ali and Sabbahi, 2001; Voerman *et al.*, 2005). However, it is important that interventions are tested in functional positions and not just positions of rest. This is especially relevant for the assessment of TRAFOs, which are intended to be used in functional positions.

The results of this study need to be interpreted in light of its limitations. Firstly, the sample size was small increasing the chance of a type II error. Secondly, the TRAFOs were investigated in able-bodied subjects who have full uncompromised descending control from the CNS over their motor systems (Kandel, Schwartz and Jessell, 2000). It may be the case that any afferent inputs by the tone-reducing features were being regulated by the healthy CNS. However, previous studies on the effects of interventions such as tendon pressure and circumferential pressure in able-bodied subjects have demonstrated that MN excitability can be altered by these interventions in normal subjects (Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Kukulka *et al.*, 1987; Robichaud *et al.*, 1992; Robichaud and Brunt, 1994). Furthermore, it was demonstrated that the results of the studies on able-bodied subjects were generalisable to subjects with spasticity.

Further investigations are required to assess the effect of TRAFOs in subjects with spasticity to determine if the results of this study can be generalised to hemiplegic subjects.

4.5 Conclusion

This study has demonstrated that the application of pressure to the Achilles tendon, the ITB and the OCG had no significant effect on the MN excitability of able-bodied subjects when applied to a solid AFO and tested in a standing position. Within the limitations of this study, TRAFOs had no significant neurophysiological tone-reducing effects as measured using the H-reflex. However, further investigations are required to determine if these results can be generalised to subjects with spasticity.

Chapter Five

The neurophysiological effect of three tone-reducing devices in subjects with spasticity[2]

5.1 Introduction

The previous study (Chapter Four) identified the need to investigate the effect of tone-reducing features on their own to ensure that they are optimally constructed before being incorporated into AFOs. Ricks and Eilert (1993, p15) stated that "it is important to identify what aspects of the inhibitory devices make them effective, so that they can be constructed to give optimal benefit". Since any AFO can be made into a TRAFO with the addition of tone-reducing features, the effect of TRAFOs is dependent upon the design and construction of its tone-reducing features. Therefore, this chapter presents a study that was conducted to assess the effects of individual tone-reducing features to determine how they may be most effectively combined with AFO management.

The previous two studies in this thesis tested able-bodied subjects. A limitation noted in the discussion of Chapter Four was that the results may not be generalisable to individuals with spasticity. The remainder of this thesis will focus on people who have spasticity as a result of stroke. Although people may experience spasticity from a number of neurological conditions, people with stroke were chosen as the subject group for this thesis as they exhibit spasticity from an easily identifiable cause. They also form the largest group of adults with spasticity (Boyd and Ada, 2001; Carr and Kenney, 1992).

5.1.1 Spasticity and stroke

A stroke is defined as an acute onset neurological dysfunction, due to an abnormality in the arterial supply of the brain following haemorrhage or thrombus, usually only affecting one side of the brain (Olney and Richards, 1996; Wang, Yen, Lee *et al.*, 2005). The pathophysiology behind stroke will not be detailed in this thesis as this information

[2] A manuscript based on this chapter has been published in Prosthetics and Orthotics International (Ibuki *et al*., 2010). A copy of the published manuscript is included as Appendix B.

can be found elsewhere in literature. However, details regarding stroke and its relationship with spasticity will be discussed.

Stroke is a leading cause of morbidity and mortality that creates a huge economic burden due to the numerous disabilities that can result following the neurological attack (Lennon, 1996; Lundstrom, Terent and Borg, 2008). Considerable health care resources are directed towards stroke survivors and this is likely to increase with the growing number of elderly individuals in the population (Lennon, 1996). Although the growing awareness of stroke and advances in medicine have helped to decrease the mortality rates following stroke, the number of stroke survivors left with permanent disabilities is increasing (Leathley, Gregson, Smith, Sharma and Watkins, 2004).

Following a stroke, the area of the brain that is damaged and the extent of the damage determine the resultant impairments, which usually only affect one side of the body (Fatone, 2009a). This is known as hemiplegia. There is an almost infinite range of possible permutations of functional deficits following a stroke with each individual's resultant impairments being unique (Carr and Kenney, 1992; Lundstrom *et al.*, 2008). If the motor cortex is damaged by a stroke, the motor system for that individual will be affected to some extent and this may lead to spasticity in the lower limbs. Motor deficits are the most common impairment acutely after stroke (Cramer, 2004) and according to Kumar *et al.* (2006) motor deficits are present in 80% of surviving stroke patients. These deficits are often persistent if not permanent (Carr and Kenney, 1992).

Studies have indicated that about 36-38% of stroke patients develop spasticity during the first year following a stroke (Voerman *et al.*, 2005). Others have reported spasticity to occur in approximately 60% of patients following stroke, however, the time since onset of stroke for this estimation is not stated (Wallesch *et al.*, 1997). Spasticity may be considered one of the most physically debilitating consequences of a stroke, and it has been shown that stroke patients with spasticity spend three times longer as rehabilitation inpatients than those without spasticity, because they are functionally more impaired (Fleuren, Nederhand and Hermens, 2006; Harburn *et al.*, 1992).

Recovery following stroke is a complex process. Richards and Olney (1996) identified six stages of motor recovery:

Stage 1 Immediately after stroke flaccidity is present and no movement of limbs can be initiated.

Stage 2 Basic limb synergies or some of their components may appear as associated reactions or minimal voluntary movement responses may be present and spasticity begins to develop.

Stage 3 Voluntary control of movement synergies appear though full range may not be attained and spasticity develops further.

Stage 4 Possible to execute some voluntary movement combinations outside the synergist patterns while spasticity begins to decline.

Stage 5 More difficult movement patterns learnt as basic synergies lose their dominance over motor acts.

Stage 6 Spasticity disappears, individual joint movements become possible and coordination approaches normal.

Most motor recovery tends to occur within three months of a stroke, however, it may take up to five years (Richards and Olney, 1996). Active rehabilitation procedures and therapeutic aids (such as orthoses) can augment the natural recovery process (Richards and Olney, 1996). Unfortunately, not all stroke survivors will experience all six stages of motor recovery and some are left with permanent disabilities and persistent ongoing problems like spasticity.

5.1.2 Tone-reducing orthoses

AFOs have been prescribed to manage spasticity in the lower limbs of people with spasticity following stroke. These devices aim to enhance the biomechanics of a limb in order to improve position, movement and function. Information regarding the number of AFOs prescribed and fitted following stroke in Australia is difficult to obtain as patients can be treated in either the public or private healthcare sectors. The value of orthoses for patients with stroke is still a matter of debate (Wang *et al.*, 2005), however, there is evidence that the biomechanical effects of orthoses are useful for hemiplegic stroke

patients (Hesse *et al.*, 1999; Mojica *et al.*, 1988; Tyson and Thornton, 2001; Wang *et al.*, 2007).

As detailed in section 2.4.1 of this thesis, TRAFOs are simply AFOs with the addition of tone-reducing features to target and reduce muscle spasticity. Therefore, TRAFOs are only as effective as the tone-reducing features that are incorporated into the standard AFO design. The tone-reducing features that were assessed in this study were the tone-reducing footplate, circumferential compression around the leg, orthokinetics and stretch of the plantarflexor muscles. These features were chosen as they represent common mechanisms by which tone-reducing features are purported to work:

1. The stimulation or inhibition of particular reflexogenous areas on the sole of the foot (Lima, 1990);
2. The stimulation or inhibition of cutaneous receptors (Bronkhorst and Lamb, 1987); and
3. The stimulation of GTOs (Smelt, 1989).

The effect of these tone-reducing features can be assessed individually using specific orthotic devices (Table 5.1).

Table 5.1 The tone-reducing mechanisms and features tested in this study. See text for discussion.

Tone-reducing neurophysiological mechanism	**Tone-reducing feature**	**Orthotic device to test the tone-reducing feature**
Stimulation or inhibition of reflexogenous areas on the sole of the foot	Tone-reducing footplate	Dynamic foot orthosis
Stimulation or inhibition of cutaneous receptors	Circumferential compression Orthokinetics	Orthokinetic compression garment
Stimulation of GTOs	Muscle stretch	ROM walker

5.1.2.1 Dynamic foot orthoses

In the previous study (Chapter Four), ITBs were evaluated with AFOs as they are said to alter spasticity by extending the toes and decreasing pressure on the reflexogenous area of the foot that is responsible for eliciting the toe grasp reflex (Figure 4.1). However, there are other reflexogenous areas on the sole of the foot that are thought to be responsible for tonic reflexes that may interfere with movement and control of the lower limb. These have been previously discussed (see section 4.1.1.1). In order to prevent the stimulation of these tonic reflexes, tone-reducing footplates are used to provide an optimal weight-bearing surface for the whole foot (Charlton and Ferguson, 2001). Tone-reducing footplates are often incorporated into TRAFOs and they are a key tone-reducing feature of dynamic AFOs (Hylton, 1990a; Hylton, 1990b). To assess the effects of the tone-reducing footplate on its own, a dynamic foot orthosis (DFO) can be used.

DFOs are foot orthoses that accommodate the contours and arches of the foot to provide an optimal weight-bearing surface, which is said to settle the positive support reaction (Charlton and Ferguson, 2001). DFOs reinforce the three dynamic arches of the foot (transverse, medial and lateral longitudinal arches) and relieve pressure under the metatarsal heads and the calcaneal fat pad. The key features of DFOs are (Dieli *et al.*, 1997; Hylton, 1990a; Hylton, 1990b; Pratt, 2000; Radtka *et al.*, 1997):

1. Unloading of the metatarsal heads and the centre of the heel by creating recessed areas in the footplate;
2. Pressure applied to the medial and lateral longitudinal arches as well as the peroneal notch;
3. Pressure applied behind the second through fourth metatarsal heads via a metatarsal dome that further unloads the metatarsal heads; and
4. Pressure applied to the sulcus of the toes in a manner similar to the ITBs.

These features are consistent with the theory of reflexogenous areas on the sole of the foot, except for the feature of unloading the centre of the heel. It is said that pressure applied to the centre of the heel stimulates the dorsiflexion reflex (Duncan, 1960; Duncan and Mott, 1983) which would be useful to counteract the spastic plantarflexor muscles. Earlier descriptive papers and investigations on TRAFOs utilised this reflex by using internal heel pads (approximately ¼ inch thick) within TRAFOs to increase pressure on

the heels (Bronkhorst and Lamb, 1987; Lohman and Goldstein, 1993). However, Pratt (2000), Hylton (1990a; 1990b) and Radtka *et al.* (1997) recommend unloading the centre of the heel by creating a depression in the footplate under the heel. While the use of internal heel pads by previous investigators is justified by the theory of reflexogenous areas on the sole of the foot, an internal heel pad may increase the plantarflexion angle of the ankle and subsequently increase the amount of pressure borne through the metatarsal heads which may stimulate the toe grasp reflex (Figure 5.1). The discontinued use of internal heel pads can be justified for this reason, however, investigators who advocate unloading the centre of the heel do not explain the rationale for this feature. This reflects the lack of literature in this area.

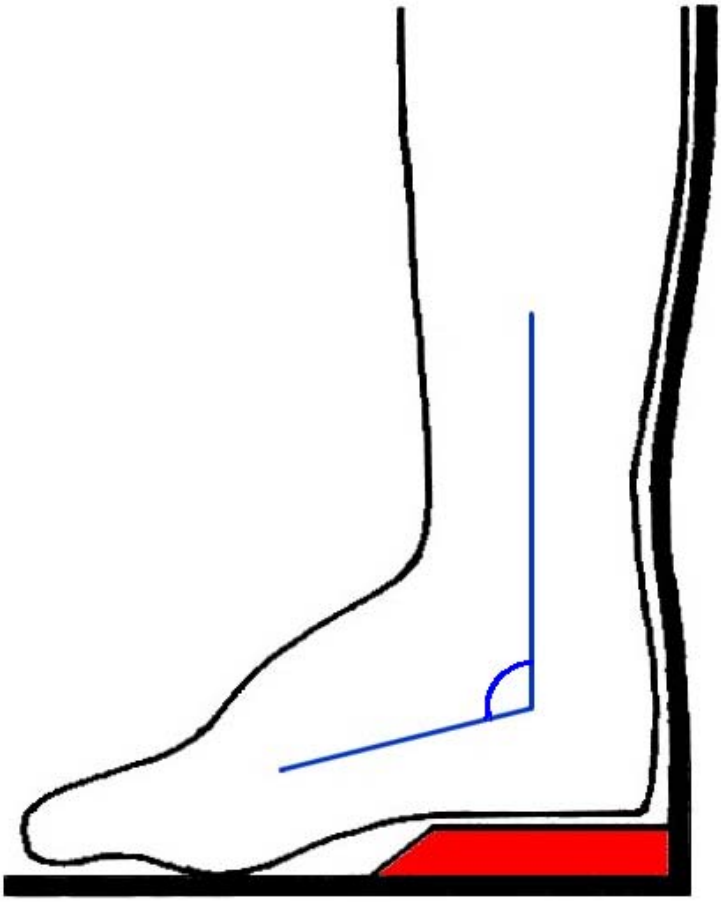

Figure 5.1 An AFO with an internal heel pad.
This illustration demonstrates the potential of the internal heel pad (coloured in red) to increase the plantarflexion angle of the ankle (adapted from Bronkhorst and Lamb, 1987, p23).

According to Pratt (2000), there are claims that DFOs have been shown to reduce spasticity, improve stability, balance and symmetry, reduce muscle imbalance, and improve function in the entire body. Pitetti and Wondra (2005) reported that DFOs also improve proprioceptive feedback. However, it is not clearly explained how these effects have been demonstrated in DFOs and furthermore, it is uncertain how such effects are able to be produced. The literature on DFOs is very limited, and evidence to support the effects that have been claimed by authors is lacking.

5.1.2.2 Orthokinetic compression garments

The stimulation of cutaneous receptors is believed to have tone-reducing effects on underlying muscles (Lohman and Goldstein, 1993; Ushiba *et al.*, 2004). Cutaneous receptors may be stimulated by the close contact of certain materials against the skin (Blashy and Fuchs, 1959; Lohman and Goldstein, 1993) as well as the application of circumferential pressure around a limb (Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992). OCGs can be used to assess these tone-reducing mechanisms. An overview of OCGs was provided in the previous study (see section 4.1.1.3).

Despite the fact that orthokinetics were first described in the literature several decades ago (Blashy and Fuchs, 1959), they have received very little research attention. In more recent times (mid 1990s) a similar concept has emerged with the development of Lycra orthoses (also known as soft splints, Lycra garments or dynamic splints). Lycra orthoses are predominantly made of Lycra segments sewn together in specially placed orientation appropriate to produce a directional stretch on a limb (Corn *et al.*, 2003; Gracies, Fitzpatrick, Wilson, Burke and Gandevia, 1997). Lycra orthoses aim to correct deformity, improve joint stability, inhibit spasticity and encourage more normal function (Blair, Ballantyne, Horsman and Chauvel, 1995; Corn *et al.*, 2003). They are believed to achieve this by applying low-level stretching forces to the limb and by providing firm circumferential pressure around the limb to stimulate cutaneous receptors (Gracies *et al.*, 1997; Gracies *et al.*, 2000).

Lycra orthoses can be applied to the upper limbs, lower limbs and trunk, but their effects on spasticity and function remain unclear. The majority of research that has been

conducted on Lycra orthoses has focused on their effects in the upper limbs. There do not appear to be any published studies on the effects of Lycra orthoses specifically for the lower limbs. Two studies have assessed the effects of full body Lycra orthoses using three-dimensional gait analysis and the Paediatric Evaluation of Disability Index (PEDI) in children with CP and Duchennes muscular dystrophy (Nicholson, Morton, Attfield and Rennie, 2001; Rennie, Attfield, Morton, Polak and Nicholson, 2000). While it was observed that some children could benefit from the Lycra orthoses with improvements in their PEDI scores and gait patterns, the disadvantages of the full body Lycra orthoses (mainly associated with toileting) were a significant issue. Further research is required to determine whether Lycra orthoses that only cover the lower limbs have similar effects on gait and function without the disadvantages associated with the full body Lycra orthoses.

Studies on Lycra orthoses for the upper limbs have found mixed results (Corn *et al.*, 2003). Some authors have demonstrated improvements in various outcomes with the use of Lycra orthoses such as improved resting position, improved passive and active ROM and decreased spasticity in the wrist and finger flexor muscles (Gracies *et al.*, 2000; Watson, Crosby and Matthews, 2007). Other authors have demonstrated no differences in outcomes, or a decline in function with the use of Lycra orthoses (Corn *et al.*, 2003). These mixed results highlight the need for continued research on the effects of Lycra orthoses especially in managing lower limb spasticity.

This study assessed the effects of OCGs rather than Lycra orthoses, as Lycra orthoses incorporate the added element of muscle stretch. OCGs were used to assess the tone-reducing mechanism of cutaneous stimulation by the close contact of elastic and inelastic materials with the skin and circumferential pressure applied around the limb. Muscle stretch was evaluated as a separate tone-reducing feature, which will now be discussed.

5.1.2.3 Muscle stretch

In the previous study (Chapter Four), pressure was applied to the Achilles tendon in an attempt to activate GTOs which have an inhibitory effect on alpha MNs. However, tendon pressure may not be the most effective method for activating GTOs when it comes to AFO management, due to the risk of causing skin breakdown or pressure sores

over the area of pressure application. An alternative method for activating plantarflexor GTOs would be to stretch the plantarflexor muscles and the Achilles tendon by dorsiflexion of the ankle joint (Middleton, Hurley and MeIlwain, 1988; Robinson *et al.*, 1982).

The stretching of muscles is frequently prescribed for patients with spasticity, either in the form of physical therapy or through the application of orthotic devices. The stretching of muscles is thought to have immediate tone-reducing effects on the muscle being stretched (Vattanasilp *et al.*, 2000). For example, holding an ankle in slight dorsiflexion may inhibit spasticity in the lower limb extensor muscles (Charlton and Ferguson, 2001). Muscle stretch can also prevent muscle shortening and contracture development, which is important to prevent increased stretch reflex responses that could occur at smaller joint angles in shorter muscles (Gracies *et al.*, 1997).

Selles *et al.* (2005) conducted a study to assess the effects of muscle stretch on ten hemiplegic subjects with chronic plantarflexor spasticity and/or contracture following stroke. The mean time since stroke onset of the subjects was 7.7 years (range 1.8 to 21.3 years). The authors found that 45 minutes of stretching the plantar- and dorsiflexor muscles three times a week for four weeks significantly improved passive ankle joint resistance, passive plantar- and dorsiflexion ROM, maximum voluntary contraction of the plantarflexors and comfortable walking velocity. In addition, subjects' subjective evaluation of the stiffness of their ankles improved significantly. No significant differences were demonstrated for active ROM or reflex excitability as assessed by observing EMG and joint torque responses to Achilles tendon tapping. The authors did not provide any physiological explanation to explain their findings (Selles *et al.*, 2005).

It has been demonstrated that passively stretching the soleus muscle significantly depresses the soleus H-reflex amplitude in healthy subjects (Hwang, 2002a; Kanter, Zhu, McNulty and Weber, 2006; Vujnovich and Dawson, 1994) and subjects with spasticity (Burke, Andrews and Ashby, 1971). Kanter *et al.* (2006) reported reductions in H-reflex amplitudes of 62% while Robinson *et al.* (1982) reported reductions of 47%. Vujnovich and Dawson (1994) found that both static and ballistic applied stretch reduced soleus H-reflex amplitudes with ballistic stretch having a greater inhibitory effect. This inhibitory

effect on MN excitability has been attributed to presynaptic inhibition, reduced excitability of intramuscular stretch receptors and inhibitory influences to alpha MNs by GTOs (Odeen and Knutsson, 1981b; Robinson *et al.*, 1982; Vujnovich and Dawson, 1994).

5.1.3 Summary and aims

The previous study (Chapter Four) assessed the effects of three tone-reducing features (ITB, OCG and the application of pressure to tendons) when incorporated into TRAFOs. The effect of TRAFOs on spasticity is dependent upon the tone-reducing features that are incorporated into the TRAFOs. The results of the previous study highlighted the fact that the individual tone-reducing features needed to be assessed to determine how they may be optimally designed and constructed to produce the greatest effect before being incorporated into AFOs. There is very little information in the literature regarding the optimal design and construction of TRAFOs. The reason for this is that the optimal design and construction of individual tone-reducing features has not previously been investigated.

The ITB and tendon pressure pad that were assessed in the previous study may not have been the most effective devices for stimulating the inhibitory afferent fibres that are required to alter MN excitability. Therefore, the DFO and muscle stretch were used to target the same inhibitory afferent fibres. The OCG was still used to assess the effect of cutaneous stimulation in this study. However, the design of the OCGs was changed so that the level of pressure applied by the garments could be standardised.

The purpose of this study was to evaluate the effect of three tone-reducing devices on the MN excitability of the soleus muscle in subjects with spasticity following stroke while standing. The three tone-reducing orthotic devices used in this study were the:

1. DFO;
2. OCG; and
3. ROM walker.

It was hypothesised that use of the devices would result in decreased MN excitability in subjects with stroke while standing.

5.2 Methods

5.2.1 Subjects

Thirteen subjects who had previously suffered stroke were recruited from the community for this study. The subjects were recruited via a number of different methods including advertisements in print media, the Stroke Association of Victoria, community stroke support groups, the National Stroke Research Institute (Victoria) and outpatient rehabilitation clinics. The subject characteristics can be found in Table 5.2.

Subjects were included in the study if they met the following criteria:

- Hemiparesis secondary to a stroke suffered at least 12 months prior to the study;
- Independent community ambulators (gait aids acceptable);
- A good current level of health;
- Spasticity in the soleus muscle as determined by a grade greater than or equal to two on the Tardieu Scale adapted for stroke (Table 5.3);
- Able to passively dorsiflex the affected ankle joint at least 7.5 degrees with the knee extended; and
- Able to comply with instructions and give informed consent.

The Tardieu Scale was only used to determine the presence and severity of spasticity. It was not used as an outcome measure. Therefore, it was not considered necessary to report and perform all components of the Tardieu Scale which have been described by previous authors (Boyd and Ada, 2001; Morris, 2002).

Subjects were excluded from the study if:

- They stated that their stroke had had no effect on their walking ability;
- They had never experienced muscle tightness or spasm; or
- They had other serious medical problems.

No subjects were taking oral antispastic drugs or local antispastic drugs at the time of the study, although this was not an exclusion criteria. Some subjects were undergoing physiotherapy or hydrotherapy at the time of the study which they were allowed to continue. All subjects were required to give informed consent before being included in the study.

Table 5.2 Subject characteristics.

Subject	Gender	Age (years)	Number of strokes experienced	Time since most recent stroke (years)	Stroke type	Side of hemiplegia	Tardieu Scale (X, Y)	Sensory deficit
1	Male	50	2	18	Haemorrhagic	Left	3, 18º	No
2	Male	52	1	26	Haemorrhagic	Right	2, 23º	Yes
3	Male	65	2	2	Ischemic	Right	2, 16º	Yes
4	Male	62	1	5	Haemorrhagic	Left	4, 15º	Yes
5	Male	59	1	4	Haemorrhagic	Right	3, 20º	No
6	Male	54	1	4	Haemorrhagic	Left	4, 17º	No
7	Female	49	1	14	Haemorrhagic	Right	2, 26º	Yes
8	Male	73	2	2	Unknown	Left	2, 10º	No
9	Male	50	5	5	Ischemic	Left	4, 25º	No
10	Female	60	1	8	Haemorrhagic	Left	2, 15º	Yes
11	Female	59	1	4	Haemorrhagic	Left	2, 24º	No
12	Female	46	1	12	Haemorrhagic	Left	3, 12º	Yes
13	Female	63	1	11	Ischemic	Right	3, 10º	No
Total / Mean	8 Males, 5 Females	57.67	1.50	8.08	9 Haemorrhagic, 3 Ischemic, 1 Unknown	8 Left, 5 Right	2.77, 17.77º	6 Yes, 7 No

X= Quality of muscle reaction; Y= Angle of catch.

Table 5.3 The Tardieu Scale adapted for stroke (Boyd and Ada, 2001).

Score	Quality of muscle reaction
0	No resistance throughout the course of the passive movement.
1	Slight resistance throughout the course of the passive movement with no clear catch at a precise angle.
2	Clear catch at a precise angle, interrupting the passive movement, followed by release.
3	Fatigable clonus (less than 10 seconds when maintaining the pressure) appearing at a precise angle.
4	Infatigable clonus (more than 10 seconds when maintaining the pressure) at a precise angle.

5.2.2 Apparatus

The apparatus used for this study were exactly the same as those used for the previous study (see section 4.2.2).

5.2.3 Procedures

Ethics approval for this study was obtained from the Health Sciences Faculty Human Ethics Committee at La Trobe University (ethics approval number FHEC06-045). A telephone interview was used to screen potential subjects to determine their eligibility for the project. Eligible subjects were then brought to the University for a detailed subjective and objective assessment. During the assessment, the level of impairment following stroke was determined for each subject with particular attention to the presence and degree of spasticity. Subjects who fulfilled all of the criteria and who agreed to participate in the study then had measurements taken of their affected leg to determine the correct sizing for the ROM walker and compression garments.

5.2.3.1 Fabrication of the orthotic devices

A partial weight-bearing foot impression was taken using a compressible foam impression box (Gotz Service, Goppingen, Germany, 73037) for the fabrication of the

custom-made DFOs. The foam impression boxes were filled with plaster of Paris to create positive moulds which were then modified according to instructions detailed in the literature (Hylton, 1990a; Pratt, 2000; Radtka *et al.*, 1997). Plaster removal and additions were performed in designated areas to a depth of 3-10 mm depending upon the compressibility and flexibility of the patient's tissues and the amount of force necessary to create the desired pressure system (Figure 5.2). The modified casts were vacuum moulded with sheets of high density EVA (Ethylene vinyl acetate) foam (250 $kg.m^3$) and the DFOs were shaped to fit within post-operative (post-op) shoes (OTS, Victoria, Australia, 3199).

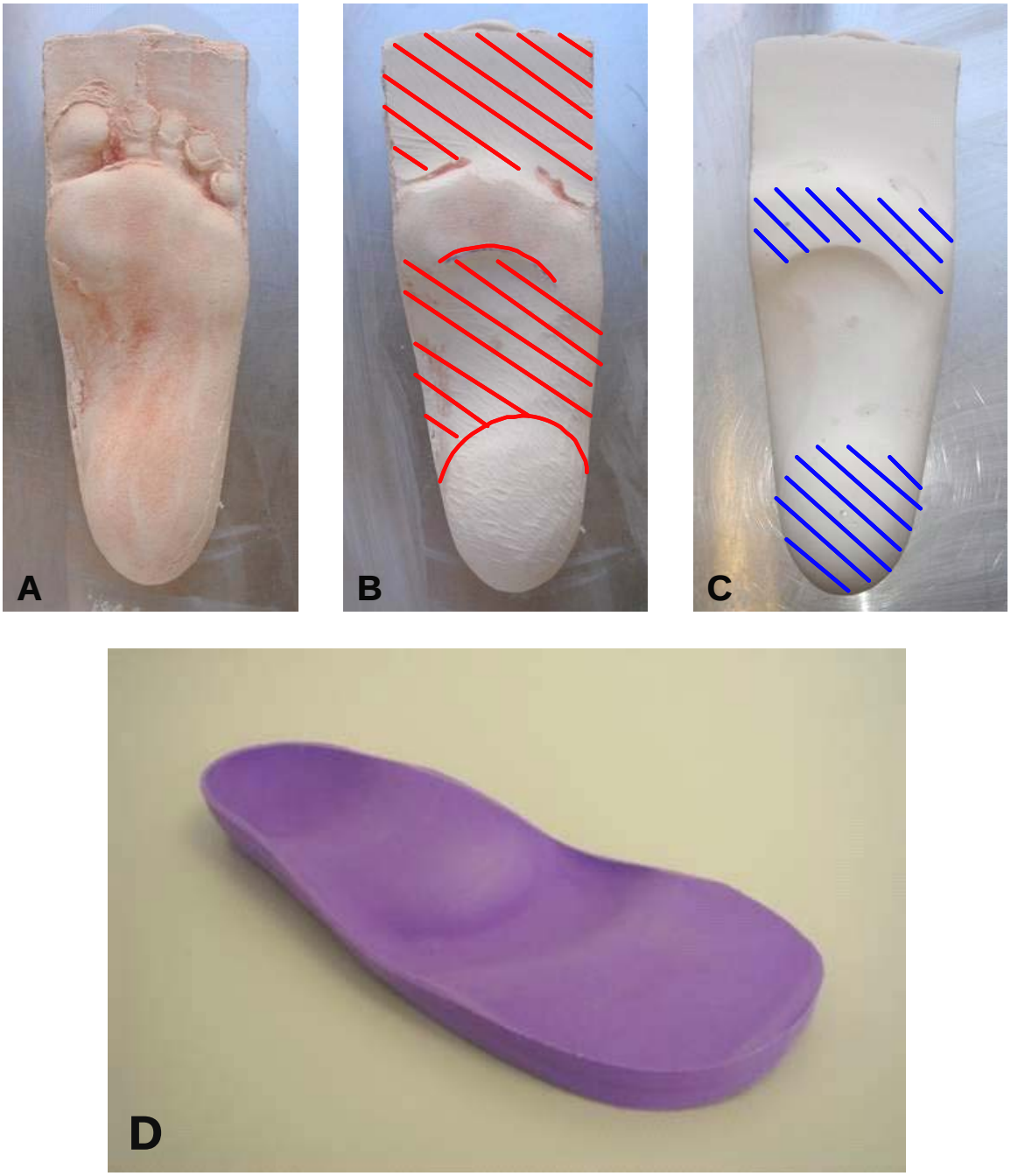

Figure 5.2 The dynamic foot orthosis.
(A) Positive foot mould. (B) Areas of plaster removal to increase load on the toes, metatarsal shafts, medial and lateral longitudinal arches. (C) Areas of plaster addition to unload the metatarsal heads and the centre of the heel. (D) The completed DFO.

In the previous study (Chapter Four), the OCGs were custom-made for each subject making it difficult to standardise the amount of compression that they applied to the limb (see section 4.2.3.1). Therefore, Venosan (Salzmann AG, St. Gallen, Switzerland, 9000) below knee compression garments without toes were used to create the OCGs in this study. 4001 (18-21 mmHg) and 4002 (23-32 mmHg) range garments were used to ensure a standard level of compression between subjects, and to assess the effects of two different levels of compression. These classes of compression were chosen as they represent standard levels used in compression therapy and are similar to those applied by Lycra garments for the management of spasticity. Pieces of smooth inelastic vinyl were specifically cut from measurements of the subjects' legs to create inactive fields over the plantarflexor muscles. The vinyl segments were fitted directly against the subjects' legs inside the compression garments. The elastic fabric of the compression garments acted as the active field over the dorsiflexor muscles.

Adjustable ROM walkers (Ossur, Northmead, NSW, Australia, 2152) were used to maintain a constant stretch on the plantarflexor muscles by immobilising the ankle joint at two specified angles; plantargrade (0 degrees) and 7.5 degrees of dorsiflexion. These angles were chosen as the ROM walker joints were only adjustable in 7.5 degree increments, and because these angles may be applicable for use in functional AFOs.

The subjects wore post-op shoes with all of the conditions (except on the affected foot for the ROM walker condition) to standardise footwear. Due to the thickness of the DFOs and the soles of the ROM walkers, full-length internal raises were worn in the post-op shoe of the unaffected foot to ensure that the height of the standing surfaces was the same under both legs.

5.2.3.2 Testing procedures

Details regarding the procedures for subject preparation and the procedures for testing have been previously described (see sections 3.2.3.1 and 3.2.3.2). The design of this repeated-measures intervention study followed an ABA format. There were seven conditions tested in this study:

A_1 - Shoes only (baseline);

B_1 - DFO;

B_2 - OCG class 1;

B_3 - OCG class 2;

B_4 - ROM walker set in neutral (plantargrade);

B_5 - ROM walker set in 7.5 degrees of dorsiflexion; and

A_2 - Shoes only (baseline return).

The order of the interventions (B_1 to B_5) was randomised by pulling pieces of paper with the interventions written on them out of a box to prevent a series effect. Donning and doffing of the devices was performed carefully to minimise electrode movement. For each of the conditions, H-reflex and M-response recruitment curves were constructed from an average of four trials at each level of stimulus intensity. Subjects were able to rest between each condition, and a short accommodating period was given at the beginning of each of the conditions to allow subject to adopt a comfortable standing position.

5.2.4 Statistical analyses

The results of the previous study (see section 4.3) were used to calculate the sample size and power required for the current study. It was estimated that a sample of 14 subjects would be required to provide 80% power for detecting a difference of 0.225 (the $R_{95\%}$ interval from the previous study) between the baseline and intervention H:M ratio means, assuming a SD of 0.21. Details regarding the statistical analyses have been previously provided (see section 4.2.4).

5.3 Results

Of the 13 subjects who were recruited for the study, data for only ten of the subjects were able to be analysed as three subjects (subjects 1, 6 and 7) had adverse reactions to the electrical stimulations. One subject was unable to complete the testing as the artificial stimulations activated clonus in their ankle plantarflexor and knee extensor muscles. The clonus lasted more than five seconds which was the period between stimulations. This caused the subject to become very frustrated and uncomfortable. Another subject was unable to complete the testing as the tone in their plantarflexor muscles increased as the intensity of the electrical stimulations increased. This continued to the point where the subject's ankle was plantarflexed and only the forefoot was in contact with the ground. The subject was unable to lower the heel back down to the ground unless allowed to sit down and rest. In both of these cases, it was deemed unsafe for the subjects to continue the testing. The third subject was unable to be included as their H-reflex and M-response amplitudes were very inconsistent and did not follow the standard recruitment curve pattern. As a result, it was not possible to determine the maximum H-reflex and M-response amplitudes with certainty.

The ANOVA revealed that there were no statistically significant differences in the H:M ratios between any of the conditions (F=1.208, df=3.232, p=0.328) (Figure 5.3). The $R_{95\%}$ was used to further examine the data to determine whether any substantial changes within individual subjects had been masked by the averaging process. The individuality of the impairments of subjects with stroke makes it reasonable to analyse the data on a subject-by-subject basis to determine individual changes. The $R_{95\%}$ interval for the H:M ratio was found to be 0.182. The DFO and OCG of class 1 compression both caused the H:M ratio to fall below the $R_{95\%}$ for subject 4, and the DFO caused the H:M ratio to fall below the $R_{95\%}$ for subject 11. Table 5.4 includes the lower and upper bounds for the $R_{95\%}$ for each individual subject.

It is worth noting that H:M ratios greater than one were recorded for subjects 10 and 11 (Table 5.4). The H:M ratios for these two subjects were much higher than those of the other subjects (H:M ratios were greater than 0.90 in baseline conditions). The greater than one ratios were due to the maximum H-reflex amplitudes being slightly greater than the maximum M-response amplitudes, suggesting that a greater number of MNs were

excited by reflex activation than were excited by direct stimulation of the motor nerve. Based on our current knowledge of the H-reflex, it should not be possible to obtain a H:M ratio greater than one which indicates that 100% of the MN pool is being activated (Biering-Sorensen *et al.*, 2006; Palmieri *et al.*, 2004; Schiepatti, 1987). Reasons for the greater than one H:M ratios are presented in the discussion.

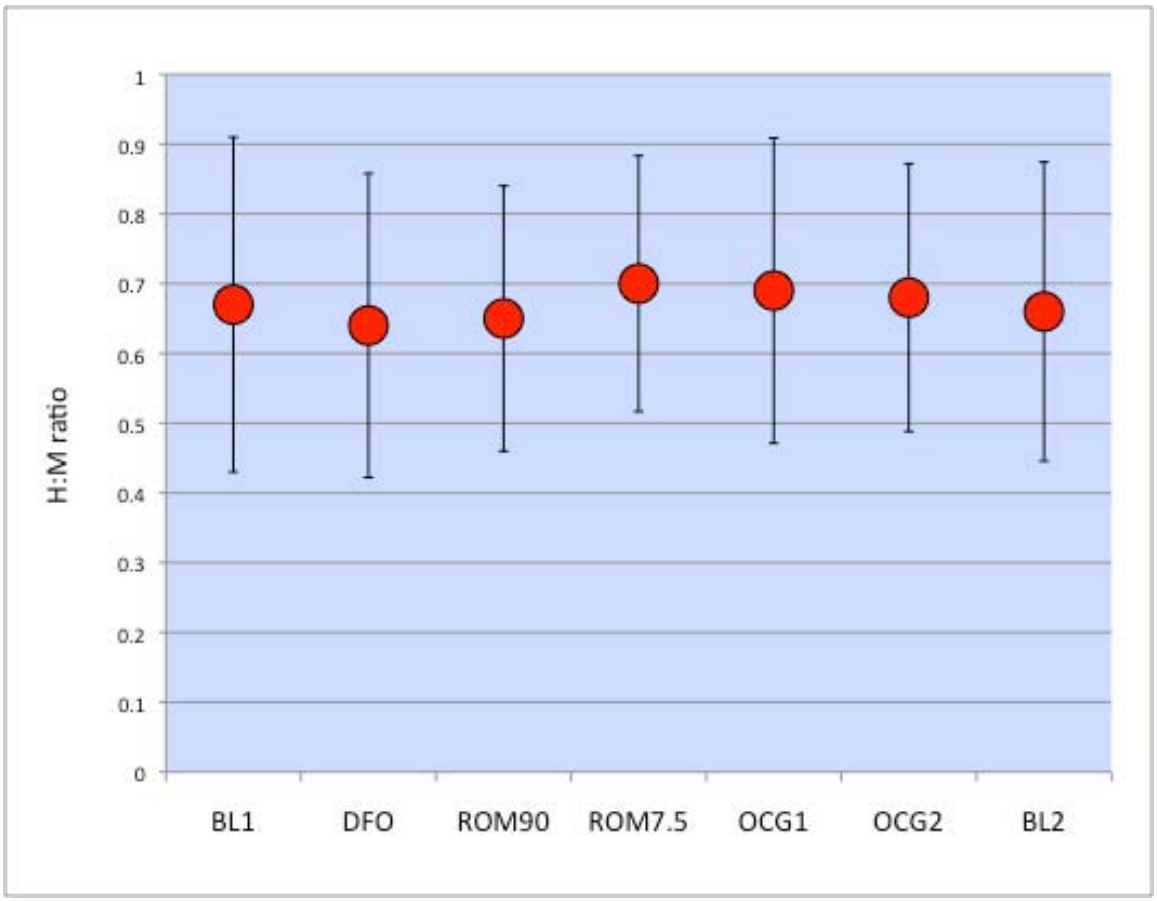

Figure 5.3 Mean (±1 SD) H:M ratios for all subjects across all conditions.

BL1= Shoes only initial baseline; DFO= Dynamic foot orthosis; ROM90= ROM walker set at 90 degrees (plantargrade); ROM7.5= ROM walker set at 7.5 degrees; OCG1= Orthokinetic compression garment class 1; OCG2= Orthokinetic compression garment class 2; BL2= Shoes only baseline return.

Table 5.4 Mean H:M ratios and the $R_{95\%}$ for each subject.

Subject	BL1	DFO	ROM90	ROM7.5	OCG1	OCG2	BL2	Mean of BL1 and BL2	$R_{95\%}$
2	0.55	0.61	0.73	0.70	0.70	0.67	-	0.55	0.37 - 0.73
3	0.20	0.19	0.17	0.26	0.24	0.25	0.19	0.20	0.02 - 0.38
4	0.71	**0.48**	0.60	0.73	**0.50**	0.57	0.68	0.70	0.52 - 0.88
5	0.67	0.70	0.64	0.77	0.65	0.67	0.67	0.67	0.49 - 0.85
8	0.49	0.54	0.56	0.66	0.68	0.66	0.63	0.56	0.38 - 0.74
9	0.74	0.75	0.68	0.70	0.77	0.76	0.70	0.72	0.54 - 0.90
10	0.96	0.96	0.80	0.89	1.02	0.93	0.95	0.96	0.78 - 1.14
11	1.07	**0.74**	0.87	0.94	0.91	0.93	0.90	0.99	0.81 - 1.17
12	0.65	0.67	0.70	0.61	0.67	0.69	0.64	0.65	0.47 - 0.83
13	0.66	0.66	0.71	0.69	0.57	0.63	0.62	0.64	0.46 - 0.82

BL1= Shoes only baseline 1; DFO= Dynamic foot orthosis; ROM90= ROM walker set at 90 degrees (plantargrade); ROM7.5= ROM walker set at 7.5 degrees; OCG1= Orthokinetic compression garment class 1; OCG2= Orthokinetic compression garment class 2; BL2= Shoes only baseline 2.
Subject 2 did not complete the final baseline condition due to fatigue.
H:M ratios in large, bold font are outside of the $R_{95\%}$.

5.4 Discussion

The results of this study indicated that overall, the tone-reducing devices were ineffective in altering soleus MN excitability in the standing position in subjects with spasticity following stroke. This is consistent with the results of the previous study (Chapter Four) conducted on able-bodied subjects. However, when the results were analysed on an individual basis, two subjects (4 and 11) responded to the DFO condition (as indicated by the significant decrease in the H:M ratio) and one of those subjects (subject 4) also responded to the OCG of class 1 compression.

Naslund *et al.* (2005) reported similar findings when examining the effects of dynamic AFOs on children with spastic diplegia. On a group level, the dynamic AFOs did not appear to have any effects on posture, distribution of body weight or force application beneath the feet. However, when analysed subject-by-subject, the results demonstrated that some children could benefit from the orthoses. Matthews, Watson and Richardson (2009) and Rennie *et al.* (2000) also found that the results of objective measures on the effects of Lycra orthoses in children with spastic diplegia were varied between individuals and could not be generalised to the whole subject group. Rennie *et al.* (2000) suggested that the heterogeneous nature of subjects with spasticity dilute any perceptible differences making the collective statistical analysis non-significant. These findings are consistent with Moore's (1998) statement that the way in which spasticity responds to treatment is unpredictable and that no two patients with spasticity are alike (Wyke, 1976). What might cause an effect in one patient may not necessarily work for another.

Upon closer inspection of subjects 4 and 11 (Table 5.2), there do not appear to be any distinguishing characteristics that set them apart from the subject group to explain why their H:M ratios decreased in response to the interventions. It is difficult to determine why the DFO and OCG of class 1 compression reduced their MN excitability when the other devices had no statistically significant effects. Likewise, it is difficult to explain why the OCGs of class 1 compression had an effect in one subject while the OCGs of class 2 compression did not. It remains possible that the observed differences were chance occurrences.

The overall ineffectiveness of the devices challenges the claims of previous authors who have supported the use of tone-reducing orthotic devices to reduce spasticity. Questions may arise regarding the specific design of the tone-reducing devices that were used in this study. Pitetti and Wondra (2005) are possibly the only authors who have quantitatively assessed the efficacy of DFOs in an experimental study. They conducted a repeated measures cohort study on 25 children with gross motor delay (including three children with CP), and found that prefabricated DFOs were able to improve the gross motor skills of children with motor delay as determined by the locomotion section of the Peabody developmental motor scales test (2nd edition). This test is used to determine the motor skills in children up to six years of age, and includes numerous items such as standing, stair climbing, jumping and running. The authors did not provide any insight into how the DFOs were able to improve motor capacities. The authors also compared the improvements between disability categories and found that children who were developmentally delayed (n=20) demonstrated the largest improvement whereas small but non-significant improvements were seen in children with CP (n=3) and Down syndrome (n=2) (Pitetti and Wondra, 2005). These results need to be carefully interpreted due to the different sample sizes of the subject groups.

The design and construction of the DFOs used in this study carefully followed detailed instructions in descriptive literature, therefore, poor orthosis design is unlikely to be a contributing factor (Hylton, 1990a; Hylton, 1990b; Pratt, 2000; Radtka *et al.*, 1997). DFOs are frequently described in the literature as features of dynamic AFOs rather than as tone-reducing devices used on their own. It may be possible that the effects of DFOs are dependent upon the positioning of the subtalar joint which can be controlled by dynamic AFOs to ensure that the foot is correctly aligned and positioned on top of the DFOs. It is also possible that the DFOs may be most effective in altering MN excitability during dynamic activities such as walking, and this warrants further investigation.

OCGs are said to have inhibitory effects on MN excitability presumably through the stimulation of cutaneous mechanoreceptors (Blashy and Fuchs, 1959; Robichaud *et al.*, 1992). Literature regarding OCGs is quite outdated and experimental studies are few. One of the only experimental studies found that the use of a OCGs on the upper limb of 20 subjects with spastic hemiplegia following stroke resulted in improved neuromuscular function assessed through postural carriage, reaction speed, muscle strength and range of

active motion (Whelan, 1964). The authors provided no explanation for how the OCGs were able to improve function except to suggest that OCGs may improve exteroceptive and proprioceptive facilitation.

There have been a number of experimental studies conducted on the effects of Lycra orthoses which have found varied results. Corn *et al.* (2003) assessed Lycra orthoses on the upper limbs of four children with spasticity. One child experienced a decline in the quality of his upper limb movements, another child showed initial improvements that were not maintained over time, and the other two children showed no significant changes. The authors gave little explanation for their non-significant findings but suggested that the effects of Lycra orthoses can be variable and individual. On the other hand, a study conducted by Gracies *et al.* (2000) on the effects of upper limb Lycra orthoses in adults with spasticity following stroke demonstrated that the Lycra orthoses were effective in reducing wrist and finger flexor spasticity as assessed by the Tardieu Scale. They were also effective in decreasing swelling, improving the resting position of the wrist, and increasing the passive ROM of the fingers in extension. The authors attributed the improvements to the tonic stretch applied by the garments rather than cutaneous stimulation. Most of the literature on Lycra orthoses focuses on the effects of garments for the upper limb or the whole body. A thorough literature search revealed no published studies that assess the effects of Lycra orthoses solely in the lower limbs.

One of the main components of OCGs is the circumferential pressure applied around the limb. It has been demonstrated that circumferential pressure decreases the soleus H-reflex in able-bodied subjects and subjects with spasticity (Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992). The pressures applied to the leg in previous investigations ranged from 36.7 to 40.8 mmHg which is greater than the pressures applied in the present study. According to standards for compression socks (British Standards Institution, 2001), compression up to 32 mmHg should have been achieved with the class 2 OCGs in the present study, although direct pressure measurements were not made. It is possible that higher pressures are required to influence spasticity, although in the one subject who responded to the OCG condition, the effect was noted for the lower compression OCG and not for the higher compression OCG. There is limited information about the

optimum level of circumferential pressure for reducing spasticity in the leg. The results of this study suggest that OCGs that apply less than 32 mmHg do not have a significant neurophysiological effect on spasticity. Further research is required to determine the optimum level of compression for the treatment of spasticity.

The amount of muscle stretch provided by the ROM walkers used in this study was relatively small compared to the amount of stretch applied by previous investigators. Kanter *et al.* (2006) and Vujnovich and Dawson (1994) both tested soleus MN excitability in healthy subjects with the soleus muscle stretched to its full range of dorsiflexion. These authors found that maximum H-reflex amplitudes were significantly depressed during the stretch. Hwang (2002a) stretched the soleus muscle in able-bodied subjects with the ankle in 20 degrees of dorsiflexion and found that the H:M ratio was significantly depressed compared with the ankle in a neutral position and in 20 degrees of plantarflexion (p=0.038).

Other authors have demonstrated H-reflex suppression with much smaller changes in ankle joint angles. Burke *et al.* (1971) assessed patients with spasticity and found that increasing passive dorsiflexion of the ankle resulted in progressive diminution of the H-reflex of the triceps surae muscles by approximately 30%. However, inhibition of the H-reflex amplitude could also be shown with passive dorsiflexion movements as small as five degrees (Burke *et al.*, 1971). Robinson *et al.* (1982) did not clearly state how much stretch they placed to the soleus muscle, however, they found that an average of four degrees of dorsiflexion reduced soleus H-reflex amplitudes in able-bodied subjects by 37%. In three out of ten subjects, only two degrees of dorsiflexion was sufficient to reduce H-reflexes by 50%.

The dorsiflexion angle of 7.5 degrees that was used in the present study may have been smaller than the angles used in some previous studies, however, the subjects in this study had chronic plantarflexor spasticity. While all of the subjects could dorsiflex their ankle to 90 degrees with their knee extended, their passive dorsiflexion ranges were less than normal. This means that 7.5 degrees of dorsiflexion should have been sufficient to apply a decent stretch on the plantarflexor muscles. The reason why greater dorsiflexion angles were not investigated in the present study is because they would not be functional or safe

for use in AFOs for ambulation. All of the previous studies tested subjects in non-weight-bearing positions, therefore, subject comfort, safety and stability in weight-bearing were not important considerations.

One key feature of this study was that the effects of the tone-reducing devices were examined in full weight-bearing. This is important as it has been shown that subject positioning can significantly affect the H-reflex in hemiplegic subjects as well as able-bodied subject, as the H-reflex is a task and posture dependent measure (Chalmers and Knutzen, 2002; Kawashima *et al.*, 2003). Kawashima *et al.* (2003) demonstrated that H-reflex amplitudes were reduced in patients with complete SCI in a standing position compared with sitting. As the patients had complete spinal lesions, peripheral sensory inputs were the most likely cause for the inhibition in standing as opposed to descending commands from supraspinal centres. The authors suggested that graviception (the load at the vertebral column and lower limb joints, and/or cutaneous information from the sole of the foot) played an important role in reducing the excitability of the spinal MNs to the Ia afferent inputs while standing upright.

As was suggested in the previous study, it is possible that evaluating the tone-reducing devices in the standing position resulted in the overall non-significant findings. This may be particularly true as postural perturbations were caused by the electrical stimulations potentially increasing the effects of peripheral and/or cortical influences required to maintain postural standing balance. Nevertheless, it is important that these devices are tested in real life circumstances to gauge their effects on functional activities. It may be that effects produced in laboratory conditions, where subjects are relaxed in non-weight-bearing positions, may not be achievable in real life orthotic applications. Whether TROs are less effective in altering MN excitability in weight-bearing positions compared with non-weight-bearing position warrants investigation.

5.4.1 H:M ratios greater than one

H:M ratios greater than one were recorded for two subjects which should not have been possible as H:M ratios of 1.0 indicate that the entire MN pool is being activated by the reflex. Firstly, it is possible that the maximum M-response amplitudes were not recorded

accurately as increases in the stimulus intensity were ceased too early. Testing was terminated when increases in the M-response amplitudes had apparently ceased, despite an increase in stimulus intensity. Further increases in stimulus intensity to confirm that the M-response had indeed reached its maximum amplitude were not performed. This decision was made to reduce the effects of subject fatigue and discomfort which would have been caused by further increases in stimulus voltage. Because of variability in responses from trial to trial, it is possible that in these cases stimulus voltage was not increased sufficiently to record the true maximum M-response amplitudes.

Secondly, the maximum M-response amplitudes may have been missed because the increments between stimulus intensities were too big. Brinkworth *et al.* (2007) and Palmieri *et al.* (2002) recommended that stimulus intensity be increased in very small increments to determine the stimulus intensity required to produce the H-reflex and M-response maximum amplitudes with sufficient accuracy. Brinkworth *et al.* (2007) suggested that at least 15 stimulus steps were required to determine the H-reflex peak. It could then be assumed that at least 30 stimulus steps are required to determine the M-response peak since the M-response peak occurs at a much higher stimulus voltage. About 20 stimulus steps were made for each condition to construct the recruitment curves in the present study which may have resulted in an underestimation of the M-response maximum amplitudes for those two subjects.

However, decreasing the increment size between stimulus intensities would have greatly increased the length of the testing session and the effect of subject fatigue. In the present study, the testing sessions lasted an average of three hours for each subject which is already quite long. The errors in estimates of the H:M ratios were small and did not affect the overall results. Therefore, the methods for recording the H-reflex and M-response amplitudes were considered appropriate for future studies. As the H:M ratios were only slightly greater than one, they were reported in the data as they were recorded.

One of the questionable H:M ratios was the initial baseline measure for subject 11. This could raise concerns regarding the calculation of the $R_{95\%}$, so more conservative numbers were substituted for the initial baseline measure to determine if they would alter the overall results. When the initial baseline H:M ratio was amended to one, the $R_{95\%}$

decreased to 0.148. The DFO condition for subject 11 was still outside of the $R_{95\%}$. To be even more conservative, the M-response of the second baseline condition was used to recalculate the H:M ratio for the initial baseline measure. This changed the second baseline H:M ratio to 0.86. When the $R_{95\%}$ was recalculated with the baseline measure amended to 0.86, the overall results remained the same. As the discrepancies were very small and substituting other estimates of the maximum M-response amplitude did not alter the conclusions, it was concluded that they did not significantly affect the validity of the measurements or the results of the statistical analysis.

5.4.2 Limitations

The data for three subjects were unable to be analysed (as discussed in section 5.3). For two of those subjects, the electrical stimulations caused increases in clonus and tone suggesting that those subjects had abnormally high levels of MN excitability. It is possible that the repeated electrical stimulations also induced a less obvious increase in MN excitability in the other subjects and it could be suggested that the tone-reducing effects of the orthoses were masked by the effects of the electrical stimulations. However, there was no evidence of augmentation of the H-reflex curve with repeated electrical stimulation and the two baseline conditions which occurred at either end of the testing period were not significantly different.

One of the limitations of this study was the small sample size which was exacerbated by the three subjects who were unable to complete the testing. According to the results of the power analysis, it was estimated that the sample size of 10 subjects gave 65% power. The risk of subjects being unable to complete the testing needs to be anticipated for future studies to ensure that the sample size is sufficient for data analysis. There is no easy way to screen subjects to ensure that usable H-reflex and M-response measures can be obtained from them, so a greater number of subjects need to be recruited to account for potential dropouts.

The use of an unblinded assessor may be a potential criticism of this study. However it was impractical for the assessor to be blinded as the interventions were clearly visible and distinguishable. The measurement of MN excitability is highly objective and a strict

protocol was followed for each condition. Therefore, the results of this study are not believed to be affected by assessor bias.

The lack of a prolonged accommodation period for each of the devices could be proposed as a potential reason for the observed ineffectiveness. However, previous studies have shown that spinal reflex activity responds immediately to interventions (Nishikawa and Grabiner, 1999b; Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992; Robichaud and Brunt, 1994). Therefore, lengthy accommodation periods should not be necessary. The procedures of this study have provided valuable information regarding the immediate effects of the tone-reducing devices.

5.5 Conclusion

This study has demonstrated that overall, the DFO, OCG and stretch of the plantarflexor muscles had no significant effect on soleus MN excitability in subjects with spasticity while standing. Therefore, the study hypothesis was not supported. Further testing is required to examine the effect of such devices when combined with biomechanical AFO management and assessed during functional activities in hemiplegic subjects.

Chapter Six

The neurophysiological effect of TRAFOs in subjects with spasticity[3]

6.1 Introduction

The study reported in the previous chapter of this thesis was conducted with a view towards assessing the effects of TRAFOs in subjects with spasticity. The previous study was required to investigate the effects of tone-reducing devices on their own, prior to being incorporated into AFOs. While the previous study found overall non-significant effects of the tone-reducing devices on MN excitability, their effects when combined with biomechanical AFO support and control needed to be investigated in order to meet the aim of this thesis, which was to determine the effects of TRAFOs. It was suggested in the previous study that the effects of the tone-reducing devices may differ when used in conjunction with AFOs which provide biomechanical support and control.

The three tone-reducing features that were examined in the previous study (tone-reducing footplates, OCGs and muscle stretch) can be incorporated into AFOs to produce TRAFOs. A review of these tone-reducing features has been previously presented (see section 5.1.2). In order to determine the tone-reducing effects of TRAFOs, they need to be compared with identical AFOs that do not have tone-reducing features. In other words, the biomechanical effects of the two orthoses need to be controlled so that any differences in the results can be confidently attributed to the tone-reducing effects of the TRAFOs.

[3] A manuscript based on this chapter has been published in Prosthetics and Orthotics International (Ibuki *et al*., 2010). A copy of the published manuscript is included as Appendix C.

The purposes of this study were:

1. To examine the neurophysiological effect of articulated AFOs and TRAFOs on the MN excitability of subjects with spasticity following stroke while standing; and
2. To compare the neurophysiological effect of articulated AFOs and TRAFOs to determine if the TRAFOs offer any neurophysiological benefits over standard AFO designs.

The TRAFOs tested in this study included an articulated ankle joint with a 90 degree plantarflexion stop, a DFO, and the addition of an OCG in an attempt to further enhance the speculated tone-reducing effects. It was hypothesised that the H:M ratio would be reduced with the TRAFO conditions.

6.2 Methods

6.2.1 Subjects

Fifteen subjects who had previously suffered stroke were recruited from the community for this study. All of the subjects who had been involved in the previous study were invited to participate in the current study. Six subjects volunteered to participate. The remaining subjects were recruited through advertisements in print media, the Stroke Association of Victoria, community stroke support groups, the National Stroke Research Institute (Victoria) and outpatient rehabilitation clinics. The inclusion criteria were the same as those for the previous studies (see section 5.2.1). The subject characteristics are summarised in Table 6.1.

6.2.2 Apparatus

The apparatus used for this investigation were exactly the same as those used for the previous studies (see section 4.2.2).

Table 6.1 Subject characteristics.

Subject	Gender	Age (years)	Time since stroke (years)	Stroke type	Side of hemiplegia	Tardieu test (X, Y)	AFO User	Type of AFO	Gait aids required	Sensory deficit
1	M	52	26	H	Right	2, 20º	Yes	LSAFO	Yes	Yes
2	M	58	10	I	Right	2, 42º	Yes	Solid AFO	Yes	No
3	M	65	3	I	Right	2, 12º	No		No	Yes
4	M	63	6	H	Left	3, 20º	No		Yes	Yes
5	M	60	5	H	Left	3, 7º	Yes	Hinged AFO with PF stop	Yes	No
6	F	57	2	I	Right	2, 24º	Yes	Hinged AFO	Yes	No
7	F	50	15	H	Right	3, 8º	No		Yes	Yes
8	F	60	9	H	Left	3, 22º	Yes	Solid AFO	Yes	Yes
9	F	41	2	H	Right	2, 14º	No		No	No
10	F	66	4	I	Right	2, 18º	No		Yes	Yes
11	M	59	17	H	Right	4, 36º	No		Yes	Yes
12	F	60	5	H	Right	2, 30º	No		No	No
13	M	71	6	I	Left	3, 22º	No		No	No
14	M	71	35	H	Right	3, 22º	No		Yes	No
15	F	38	4	H	Left	3, 26º	Yes	Prefabricated ankle brace	Yes	No
Total / Mean	8 Male, 7 Female	58.07	9.93	10 H, 5 I	10 Right, 5 Left	2.6, 21.53º	6 Yes, 9 No		11 Yes, 4 No	7 Yes, 8 No

X= Quality of muscle reaction; Y= Angle of muscle reaction in plantarflexion; H= Haemorrhagic; I= Ischemic; LSAFO= Leaf-spring ankle-foot orthosis; AFO= Ankle-foot orthosis; PF= Plantarflexion.

6.2.3 Procedures

Ethics approval for this study was obtained from the Health Sciences Faculty Human Ethics Committee at La Trobe University (ethics approval number FHEC07/187). All subjects provided written informed consent before being included in the study. This study was conducted as a separate study following the conclusion of the study reported in Chapter Five. All subjects (including those who were involved in the previous study) were required to attend two sessions, the first for assessment and the second for testing.

During the assessment session, detailed assessments were completed on each subject by the same qualified orthotist to determine each subject's level of impairment with particular attention to the presence and degree of spasticity. A plaster-of-Paris impression cast was taken of each subject's affected leg with the ankle joint held in a neutral position (tibia and foot aligned at 90 degrees and the subtalar joint neither inverted nor everted as determined by visual examination of the position of the calcaneus in relation to the leg). The plaster impression casts were sealed and filled with liquid plaster to produce positive models of each subject's leg.

6.2.3.1 Orthosis fabrication

Custom-made AFOs and TRAFOs were fabricated from the same positive cast for each subject (Figure 6.1). This ensured that the devices were identical except for the tone-reducing features of the TRAFOs. Both the AFOs and TRAFOs were moulded with 5 mm thick polypropylene plastic and were articulated with Tamarack flexure ankle joints (free motion model 740, Becker Orthopedic, Michigan, USA, 48083-4576). Both of the AFOs had plantarflexion stops at 90 degrees but allowed free dorsiflexion.

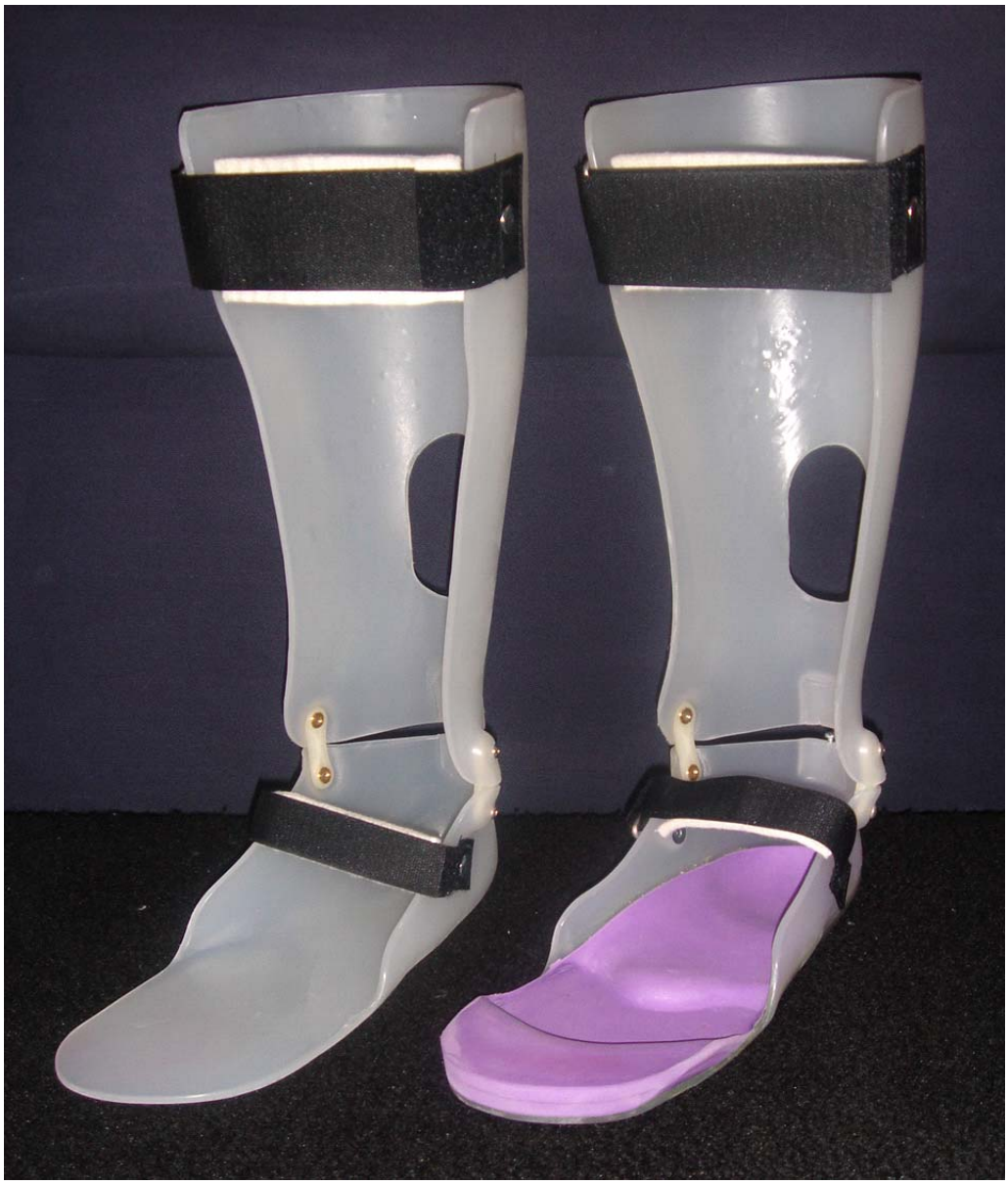

Figure 6.1 The custom-made articulated AFO and TRAFO.

The AFOs were fabricated first following standard cast modifications for AFOs with full length footplates (Weber, 1990) (Figure 6.2). The AFOs were all to be worn with firm-soled post-op shoes with 1 cm pitch at the heels. No pitch was modified into the casts as the 1 cm heel height of the post-op shoes was used to allow slight anterior inclination of the AFOs to relieve tension in the plantarflexor muscles by encouraging slight knee flexion. Further details of the fabrication process have been previously provided (see section 4.2.3.1). Once the AFOs had been fabricated, the plaster casts were repaired and modified with the tone-reducing footplate features to fabricate the articulated TRAFOs

(Figure 6.3). These tone-reducing footplate features were the same as those used in the previous study (Hylton, 1990a; Hylton, 1990b; Radtka *et al.*, 1997) (see section 5.2.3.1). The DFOs were fabricated using sheets of high density EVA foam (250 kg.m^3) that were vacuum moulded onto the soles of the casts and then shaped. This was done instead of directly vacuum moulding the plastic onto the casts, as EVA is able to contour more closely to the shape of the casts than plastic, which can bridge over the concavities of the positive casts. The EVA was also moulded first so that the base of the TRAFOs could be levelled to provide a stable plantar base of support. It was also felt that patients would be more comfortable standing on the footplate modifications if they were fabricated from EVA which is softer than the hard polypropylene plastic. The DFOs were held onto the casts with nylon stockinette and plastic was moulded over the casts to fabricate the TRAFOs.

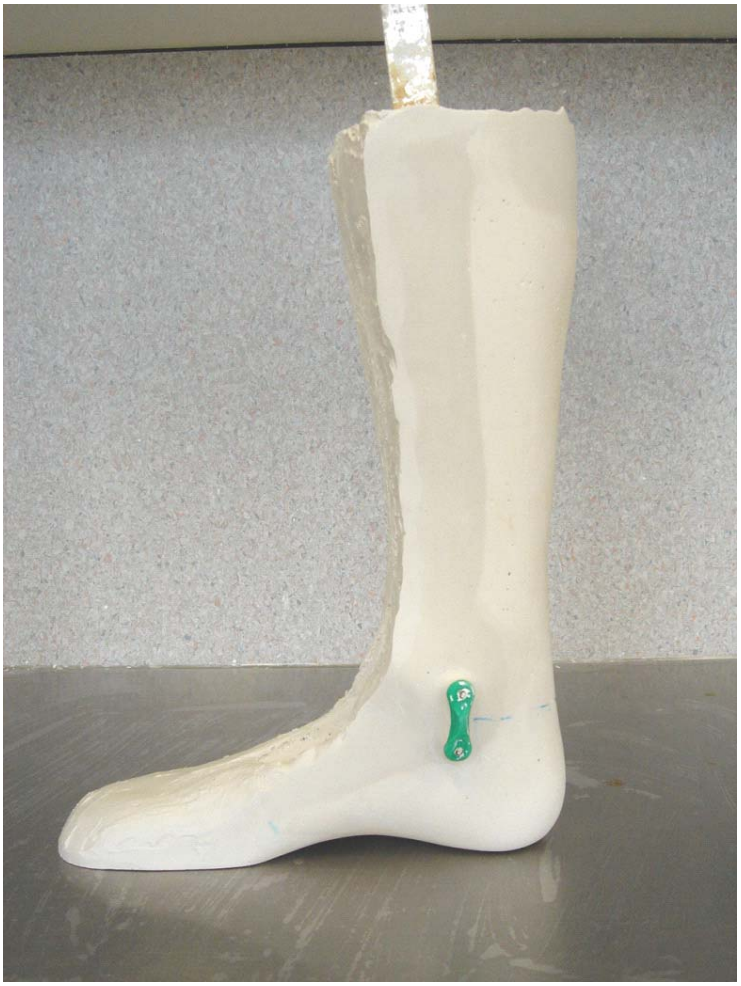

Figure 6.2 A modified positive cast for the fabrication of a standard articulated AFO.

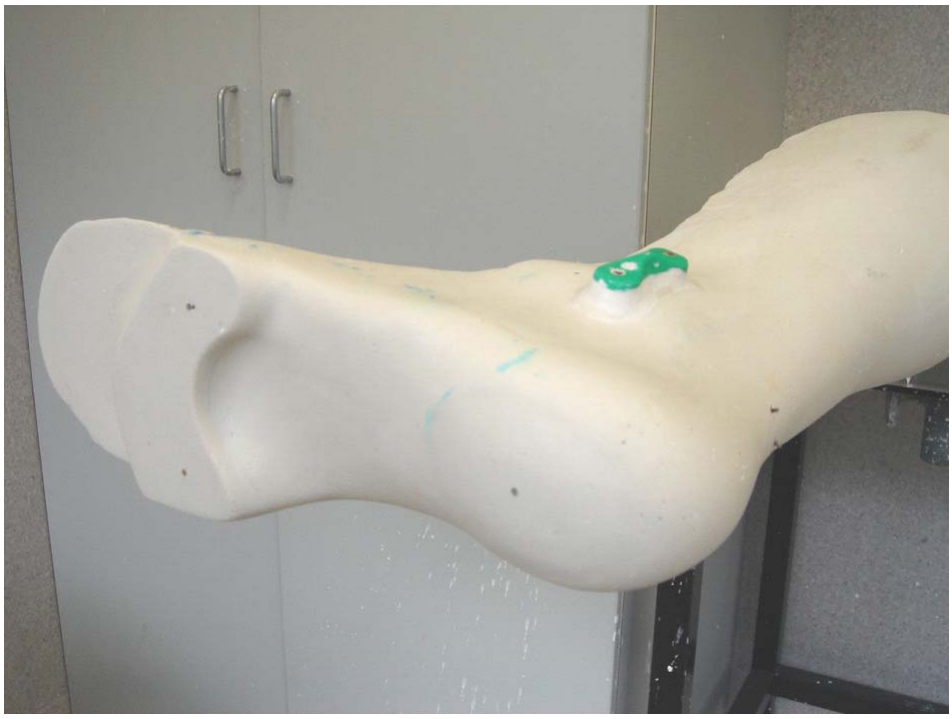

Figure 6.3 A modified positive cast for the fabrication of an articulated TRAFO.

Rectangular cut-outs (30x50 mm) were made in the posterior shells of the AFOs and TRAFOs to allow for EMG electrode placement over the soleus muscles (Figure 6.1). Calf and ankle straps were attached and threaded screws were drilled into the plantarflexion stops to adjust the stops to 90 degrees. It was decided not to set the ankle joints of the TRAFOs into dorsiflexion to stretch the plantarflexor muscles as this would have changed the biomechanical effect of the TRAFOs which needed to be controlled between the AFOs. As subjects were only tested in a static standing position in this study, the articulated ankle joints did not have a significant function. The orthoses were all fabricated with articulating ankle joints as they were intended to be used for the following study (Chapter Seven) which required subjects to walk while wearing the AFOs.

Class 1 (18-21 mmHg) Venosan (Salzmann AG, St. Gallen, Switzerland, 9000) below-knee compression garments without toes were used to create the OCGs, which were an added tone-reducing feature (Blashy and Fuchs, 1959; Robichaud and Agostinucci, 1996;

Whelan, 1964). The previous study demonstrated that there were no significant differences between the effects of the class 1 garments and the class 2 garments, therefore, class 1 garments were chosen for use in this study as they were easier to don and doff minimising the risk of electrode movement. Pieces of smooth inelastic vinyl were specifically cut from measurements of the subjects' legs to create inactive fields over the plantarflexor muscles. The pieces of vinyl were fitted directly against the subjects' legs inside the compression sleeves which were worn like socks under the TRAFOs.

Prefabricated post-op shoes were worn with the orthoses. The post-op shoes were modified with additional ankle straps to ensure that they were adequately supportive and held the AFOs firmly in the shoes (Figure 6.4).

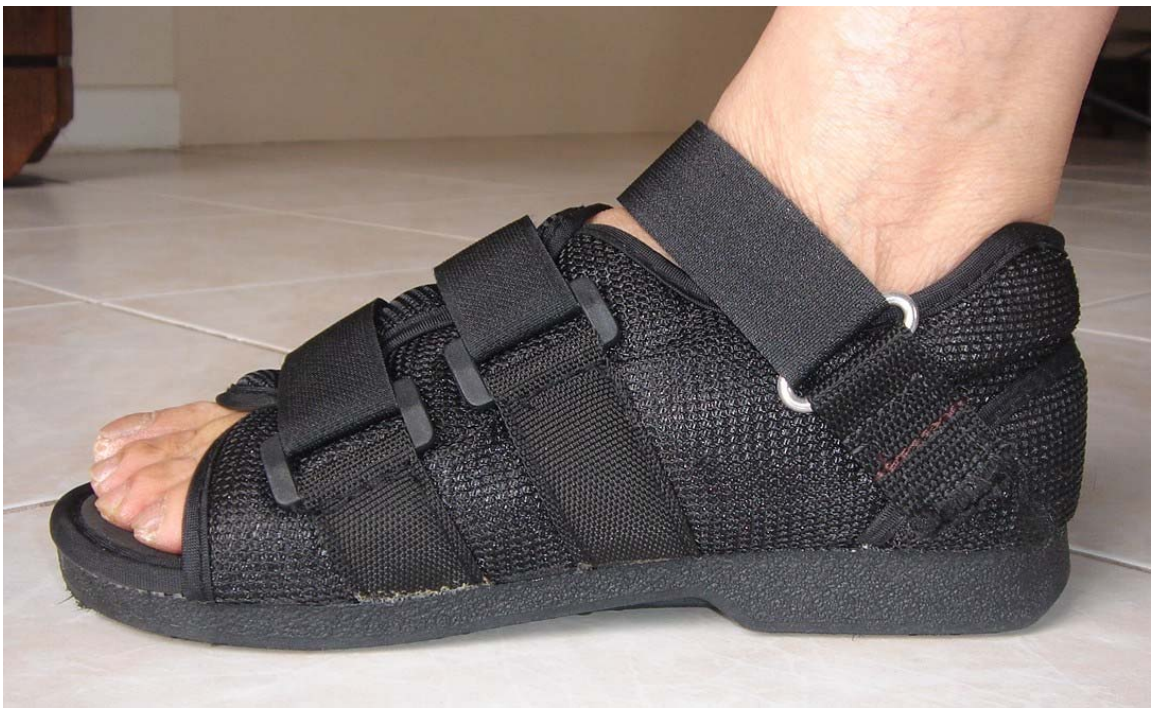

Figure 6.4 A prefabricated post-op shoe with the addition of an ankle strap.

6.2.3.2 Testing procedures

Details regarding the procedures for subject preparation have been previously described (see section 3.2.3.1). Details regarding the testing procedures have also been previously described (see section 3.2.3.2). All of the subjects were required to verbally confirm that the AFOs and TRAFOs were comfortable to stand in before the testing could begin. Any areas of discomfort or issues with the fitting of the orthoses were rectified before the testing commenced.

The design of this repeated-measures intervention study followed an ABA format. There were five conditions tested in this study:

A_1 - Shoes only (baseline);

B_1 - Standard AFO;

B_2 - TRAFO;

B_3 - TRAFO with OCG;

A_2 - Shoes only (baseline return).

For all of the conditions, modified post-op shoes were worn by the subjects to standardise the footwear. The order of the intervention conditions (B_1 to B_3) was randomised to prevent series effects, and a brief accommodation period was given at the beginning of each condition to allow subjects to assume a comfortable standing position. For each of the five testing conditions, H-reflex and M-response recruitment curves were constructed from an average of four trials at each level of stimulus intensity.

6.2.4 Statistical analyses

The results of the previous study (see section 5.3) were used to calculate the sample size and power required for the current study. It was estimated that a sample of 18 subjects would be required to provide 80% power for detecting a difference of 0.182 (the $R_{95\%}$ interval from the previous study) between the baseline and intervention H:M ratio means, assuming a SD of 0.20. Details regarding the statistical analyses have been previously provided (see section 4.2.4). The critical value for t with 14 degrees of freedom (t=2.1448) was used as the multiplier in the calculation of the $R_{95\%}$.

6.3 Results

The ANOVA revealed that there were no significant differences in H:M ratios between any of the conditions (F=0.992, df=2.167, p=0.388) (Figure 6.5). The $R_{95\%}$ was used to further examine the data to determine whether any substantial changes within individual subjects had been masked by the averaging process. The $R_{95\%}$ interval for the H:M ratio was found to be 0.12. Four subjects had significant responses to one or more of the interventions when analysed using the $R_{95\%}$ (Table 6.2). All of the significant responses were increases in the H:M ratios demonstrating increases in the level of MN excitability. Subject 9 responded to the AFO condition, subjects 5 and 11 responded to the TRAFO condition and subjects 5 and 8 responded to the TRAFO with the addition of the OCG.

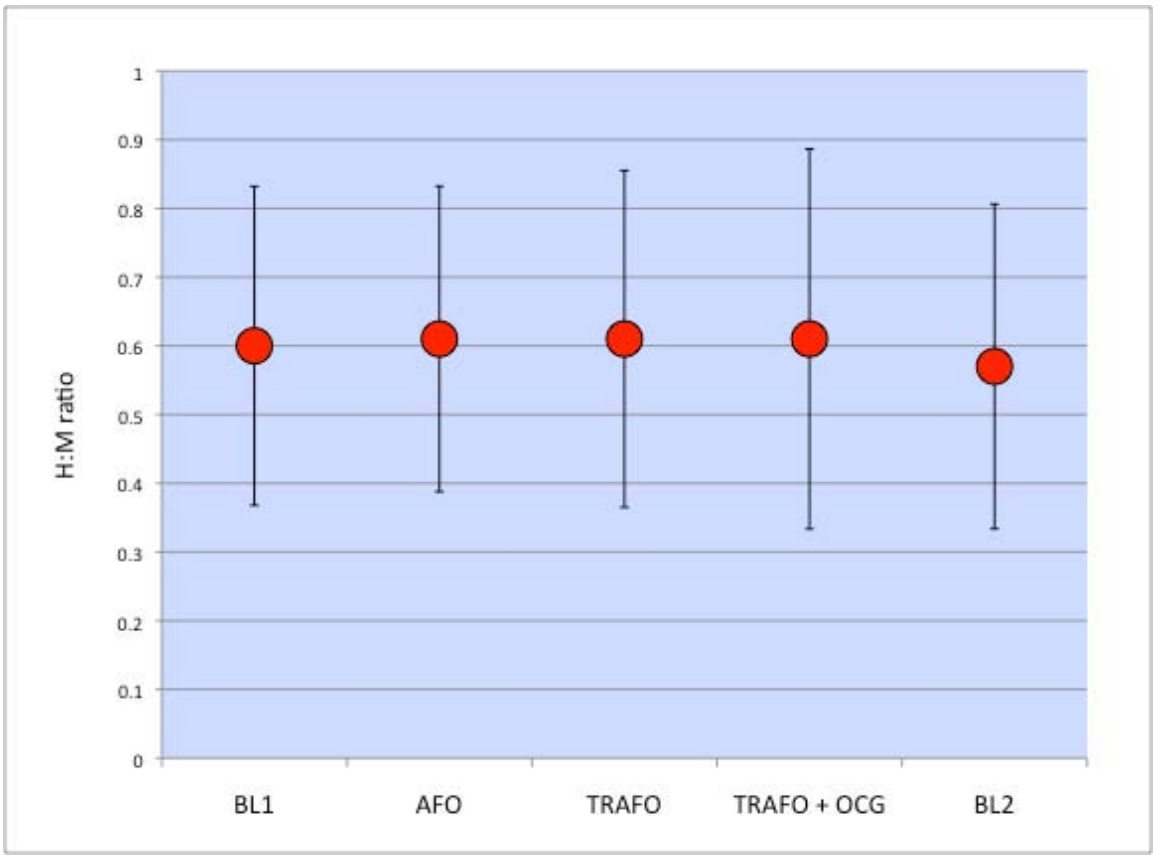

Figure 6.5 Mean (±1 SD) H:M ratios for all subjects across all conditions.

BL1= Shoes only initial baseline; AFO= Ankle-foot orthosis; TRAFO= Tone-reducing ankle-foot orthosis; TRAFO+OCG= Tone-reducing ankle-foot orthosis with the addition of an orthokinetic compression garment; BL2= Shoes only baseline return.

Table 6.2 Mean H:M ratios and the $R_{95\%}$ for each subject.

Subject	BL1	AFO	TRAFO	TRAFO+OCG	BL2	Mean of BL1 and BL2	$R_{95\%}$
1	0.57	0.60	0.59	0.60	0.54	0.56	0.44 - 0.68
2	0.60	0.47	0.44	0.46	0.42	0.51	0.39 - 0.63
3	0.20	0.22	0.24	0.25	0.20	0.20	0.08 - 0.32
4	0.59	0.58	0.61	0.62	0.55	0.57	0.45 - 0.69
5	0.69	0.75	**0.85**	**1.03**	0.69	0.69	0.57 - 0.81
6	0.61	0.62	0.58	0.59	0.58	0.60	0.48 - 0.72
7	0.64	0.62	0.62	0.59	0.67	0.66	0.54 - 0.78
8	0.91	0.92	0.98	**1.04**	0.89	0.90	0.78 - 1.02
9	0.29	**0.46**	0.29	0.21	0.19	0.24	0.12 - 0.36
10	0.39	0.42	0.41	0.35	0.38	0.39	0.27 - 0.51
11	0.68	0.77	**0.80**	0.79	0.66	0.67	0.55 - 0.79
12	1.00	0.97	0.99	0.92	0.99	1.00	0.88 - 1.12
13	0.30	0.24	0.29	0.24	0.35	0.33	0.21 - 0.45
14	0.58	0.60	0.58	0.58	0.62	0.60	0.48 - 0.72
15	0.90	0.84	0.84	0.86	0.85	0.88	0.76 - 1.00

BL1= Baseline (Shoes only); AFO= Ankle-foot orthosis; TRAFO= Tone-reducing ankle-foot orthosis; TRAFO+OCG= Tone-reducing ankle-foot orthosis with the addition of an orthokinetic compression sleeve; BL2= Baseline return (Shoes only).
H:M ratios in large, bold font are outside of the $R_{95\%}$.

6.4 Discussion

The results of this study indicated that overall, the AFOs and TRAFOs had no significant neurophysiological effect on the MN excitability of subjects with spasticity while standing. These results also demonstrated that TRAFOs had no tone-reducing benefits when compared with identical standard AFOs that did not have any tone-reducing features. The TRAFOs included the inhibitive footplate design advocated by a number of investigators (Dieli *et al.*, 1997; Hylton, 1990a; Pitetti and Wondra, 2005; Pratt, 2000) and were tested both with and without the addition of OCGs, which are also thought to have tone-reducing effects (Blashy and Fuchs, 1959; Dieli *et al.*, 1997; Lohman and Goldstein, 1993; Nash *et al.*, 2008; Pratt, 2000; Whelan, 1964).

Previous investigators have found improvements in gait (Diamond and Ottenbacher, 1990; Dieli *et al.*, 1997; Iwata *et al.*, 2003; Nash *et al.*, 2008; Zachazewski *et al.*, 1982), EMG activity (Nash *et al.*, 2008), posture (Zachazewski *et al.*, 1982), and joint position (Mills, 1984) with the use of TRAFOs. However, most of these studies compared the TRAFOs with a no-orthosis condition or compared the TRAFOs with different AFO designs, making it impossible to separate the neurophysiological and biomechanical effects of the orthoses. No evidence was found of inhibitory effects of the TRAFO features on MN excitability in the present study. It seems possible and even probable that improvements in function observed by previous investigations were due to the biomechanical effect of the orthoses rather than any neurophysiological effect.

In individual subjects who demonstrated changes in their MN excitability with the TRAFOs conditions in the present study, the excitability was increased in all cases. There were no cases where the TRAFO features caused a significant decrease in MN excitability. These results are the opposite of those found in the previous study (Chapter Five) in which subjects who responded to the tone-reducing devices all demonstrated decreases in their MN excitability (Table 5.4). It remains possible that some individuals may exhibit reductions in MN excitability as a result of TRAFO modifications, although none were observed in the present study.

In the four subjects who responded to one or more of the orthosis conditions, it appears that the effects of the orthoses on MN excitability did not last once the orthoses had been

removed (Table 6.2). This is evidenced by the similarity between the H:M ratios for the initial baseline condition and the final baseline condition. This indicates that any effects of the orthoses were only present when the orthoses were being worn.

Only one other study has examined the effect of AFOs on lower limb MN excitability using H-reflex measurements. Nishikawa and Grabiner (1999b) examined the effect of semi-rigid prefabricated ankle braces compared with no-brace conditions in 11 able-bodied subjects. They measured peroneal MN excitability using H-reflex amplitudes and found immediate increases in H-reflex amplitudes (approximately 10%) with the application of the ankle braces. The authors attributed the results to the stimulation of mechanoreceptors, particularly cutaneous receptors. The results of Nishikawa and Grabiner (1999b) and the results of the analysis of individual subjects in the present study suggest that AFOs may have a predominantly excitatory effect on lower limb MNs.

Previous studies that have been conducted on specific tone-reducing features such as muscle stretch, the application of tendon pressure and circumferential pressure, found significant decreases in MN excitability due to the stimulation of specific afferent pathways (Hwang, 2002a; Kanter *et al.*, 2006; Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988; Robichaud *et al.*, 1992; Vujnovich and Dawson, 1994). It appears that it may not be possible to replicate these inhibitory effects in AFOs by incorporating these tone-reducing features as the apparent excitatory effect of the AFOs themselves may overshadow any inhibitory effect of the tone-reducing features.

This study highlights the need for care in prescribing orthoses for patients with spasticity. Despite the requirement for biomechanical control, it is possible that orthoses with or without tone reducing features may have excitatory effects on MN excitability, causing increases in the level of muscle spasticity. It has been previously suggested that the use of orthoses for patients with spasticity may be detrimental, due to the activation of reflexes which may result in undesirable muscle activity (Pratt, 2000; Smelt, 1989). Careful consideration needs to be given to the design of orthoses for patients with spasticity to minimise any potential detrimental effects. This can be done by carefully selecting the materials used for fabrication, considering the trimlines of the orthoses, considering areas where the orthosis contacts the limb and selecting appropriate joint ranges of motion allowed by the orthoses.

6.4.1 Limitations

In this study, subjects were only assessed during static standing. However, it is during walking when changes in plantar pressures, joint angles and muscle length are believed to have the greatest affect on spasticity (Duncan, 1960; Rossi, 1992). The TRAFOs may have been ineffective in this study because subjects were only standing in them and not walking. The effects of the TRAFOs during walking warrants further investigation. However, previous authors have stated that significant inhibitory changes can be observed when a patient with spasticity simply stands on a tone-reducing footplate (Pratt, 2000). This is said to be due to the inhibition of reflexogenous areas on the sole of the foot by the diminution of plantar pressures (Pratt, 2000). No such changes were observable in the present study.

Despite the fact that subjects were instructed to bear equal weight through both limbs during the testing period, the actual weight distribution was not measured. It is known that stroke patients generally favour their unaffected side when weight-bearing (Mojica *et al.*, 1988). It is possible that subjects were not bearing enough weight through their affected limbs to achieve the presumed tone-reducing effects of the footplates. Future investigations should measure the amount of weight-bearing between the affected and unaffected legs to ensure that the footplates are adequately loaded, and for consistency between the conditions.

It must be noted that in two subjects (subject 5 and 8), H:M ratios greater than one were recorded (Table 6.2). Subject 8 was one of the subjects from the previous study (subject 10 in the previous study) who recorded an H:M ratio greater than one (Table 5.4). Reasons for the greater than one ratios were explained in the previous study (see section 5.4.1). The ratios were only just above one, and for the same reasons as those outlined in the previous chapter, it was not believed that they affected the validity of the measurements or the results of the statistical analysis.

A further limitation of this study was the small sample size which increased the chance of a type II error. Difficulty in recruiting subjects who met all of the criteria for the study resulted in a smaller sample size than was initially planned. According to the results of the power analysis, it was estimated that the sample size of 15 subjects gave 70% power.

Based on the results of this study and the results of the previous study (Chapter Five), the addition of three more subjects is not likely to alter the overall results.

6.5 Conclusion

Overall, the TRAFOs and AFOs had no significant effects on soleus MN excitability in subjects with spasticity while standing. There were also no significant differences between the TRAFOs and the standard AFOs suggesting that TRAFOs have no additional tone-reducing benefits when compared to standard AFOs. The study hypothesis was not supported by the results. Further testing is required to determine the effect of the TRAFOs during functional activities such as walking.

Chapter Seven

The effect of TRAFOs on gait and soleus muscle activity in hemiplegic subjects with spasticity

7.1 Introduction

So far, the studies described in this thesis have focused on examining the neurophysiological effect of TRAFOs on spasticity by measuring MN excitability in the standing position. The results of previous studies in this thesis have suggested that overall, TRAFOs have no significant neurophysiological effect on the excitability of MNs that innervate the spastic soleus muscle. However, it is possible that TRAFOs have more substantial effects during dynamic activities such as walking. This warrants investigation especially since one of the aims of prescribing TRAFOs is to improve gait and function. Examining the effects of TRAFOs in the standing position alone is likely to underestimate their potential effects. Therefore, the focus in this chapter was to determine whether TRAFOs have any effect on gait parameters and muscle activation during walking.

7.1.1 Hemiplegic gait

Spasticity in the lower limbs may affect gait and muscle function in a detrimental manner. Normal walking relies on co-ordinated and controlled muscle function to produce an effective, efficient and safe gait. Spasticity affects muscle co-ordination and control thus causing gait to become ineffective, inefficient and unsafe (Fatone, Gard and Malas, 2009b; Mauritz, 2004; Rossi, 1992; Thijssen, Paulus, Van Uden, Kooloos and Hopman, 2007). Spasticity in lower limb muscles may also cause significant gait deviations, which if left unmanaged, may potentially increase in severity (Lima, 1990). Some of these gait deviations will now be discussed.

Hemiplegic gait is typically slow, laboured and asymmetric with smaller step lengths and more frequent steps (Fatone and Hansen, 2007; Lin, Yang, Cheng and Wang, 2006; Rossi, 1992; Tyson *et al.*, 1998). There may also be problems with trunk-limb, inter-limb, and intra-limb co-ordination (Thijssen *et al.*, 2007). Hemiplegic gait abnormalities

may result from one or more problems such as impaired sensorimotor control, balance dysfunction, neglect of the affected limb, weakness in individual muscles (particularly flexor muscles), decreased joint ROM, abnormal joint posturing, co-contractions, hyper-reflexive movements, or exaggerated primitive reflexes with loss of selective control of fine motor movement (Abe *et al.*, 2009; Lin *et al.*, 2006; Mojica *et al.*, 1988; Rossi, 1992; van der Salm *et al.*, 2005; Yelnik *et al.*, 1999). Sensory and proprioceptive impairments add to gait difficulties as patients may lack normal feedback with respect to limb position and limb placement (Lin *et al.*, 2006; Rossi, 1992). Hemiplegic gait is also associated with abnormalities in EMG activity with alterations in the timing, duration and profile of muscle activity (Stewart, Barbeau and Gauthier, 1991). While many hemiplegic gait deviations can be directly related to reductions in walking speed, there are many other changes that are independent of walking speed and are unique to hemiplegic gait (Leung and Moseley, 2003).

The gait patterns of hemiplegic subjects are not homogenous and can depend on the severity of neuromuscular impairment, the overall fitness and activity level of the individual, other co-morbidities or disabilities, and variations in the individual's gait pattern prior to the stroke (Kinsella and Moran, 2008; Titianova, Pitkanen, Paakkone, Sivenius and Tarkka, 2003). Further diversity in hemiplegic gait patterns is also expected due to variations in the type, location and severity of the brain lesion and variations in neuromuscular impairment associated with aging irrespective of the stroke (Kinsella and Moran, 2008; Wade, Wood and Hewer, 1985). Due to the variability in hemiplegic gait patterns following stroke, attempts to develop classifications for the various gait patterns is very difficult. Unlike the classifications that have been developed for spastic CP gait patterns (Rodda and Graham, 2001), there are no classifications for hemiplegic gait following stroke that are currently widely accepted.

Attempts by various investigators to classify hemiplegic gait patterns have differed depending upon the specific parameters considered in the classification and the recovery stage of the subjects examined. Some investigators have developed classifications based only on temporo-spatial and kinematic data (Kinsella and Moran, 2008) while others have included muscle strength, spasticity and muscle activity in their classifications (Kramers de Quervain, Simon, Leurgans, Pease and McAllister, 1996; Mulroy, Gronley,

Weiss, Newsam and Perry, 2003). While each study has been able to develop classifications for hemiplegic gait patterns depending upon the parameters considered and the characteristics of the subject groups studied, variability between the different classifications exists. This highlights the wide range of walking abilities in hemiplegic stroke subjects and the difficulty in developing a universally accepted classification system for hemiplegic gait following stroke. To add to this difficulty, it has also been demonstrated that the characteristics of hemiplegic gait will change depending upon the recovery stage of the subject following stroke (Mulroy *et al.*, 2003). Therefore, a particular classification system may only be relevant for a particular recovery period following stroke.

The possible gait deviations following stroke are numerous. Regarding temporal changes in hemiplegic gait, stance phase is generally longer on both the affected and unaffected limbs (Olney and Richards, 1996; Rossi, 1992; Titianova *et al.*, 2003). Stance phase duration is usually longer on the unaffected limb compared with the affected limb (Olney and Richards, 1996). These changes in stance phase lead to a decreased single limb support time on the affected limb (Lin *et al.*, 2006) and increase the percentage of the gait cycle spent in double support (Nash *et al.*, 2008; Olney and Richards, 1996; Stewart *et al.*, 1991).

Common gait deviations in the stance phase on the affected limb can include hip retraction, toe-heel or flatfoot initial contact, lack of tibial progression over the foot, decreased loading, lack of push-off and increased step width (Chen, Patten, Kothari and Zajac, 2005b; Fatone and Hansen, 2007; Mauritz, 2002; Roehrig and Yates, 2008; Rossi, 1992). Gait deviations of the affected limb during swing phase include circumduction of the hip, hiking of the pelvis, stiff knee and foot drop or toe drag causing poor foot clearance (Roehrig and Yates, 2008; Rossi, 1992; van der Salm *et al.*, 2005). Inadequate propulsion by the affected plantarflexors or hip flexors can cause lack of power production at toe-off and limit the speed of paretic limb advancement during swing (Chen, Patten, Kothari and Zajac, 2005a).

A significant number of these gait deviations are due to spasticity of the plantarflexor muscles. It has been estimated that the incidence of equinus deformity at the ankle in

stroke patients is between 10% and 20% (Kinsella and Moran, 2008). In normal slow walking the single largest burst of positive work is performed by the ankle plantarflexors during push off (Olney and Richards, 1996). Spasticity in these muscles prevents this work from occurring effectively. The lack of heel contact due to plantarflexor spasticity can be especially problematic as loading of the forefoot may activate the plantarflexion reflex and toe grasp reflex keeping the heel off the ground, the triceps surae contracted throughout stance and the toes painfully curled (Brunner, Meier and Ruepp, 1998; Duncan, 1960; Rossi, 1992). Loading the affected limb may also trigger the onset of clonus in the spastic plantarflexor muscles further impeding gait (Nash *et al.*, 2008).

Regarding joint angles there is typically decreased hip flexion at initial contact, increased hip flexion during mid-stance (Rossi, 1992), increased hip flexion at toe-off and decreased hip flexion during swing (Burdett, Borello-France, Blatchly and Potter, 1988; Olney and Richards, 1996; Yelnik *et al.*, 1999). There is either increased knee flexion or knee hyperextension at initial contact and less knee flexion at toe-off and during swing (Burdett *et al.*, 1988; Olney and Richards, 1996; Rossi, 1992; Yelnik *et al.*, 1999). Ankle dorsiflexion at initial contact, mid-stance and mid-swing is reduced and there is less ankle plantarflexion at toe-off (Burdett *et al.*, 1988; Olney and Richards, 1996; van der Salm *et al.*, 2005).

Controversy exists as to exactly how spasticity contributes to gait abnormalities in hemiplegic gait. Some authors have suggested that spasticity negatively affects gait (Hesse, Krajnik, Luecke *et al.*, 1996b; Hesse, Lucke, Bertelt *et al.*, 1994; Krawetz and Nance, 1996; Lamontagne, Malouin and Richards, 2001) while others have been unable to find a significant correlation between spasticity and gait or function (Ada, Vattanasilp, O'Dwyer and Crosbie, 1998; Galiana *et al.*, 2005; Nadeau, Arsenault, Gravel and Bourbonnais, 1999; O'Dwyer and Ada, 1996a; O'Dwyer *et al.*, 1996b; Sommerfeld, Eek, Svensson, Holmqvist and Von Arbin, 2004). In theory, inhibiting lower limb spasticity should result in improvements in gait and muscle function, however, results of studies that have used antispasmodic drugs to reduce spasticity in adults have been mixed (Yelnik *et al.*, 1999).

Hesse *et al.* (1996a; 1994) conducted two studies on the effects of Botulinum toxin-A (BTX-A) on hemiplegic gait. The investigators found overall improvements in gait function (Hesse *et al.*, 1994) and muscle activity (Hesse *et al.*, 1996a) following BTX-A injections. The authors injected BTX-A into the soleus, tibialis posterior, and medial and lateral heads of the gastrocnemius muscles of subjects with a mean stroke interval of 23 months. In the initial study, ten out of 12 subjects experienced a reduction in plantarflexor spasticity after the injections as assessed by the MAS (Hesse *et al.*, 1994). Those subjects also demonstrated improvements in walking velocity, stride length, stance symmetry and push-off of the affected limb at terminal stance. In the second study, nine out of 12 subjects benefited from the injections with reduced plantarflexor spasticity and subsequent improvements in gait and soleus muscle activity (Hesse *et al.*, 1996a). However, it was also noted that one subject experienced no benefits following the injection while two subjects experienced deterioration through decreased walking velocity and premature activity of the soleus muscle.

McLellan (1977) assessed the effects of a dose of oral baclofen on knee joint movements in subjects with chronic spasticity subsequent to a number of different neurological pathologies. The chronicity of the subjects was not stated. The author reported that baclofen successfully suppressed the muscle response to passive stretch over a period of four hours. However, the majority of subjects experienced no significant improvements in voluntary movement control of the knee. Upon observing individual data, there were some subjects who appeared to benefit from the baclofen with reduced clonus, spasms and co-contraction during voluntary movements although this was not noticed at a group level.

The difficulty in determining the relationship between spasticity and function is due to the fact that spasticity is comprised of both reflex and mechanical elements. This was discussed earlier in Chapter 2 (see section 2.2.3). While treatments like antispastic drugs may be instantly effective to reduce the reflex elements of spasticity like spasm, clonus and reflex stiffness, they may not be effective to treat the mechanical elements of spasticity like muscle contracture and passive stiffness. Therefore, the results of previous studies need to be interpreted with caution as the aspects of spasticity measured in the

studies differed with some measuring reflex components and others measuring mechanical components.

The time since onset of stroke of the subjects tested also varied greatly between studies from immediately post-stroke (Sommerfeld *et al.*, 2004) to several years post-stroke (Nadeau *et al.*, 1999). This is an important factor as spasticity evolves over time. Mechanical elements of spasticity such as muscle stiffness may have a greater effect on the overall phenomenon of spasticity in chronic subjects than in subjects who exhibit spasticity in the early stages post-stroke. The non-significant results of functional outcomes in studies that have inhibited spasticity in more chronic subjects may not be meaningful as only the reflex element of spasticity was treated. Intensive physiotherapy and gait retraining may be required following antispasticity treatments to also treat the mechanical elements of spasticity before functional outcomes can be adequately assessed. There is still much research required to determine the exact relationship between spasticity, gait and muscle function and future investigations should consider both the reflex and mechanical aspects of spasticity as well as the time since stroke onset of the subjects tested.

Hemiplegic gait very often results in long-term handicap and causes difficulties such as the inability to perform specific tasks and the inability to manoeuvre particular obstacles and environments (Lamontagne, Stephenson and Fung, 2007; Mauritz, 2004). The inefficiency of hemiplegic gait also means that many patients are unable to walk the distances necessary for community ambulation and their risk of falling is increased (Fatone *et al.*, 2009b; Roehrig and Yates, 2008). Furthermore, walking impairments can interfere with an individual's ability to participate in ADLs (Perry, Garrett, Gronley and Mulroy, 1995). Fortunately, hemiplegic gait can often be improved by use of an AFO and this will now be discussed.

7.1.2 The effect of ankle-foot orthoses on hemiplegic gait

AFOs are often prescribed for stroke patients in an attempt to improve the many gait impairments that result following stroke (Yamanaka *et al.*, 2004b). There have been numerous observational and cohort studies conducted examining the effects of standard

AFOs on hemiplegic gait. The majority of studies have reported favourable results with improvements in various temporo-spatial gait parameters (Abe *et al.*, 2009; Hesse *et al.*, 1999; Mojica *et al.*, 1988; Tyson and Thornton, 2001; Wang *et al.*, 2007), energy expenditure (Mauritz, 2004), balance and weight bearing on the affected limb (Mojica *et al.*, 1988; Wang *et al.*, 2005) and proprioception (Feuerbach, Grabiner, Koh and Weiker, 1994). Studies have demonstrated that AFOs can decrease the time taken for subjects to regain standing and walking function during rehabilitation (Yamanaka *et al.*, 2004b), minimise gait deviations (Teasell, McRae, Foley and Bhardwaj, 2001), reduce the risk of falling (Rao, Chaudhuri, Hasso *et al.*, 2008), provide a more stable base of support (Teasell *et al.*, 2001) and even reduce ankle clonus (Hesse *et al.*, 1996c).

However, the majority of these studies only present low levels of evidence. One study that claimed to be a randomised controlled trial investigating the effects of AFOs on the walking ability of subjects with chronic stroke was actually just a repeated measures study with subjects randomly assigned to two different groups that both received the same interventions, but in different orders (De Wit, Buurke, Nijlant, Ijzerman and Hermens, 2004). The only real randomised controlled trial that has been conducted on AFOs for hemiplegic gait assessed placebo AFOs (free motion) and treatment AFOs (fixed in five degrees of dorsiflexion) compared with percutaneous radiofrequency thermocoagulation of the tibial nerve at the level of the popliteal fossa (Beckerman, Becher, Lankhorst, Verbeek and Vogelaar, 1996b). Both treatments were aimed at reducing plantarflexor spasticity to improve lower limb function, passive ankle ROM and walking ability. The results demonstrated that one treatment of thermocoagulation of the tibial nerve was more effective in reducing plantarflexor spasticity than the use of AFOs. However, the authors recognised that there was a high percentage of non-compliance with AFO use during the study which lasted 15 weeks. The issue of non-compliance suggested that there were problems related to the fitting of the AFOs which were not adequately followed up. These issues lead to a bias in the results clearly favouring the tibial nerve thermocoagulation.

The paper with the strongest level of evidence was a systematic review on the effect of AFOs on gait and muscle activity in hemiplegic adults (Leung and Moseley, 2003). Thirteen articles were included in the review regarding the effect of AFOs on hemiplegic

gait, and four were included regarding the effect of AFOs on lower limb muscle activity. The authors acknowledged that the analysis was made difficult by the wide range of AFO designs tested and the heterogeneity of the subject groups tested in each study. The conclusion of the systematic review was that AFOs may be immediately effective in improving equinus and equinovarus ankle problems as well as improving kinematic and temporal gait parameters (Leung and Moseley, 2003). The effects on muscle activity were inconclusive.

Studies that have assessed the effect of AFOs on hemiplegic gait have tested several AFO designs including leaf-spring AFOs (Wang *et al.*, 2005; Wang *et al.*, 2007), solid AFOs (De Wit *et al.*, 2004; Mojica *et al.*, 1988), articulated AFOs (Fatone and Hansen, 2007; Hesse *et al.*, 1999; Roehrig and Yates, 2008; Tyson and Thornton, 2001; Tyson *et al.*, 1998) and some more original designs (Chen, Yeung, Wang, Chu and Yeh, 1999; Danielsson and Sunnerhagen, 2004; Pohl and Mehrholz, 2006). Different design features such as footplate length have also been investigated with Fatone *et al.* (2009b) recommending full-length footplates in conjunction with plantarflexion stops to limit internal knee flexor moment in early stance for hemiplegic subjects. Lehmann *et al.* (1987) investigated the effect of orthosis ankle angles on hemiplegic gait and suggested that AFOs set in slight plantarflexion were most effective.

Despite the positive results that have been demonstrated with the use of AFOs for hemiplegic patients, whether TRAFOs are able to provide further benefits by reducing spasticity neurophysiologically is unknown. The few studies that have been conducted on TRAFOs for hemiplegic gait have generally demonstrated favourable results. Iwata *et al.* (2003) tested solid AFOs on their own and then with the addition of ITBs and found that the addition of the ITBs significantly increased walking speed by 13.8%, stride length by 8% and cadence by 6.1%. Interestingly, these results only applied to those subjects who exhibited an active toe grasp reflex. The ITBs had no effect on the gait of hemiplegic subjects who did not exhibit the toe grasp reflex.

Diamond and Ottenbacher (1990) and Dieli *et al.* (1997) both conducted similar studies comparing the use of custom-made dynamic AFOs with barefoot conditions and prefabricated leaf-spring AFOs in subjects with hemiplegic spasticity. Both studies found

improvements in temporo-spatial gait parameters with the dynamic AFOs compared with the other conditions. Mueller *et al.* (1992) found greater foot stability and faster progression of the centre of mass over the foot during stance with the use of a custom-made dynamic AFO compared with shoes in their single male stroke subject.

Although these three studies found positive results following TRAFO use, they do not provide evidence that TRAFOs are more effective than standard AFO designs. This is because these studies have tested the TRAFO with no other brace for comparison (Mueller *et al.*, 1992) or have compared TRAFOs with AFOs of seemingly inferior designs (Diamond and Ottenbacher, 1990; Dieli *et al.*, 1997). Biomechanical differences in lever arms, control of the ankle joint and a more intimate custom-made fit mean that the effect of the tone-reducing features of the TRAFOs cannot be isolated and proven to be responsible for the improvements observed.

Only two studies have examined the effects of TRAFOs on spastic plantarflexor muscle activity in hemiplegic adult subjects (Mills, 1984; Nash *et al.*, 2008). Neither of the studies found any significant effects on muscle activity following TRAFO use during rest or during ambulation. Mills (1984) conducted a study on seven subjects with spasticity secondary to ABI and one subject with spasticity secondary to stroke using what she termed "inhibitive splinting". The author did not give sufficient details regarding the inhibitive splints used in the study except to state that they were made of either orthoplast or plaster, were bivalved and lined with polycushion. The splints were held onto the limb using Ace bandages so that skin contact was complete around the extremity. There is no detail provided regarding features that made the splints "inhibitive" except that they held the ankle joint within 5 to 10 degrees of its full passive dorsiflexion range. Stretch and circumferential contact appear to be the only tone-reducing features of the inhibitive splints. The author tested subjects in resting gravity-eliminated positions, and monitored muscle activity during rest over a two hour period. The lack of effect on muscle activity during rest was perhaps not surprising. Biering-Sorensen *et al.* (2006, p718) stated that the "measurement of EMG from a relaxed muscle without some kind of manipulation makes no sense, if the aim is to evaluate spasticity".

Nash *et al.* (2008) reported a case study of a 25 year old male subject with SCI, 16 months post accident. The authors analysed the subject's walking ability and gastrocnemius muscle activity during five conditions:

1. Barefoot;
2. Shoes;
3. Bilateral tone-reducing footplates;
4. Articulated AFO on the left leg and solid TRAFO on the right leg; and
5. Bilateral solid TRAFOs.

The five conditions were tested during two sessions one week apart where the order of the conditions was changed for the second session. The authors suggested that the results of the EMG analysis demonstrated that the bilateral TRAFO conditions caused the least amount of EMG activity in the gastrocnemius muscles compared with the other conditions. However, closer inspection of the EMG data reveals that these results were not consistent between sessions or between the affected and unaffected legs reducing confidence in the effects reported by the authors.

7.1.3 Summary and aims

The biomechanical effect of AFOs for hemiplegic subjects has received considerable attention in the literature, however, the effects of additional tone-reducing features to standard AFO designs remains unclear. In order to assess this adequately, a TRAFO must be assessed alongside an otherwise identical AFO, or the tone-reducing features need to be assessed while being added to the same AFO so that the biomechanical effect of the orthosis are controlled. In doing this, any differences observed in gait parameters or muscle activations can be attributed solely to the tone-reducing features and not to the biomechanical effect of the orthoses.

Apart from the study conducted by Iwata *et al.* (2003) who only examined the effects of ITBs, there has been one other study conducted in this manner. Crenshaw *et al.* (2000) assessed the effects of standard articulated AFOs with and without tone-reducing features in subjects with spasticity due to CP. They found no significant differences in gait parameters with the addition of tone-reducing features. This study provides evidence that tone-reducing features have no effect in subjects with CP, however, the results are in

contrast with the results of Iwata *et al.* (2003) who found that some subjects could benefit from the tone-reducing features.

The aims of this study were:

1. To examine the effect of articulated TRAFOs on temporo-spatial gait parameters and joint kinematics in subjects with spasticity following stroke; and
2. To examine the effect of articulated TRAFOs on soleus muscle activity in subjects with spasticity following stroke while walking.

It was hypothesised that the articulated TRAFOs would improve temporo-spatial gait parameters, joint kinematics and soleus muscle activity in subjects with spasticity when compared with standard articulated AFOs.

7.2 Methods

7.2.1 Subjects

Seventeen subjects who had previously suffered stroke were recruited from the community for inclusion in this study. All of the subjects who had been involved in the previous study (Chapter Six) were invited to participate in the current study. Fifteen of those subjects volunteered to participate. The remaining subjects were recruited via a number of different methods including the media, the Stroke Association of Victoria, community stroke support groups, the National Stroke Research Institute (Victoria) and community outpatient rehabilitation clinics. The inclusion criteria for this study were the same as those for the previous studies (see section 5.2.1). The subject characteristics can be found in Table 7.1. The functional walking handicap of the subjects was classified according to the Modified Functional Walking Categories for stroke (Perry *et al.*, 1995) (Table 7.2).

Table 7.1 Subject characteristics.

Subject	Gender	Age (years)	Time since stroke (years)	Stroke type	Side of hemi	Tardieu score (X, Y)	AFO user	Type of AFO	Gait aids	Sensory deficit	Modified Functional Walking Category
1	M	52	26	H	R	2, 20º	Yes	LSAFO	SPS	Yes	3
2	M	58	10	I	R	2, 42º	Yes	SAFO	SPS	No	4
3	M	65	3	I	R	2, 12º	No	–	–	Yes	6
4	M	56	4	I	R	3, 24º	Yes	LSAFO	SPS	Yes	3
5	M	63	6	H	L	3, 20º	No	–	SPS	Yes	3
6	M	60	5	H	L	3, 7º	Yes	HAFO with PF stop	SPS	No	4
7	F	62	15	H	L	2, 30º	Yes	HAFO with PF stop	SPS	Yes	3
8	F	57	2	I	R	2, 24º	Yes	HAFO	SPS	No	3
9	F	50	15	H	R	3, 8º	No	–	1xFAC	Yes	3
10	F	60	9	H	L	3, 22º	Yes	SAFO	SPS	Yes	4

Table 7.1 Continued.

Subject	Gender	Age (years)	Time since stroke (years)	Stroke type	Side of hemi	Tardieu score (X, Y)	AFO user	Type of AFO	Gait aids	Sensory deficit	Modified Functional Walking Category
11	F	41	2	H	R	2, 14º	No	–	–	No	6
12	F	66	4	I	R	2, 18º	No	–	SPS	Yes	5
13	M	59	17	H	R	4, 36º	No	–	SPS	Yes	3
14	F	60	5	H	R	2, 30º	No	–	–	No	4
16	M	71	35	H	R	3, 22º	No	–	2xFAC	No	4
15	M	71	6	I	L	3, 22º	No	–	–	No	5
17	F	38	4	H	L	3, 26º	Yes	Soft prefab AFO	SPS	No	4
Total / Mean	9 M, 8 F	58.18	9.88	11 H, 6 I	11 R, 6 L	2.59	8 Y, 9 N		11 SPS, 2 FAC	9 Y, 8 N	3.94

Hemi= Hemiplegia; X= Quality of the muscle reaction; Y= Angle of catch in plantarflexion; M= Male; F= Female; H= Haemorrhagic; I= Ischaemic; LSAFO= Leaf spring ankle-foot orthosis; SAFO= Solid ankle-foot orthosis; HAFO= Hinged ankle-foot orthosis; PF stop= Plantarflexion stop; Prefab= Prefabricated; SPS= Single point stick; FAC= Forearm crutch.

Table 7.2 The Modified Functional Walking Categories for stroke (Perry *et al.*, 1995).

Grade	Category	Description
1	Physiological walker	• Walks for exercise only either at home or in parallel bars during physical therapy • Uses a wheelchair for both bathroom and bedroom mobility
2	Limited household walker	• Relies on walking to some extent for home activities • Requires assistance for some walking activities, uses a wheelchair, or is unable to perform others • If a wheelchair is needed for either bedroom or bathroom mobility, the other activity can be performed with supervision only
3	Unlimited household walker	• Able to use walking for all household activities without any reliance on a wheelchair • Can perform bathroom mobility without assistance (may need supervision) • If supervision is required for both bedroom and bathroom mobility, then can enter/exit the home without a wheelchair • Encounters difficulty with stairs and uneven terrain • Needs at least supervision for both enter/exiting the house and managing curbs
4	Most-limited community walker	• Independent (without supervision) in either entering/exiting the home or managing curbs • Can manage both entering/exiting the home and curbs without assistance • Requires some assistance in both local store and uncrowded shopping centres
5	Least-limited community walker	• Can perform all moderate community activities without use of wheelchair • Needs at least some assistance in a crowded shopping centre • Can perform without assistance (but may need supervision) in one of the following: local stores, or uncrowded shopping centres
6	Community walker	• Independent in all home and moderate community activities • Can accept uneven terrain • Can negotiate a crowded shopping centre with supervision only

7.2.2 Apparatus

All gait data were acquired in the Musculoskeletal Research Centre's permanent motion analysis laboratory. A schematic diagram of the laboratory is depicted in Figure 7.1. The laboratory had a level vinyl floor with an AMTI force platform (Model BP400600HF-2000, Advanced Mechanical Technology Inc., Massachusetts, USA, 02472-4800) and a Kistler force platform (Model 9281B, Kistler Instrument Corp., New York, USA, 14228-2171) embedded flush in the floor in the middle of a 12 metre walkway. The calibration space for the motion analysis system includes the middle 6 metre of the walkway. The two force platforms were both 40x60 cm in size and were clearly visible in the floor. Objective kinematic and kinetic gait measurements were acquired with a motion analysis system (Vicon Motion Systems, Oxford, UK, OX2 0JB) utilising ten Vicon MX infrared cameras. Ground reaction forces (GRF) were recorded using the two force platforms in the centre of the calibration volume. Kinematic data were recorded at 120 Hz and GRFs were recorded at 960 Hz.

The three-dimensional marker coordinates and force platform data were used as inputs to Plug-In Gait (Vicon Motion Systems, Oxford, UK, OX2 0JB) to calculate joint kinematics and kinetics. Gait cycle events (heel contact and toe off) were identified from the force platforms for strikes which struck one of the force platforms cleanly. An autocorrelation routine using marker coordinates was used to identify similar events for strides not on the force platforms. All gait events were expressed as a percentage of the gait cycle to yield a normalised gait cycle irrespective of the actual time for a stride.

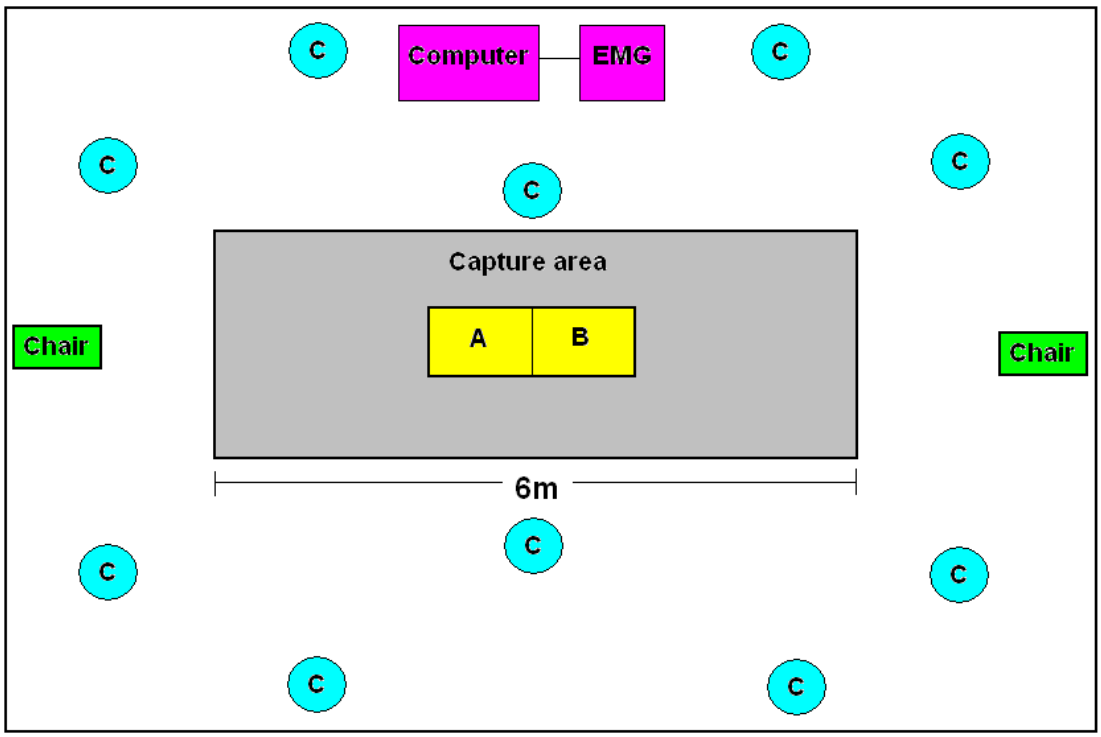

Figure 7.1 A schematic diagram of the motion analysis laboratory.
A= AMTI force platform; B= Kistler force platform; C= Infrared cameras.

The EMG activity of both affected and unaffected soleus muscles was concurrently monitored as the subjects completed each of the testing conditions. A Motion Analysis MA-100 system (Motion Lab Systems, Baton Rouge, USA, 70816) was used to monitor muscle activity. Bipolar electrodes (10 mm diameter) embedded in a preamplifier (Z03, Motion Lab Systems, Baton Rouge, USA, 70816) with a fixed inter-electrode distance of 2 cm between recording surfaces were used. Each of the electrodes was connected by cables to a backpack unit (Figure 7.2). The backpack was connected by an umbilical cord to the MA-100 system amplifier which was connected to the analogue to digital converter that was part of the laboratory computer system. Subjects walked with a cable trailing behind them from the backpack unit at all times during the testing. Data were high pass filtered with a cut-off frequency of 20 Hz, full wave rectified and low pass filtered with a cut-off frequency of 10 Hz to create linear envelope EMG waveforms. Data were captured at 1000 Hz using the Vicon motion capture system so that EMG data could be synchronised with kinematic and kinetic data.

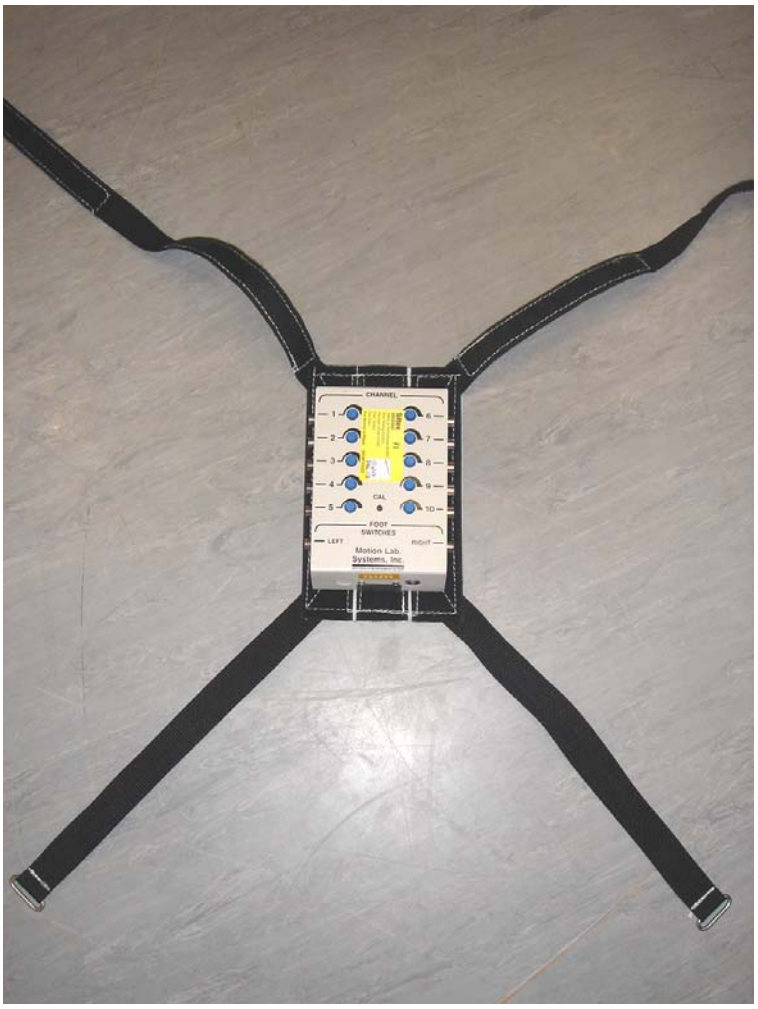

Figure 7.2 The EMG backpack unit.

7.2.3 Procedures

Ethics approval for the study was obtained from the Health Sciences Faculty Human Ethics Committee at La Trobe University (ethics approval number FHEC07/187). All subjects were required to give written informed consent before being included in the study. The 15 subjects who were involved in the previous study were only required to attend the testing session in this study. For the remaining subjects, detailed subjective and objective assessments were completed beginning with a phone interview to screen the subjects to determine their eligibility for the project. Subjects were immediately excluded if they stated that their stroke had had no affect on their walking ability, if they had never experienced muscle tightness or spasm, or if they had other serious medical

problems. Subjects who were not excluded after the phone interview were then brought to the University for a physical assessment conducted by a qualified orthotist. During the assessment, subjects underwent a subjective and objective assessment to determine their level of impairment following the stroke with particular attention paid to the presence and degree of spasticity in the lower limbs. Subjects then had plaster impression casts taken of their affected leg with the ankle joint held in a neutral position (tibia and foot aligned at a 90 degree angle and the subtalar joint neither inverted nor everted) for the fabrication of custom-made standard articulated AFOs and TRAFOs (Figure 6.1). All subjects were assessed and cast by the same qualified orthotist.

Details of the fabrication and design of the custom-made articulated AFOs and articulated TRAFOs have been previously provided (see section 6.2.3.1). OCGs were worn with the TRAFOs to increase the speculated tone-reducing effects. Details regarding the fabrication of the OCGs have been previously provided (see section 6.2.3.1). Modified prefabricated post-op shoes were also used to standardise footwear between subjects (Figure 6.4).

7.2.3.1 Subject preparation

Subjects were prepared for testing with the motion analysis system according to requirements for analysis using Plug-in-Gait with the lower body model (Vicon Motion Systems, Oxford, UK, OX2 0JB). The required subject measurements for Plug-in-Gait were recorded for each subject:

- Weight (kg);
- Height (cm);
- Inter-ASIS (anterior superior iliac spine) distance (cm);
- Leg length (cm) measured between the ASIS and the medial malleolus via the knee joint;
- Knee width (cm); and
- Ankle width (cm).

Reflective markers (14 mm diameter) were attached to the lower body (pelvis, thigh, shank and foot) with double-sided hypoallergenic adhesive tape over palpable anatomic landmarks. Wand markers (60 mm long with 14 mm reflective markers on the end) were

placed on the lateral aspects of the thigh and leg. As subjects wore post-op shoes for all of the conditions, the toe and heel markers were adhered to the post-op shoes over the anatomical landmarks (Figure 7.3).

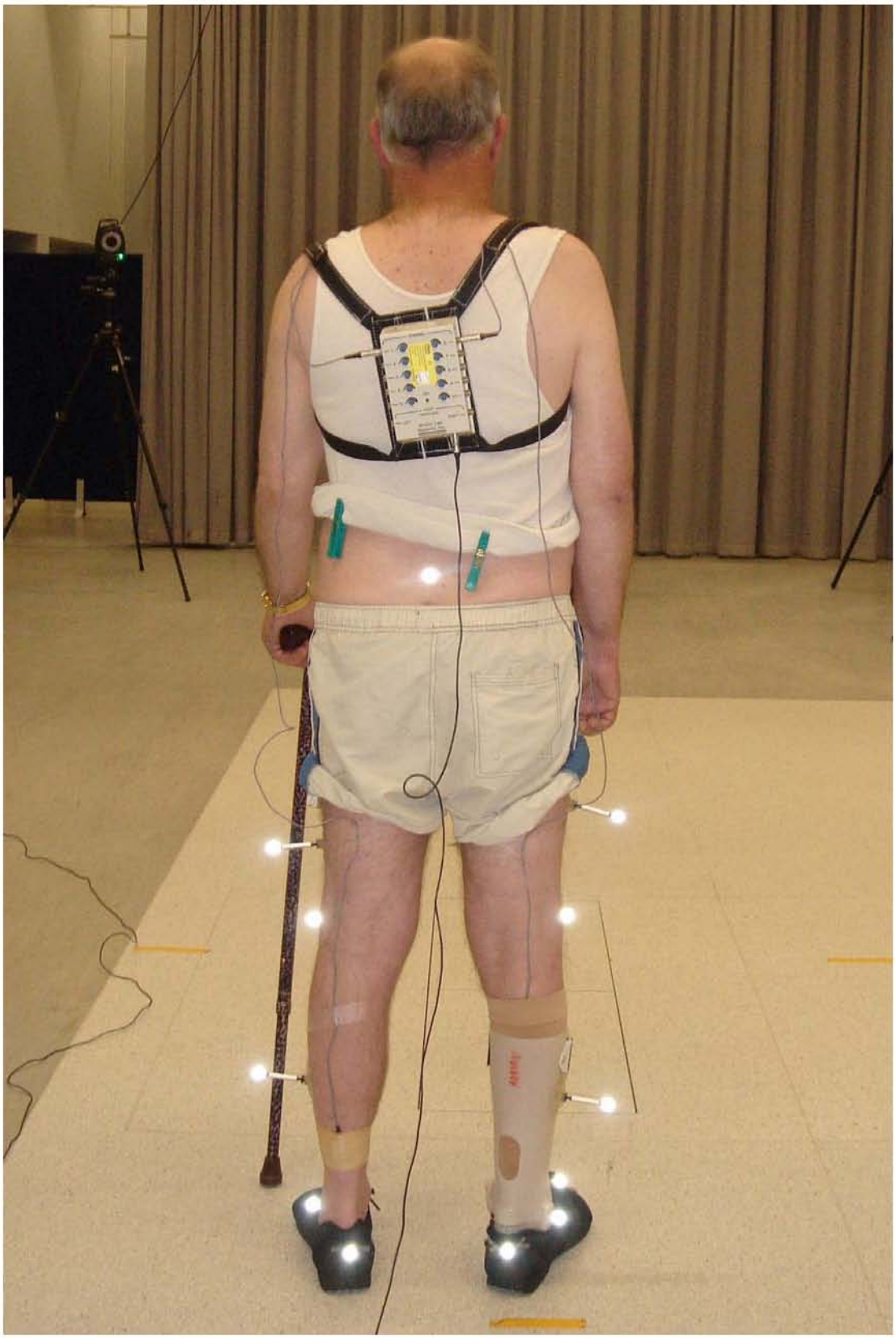

Figure 7.3 A subject prepared for testing.

Where orthosis componentry interfered with placement of markers directly onto the skin, markers were attached to the orthosis directly over the bony landmarks (Figure 7.3). The ankle marker was placed over the proximal screw of the orthosis ankle joint as has been done by previous investigators (Fatone *et al.*, 2009b). Between the conditions, the markers that required reattaching were the toe, heel, ankle and leg markers on the affected foot, and the heel and toe markers on the unaffected foot. For consistency, the same person placed all markers on all subjects.

EMG recording electrodes were placed over the soleus muscles of both legs. The method for determining the position for the recording electrodes was Hugon's method (Hugon, 1973b) which is the same method that was used to place the recording electrodes for the previous studies (see section 3.2.3.1). The EMG electrodes were worn under the orthoses and therefore, did not need to be moved throughout the testing session.

7.2.3.2 Testing procedures

All subjects were required to verbally confirm that their AFO and TRAFO were comfortable to walk in before testing could commence. Any areas of discomfort or issues with the fitting of the orthoses were rectified before the testing session began. Subjects were allowed to use their own gait aids during the testing befitting their ambulatory status and confidence. The use of gait aids was kept constant across all conditions if subjects chose to use them. Subjects were given time to accommodate to the testing environment and to practice walking over the two force platforms along a 6 metre walkway in the capture area. According to Abe *et al.* (2009), a 5 metre long walkway is sufficient for patients with hemiplegia. Some subjects were unable to strike the force platforms in sequence due to a "step-to" gait pattern or insufficient step length, so they were instructed to walk along one side of the force platforms and to strike both force platforms with the same foot (Figure 7.4). It also appeared that walking along the side of the force platforms and striking them with the same foot was easier to comprehend and perform for some subjects who had cognitive difficulties.

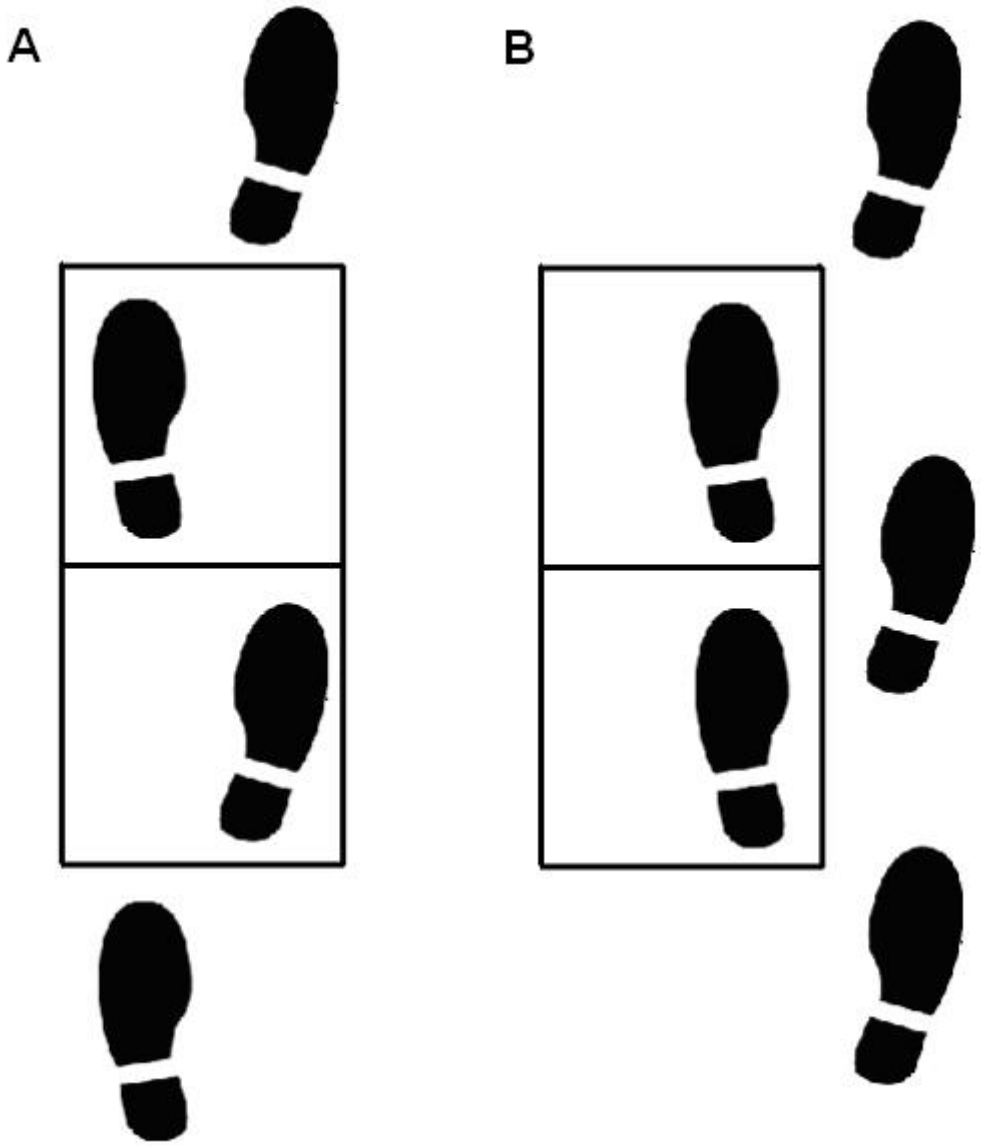

Figure 7.4 Walking patterns over the force platforms.
(A) Down the centre of the force platforms striking one foot on each force plate.
(B) Along the side of the force platforms striking the same foot on each force plate.

Once subjects were comfortable with the testing procedures, a static trial of 3 seconds was recorded prior to each condition. Subjects were then instructed to perform a number of walking trials at their own comfortable walking speed. Subjects were allowed to rest as required between the trials. The study adopted an ABC design where three conditions were tested in random order by pulling pieces of paper with the conditions written on them out of a box:

A - Shoes;

B - AFO; and

C – TRAFO.

An OCG was worn under the TRAFO to increase the speculated tone-reducing effect. If it was considered a risk to a subject's safety for them to walk without an orthosis, the shoes condition was not performed. During the AFO and TRAFO conditions, full length sheets of EVA were worn in the post-op shoe of the unaffected leg to counterbalance the thickness of the orthosis footplates.

Trials were collected until three clean force platform strikes were recorded for each foot. A clean strike was defined as a single foot in contact with the force platform and entirely within the force platform boundaries. The total number of trials recorded for each subject varied depending upon the number of trials deemed successful and unsuccessful.

7.2.4 Statistical analyses

Polygon software (Vicon Motion Systems, Oxford, UK, OX2 0JB) was used to analyse kinematic, GRF and EMG data. For each trial, ensemble averages of temporo-spatial, kinematic and EMG data were computed from as many complete steps as possible in each trial. Time was expressed as a percentage of cycle time and averages were obtained at 2% intervals throughout the cycle. For each subject, three trials of each condition were then averaged.

The dependent variables measured for temporo-spatial parameters included cadence (steps/min), velocity (m/sec), total double support time (sec) and stride length (m). Step length (m) and single support time (sec) were recorded for both the affected and unaffected limbs. The first and second peak vertical GRFs for both the affected and

unaffected legs were recorded. The kinematic dependent variables included the maximum angles of ankle dorsiflexion and plantarflexion, knee flexion and extension and hip flexion and extension over the whole gait cycle.

The dependent variables measured for soleus muscle activity were the peak EMG (highest point on the curve) and average EMG (the mean value of all of the data points of the EMG curve) for each condition. A 200 μV calibration signal (cal) was recorded separately for each subject at the end of each testing period in order to calibrate the recorded signals at the skin surface. All of the EMG values (in computer units, cu) were converted to microvolts using the following formula:

$$EMG\mu V = \frac{EMG(cu)x200\mu V}{cal(cu)}$$

Repeated measures one-way ANOVAs were used to test for differences between the three conditions (shoes, AFO and TRAFO) for each of the dependent variables. All statistical tests were conducted using SPSS and significance was set at α=0.05. There are no proper post hoc tests for repeated measures ANOVAs in SPSS. Therefore, any significant ANOVA tests were analysed with the paired *t*-test procedure (that is provided for repeated measures ANOVAs in SPSS) to determine which pair-wise conditions were significantly different. A Bonferroni correction was applied to the post hoc tests and significance for these tests was set at α=0.05.

Due to the lack of homogeneity in the subject group, two-way ANOVAs were also performed to analyse the data based on whether subjects were experienced AFO users, to determine whether subjects responded differently to the conditions based on previous AFO use. This was done as it appeared that some subjects improved in their walking ability with the orthosis conditions while others deteriorated in their walking ability with the orthosis conditions. Eight subjects were experienced AFO users, and nine subjects were not (Table 7.1). A summary of the demographic data for the subjects in each of the two groups is presented in Table 7.3.

Table 7.3 Summary of the demographic data for subjects in group 1 and 2 (mean ± SD).

	No.	Age (years)	Gender	Time since stroke (years)	Side of hemiplegia	Tardieu score	Gait aids	Sensory deficit	Modified functional walking category
Group 1 AFO users	8	55.38 ± 7.65	Males (4) Females (4)	9.38 ± 7.93	Right (4) Left (4)	2.5 ± 0.53	Yes (8) No (0)	Yes (4) No (4)	3.5 ± 0.53
Group 2 Non-AFO users	9	60.67 ± 9.81	Males (5) Females (4)	10.33 ± 10.63	Right (7) Left (2)	2.66 ± 0.71	Yes (6) No (4)	Yes (5) No (4)	4.33 ± 1.22

7.3 Results

Two subjects (subject 4 and 7) were unable to walk confidently without an orthosis and therefore, did not complete the shoes condition. Thirteen subjects were tested while using gait aids; 11 used a single point stick, one used a forearm crutch and one used two forearm crutches. Only two subjects (subject 3 and 11) could walk over the force platforms with one foot striking each platform (Figure 7.4A). The rest of the subjects walked along the side of the force platforms striking both platforms with the same foot. The EMG for subject 12 could not be used as there appeared to be a problem with the instrumentation which affected the recording of the data. This was only noticed after the testing session when the data were being analysed.

When all of the subjects' data were analysed as a group, there were no significant differences in temporo-spatial variables between any of the conditions (Table 7.4). Analysis of the kinematic data revealed that maximum ankle plantarflexion angles of the affected legs were the only angles to be significantly affected by the orthosis conditions (F=14.91, df=2.00, p=0.00). Post hoc comparisons revealed significant differences between the shoe and AFO conditions and the shoe and TRAFO conditions. The AFO condition reduced plantarflexion angles from an average of -2.24º (SD 5.77º) for the shoe condition to 2.42º (SD 3.54º) of dorsiflexion (p=0.01). The TRAFO condition reduced plantarflexion angles to 3.18º (SD 3.74º) of dorsiflexion (p=0.01). The two-way ANOVA revealed that there were no significant condition by group interactions. The only significant group effects were for affected knee flexion angles (p=0.03) (Table 7.5).

Table 7.4 Mean dependent variables for the three conditions, including results of the one-way ANOVA (mean ± SD).

Dependent Variables	Shoes	AFO	TRAFO	One-way ANOVA
Cadence (steps/min)	86.13 ± 20.84	82.78 ± 20.44	83.35 ± 20.44	F=1.44, df=1.43, p=0.25
Velocity (m/s)	0.63 ± 0.29	0.59 ± 0.31	0.59 ± 0.30	F=0.19, df=2.00, p=0.83
Double support (s)	0.54 ± 0.22	0.60 ± 0.27	0.60 ± 0.27	F=2.45, df=2.00, p=0.10
Stride length (m)	0.85 ± 0.20	0.82 ± 0.23	0.81 ± 0.24	F=0.39, df=2.00, p=0.68
	Affected			
Step length (m)	0.45 ± 0.10	0.43 ± 0.12	0.44 ± 0.13	F=2.71, df=1.46, p=0.10
Single support (s)	0.38 ± 0.06	0.38 ± 0.08	0.39 ± 0.07	F=0.70, df=2.00, p=0.51
GRF 1st peak	9.11 ± 0.98	8.99 ± 0.79	9.15 ± 0.84	F=0.62, df=1.36, p=0.49
GRF 2nd peak	9.09 ± 0.82	9.03 ± 0.69	10.01 ± 0.69	F=0.76, df=1.29, p=0.43
Ankle dorsiflexion	12.78 ± 5.68	12.32 ± 5.51	13.12 ± 6.03	F=1.44, df=1.43, p=0.26
Ankle plantarflexion	-2.24 ± 5.77	2.42 ± 3.54	3.18 ± 3.74	F=14.91, df=2.00, p=0.00*
Knee flexion	33.88 ± 15.05	32.68 ± 15.70	33.62 ± 14.86	F=1.61, df=2.00, p=0.22
Knee extension	-3.15 ± 10.14	-1.26 ± 8.39	-1.07 ± 8.22	F=1.87, df=1.33, p=0.19
Hip flexion	32.98 ± 10.63	30.99 ± 10.17	33.64 ± 9.63	F=1.34, df=2.00, p=0.28
Hip extension	0.49 ± 15.03	0.43 ± 12.58	2.62 ± 12.90	F=0.24, df=1.22, p=0.68
EMG peak (μV)	72.91 ± 33.81	68.49 ± 22.18	62.79 ± 21.50	F=1.86, df=1.41, p=.019
EMG average (μV)	30.52 ± 10.82	31.61 ± 8.89	29.60 ± 8.35	F=0.36, df=2.00, p=0.70

Table 7.4 Continued.

Dependent Variables	Shoes	AFO	TRAFO	One-way ANOVA
			Unaffected	
Step length (m)	0.40 ± 0.12	0.39 ± 0.13	0.38 ± 0.13	F=0.92, df=2.00, p=0.41
Single support (s)	0.56 ± 0.14	0.56 ± 0.11	0.55 ± 0.12	F=0.45, df=1.26, p=0.56
GRF 1st peak	9.15 ± 0.87	9.36 ± 0.64	10.01 ± 0.72	F=2.83, df=1.27, p=0.10
GRF 2nd peak	9.96 ± 0.57	9.92 ± 0.60	10.01 ± 0.65	F=0.50, df=2.00, p=0.61
Ankle dorsiflexion	19.06 ± 4.65	18.87 ± 3.78	18.19 ± 5.11	F=0.26, df=2.00, p=0.77
Ankle plantarflexion	-4.32 ± 5.67	-2.49 ± 6.14	-3.36 ± 5.98	F=1.07, df=2.00, p=0.36
Knee flexion	60.23 ± 9.52	58.81 ± 9.90	58.10 ± 8.12	F=1.69, df=2.00, p=0.20
Knee extension	2.15 ± 6.94	2.41 ± 6.27	3.15 ± 5.91	F=0.53, df=2.00, p=0.59
Hip flexion	40.30 ± 9.60	39.13 ± 9.83	38.57 ± 9.17	F=1.24, df=1.46, p=0.30
Hip extension	-5.66 ± 8.88	-5.14 ± 7.92	-4.56 ± 8.13	F=0.18, df=2.00, p=0.84
EMG peak (μV)	90.21 ± 23.68	87.33 ± 22.12	84.50 ± 21.63	F=0.86, df=1.28, p=0.40
EMG average (μV)	40.60 ± 11.87	39.47 ± 9.96	38.19 ± 8.47	F=2.00, df=2.00, p=0.16

Joint angles are all recorded in degrees and negative values represent degrees in extension.
* significant at p<0.05

Table 7.5 Mean dependent variables for the three conditions according to groups, including results of the group effect (mean ± SD).

Dependent Variables	Shoes		AFO		TRAFO		Difference between groups
Group	**1**	**2**	**1**	**2**	**1**	**2**	***p*-value**
Cadence (steps/min)	82.20 ± 15.25	88.74 ± 24.40	81.30 ± 13.09	84.10 ± 26.10	79.86 ± 12.75	85.84 ± 25.96	0.73
Velocity (m/s)	0.51 ± 0.12	0.71 ± 0.35	0.48 ± 0.16	0.69 ± 0.38	0.46 ± 0.14	0.70 ± 0.37	0.27
Double support (s)	0.57 ± 0.20	0.52 ± 0.24	0.59 ± 0.16	0.60 ± 0.35	0.63 ± 0.17	0.59 ± 0.34	0.96
Stride length (m)	0.74 ± 0.09	0.92 ± 0.22	0.70 ± 0.17	0.93 ± 0.24	0.69 ± 0.16	0.93 ± 0.24	0.11
				Affected			
Step length (m)	0.40 ± 0.04	0.48 ± 0.12	0.38 ± 0.08	0.49 ± 0.12	0.37 ± 0.10	0.50 ± 0.12	0.14
Single support (s)	0.37 ± 0.06	0.39 0.06	0.34 ± 0.05	0.42 ± 0.09	0.35 ± 0.05	0.41 ± 0.08	0.21
GRF 1st peak	8.78 ± 1.05	9.32 ± 0.93	8.82 ± 0.63	9.15 ± 0.91	8.81 ± 0.67	9.02 ± 1.00	0.49
GRF 2nd peak	8.82 ± 0.87	9.27 ± 0.78	8.80.± 0.45	9.23 ± 0.82	8.86 ± 0.70	9.08 ± 0.70	0.24
Ankle dorsiflexion	9.83 ± 5.48	14.75 ± 5.18	10.62 ± 5.71	13.82 ± 5.18	10.83 ± 7.18	15.02 ± 4.27	0.31
Ankle plantarflexion	-3.08 ± 7.07	-1.68 ± 5.11	2.14 ± 3.13	2.67 ± 4.04	2.83 4.92	3.86 ± 2.50	0.99
Knee flexion	24.54 ± 10.75	40.11 ± 14.66	23.00 ± 8.98	41.29 ± 15.66	23.92 ± 7.32	42.16 ± 14.79	0.03*
Knee extension	-7.24 ± 9.02	-0.43 ± 10.40	-3.23 ± 8.57	0.50 ± 8.32	-2.42 ± 7.84	0.06 ± 8.83	0.30
Hip flexion	33.12 ± 9.49	32.89 ± 11.90	30.30 ± 10.57	31.61 ± 10.40	30.85 ± 10.07	31.08 ± 9.83	0.82
Hip extension	3.91 ± 18.52	-1.79 ± 12.91	4.08 ± 15.46	-2.81 ± 9.08	4.92 ± 15.71	-3.08 ± 9.11	0.37
EMG peak (μV)	77.52 ± 48.56	69.46 ± 20.17	72.85 ± 23.72	64.14 ± 21.17	60.75 ± 23.39	64.82 ± 20.84	0.71
EMG average (μV)	27.95 ± 15.08	31.30 ± 10.66	32.39 ± 9.71	30.83 ± 8.59	28.04 ± 9.34	31.15 ± 7.53	0.74

Table 7.5 Continued.

Dependent Variables	Shoes		AFO		TRAFO		Difference between groups
Group	**1**	**2**	**1**	**2**	**1**	**2**	***p*-value**
				Unaffected			
Step length (m)	0.34 ± 0.12	0.44 ± 0.11	0.33 ± 0.13	0.44 ± 0.12	0.32 ± 0.12	0.43 ± 0.13	0.14
Single support (s)	0.58 ± 0.10	0.54 ± 0.17	0.58 ± 0.09	0.55 ± 0.13	0.56 ± 0.09	0.54 ± 0.15	0.74
GRF 1st peak	8.93 ± 0.64	9.29 ± 1.00	9.21 ± 0.42	9.49 ± 0.80	9.16 ± 0.50	9.38 ± 0.89	0.46
GRF 2nd peak	9.73 ± 0.22	10.11 ± 0.69	9.65 ± 0.41	10.16 ± 0.67	9.62 ± 0.36	10.20 ± 0.74	0.20
Ankle dorsiflexion	21.64 ± 4.59	17.34 ± 4.05	20.20 ± 4.45	17.69 ± 2.83	19.38 ± 5.71	16.98 ± 4.55	0.08
Ankle plantarflexion	-1.10 ± 5.85	-6.47 ± 4.69	0.56 ± 5.45	-5.20.± 5.64	0.12 ± 5.23	-6.89 ± 4.63	0.05
Knee flexion	62.01 ± 7.92	59.04 ± 10.74	60.08 ± 8.05	57.68 ± 11.67	59.11 ± 6.73	57.74 ± 9.55	0.50
Knee extension	4.46 ± 6.80	0.61 ± 6.99	4.48 ± 5.72	0.58 ± 6.47	4.75 ± 5.79	0.72 ± 5.66	0.35
Hip flexion	43.88 ± 10.42	37.92 ± 8.80	41.60 ± 11.31	36.95 ± 8.34	40.61 ± 10.69	37.52 ± 7.95	0.26
Hip extension	-0.82 ± 7.41	-8.89 ± 8.62	-1.53 ± 5.93	-8.35 ± 8.36	-1.11 ± 6.46	-8.20 ± 8.31	0.11
EMG peak (μV)	97.25 ± 27.33	2.00 ± 20.83	89.59 ± 24.44	85.08 ± 20.98	81.18 ± 20.27	87.81 ± 23.80	0.59
EMG average (μV)	37.06 ± 20.70	35.41 ± 8.90	44.03 ± 9.77	34.92 ± 8.35	40.78 ± 8.80	35.59 ± 7.81	0.09

Group 1= AFO users; Group 2= Non-AFO users.
Joint angles are all recorded in degrees and negative values represent degrees in extension.
*significant at $p<0.05$.

There were no significant differences in peak EMG or average EMG between any of the conditions for the affected or unaffected soleus muscles when the data were analysed for the whole group (Table 7.4) or when the group was partitioned into AFO users and non-AFO users sub-groups (Table 7.5). Visual inspection of the EMG curves, however, suggested that the orthosis conditions may have had a notable effect in some individuals. As no return to baseline condition was tested in this study, the $R_{95\%}$ could not be calculated to determine if any individual responses were statistically significant as was done for in the analyses of reflex excitability describe in previous chapters.

Based on visual inspection of the EMG data, subject 8 appeared to have experienced the greatest benefit from the orthosis conditions with the most noticeable reduction in peak and average EMG compared to the shoe condition (Figure 7.5). The TRAFO condition appeared to be more effective than the AFO condition causing the greatest reduction in peak and average EMG of the three conditions. Interestingly, the unaffected soleus muscle for this subject appeared to have been equally affected by the TRAFO condition. Subject 7 also appeared to benefit from the TRAFO condition compared with the AFO condition with reduced peak and average EMG, however, both orthosis conditions increased EMG activity compared with the shoe condition (Figure 7.6). The unaffected leg of this subject did not appear to have been affected by either of the orthosis conditions. Subject 5 appeared to have higher EMG in the TRAFO condition with increases in the average EMG activity compared with the shoe condition and increases in both the peak and average EMG compared with the AFO condition (Figure 7.7). Both orthosis conditions reduced the peak EMG of the unaffected leg.

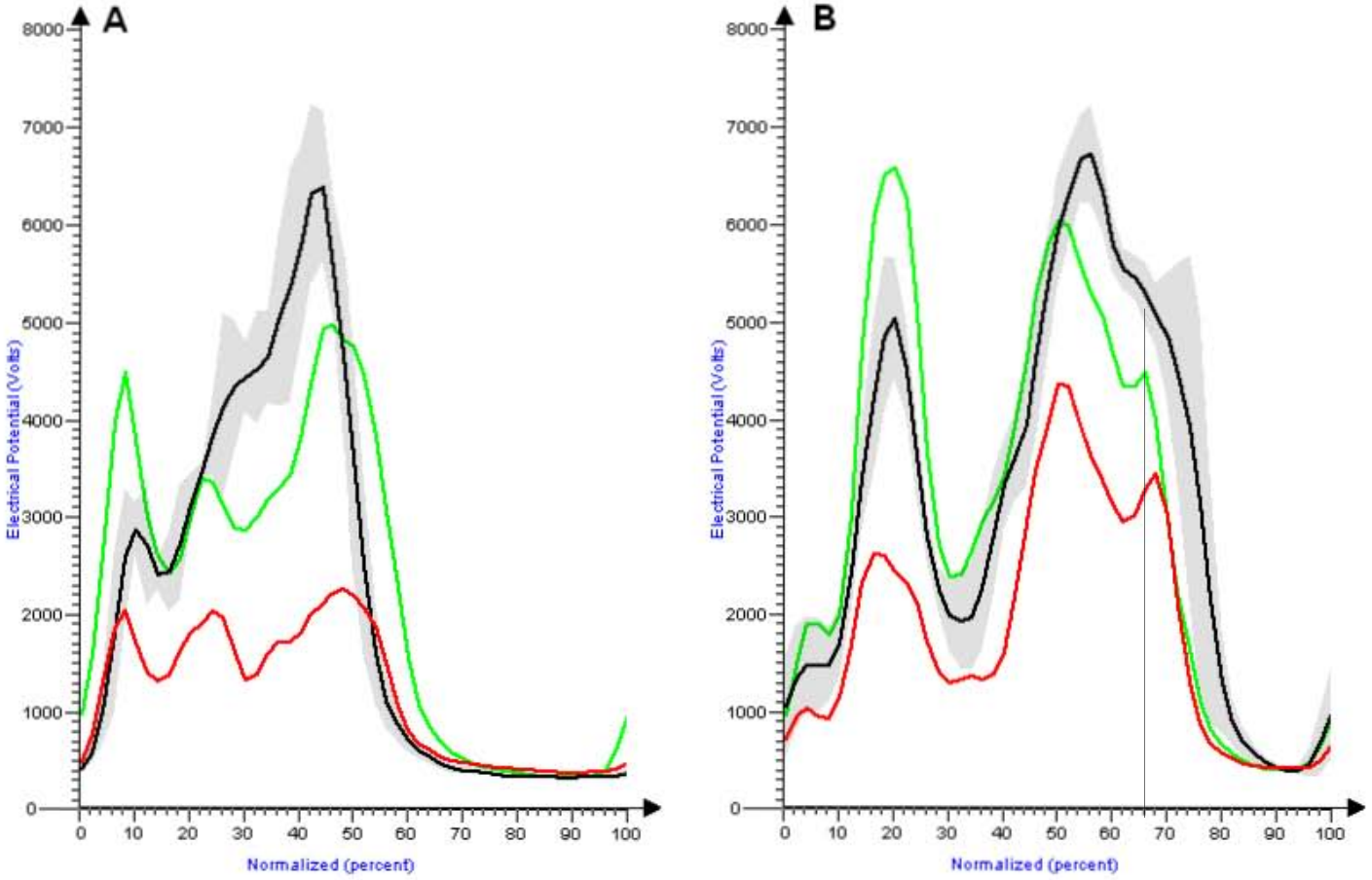

Figure 7.5 EMG curves for the affected (A) and unaffected (B) soleus muscles of subject 8.

The black line represents the shoes condition, the green line represents the AFO condition and the red line represents the TRAFO condition. The shaded area represents the SD for the shoes condition.

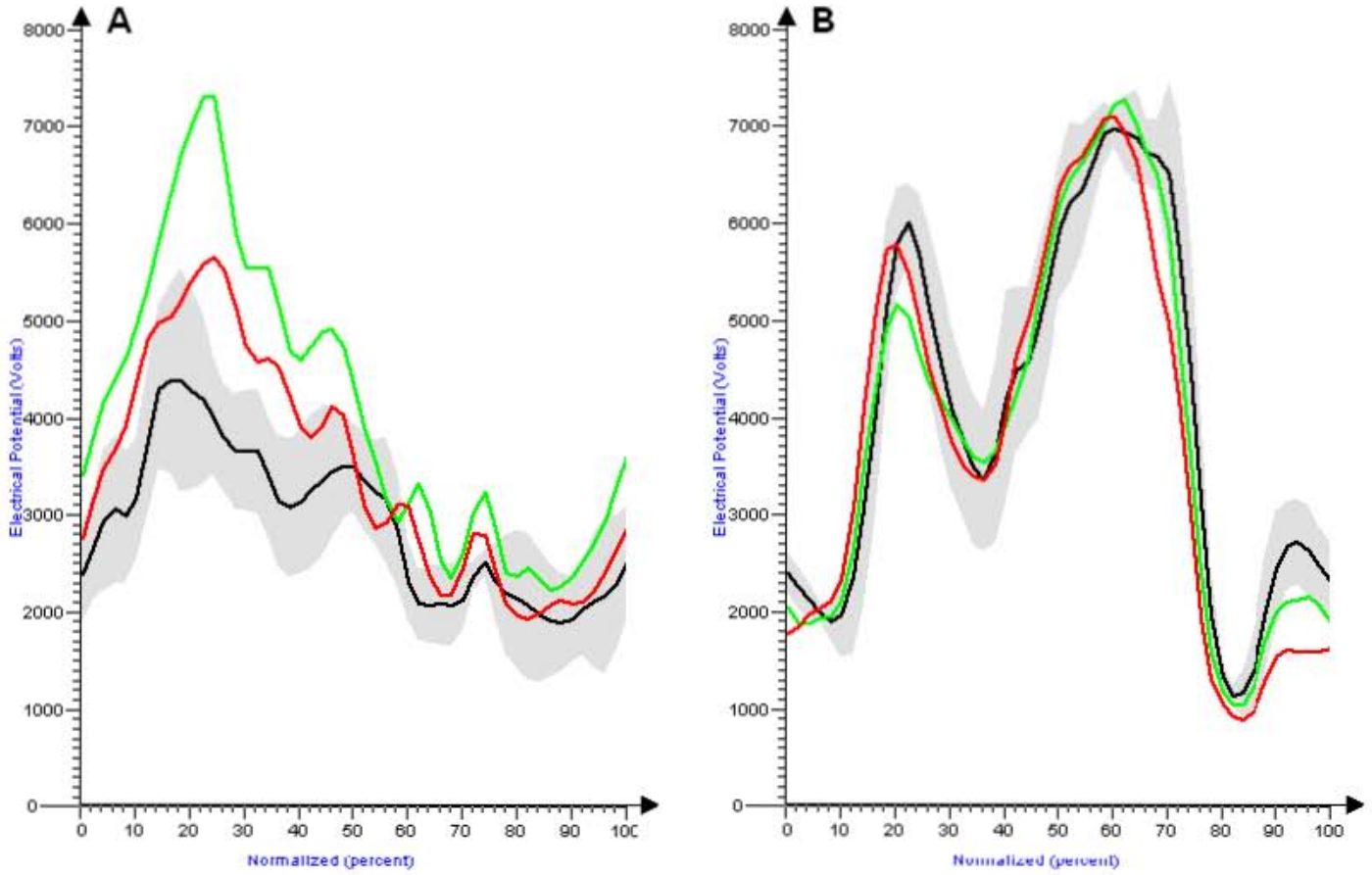

Figure 7.6 EMG curves for the affected (A) and unaffected (B) soleus muscles of subject 7.

The black line represents the shoes condition, the green line represents the AFO condition and the red line represents the TRAFO condition. The shaded area represents the SD for the shoes condition.

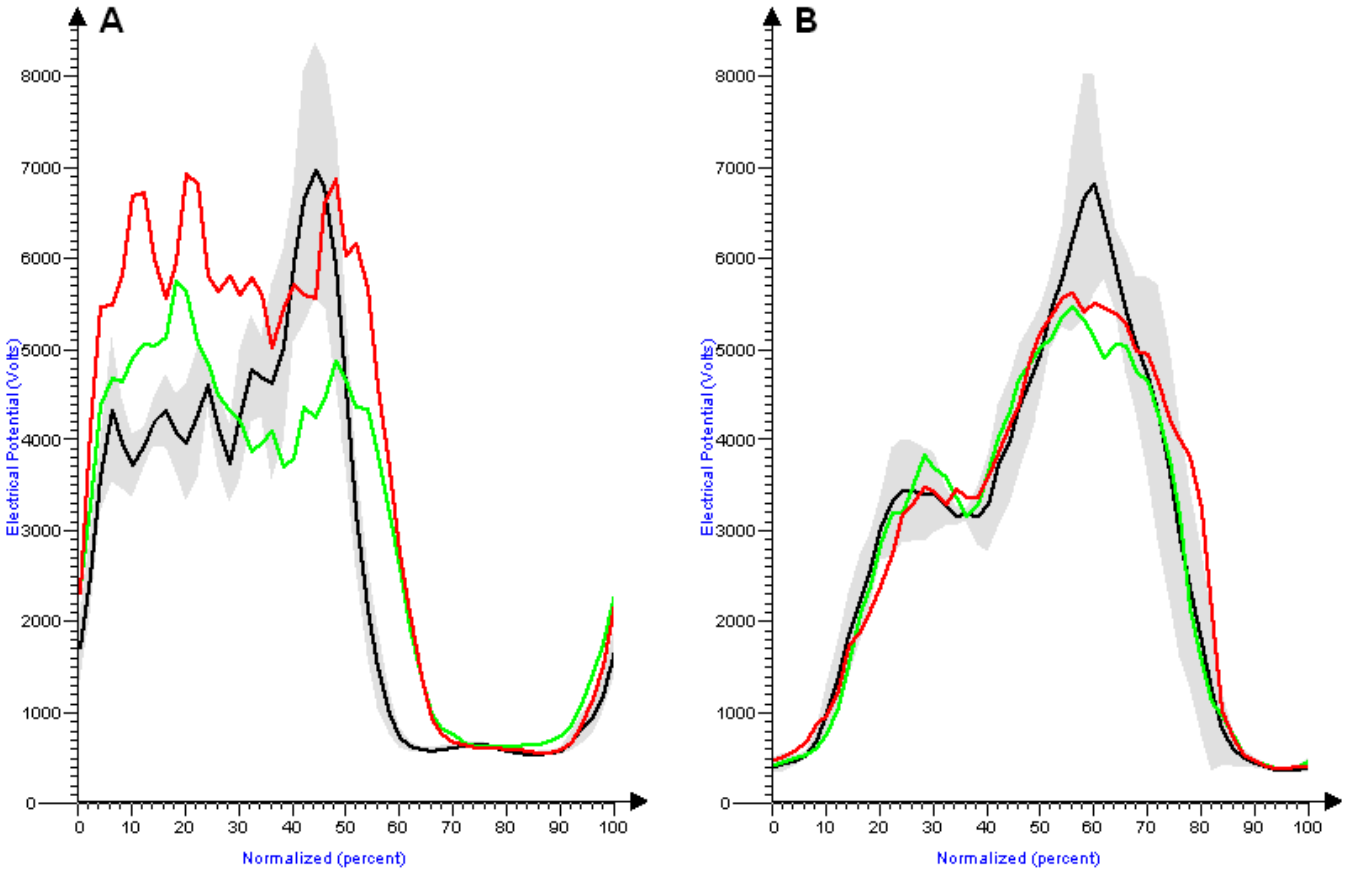

Figure 7.7 EMG curves for the affected (A) and unaffected (B) soleus muscles of subject 5.

The black line represents the shoes condition, the green line represents the AFO condition and the red line represents the TRAFO condition. The shaded area represents the SD for the shoes condition.

7.4 Discussion

The results of this study demonstrated that overall there were no significant differences in temporo-spatial gait parameters, joint angles or soleus muscle activity between the AFO and TRAFO conditions. These results suggest that TRAFOs have no significant benefits over standard AFO designs to alter gait and soleus muscle activity. These results will be discussed in further detail in sections below.

7.4.1 Gait data results

The only significant difference demonstrated between the three conditions was for affected ankle plantarflexion angles which were reduced by the two orthosis conditions compared with the shoe condition. This result is consistent with the results of previous investigations that have assessed the effects of AFOs with plantarflexion stops (Burdett *et al.*, 1988; Fatone *et al.*, 2009b; Fatone and Hansen, 2007; Hesse *et al.*, 1999). The reduced plantarflexion angles can be directly attributed to the mechanical effect of the plantarflexion stops in the AFOs and TRAFOs which did not allow plantarflexion beyond 90 degrees.

Apart from the results of the ankle plantarflexion angles of the hemiplegic leg, there were no other significant differences observed between the orthosis conditions and the shoe condition. This is not consistent with the results of previous investigations which have reported significant improvements in hemiplegic gait with the use of AFOs compared with no orthosis conditions (Abe *et al.*, 2009; Burdett *et al.*, 1988; De Wit *et al.*, 2004; Franceschini *et al.*, 2003; Gok *et al.*, 2003; Hesse *et al.*, 1999; Mojica *et al.*, 1988; Rao *et al.*, 2008; Tyson and Thornton, 2001; Tyson *et al.*, 1998; Wang *et al.*, 2007). The heterogeneity of the subject group and the large variability between individual responses may have contributed to these non-significant results. When the subjects were allocated to groups based on previous AFO use, the statistical analysis revealed that group effects were significant for affected knee flexion angles. This demonstrated that subjects who were AFO users did not flex their affected knees as much as subjects who were not AFO users (Table 7.5). Table 7.3 demonstrates that subjects in the AFO users group had a mean Modified Functional Ambulation Category of 3.5, which was lower than subjects in the non-AFO users group (mean category of 4.33). The poorer walking category of

subjects in the AFO users group and their need to wear an AFO for ambulation may explain the differences in knee joint angles between the groups.

It is interesting that there were no other significant differences observed between the two groups, however, this may have been due to the small number of subjects in each group (n= 8 and n=9). Differences between groups for unaffected ankle plantarflexion angles were close to reaching significance (p=0.05, Table 7.5) and a larger sample of subjects would likely have resulted in a significant difference for this variable. The overall lack of significant differences between AFO users and non-AFO users suggests that AFO users walk in a similar way to non-AFO users or that the individuality of the hemiplegic subjects means that analyses need to be conducted subject-by-subject due to the many other individual factors (such as gait velocity and time since stroke onset) which may cause individual differences between subjects.

Overall, the ineffectiveness of the TRAFOs compared with the otherwise identical AFOs reflects the results of previous investigations that have assessed the effects of TRAFOs while controlling the biomechanical effect of the orthoses (Crenshaw *et al.*, 2000; Iwata *et al.*, 2003). These studies suggest that the tone-reducing features of TRAFOs add no significant benefits to standard AFOs for the treatment of hemiplegic gait. Only subjects who exhibited an active toe grasp reflex were found to have benefited from the TRAFOs in the study conducted by Iwata *et al.* (2003). The subjects in the present study were not separated into groups depending on whether or not they exhibited an active toe grasp reflex as this was not an aim of the study.

7.4.2 EMG data results

The overall results of the effect of TRAFOs on soleus muscle activity are in line with those of previous investigations that also failed to find significant differences in plantarflexor muscle activity following TRAFO use (Lam *et al.*, 2005; Mills, 1984; Radtka *et al.*, 1997). However, upon inspection of individual changes, there appeared to be some individuals who possibly benefited from TRAFO use and exhibited decreased muscle activity compared to the AFO conditions. This is consistent with the results of

previous studies reported in this thesis that demonstrated individual responses to particular tone-reducing features.

Visual examination of individual subject EMG data demonstrated that despite the overall non-significant findings of the ANOVAs, the AFO and TRAFO conditions may have substantially altered the soleus muscle activity for some subjects. Subject 8 appeared to have experienced the greatest benefit from the TRAFO condition with reduced soleus activity compared with both the AFO and shoe conditions (Figure 7.5). Perhaps the most interesting observation was that the effect was bilateral. The reduced soleus muscle activity was not due to a reduction in gait velocity for this subject with mean velocity only reducing slightly from 0.53 ± 0.031 m/s for the shoes condition to 0.50 ± 0.011 m/s for the TRAFO condition. The reduced soleus muscle activity appeared to be a direct result of the orthosis conditions, however, the exact mechanisms behind the cause of the reduced muscle activity are unknown. This subject was also involved in the previous investigation on MN excitability (subject 6 in the previous study). Although there were no significant differences in MN excitability with the use of orthoses for this subject, there was a slight decrease in mean H:M ratios from 0.61 for the shoes condition to 0.59 for the TRAFO+OCG condition. The AFO condition caused a slight increase in the H:M ratio to 0.62, so there was no correlation between the EMG results and H:M ratio results.

There were other subjects who also appeared to have benefited from the TRAFO conditions compared to the AFO conditions (Figure 7.6), and it may be suggested that for those subjects, providing them with a TRAFO would be better than a standard AFO if one was required. On the other hand, subjects who demonstrated greater soleus muscle activity with the TRAFO condition than the AFO condition (Figure 7.7) may not be good candidates for TRAFO prescription. However, caution needs to be exercised when interpreting results based primarily on visual inspection. Future studies of this nature should investigate methods for statistically analysing data individually to determine if differences between conditions are significant for individuals. The $R_{95\%}$ could be used to do this in future studies if a return to baseline condition is recorded. A return to baseline condition was not recorded in this study to minimise the length of the testing session and the effects of subject fatigue. The need to analyse EMG data on an individual basis was not anticipated when designing this study.

7.4.3 Relationship between the results of this study with those from the previous study on MN excitability

The subjects who responded to one or more of the conditions in the previous study (Chapter Six) with significant increases in their H:M ratios were subjects 6, 10, 11 and 13. Visual examination of the gait data and soleus muscle activity for these subjects did not suggest any correlation between the results of the two studies.

For subjects who participated in both studies, changes in their H:M ratios and average EMGs between the shoe and TRAFO conditions and the AFO and TRAFO conditions were calculated. These change scores were used to determine if there was a correlation between the responses to the TRAFOs between the H:M ratios and the average EMGs. A Pearson's correlation revealed a poor correlation between the scores for changes between the shoe and TRAFO conditions (r=-0.23) and between the AFO and TRAFO conditions (r=-0.12). This suggests that there was no significant relationship between the response of the soleus MN excitability to the TRAFO condition during standing and the response of the soleus muscle activity to the TRAFO condition during gait. Therefore, the response of soleus MN excitability to a TRAFO does not appear to predict the response of soleus muscle function in walking with a TRAFO.

7.4.4 Other factors that may have affected the results

The AFO and TRAFO designs used in this study were chosen for evaluation purposes without first establishing if they were the most appropriate prescription for each individual subject. The heterogeneity of the subject group meant that the AFO design used (free dorsiflexion with a 90 degree plantarflexion stop) may not have been the most appropriate design for each individual. A solid ankle AFO or a free ankle motion AFO may have suited some subjects better. The AFO design used in this study was chosen as the most appropriate design for hemiplegic subjects with plantarflexor spasticity and it would have been the most appropriate design for the majority of subjects. For consistency and simplicity, only one design of AFO was tested.

Perhaps to accurately evaluate the biomechanical effect of TRAFOs the subject group should be selected with much stricter inclusion criteria so that only subjects with the

most likely chance of benefiting from the TRAFOs are included. These subjects are likely to have a poorer Modified Functional Walking Category (3 or less), an equinus deformity at the ankle joint preventing adequate toe clearance during the swing phase and preventing heel contact at the beginning of stance, weakness of the ankle dorsiflexor muscles, a slower walking velocity and require the use of a gait aid. A study by Kinsella and Moran (2008) conducted to identify gait patterns for stroke affected subjects suggested that subjects in their subgroup 3, termed the "slow walking and knee hyperextension group" would benefit the greatest from use of ankle-foot orthoses with plantarflexion stops.

The shortness of the accommodation period for each of the conditions in this study may have also contributed to the failure to observe significant differences between the conditions. A longer accommodation time and the provision of gait training may have resulted in greater effects of the orthosis conditions. This may be especially relevant since the majority of subjects tested in this study were chronic stroke sufferers with an average of ten years post-stroke (Table 7.1). However, the short accommodation period provided valuable information regarding the immediate effects of the TRAFOs and this is important as the tone-reducing effects of TRAFOs are said to occur immediately (Hylton, 1990a).

A study conducted by Wang *et al.* (2005) assessed the effects of prefabricated AFOs (possibly leaf-spring AFOs although not well described) on the balance and walking ability of both short duration (less than six months post-stroke) and long duration (over 12 months post-stroke) hemiplegic subjects. They found significant improvements in gait and balance of the short duration group with the use of AFOs, but there were no differences found in any of the dependent variables in the long duration group. The authors suggested that structural changes in joints such as the loss of ROM and muscle shortening, contributed to the lack of significant effects in this group. The average time since stroke for the long duration group was 2.9 years, considerably less than subjects in the present study.

It is believed that subjects with chronic spasticity exhibit more significant mechanical changes in joints and soft tissues that may not respond immediately to just one form of

antispasticity treatment (Barnes, 2001a). Previous authors have stated that reducing spasticity will not automatically improve movement or function without the addition of specific therapies and training, and this may be especially true in more chronic subjects (Boyd and Ada, 2001; Lennon, 1996). In order to achieve the most optimal outcome for a chronic hemiplegic subject, specific physiotherapy and gait retraining may be required along with the provision of orthotic devices. This is the case with other interventions such as antispasticity drugs or electrical nerve stimulation devices such as Walkaide® which are generally never administered without complimentary physical therapy (Losseff and Thompson, 1995). Whether the TRAFOs would have greater immediate effects in subjects of shorter duration following stroke onset requires further investigation.

7.4.5 Limitations

There were a number of limitations in this study and these are discussed in the following paragraphs. There were a large number of statistical tests performed which increased the chance of experiment-wise error. It was decided to maintain the alpha level at 0.05 for all of the statistical tests and not to adjust it to account for the experiment-wise error for greater exploration of the data. Based on earlier experiments described in this thesis, it seemed that the effect of the TRAFOs was minimal. Adopting a more conservative alpha level would have greatly reduced the probability of finding any differences attributable to the TRAFOs. The conclusion of the present investigation would not have changed if a more conservative alpha level had been adopted. The only significant difference observed was in the ankle plantarflexion angle between the three conditions ($p=0.00$) on the affected side, a difference which would have been significant even if a more conservative alpha level had been chosen.

The removal and reattachment of markers between the conditions may have led to discrepancies in marker placements affecting the overall results. As this was identified as a potential source of error prior to the commencement of testing, care was taken to place all markers in the same position over bony landmarks, and the same investigator placed all markers on all subjects for each condition. By taking measures to minimise the effect of marker movement, it is considered unlikely that marker movement had a significant effect on the results.

The majority of subjects involved in this study had significant gait impairments. This is evident in the number of subjects who required a walking aid as well as the number of subjects in a poorer functional walking category (Table 7.1). The requirements of walking in the motion analysis laboratory in this study may have affected the subjects' ability to walk in their normal manner. It is unknown how much attention is required for subjects with hemiplegia to walk in a motion analysis laboratory, however, the instructions that subjects had to comply with in order to achieve clean strikes on the force platforms and the increasing effect of fatigue in subjects who had difficulty achieving clean strikes may have affected the overall results. Although subjects were instructed to walk as normally as possible, subjects may have intentionally walked to achieve clean strikes in order to decrease the length of the testing session. To minimise these effects, trials where subjects appeared to have targeted the force platforms to achieve clean strikes (for example, by slowing down their gait upon approaching the force platforms) were not included for analysis. Future investigations should use force platforms that are not visible in the floor to eliminate subject's ability to target the force platforms.

7.5 Conclusion

There were no significant differences between AFOs and TRAFOs for any of the dependent variables for temporo-spatial gait parameters, kinematic measure or soleus muscle activity in subjects with chronic spasticity. Therefore, the study hypothesis was not supported. Visual inspection of the EMG data suggested that individual subjects may have responded to the conditions and some subjects possibly benefited from the TRAFO condition. The only significant difference found between the shoe conditions and the two orthosis conditions were for affected ankle plantarflexion angles. This difference could be reasonably attributed to the mechanical effect of the plantarflexion stops in the AFOs and TRAFOs.

This study has provided further evidence that TRAFOs are ineffective in improving gait and soleus muscle activity in subjects with spasticity following stroke when compared with standard AFO designs. When considering the results of the previous studies in this thesis, it may be suggested that with the exception of a few cases, TRAFOs have no significant neurophysiological effect on MN excitability and no significant effect on soleus muscle function or gait in chronic stroke subjects. These results suggest that the

time, effort and cost of incorporating tone-reducing features into AFO designs is ineffective and unnecessary.

Chapter Eight

Overall discussion

8.1 Introduction

TRAFOs have been described in the literature since the early 1980s. However, almost 30 years later it remains unknown whether or not they are effective in reducing muscle spasticity. This continued gap in knowledge is due to the limited research attention that TRAFOs have received, and the fact that the majority of TRAFO literature is comprised of studies that provide only low levels of evidence. The assessment of TRAFOs is especially difficult due to the lack of clarity regarding exactly what spasticity is and how it can best be measured. However, as knowledge of spasticity increases, the evaluation of treatments can be performed with greater understanding and accuracy.

The main purpose of this thesis was to determine the effect of TRAFOs on spasticity. To achieve this aim a series of studies were conducted to determine the neurophysiological tone-reducing effect of TRAFOs on various aspects of spasticity. Subjects with chronic spasticity secondary to stroke were the main subject group studied in this thesis and the main outcome measures were the H-reflex, three-dimensional gait analysis and soleus muscle activity. The studies in this thesis represent foundational work in building an evidence base for TRAFO use. This chapter will summarise the main findings of the studies in this thesis and discuss their limitations. The clinical implications will be presented as well as recommendations for further research into tone-reducing orthoses.

8.2 Summary of the main findings

The main hypotheses addressed in this thesis were:

1. TRAFO use results in a reduction of MN excitability of the soleus muscle in subjects with spasticity while standing;
2. TRAFO use results in improved temporo-spatial gait parameters and joint kinematics in subjects with spasticity; and
3. TRAFO use results in improved soleus muscle function in subjects with spasticity while walking.

The studies in this thesis were conducted to allow the neurophysiological tone-reducing effect of the TRAFOs to be evaluated separately from their biomechanical effect. In doing this, any differences observed in the outcome measures as a result of the TRAFOs could be attributed solely to their neurophysiological effect.

The studies conducted in Chapters Four, Five and Six all contributed to testing the first hypothesis. The main conclusion of these studies was that the TRAFOs were ineffective in altering soleus MN excitability. Therefore, the first hypothesis was not supported. No evidence of reductions in MN excitability of the soleus was found in any of the subjects with the use of TRAFOs while standing. In fact, for some subjects the opposite effect was found where MN excitability increased significantly with TRAFO use. The study conducted in Chapter Five demonstrated that for some individuals, MN excitability could be decreased significantly with the use of certain tone-reducing features. However, these results were not repeated when the tone-reducing features were incorporated into AFO designs.

The second and third hypotheses were also not supported by the study reported in Chapter Seven, which concluded that there were no significant differences in temporo-spatial gait parameters, joint kinematics or soleus muscle function with the use of TRAFOs in subjects with spasticity while walking. The soleus muscle function appeared to have improved with the use of TRAFOs for some subjects, however, these results were based only on visual inspection of the EMG curves. The number of subjects who appeared to have improved with the use of TRAFOs was very small and there were some who appeared to have increased EMG when using the TRAFOs.

The main results of the studies conducted in this thesis have demonstrated that TRAFOs had no significant neurophysiological effect on soleus MN excitability of subjects with chronic hemiplegic spasticity following stroke while standing. This suggests that TRAFOs have no immediate effects on the reflex element of spasticity. Furthermore, the results demonstrated that TRAFOs had no significant effect on temporo-spatial gait parameters, joint kinematics or soleus muscle activity in subjects with chronic hemiplegic spasticity following stroke. This suggests that TRAFOs have no significant effect on functional aspects of spasticity which are affected by both reflex and mechanical elements. These main findings indicate that TRAFOs have no immediate effect in reducing spasticity in subjects with chronic hemiplegic spasticity following stroke.

It has been acknowledged by other authors that responses to tone-reducing orthoses may vary between individuals. For this reason, the responses of individual subjects were examined to identify significant responses that may have been masked by averaging results across the sample. This examination revealed that some subjects responded to the orthosis conditions with significant changes in their MN excitability. The responses of these subjects appeared to be unpredictable and unique. In the study of individual tone-reducing devices (Chapter Five) some individuals responded to the devices and all of those subjects demonstrated significant decreases in their H:M ratios. Conversely, in the study of TRAFOs (Chapter Six), some individuals responded to the orthosis conditions and all of those subjects demonstrated significant increases in their H:M ratios. It was also observed that for some individuals, the TRAFOs appeared to have substantially altered their soleus muscle activity during walking when compared to the standard AFOs (Chapter Seven). The soleus muscle activity decreased in some subjects as a direct result of the orthosis conditions while in other subjects, it increased.

Due to the small number of subjects who responded to the orthosis conditions with changes in their H:M ratios and soleus muscle activity, it was difficult to identify factors that could explain why some subjects responded to the orthosis conditions and others did not. Examination of the demographic data for those subjects did not reveal any predicting factors. Interestingly, all of the subjects who responded to the tone-reducing devices demonstrated decreases in their H:M ratios and all of the subjects who responded to the TRAFO conditions responded with increases in their H:M ratios. While these results

suggest that there were trends in the ways that subjects responded to the tone-reducing devices and to the TRAFOs, these trends may have been coincidental. Testing with a much larger number of subjects, or testing the same subjects to determine if their responses can be reproduced would be required to determine if the trends were real or simply due to chance. There were no trends in the soleus EMG results during walking, and there were no apparent reasons for differences in EMG responses between individuals.

While these main results point to the conclusion that TRAFOs have no significant benefits for subjects with spasticity, an interesting finding that emerged from the study conducted in Chapter Seven was that the same may be said of traditional AFOs. Apart from ankle plantarflexion angles, no significant differences were observed in temporo-spatial parameters or muscle activity when subjects walked with or without an AFO. This was a surprising outcome since many investigations conducted by previous authors have found significant improvements in temporo-spatial parameters (especially walking velocity) with the use of AFOs (Abe *et al.*, 2009; Burdett *et al.*, 1988; De Wit *et al.*, 2004; Franceschini *et al.*, 2003; Gok *et al.*, 2003; Hesse *et al.*, 1999; Mojica *et al.*, 1988; Rao *et al.*, 2008; Tyson and Thornton, 2001; Tyson *et al.*, 1998; Wang *et al.*, 2007). At the same time, this was not in isolated case as there have been other investigations conducted that found non significant effects (apart from the direct effect of biomechanical influences like ankle joint movement restrictions) with the use of AFOs (Fatone, 2009a; Van Peppen, Kwakkel, Wood-Dauphinee, Hendriks, Van der Wees and Dekker, 2004). The benefit of AFOs appears to be highly dependent on the needs of the individual and therefore, outcomes need to be carefully considered. For example, an individual's needs may not be to walk faster, but to adequately clear the ground during the swing phase to decrease the risk of tripping and falling.

Lastly, it is worth noting that the range of baseline H:M ratio values was similar for both the able-bodied subjects (see Table 3.3) and the subjects with spasticity (see Table 5.4 and Table 6.2). While no explicit comparisons were made (as this was not an aim of the thesis), inspection of the baseline data revealed that the range of values was very similar between the two subject groups. This was an interesting finding as it could be reasonably assumed that subjects with spasticity have a greater level of reflex excitability (i.e. higher H:M ratios) than able-bodied subjects. There has not been any research conducted to

compare the H:M ratios of able-bodied subjects and subjects with spasticity and this warrants further research. It may be that reflex responses to natural and artificial stimulations are different, where under experimental conditions, artificial stimulations elicit similar responses in subjects regardless of any underlying pathology.

8.3 Discussion of the main findings

The main results of this thesis challenge the claims of previous authors who have suggested that TRAFOs can reduce spasticity (Iwata *et al.*, 2003; Lin *et al.*, 2000; Lohman and Goldstein, 1993; Weber, 1990), improve gait (Iwata *et al.*, 2003; Nash *et al.*, 2008; Zachazewski *et al.*, 1982) and improve function (Harris and Riffle, 1986; Taylor and Harris, 1986). The claims of these authors were largely based on the results of studies that were poorly controlled and did not allow differentiation of the neurophysiological effect from the biomechanical effect of the orthoses. The biomechanical effect of AFOs has been previously studied by numerous investigators. It is therefore important that studies examining the effect of TRAFOs examine in isolation the neurophysiological effect of the orthoses. This was the starting point for the series of studies that formed this thesis, and this played a significant role in the design of the studies for this thesis.

A previous study that was conducted in a similar manner to the studies in this thesis also failed to support the use of TRAFOs. Crenshaw *et al.* (2000) tested the effects of TRAFOs compared with otherwise identical AFOs on CP gait and found no significant effects of the TRAFOs. Taken together, the results of these studies suggest that the beneficial effects of TRAFOs claimed by previous authors were a result of the biomechanical effect of the TRAFOs rather than any neurophysiological tone-reducing effect. Furthermore, previous studies conducted on the effects of TRAFOs have failed to use measurement tools that can adequately measure the neurophysiological effect of the orthoses on spasticity. The studies in this thesis are the only studies that have used the H-reflex to measure the neurophysiological effect of TRAFOs on spasticity.

One of the themes that emerged from the results of this thesis was the individuality of hemiplegic subjects. It became clear that analysing the results as a group was insufficient as this masked some significant individual responses to the conditions. Naslund *et al*. (2005) stated that due to the wide range of clinical signs and symptoms of hemiplegic subjects, group level comparisons easily lead to non-significant findings, and this was certainly the case for some of the studies in this thesis (Chapters Five and Six). Other investigators who have assessed the effect of TRAFOs on subjects with spasticity have made similar observations finding no significant differences on a group level but the

appearance of significant differences on an individual level (Naslund *et al.*, 2005; Radtka *et al.*, 1997). These authors have concluded that TRAFOs are not a universal aid but can be beneficial for selected individuals (Naslund *et al.*, 2005).

Previous investigations on individual tone-reducing features and devices such as muscle stretch (Hwang, 2002a), tendon pressure (Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988) and circumferential pressure (Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992) have found significant decreases in MN excitability in subjects with spasticity. The results of the studies in this thesis suggest that while manipulation of inhibitive afferent inputs may have an effect on MN excitability, it may be impractical to incorporate suitable tone-reducing modifications into an AFO to achieve similar changes. In some cases, it may be that the excitatory effect of the orthoses themselves (Nishikawa and Grabiner, 1999b) override any beneficial tone-reducing effect of the TRAFOs. Studies that have demonstrated significant decreases in MN excitability in the upper limb with the use of tone-reducing wrist-hand orthoses (Pizzi *et al.*, 2005a) do not appear to be generalisable to the lower limbs.

Perhaps the only benefits of tone-reducing orthoses are in their biomechanical effects such as the application of stretch to specific muscles, redistribution of pressure under the soles of the feet and restriction of joint ranges of movement. While there is no evidence that tone-reducing features alter muscle spasticity when used in an AFO, their biomechanical effects may be important to treat the mechanical elements of spasticity. It was clear in a study on the effects of ITBs that subjects who exhibited the toe grasp reflex benefited from the ITBs more than subjects who did not exhibit the reflex (Iwata *et al.*, 2003). The ITBs may not have affected the spasticity in the toe flexors but simply improved the positioning of the toes and restricted flexion of the toe joints. This may have decreased the pain and discomfort associated with walking with the toes flexed and subsequently improved walking ability. Therefore, while tone-reducing devices do not appear to have any neurophysiological benefits, they may still serve a purpose to improve the mechanics of spastic limbs. Further research is required to determine the biomechanical effect of such devices.

8.4 Limitations

The main limitations regarding the studies conducted in this thesis were discussed in the relevant chapters, but will be summarised in further detail here. Perhaps the main limitation affecting all of the studies in this thesis was the limited number of subjects recruited for testing. Despite the large number of subjects who experience stroke each year, the resultant impairments and disabilities vary greatly between subjects, and it was difficult to recruit an homogenous sample of hemiplegic subjects. Many studies conducted on TRAFOs are single subject case studies. The authors of such studies justify their study design by stating that the individuality of hemiplegic subjects makes it reasonable and appropriate to use single subject designs (Smelt, 1989). While this justification may be valid, it greatly reduces the generalisability of the results. The numbers of subjects recruited for inclusion in the studies of this thesis, although small, were similar or greater than the numbers recruited by previous investigators. Even though the small sample reduced the statistical power, there were no trends in the results to suggest that larger samples would have altered the overall conclusions of the studies.

The first study in this thesis was a reliability study conducted on able-bodied subjects to determine the reliability of the H-reflex as a measure of reflex excitability in the prone and standing positions. Following the reliability study, the subsequent studies were all conducted on subjects with spasticity following stroke. While the reliability study found the H-reflex to be a reliable measure, it is unknown whether the reliability conducted on able-bodied subjects is equivalent to reliability conducted on subjects with spasticity. Therefore, a limitation of this thesis was that the reliability study was not conducted on subjects with spasticity.

The heterogeneity of the subject group was also a limitation of the studies and may have contributed to the overall non-significant results. The heterogeneity of the subject group was due to the broadness of the inclusion criteria. While it has been identified that hemiplegic subjects are all unique (Mojica *et al.*, 1988), stricter inclusion and exclusion criteria may have led to more significant results. However, this would also have reduced the potential subject pool, further reducing the sample size and limiting the generalisability of the results.

Despite the heterogeneity of the subject group, all of the subjects received the same TRAFO design without first establishing that it would be the best design for each individual. Perhaps only subjects with the greatest chance of benefiting from the TRAFOs should have been recruited, or each subject should have received an individually prescribed TRAFO design which would have given them the best possible outcome. Unfortunately, the literature does not identify factors which can be used to select patients who are good candidates for TRAFO management, or which TRAFO designs are suited to particular patients.

The shortness of the accommodation periods given for the conditions in the studies may not have been appropriate for the subjects tested due to the chronicity of their spasticity. It is believed that changes in muscles and soft tissues (mechanical elements) have a greater effect on the overall phenomenon of spasticity over time (Thilmann, Fellows and Garms, 1991b; Wang *et al.*, 2005). For all of the studies, subjects were only given enough time to either adopt a comfortable standing position or to become familiar with the testing environment. The short accommodations periods were used as TRAFOs are said to have immediate effects on target receptors (Hylton, 1990a). Although these studies provided valuable information regarding the immediate effects of TRAFOs on subjects with chronic spasticity, the limited accommodation periods may have contributed to the non-significant effects.

Spasticity is a phenomenon that involves many signs and symptoms involving both reflex and mechanical elements. A treatment for spasticity may affect more than one aspect of the phenomenon, but this can only be determined by the use of appropriate outcome measures. This thesis used three outcome measures: MN excitability, gait analysis and muscle activity which mainly focused on the measure of body functions. A greater number of outcome measures could have been used to determine the effects of TRAFOs on other aspects of spasticity such as joint ROM and passive stiffness. Other domains of the International Classification of Functioning, Disability and Health (ICF) related to activity and participation could also have been measured. However, it is not always possible to gather as much data as desired without tiring subjects or demanding too much of their time. Furthermore, during testing sessions, consideration of a subject's capabilities and endurance is required (Tyson *et al.*, 1998). The outcome measures used in this thesis mainly addressed body function and were appropriate for the aims and

hypotheses of the thesis. Although activity and participation were not measured, it seems unlikely that TRAFOs could affect activity and participation measures if there were no measurable effects on body function.

It has been suggested that perhaps the best measure of spasticity is in the subjective report of the person who experiences the spasticity. An additional shortcoming of this thesis was the lack of subjective measurement of the effects of the TRAFOs. Clinically, it has been said that the most important measure of the effects of an intervention, is one that measures something meaningful to the person experiencing the problem (Boyd and Ada, 2001). If the purpose of treating spasticity is to improve function and quality of life, subjective reports regarding the effects of an intervention on those outcomes may be just as important as objective clinical measures.

Participant-rated spasticity is now being recognised as an increasingly useful measure that may be better at documenting minor changes in spasticity than objective clinical measures (Adams *et al.*, 2007; Skold, 2000). The results of studies on the usefulness of self-assessments compared with objective assessments have favoured self-assessments, finding them to be more sensitive to changes in aspects relevant to daily life than objective clinical assessments (Adams *et al.*, 2007).

However, studies have also questioned the reliability of participant-rated spasticity. Skold, Levi and Seiger (1999) reported that spasticity could not be elicited by movement provocation on physical examination in 40% of patients self-reported to have spasticity following SCI. Additionally, a few cases of detectable spasticity by an assessor were noted in patients who had not reported that they experienced spasticity. This could be due to either limitations of clinical assessments, limitations of the assessor's ability to provoke and grade the spasticity, inaccurate perceptions of subject's own spasticity or inconsistencies with the definition and understanding of spasticity between the assessor and the patient (Priebe *et al.*, 1996; Skold *et al.*, 1999). Poor correlations have also been found between self-reported spasticity and clinical assessments (Priebe *et al.*, 1996). This highlights a need for further investigations into the reliability, validity and usefulness of self-reports of spasticity.

8.5 Recommendations for future research

This thesis has focused on the assessment of subjects with chronic spasticity following stroke, however, there are many other pathological causes for spasticity such has SCI and CP. Further studies should determine the effects of TRAFOs in other subject groups as it has been suggested that subjects with spasticity following stroke are different to subjects with spasticity of other causes (Galiana *et al.*, 2005), especially children with CP. The majority of literature and research on TRAFOs have been conducted on children with CP. Although an investigation on TRAFOs in children with CP found no effects of the TRAFOs (Crenshaw *et al.*, 2000), the results were only based on temporo-spatial, kinematic and kinetic gait measures. There have not been any investigations on the effects of TRAFOs on MN excitability in children with CP, and this warrants further investigation.

The results of the studies in this thesis as well as those of a previous study (Wang *et al.*, 2005) suggest that TRAFOs are ineffective in treating chronic spasticity when the mechanical elements of spasticity have caused permanent joint and soft tissue changes. It has been suggested that AFOs are effective for hemiplegic subjects of recent onset but minimally effective for hemiplegic subjects of long duration (Wang *et al.*, 2007). Some researchers believe that interventions should be initiated early when the neuromuscular system is most responsive to change (Richards and Olney, 1996) and before the soft tissues have undergone fixed mechanical changes (Brown, 1994). Further investigation is required to determine if tone-reducing orthoses would be effective in reducing spasticity during the critical period of motor recovery following stroke.

It is possible that the TRAFOs were ineffective in altering MN excitability, gait and soleus muscle activity as they were only tested in subjects with chronic spasticity, and only their immediate effects were observed. A longer accommodation period may have been warranted for the subject group tested. The addition of physical therapy and gait training to compliment the TRAFOs also warrants further investigation. This would require a much longer term follow-up period and would need to be conducted with a control group to determine the effects of the TRAFOs separately from the effects of the therapy and training.

This thesis only assessed the effects of TRAFOs on the soleus muscle, however, there are other muscles in the foot and ankle affected by spasticity that may have been affected by the TRAFOs, such as the gastrocnemius muscles and toe flexor muscles. The effect of tone-reducing orthoses on other muscles and body segments warrants further investigation. A study on the effects of tone-reducing wrist-hand orthoses found significant reductions in H:M ratios following use (Pizzi *et al.*, 2005a) suggesting that tone-reducing orthoses may have different effects on different muscles and body segments.

The H-reflex was used in three studies in this thesis to assess the effects of the TRAFOs in quiet stance. Previous studies on the effects of various interventions on reflex excitability have only assessed subjects in non-weight bearing positions (Hwang, 2002a; Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988; Nishikawa and Grabiner, 1999b; Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992; Robichaud and Brunt, 1994; Robinson *et al.*, 1982). It is important to evaluate the effect of interventions like TRAFOs in functional positions and not just positions of rest. It is also important to test TRAFOs during dynamic activities and not just static activities. While the results of the study in Chapter Seven demonstrated that TRAFOs generally do not have a significant effect on muscle activity during gait, further investigations are required to determine if there are any effects on reflex excitability while walking. Investigations of modulation of reflex activity during walking have been performed (Chalmers and Knutzen, 2000; Simonsen and Dyhre-Poulsen, 1999) but the experimental methods are relatively complex and usually require that subjects ambulate on treadmills for protracted periods. It is unlikely that stroke survivors similar to those who participated in this study would be able to endure the required protocols.

Future research in the area of tone-reducing orthoses must be designed to separate the biomechanical effect of the orthoses from their presumed neurophysiological effect. Direct measures of orthotic effects on MN excitability should be used in preference to inferences based on subjective or functional changes to determine if the tone-reducing orthoses really have a neurophysiological effect on muscle spasticity. The study designs and methodologies used in the studies in this thesis provide an example for investigators who wish to further examine the effects of tone-reducing orthoses.

8.6 Clinical implications

The evidence provided in this thesis strongly suggests that TRAFOs have no additional tone-reducing or functional benefits over standard AFO designs for subjects with chronic spasticity following stroke. For a small number of subjects, the TRAFOs significantly increased their soleus MN excitability immediately following application. Although it remains possible that TRAFO use may be beneficial for some people, the difficulty in monitoring the effects of TRAFOs in a clinical setting may mean that it is not worth the time, effort and cost to prescribe and fit TRAFOs if the outcomes cannot be predicted beforehand with some certainty.

Where a subject with spasticity requires an AFO to improve their function or walking ability, prescription should be based on the evidence available to support their biomechanical effects (Fatone, 2009a). There is no evidence in this thesis that TRAFOs should be prescribed for their presumed neurophysiological effects in decreasing spasticity. The time, effort and costs associated with fabricating and fitting TRAFOs cannot be justified based on the results of immediate effects on soleus MN excitability, temporo-spatial gait parameters, joint kinematics and soleus muscle function. If time and cost is not an issue, it seems that the only way to determine the effects of a TRAFO for an individual in the clinical setting would be to try it and carefully observe the results. This is possible as there are no serious risks associated with TRAFO use. Providing the patient with an identical AFO without any tone-reducing features and seeking the patient's feedback regarding the effects of each orthosis may be the best indicator of the effects of a TRAFO in the clinical setting.

It has long been known that subjects with hemiplegic spasticity of any cause are highly variable in their presentation. The results of the studies in this thesis have emphasised the importance of treating hemiplegic subjects as individuals. This means designing and implementing treatments on an individual basis rather than following generalised treatment programs and protocols. Wyke (1976, p318) explained this well by stating that "every spastic patient is different from every other spastic patient, therefore, any rigid categorization of therapeutic regimes intended to be applied to 'the spastic patient' is based on a naïve view of a highly complex situation".

Despite previous research demonstrating that some tone-reducing devices and features are effective in reducing MN excitability (Hwang, 2002a; Kukulka *et al.*, 1986; Kukulka *et al.*, 1985; Leone and Kukulka, 1988; Robichaud and Agostinucci, 1996; Robichaud *et al.*, 1992; Robinson *et al.*, 1982), the studies in this thesis have suggested that these effects cannot be replicated by modifications to functional AFO designs. It may also be suggested that the incorporation of tone-reducing features into AFO designs may increase the risk of skin injury with applications of high pressure or increase the risk of poor functionality as a result of altered joint positions to maintain muscle stretch. The study conducted in Chapter Six also suggested that when tone-reducing features are incorporated into AFO designs, they may actually have the opposite effect of increasing MN excitability. It has previously been suggested that the presence of an orthosis may itself induce an adverse reaction that may lead to unwanted reflex activity (Pratt, 2000). While this was not the case for every subject tested in the study reported in Chapter Six, it is certainly cause for concern.

Since there is no evidence that tone-reducing orthoses are effective in reducing spasticity, perhaps they should not be called "tone-reducing". When these orthoses were first termed "tone-reducing orthoses", there was no evidence to support this terminology. As there is increasing evidence that tone-reducing orthoses (especially for the lower limbs) are ineffective in reducing spasticity, it is incorrect and misleading to call them "tone-reducing". The orthoses may be more accurately termed by their standardised nomenclature based on the principal joints that they encompass, and specific additions can be described by their function. For example, a TRAFO with an inhibitory toe bar should be called an AFO with the addition of a toe extension bar.

8.7 Final conclusions

The main conclusions of this thesis are as follows:

1. The neurophysiological effect of TRAFOs can be evaluated separately from their biomechanical effect to determine their effect on muscle spasticity.
2. TRAFOs have no significant immediate effects on soleus MN excitability in subjects with chronic spasticity following stroke while standing.
3. TRAFOs have no significant immediate effects on temporo-spatial gait parameters, joint kinematics or soleus muscle function in subjects with chronic spasticity following stroke while walking, when compared with identical standard AFOs.
4. For a small number of individuals, tone-reducing features used on their own and in conjunction with AFOs may cause a significant change in soleus MN excitability, however, the effects appear to be unique to the individual and unpredictable.
5. For a small number of individuals, TRAFO use may cause a significant change in soleus muscle activity while walking when compared with identical standard AFOs, however, this change appears to be unique to the individual and unpredictable.

This thesis has presented evidence that overall, TRAFOs have no significant neurophysiological effect on spasticity. This challenges the opinions of previous investigators and authors who have suggested that TRAFOs are superior to standard AFO designs for people with spasticity, due to their ability to reduce muscle spasticity neurophysiologically. There was evidence in the studies of this thesis that for a small number of individuals, tone-reducing features tested on their own were effective in decreasing MN excitability. However, there were also a small number of individuals who demonstrated increased MN excitability with the use of the tone-reducing devices incorporated into AFO designs. These individual variations in the results supported observations by others that individuals with spasticity are unique and may respond to treatments in unpredictable ways.

Finally, this thesis has demonstrated a method by which the neurophysiological effect of TRAFOs can be evaluated separately from their biomechanical effect to determine their

effect on spasticity. This thesis has paved the way for further studies into the effects of TRAFOs on MN excitability assessed using the H-reflex.

Appendix A

Ibuki, A. and Bernhardt, J. (2007) What is spasticity? The discussion continues. *International Journal of Therapy and Rehabilitation* 14(9):1-4.

What is spasticity? The discussion continues

Aileen Ibuki, Julie Bernhardt

Spasticity is a term that has been used by clinicians and researcher for decades, yet there is little agreement about what it actually means. Numerous definitions for spasticity have been proposed and as knowledge of the phenomenon builds, definitions that were accepted in the past may no longer be valid.

This paper does not attempt to define spasticity but instead explores how the definitions for spasticity have evolved over time and illustrates the need for authors to be cautious when using the term.

Key words: definition, hypertonia, spasticity, upper motor neurone syndrome

Ibuki A, Bernhardt J (2007) What is spasticity? The discussion continues. Int J Ther Rehabil 14(9): 1–5

'Some believe we lack definitions for terms such as "spasticity". I believe we have too many definitions'
(Rothstein, 1990)

Most clinicians seem to have little difficulty in recognizing spasticity, yet the discussion concerning what spasticity actually is continues. Spasticity is commonly present in conditions such as stroke and cerebral palsy where it causes a diminished capacity to produce useful works with the motor system. It is a condition that requires much attention from a range of medical and health professionals, from orthopaedic and neurological surgeons to physiotherapists. Despite decades of research and investigation, spasticity remains a mysterious phenomenon of the upper motor neurone syndrome. The underlying problem is that the most fundamental question regarding spasticity remains unanswered: what is it? Much attention has been placed on this simple question, but to no avail. Indeed, a seemingly acceptable definition today may not necessarily be acceptable tomorrow.

Recently, it appears that literature regarding the definition of spasticity has turned away from trying to provide an acceptable answer to this question and a new question is being asked: does spasticity need a universally accepted definition? The purpose of this paper is to explore how definitions for spasticity have evolved and to summarize where the literature currently stands.

LITERATURE REVIEW

Early definitions

Published literature discussing spasticity emerged in the mid-20th century (Bobath and Bobath, 1950). During this period, much of the literature was concerned with tonic reflexes and, although spasticity was identified as the cause of the tonic reflexes, more attention was paid to what was seen rather than what was unseen.

Early definitions of spasticity were based on the phenomenon of increased muscle tone owing to some form of increased reflex activity. Definitions included those by Bobath and Bobath (1950):

> **'An increase in muscle tone varying from a mild degree to a state approximating to decerebrate rigidity, depending upon the seat of the lesion and the extent of co-involvement of the extrapyramidal system'**

by Levine et al (1954):

> **'An exaggeration of proprioceptive reflex function due to the absence of some normal factor which conditions such function'**

and by Wyke (1976):

> **'A pathological increase in striated muscle tone due to excessive motor unit activity- and as such, is always of neurological origin'**

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Lance's definition

During these earlier years, careless use of the term spasticity in the professional literature led to confusion. Guy Tardieu, a French neurologist, sought to clarify this confusion (Morris, 2000) and proposed a revolutionary concept of spasticity as a:

> 'velocity-dependent increase in stretch reflexes'

In 1980, James Lance drafted this concept into a working definition of spasticity, which was conceived at an international consensus meeting (Morris, 2002). That definition became known as Lance's definition (Sommerfeld et al, 2004) and states that spasticity is:

> 'a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex'
> (Lance, 1980)

In 1990, Lance reiterated this definition and added that:

> 'spasticity does not include impaired voluntary movement and an abnormal posture'

These signs may be associated with spasticity but do not help to define it (Lance, 1990).

Lance's definition has been quoted unhesitatingly by most authors until very recently. The few who chose not to use Lance's exact definition simply moved the words around but essentially the definition remained the same. For example, Gottlieb and Myklebust (1993) defined spasticity as:

> 'A motor disorder in which a failure to actively inhibit velocity sensitive stretch reflexes can lead to exaggerated muscle resistance to both externally and self-imposed muscle stretch and, consequently, to impaired voluntary movement.'

Definition misuse

It was not until approximately 20 years after the introduction of Lance's definition that a problem was recognized. Despite its definition, spasticity was being used as a collective label for many of the symptoms of the upper motor neurone syndrome. Indeed, some authors had developed a tendency to regard all stiffness as spasticity, which has since been criticized (O'Dwyer et al, 1996; Ada et al, 1998; Wood et al, 2005). It could be argued that with the adoption of an accepted definition, the term came to be used without too much thought given to what it was describing. According to Wood et al (2005), the following diverse phenomena were all placed under the label of spasticity:

- Hyperreflexia
- Hypertonia
- Rigidity
- Range of movement
- Contracture
- Associated movement
- Spasm
- Clonus.

Spasticity had evolved into a term that merely indicated a group of symptoms that required further clarification.

Reassessment of spasticity

Realization that the term 'spasticity' was being misused led to scrutiny of Lance's definition in particular. Subsequent advances in understanding of the phenomena put the widely accepted definition under heavy critique. One paper in particular challenged it almost word by word stating that Lance's definition is 'narrow and limiting' because it was never fully validated (Pandyan et al, 2005).

Pandyan et al (2005) concluded that Lance's definition was inaccurate for the following reasons:

- Spasticity is not a pure motor disease
- Spasticity does not exclusively result from hyperexcitability of the stretch reflex
- Changes in the resistance to imposed passive movement cannot be uniquely related to muscle activity
- The phenomenon of velocity dependence is not exclusive to stretch reflex hyperexcitability.

These conclusions were based on key findings by researchers such as Salazar-Torres et al (2004) who conducted a study assessing the stretch reflex excitability of the biceps brachii in 14 stroke patients with elbow spasticity compared with 17 able-bodied volunteers. A biomechanical device to elicit the stretch reflex in the biceps brachii via a controlled step perturbation was used to characterize the behavior of the stretch reflex in the two subject groups. Results showed that the stretch reflex amplitude was significantly lower in the stroke group than the able-bodied group. This finding is contrary to what would be expected according to Lance's definition of spasticity.

Research furthers the debate

It has also become evident that spasticity should not be treated as a pure motor disorder. Activity in other afferent pathways may also contribute to spasticity. Alterations in spasticity following treatments that involve cutaneous pathways suggest that they have a role to play in spasticity. Such treatments include:

- Dynamic foot plates (Hylton, 1990)
- Serial plaster casts (Hinderer et al, 1988)
- Toe-reducing orthoses (Zachazewski et al, 1982)
- Lycra garments (Gracies et al, 2000)
- Transcutaneous electrical stimulation (Dewald and Given, 1994).

Several authors such as Sommerfeld et al (2004) have identified that the main problem with Lance's definition is that it does not mention anything about the contribution of intrinsic muscle and soft tissue changes to spasticity (Sommerfeld et al, 2004). Traditionally, the increased resistance encountered during passive lengthening at different velocities has been considered to result principally from inappropriate muscle activation associated with hyperexcitable tonic stretch reflexes. Over the last two decades, researchers have determined that non-reflex factors also contribute significantly to spasticity (Sinkjaer et al, 1993; Malouin et al, *1997; Singer et al, 2001). There have also been* several studies that have demonstrated a deformation of the length-tension curve of muscles at different velocities without any EMG evidence to suggest the influence of motorneurone activity (Dietz et al, 1981; Hufschmidt and Mauritz, 1985; Carey and Burghardt, 1993). This presents irrefutable evidence that velocity-dependent change in stiffness can be a characteristic response of the non-reflex structures.

Current definitions

The new findings that were emerging regarding spasticity highlighted the need to update the definition of spasticity. The latest definitions proposed include those by Ivanhoe and Reistetter (2004):

> 'A sensorimotor phenomenon related to the integration of the nervous system motor responses to sensory input. Although most commonly considered a velocity-dependent increase to tonic stretch, it is related to hypersensitivity of the reflex arc and changes that occur within the CNS, especially the spinal cord. Injury to the CNS results in loss of descending inhibition allowing for the clinical manifestation of abnormal impulses'

by Cramer (2004):

> 'A state of increased tone with exaggerated reflexes resulting from upper motor neurone injury. It is a condition of many contrasts'

by Burridge et al (2005):

> 'Disordered sensory motor control resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles'

by Barnes and Saulicz (2002):

> 'Simply stated, spasticity is stiffness of muscles that occurs when injury to the spinal cord or brain prevents nerve signals from reaching areas of the spinal cord that release the neurotransmitter gamma aminobutyric acid (GABA)'

by Thompson et al (2001):

> 'A cluster of changes in movement associated with alterations in the CNS due to developmental abnormality, trauma or disease'

Disappointingly, it appears that these new definitions are no better than the previous definitions that were so heavily criticized. Some authors have attempted to present a very detailed definition, e.g. Ivanhoe and Reistetter (2004):

> 'Hypertonia with one or both of the following present: Resistance to externally imposed movement that increases with increasing speed of stretch and varies with the direction of joint movement; and/or resistance to externally imposed movement increases above a threshold speed or joint angle'

Others authors play it safe and present a definition that is somewhat vague and general (Thompson et al, 2001; Cramer, 2004). As the understanding of spasticity increases, it seems more and more difficult to condense new knowledge into a succinct statement to define the phenomenon. This may be because there is so much about the phenomenon that remains unknown, including if or when treatment is indicated and how it should be measured (Boyd and Ada, 2001).

CONCLUSIONS

In light of all that has been said, the question remains: what is spasticity? It appears that opinion is divided into two main schools of thought. Spasticity is either considered synonymous with hypertonicity or a combination of positive and negative symptoms of the upper motor neurone syndrome.

Wood (2005) suggests that a way forward could be to abolish any attempt to conceptualize spasticity as a single entity and instead to label each of the individual, positive symptoms of the upper motor neurone syndrome accurately. Similarly, Pandyan (2005) recommends that when using the term spasticity, clinicians and researchers should define precisely which particular aspect is being treated or studied.

A precise and universally accepted definition of spasticity remains elusive. However, this should not hinder the quality of research into the symptoms of upper motor neurone syndrome. If researchers and clinicians take the time and care to specify exactly which aspects of the upper motor neurone syndrome they are examining, confusion can be avoided. Furthermore, any definitions that are

proposed in the future should be properly validated before being universally accepted. IJTR

Conflict of interest: none

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KEY POINTS

- A universal definition for spasticity remains elusive despite much research.
- Opinion and literature appears to be divided in to two schools of thought: Spasticity is either considered synonymous with hypertonicity or a combination of positive and negative symptoms of the upper motor neurone syndrome.
- When using the term spasticity, it should always be accompanied with a definition.
- Where symptoms of the upper motor neurone syndrome are being examined, individual symptoms should be properly defined.
- The lack of an accepted definition of spasticity should not hinder the quality of research into the upper motor neurone syndrome and its symptoms.

Appendix B

Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2010) The effect of tone-reducing orthotic devices on soleus muscle reflex excitability while standing in patients with spasticity following stroke. *Prosthetics and Orthotics International* 34(1):46-57.

Prosthetics and Orthotics International
March 2010; 34(1): 46–57

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The effect of tone-reducing orthotic devices on soleus muscle reflex excitability while standing in patients with spasticity following stroke

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Abstract
Orthoses are commonly prescribed for the management of spasticity but their neurophysiologic effect on spasticity remains unsubstantiated. The purpose of this study was to investigate the effect of three tone-reducing devices (dynamic foot orthosis, muscle stretch, and orthokinetic compression garment) on soleus muscle reflex excitability while standing in patients with spasticity following stroke. A repeated-measures intervention study was conducted on 13 patients with stroke selected from a sample of convenience. A custom-made dynamic foot orthosis, a range of motion walker to stretch the soleus muscle and class 1 and class 2 orthokinetic compression garments were assessed using the ratio of maximum Hoffmann reflex amplitude to maximum M-response amplitude (Hmax:Mmax) to determine their effect on soleus muscle reflex excitability. Only 10 subjects were able to complete the testing. There were no significant treatment effects for the interventions ($F = 1.208$, $df = 3.232$, $p = 0.328$); however, when analyzed subject-by-subject, two subjects responded to the dynamic foot orthosis and one of those two subjects also responded to the class 1 orthokinetic compression garment. Overall, the results demonstrated that the tone-reducing devices had no significant effect on soleus reflex excitability suggesting that these tone-reducing orthotic devices have no significant neurophysiologic effect on spasticity.

Keywords: *Lower limb orthotics, muscle spasticity, tone-reducing, soleus muscle, stroke*

Introduction

Spasticity defined as disordered sensori-motor control, presenting as intermittent or sustained involuntary muscle activation[1] has been reported to occur in two thirds of stroke patients.[2] It may be considered one of the most physically debilitating consequences following a stroke and it has been shown that stroke patients with spasticity spend three times longer as rehabilitation inpatients than those without spasticity.[3] It is for this reason that much attention has been placed on the prevention and treatment of spasticity by both researchers and clinicians. There are a number of tone-reducing orthotic devices which have been purported to reduce spasticity;[4–8] however, their effectiveness has not been

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ISSN 0309-3646 print/ISSN 1746-1553 online © 2010 ISPO
DOI: 10.3109/03093640903476802

conclusively determined. The tone-reducing orthotic devices featured in this investigation were the dynamic foot orthosis (DFO), orthokinetic compression garment (OCG) and range of motion (ROM) walker to achieve muscle stretch.

Dynamic foot orthosis (DFO)

The DFO is used to accommodate the contours of the foot to produce an optimum weight bearing surface that is said to reduce spasticity.[9] Its development was based on the finding that stimulation or inhibition of particular reflexogenous areas on the plantar surface of the foot had an effect on the level of tone in muscles in the foot and leg.[10,11]

According to Pratt,[12] Radtka and colleagues[13] and Pitetti and Wondra[14] there are claims that DFOs have been shown to reduce spasticity, improve stability, balance and symmetry, reduce muscle imbalance, improve function in the entire body, improve motions of the lower extremity, pelvis and trunk during standing and gait, and improve proprioceptive feedback. It is not clearly explained how these effects have been demonstrated and furthermore it is uncertain how such effects are able to be produced.

Orthokinetic compression garment (OCG)

An OCG is a flexible sleeve that incorporates active and inactive fields strategically placed on a limb to facilitate or inhibit muscle activity. OCGs are said to utilize the exteroceptors of the skin and indirectly the proprioceptors of muscles and tendons to decrease spasticity.[5]

The effects of OCGs on patients with spasticity have been reported as immediate mobilization, increased muscle power and increased joint ROM.[5] These results suggest that OCGs have the ability to reduce spasticity, however, these results were taken from very outdated case studies where the authors based their results entirely on clinical observations.[5]

Since then, there has been very little literature on OCGs, but recently, a similar concept has emerged in lycra orthoses. Lycra orthoses are considered to be dynamic orthoses provided to correct deformity, improve joint stability, inhibit spasticity and encourage more normal function.[15] Lycra orthoses can be seen as the evolution of orthokinetics, however despite improvements in their design and function, evidence to support their effectiveness in reducing spasticity is still lacking.

Muscle stretch

Muscle stretch is more accurately a feature of tone-reducing orthotic devices rather than a device itself. The stretching of muscles is frequently prescribed for patients with spasticity as it is thought to have immediate tone-reducing effects on the muscle being stretched.[16]

It has been demonstrated that passively stretching the soleus muscle depresses soleus reflex excitability in healthy subjects[17,18] and subjects with spasticity.[19] Kanter et al.[18] reported reductions in Hoffmann-reflex (H-reflex) amplitudes by $62.2 \pm 12.5\%$ while Robinson et al.[20] reported reductions of $46.9 \pm 19.2\%$. This inhibitory effect on reflex excitability has been attributed to reduced excitability of intramuscular receptors as well as presynaptic inhibition.[20,21] Based on these results orthoses that apply stretch to spastic muscles should have tone-reducing effects however this has not been thoroughly assessed.

Assessing the effects of tone-reducing orthoses: The H-reflex

Previous literature concerning tone-reducing orthoses is limited and most studies have failed to adequately support their use in managing spasticity. This is predominantly because

studies have failed to differentiate between the biomechanic and neurophysiologic effects of the orthoses.[4,13,22–25] Authors of such studies have inferred that observed improvements in measures apart from spasticity such as joint ROM, posture and gait reflect a decrease in spasticity. These conclusions need to be interpreted with caution as the changes in outcome measures could simply have been due to the biomechanic effects of the orthoses alone.

Tone-reducing orthotic devices are said to stimulate specific afferent fibres which have inhibitory effects on the motoneurons in the spinal cord with which they synapse.[26] These inhibitory afferent fibres are activated via the stimulation of Golgi Tendon Organs, reflexogenous areas on the plantar surface of the foot and cutaneous receptors over target muscles.[12,26,27] As one component of spasticity is manifested in the form of excessive activation of motoneurons within spinal reflexes,[28] inhibition of these motoneurons should result in reduced spastic muscle activity.

A suitable measurement tool to assess the effect of tone-reducing orthoses is the H-reflex which quantifies the excitability of motoneurons that innervate a particular muscle.[29–32] If tone-reducing orthotic devices can effectively stimulate inhibitory afferent fibres to decrease the level of innervation of spastic muscles, then the H-reflex would be able to detect this change and give a measure of the neurophysiologic effects of the devices. The H-reflex is a valid and reliable measure[33] commonly used in neurophysiology research to assess changes in spasticity;[34] however, it has been underutilized in orthotic research.

To record the soleus H-reflex, the tibial nerve is electrically stimulated to depolarize motor and afferent fibres.[29] Stimulation of the motoneurons causes a direct muscle response (M-response) in the soleus muscle approximately 10 ms after the stimulation. Simultaneously, afferent fibres are activated causing a monosynaptic depolarization of the same motoneurons leading to a second contraction (H-reflex) approximately 35 ms later. The two responses in the soleus are recorded via electromyography (EMG) and their amplitudes are used to monitor motoneuron excitability. In order to measure the H-reflex meaningfully and reliably, it is recommended that the ratio of maximum H-reflex amplitude to maximum M-response amplitude (Hmax:Mmax) be used to eliminate the influence of variability with recording or subject physiology.[34,35] The H-reflex has been widely used in studies of people with spasticity,[36–38] however it has not been used to examine the effectiveness of tone-reducing orthoses for the lower limbs of patients with spasticity.

The purpose of this investigation was to evaluate the effect of three common tone-reducing orthotic devices on the reflex excitability of the soleus muscle in subjects with spasticity during quiet standing within a single session. The three tone-reducing orthotic devices were (1) DFO; (2) OCG; and (3) ROM walker.

Methods

Subjects

Thirteen patients who had previously suffered stroke were recruited from a convenience sample after giving informed consent. The subject characteristics can be found in Table I. Subjects were included in the study if they met the following criteria:

- Suffered a stroke at least 12 months prior to the study;
- Independent community ambulators (gait aids acceptable);
- A good current level of health;
- Spasticity in the soleus muscle of the affected side as determined by a grade equal to or greater than 1 on the Tardieu scale;[39]

Table I. Subject characteristics. L = Left, R = Right, X = quality of muscle reaction, Y = angle of muscle reaction (in plantarflexion).

Subject	Gender	Age (years)	Number of strokes	Time since most recent stroke (years)	Stroke type	Side of lesion	Tardieu Scale (X,Y)	Sensory deficit
1	Male	50	2	18	Haemorrhage	R	3,18°	No
2	Male	52	1	26	Haemorrhage	R	2,23°	Yes
3	Male	65	2	2	Ischemia	R	2,16°	Yes
4	Male	62	1	5	Haemorrhage	R	4,15°	Yes
5	Male	59	1	4	Haemorrhage	R	3,20°	No
6	Male	54	1	4	Haemorrhage	R	4,17°	No
7	Female	49	1	14	Haemorrhage	L	2,26°	Yes
8	Male	73	2	2	Unknown	L	2,10°	No
9	Male	50	5	5	Ischemia	L	4,25°	No
10	Female	60	1	8	Haemorrhage	L	2,15°	Yes
11	Female	59	1	4	Haemorrhage	L	2,24°	No
12	Female	46	1	12	Haemorrhage	L	3,12°	Yes
13	Female	63	1	11	Ischemia	R	3,10°	No
	8 Males, 5 Females	57.67	1.50	8.08	9 Haemorrhage, 3 Ischemia	6 Left, 7 Right	2.77, 17.77°	6 Yes, 7 No

- Able to dorsiflex the affected ankle joint at least 7.5° with the knee extended; and
- Able to comply with instructions and give informed consent.

Subjects were excluded if:

- They stated that their stroke had had no affect on their walking ability;
- They had never experienced muscle tightness or spasm; or
- They had other serious medical problems.

Apparatus

H-reflexes were initiated percutaneously using a Grass SD9B Stimulator (Grass Instruments Co. Quincy, MA, USA) and a custom-built stimulus amplifier (Tain Electronics, Victoria, Australia). EMG signals were measured with an EMG system (La Trobe University, Victoria, Australia 3086), amplified (1000 gain), bandpass filtered between DC and 2 KHz and monitored on a four-channel oscilloscope (Medelec Ltd. Surrey, UK). Power spectral analysis of the EMG signals indicated that almost all of the signal power was below 200 Hz. A laboratory computer system (PowerLab/410 ADInstruments, Bella Vista, NSW, Australia) was used to digitize EMG and stimulator signals. Data was sampled at 20 kHz for 0.128 sec and processed using PowerLab software (Scope version 3.3). A Cardiometrix Artifact Eliminator (Cardiometrix, Bothell, WA, USA) was used to ensure that the impedance between electrodes was below 5 kohms.

Two 10 to 1 voltage reduction converters (La Trobe University, Victoria, Australia 3086) were required to reduce EMG and stimulus signals within the range of the A/D converter on the PowerLab. A Tektronix (Tektronix, Shanghai, China) pulse generator was also used to generate a trigger pulse to start the data acquisition as the trigger pulse from the Grass stimulator was too short to initiate data acquisition directly.

Procedures

Ethics approval for the project was obtained from La Trobe University's Health Sciences Faculty Human Ethics Committee (FHEC06-045). Eligible subjects underwent a detailed assessment during which the level of impairment following stroke was determined with particular attention paid to the presence and degree of spasticity.

Orthotic devices

Measurements were taken of the subject's affected leg to determine the correct sizing of the tone-reducing devices and a partial weight-bearing foot impression was taken using a foam impression box for the fabrication of a custom-made DFO. Positive plaster molds were modified according to the instructions of Hylton[8] and vacuum molded with EVA (250 kg.m^3) to create the DFOs which were shaped to be worn within post-operative shoes (OTS, Victoria, Australia).

Venosan (Salzmann AG, St Gallen, Switzerland) below knee compression garments were used to create the OCGs. Class 1 (18–21 mmHg) and class 2 (23–32 mmHg) garments were used to ensure a standard level of compression between subjects and to assess the effect of two different levels of compression. These classes of compression were chosen as they represent standard levels used in compression therapy and are similar to those applied by lycra garments for the management of spasticity. Pieces of smooth inelastic vinyl were specifically cut from measurements of the subjects' legs to create inactive fields over the posterior half of the legs as this is said to further inhibit muscle activity.[5,6] The pieces of vinyl were fitted directly against the subjects' legs inside the compression sleeves.

ROM walkers (Ossur, Northmead, NSW, Australia) were used to maintain a constant stretch on the plantarflexor muscles by immobilizing the ankle joints at two specified angles, plantargrade (0°) and 7.5° of dorsiflexion. These angles were determined by the available range of adjustability of the ROM walkers and what might be applicable for use in functional orthoses.

The subjects wore post-operative shoes with all of the conditions to standardize footwear. Due to the thickness of the dynamic footplates and the soles of the ROM walkers, full-length internal raises were worn in the contralateral shoes to ensure that the height of the standing surfaces were the same under both legs.

Testing

In preparation for electrode placement, a small area of skin over the soleus muscle on the affected leg was shaved, abraded, and cleansed with alcohol. Surface electrodes (Ag/AgCl 10 mm disk monitoring electrodes) were placed using Hugon's method.[40] The center-to-center electrode distance was 30 mm and the ground electrode was positioned over the fibular head. The anode was positioned on the anterior thigh just proximal to the patella and the cathode was positioned in the popliteal fossa to stimulate the tibial nerve.

Subjects were given an accommodating period to become accustomed to the sensation of the percutaneous electrical stimulations in the standing position before testing began. A walking frame was positioned in front of the subjects and a chair was placed behind them. Subjects were instructed to bear equal weight through both limbs, to use the frame only as a balance aid, to remain relaxed and to fix their gaze on a target placed on a wall three meters

in front of them. For each of the testing conditions, H-reflex and M-response recruitment curves were constructed.[29] There were seven conditions tested of which the interventions were presented in random order:

1. Shoes only (baseline);
2. DFO;
3. OCG class 1;
4. OCG class 2;
5. ROM walker set at neutral (plantargrade);
6. ROM walker set at 7.5° of dorsiflexion;
7. Shoes only (baseline return).

The stimulus duration was 1 msec and the stimulus frequency was 0.2 Hz.[34] Four stimulations were recorded at each level of stimulus voltage before the voltage was increased by an increment of approximately 1.5 volts. Stimulus voltage was progressively increased in this manner until the H-reflex had become extinct and the maximum M-response amplitude had been reached. The mean trace at each level of stimulus intensity was obtained and used to graph the recruitment curve for the H-reflex and the M-response.

Statistical analysis

A one-way repeated measures Analysis of Variance (ANOVA) was used to calculate differences between the conditions. Mauchly's test of sphericity was used to determine whether distributions were normal, and if they were not, a Greenhouse-Geiser adjustment was used.[41] All statistical analyses were performed using the SPSS Statistics 17 (SPSS Inc, Chicago) and significance was set at $\alpha = 0.05$.

In addition, the 95% range for change was calculated to determine which within subjects' measures were significantly different.[42] According to Chinn[43] the 95% range of change ($R_{95\%}$) can be calculated as:

$$R_{95\%} = 1.96SD(y_1 - y_2)$$

where y_1 and y_2 are the mean Hmax:Mmax ratios for the two baseline conditions and SD is the standard deviation of the means. However, using 1.96 as the critical value for *z* assumes a large sample and is highly optimistic. For a more conservative estimate, the critical value for *t* with 9° of freedom ($t = 2.306$) was used as the multiplier. Any measures that fell outside of the range were considered to be indicative of a significant individual response to the intervention.

Results

Of the 13 subjects who were recruited for the study, the data for only 10 subjects was able to be analyzed as three subjects had adverse responses to the stimulations which would have confounded the results or compromised the safety and comfort of those subjects.

The ANOVA revealed that there were no significant differences in the Hmax:Mmax ratios between any of the conditions ($F = 1.208$, $df = 3.232$, $p = 0.328$) (Figure 1). The $R_{95\%}$ was used to further examine the data to determine whether any substantial changes within individual subjects had been masked by the averaging process. The individuality of the impairments of stroke survivors makes it reasonable to analyze the

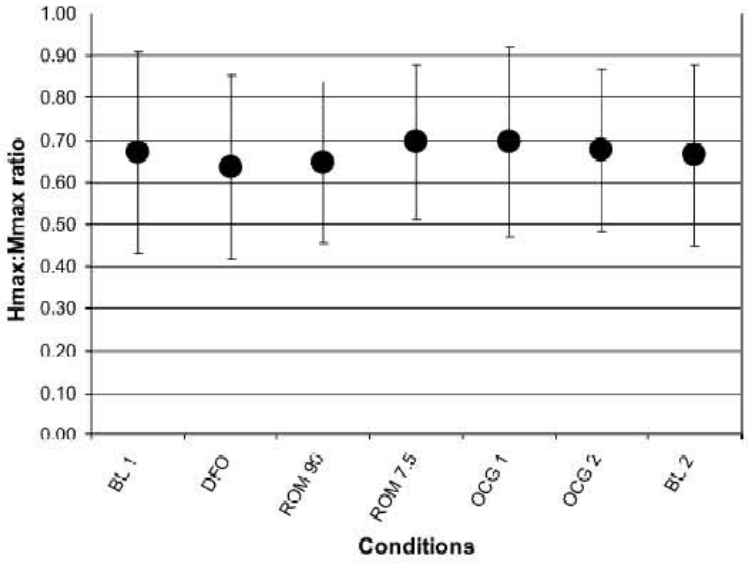

Figure 1. Mean Hmax:Mmax ratios for all subjects. BL = Baseline (shoes only), ROM 90 = ROM walker set at 90°, ROM 7.5 = ROM walker set at 7.5°, OCG1 = Orthokinetic compression garment class 1, OCG2 = Orthokinetic compression garment class 2.

Table II. Hmax:Mmax ratios for each subject across all conditions including the $R_{95\%}$ for each subject. Subject number two did not complete the final baseline condition due to fatigue. BL1 = Baseline 1 (shoes only), BL2 = Baseline 2 (shoes only), DFO = Dynamic foot orthosis, ROM 90 = Range of motion walker set at plantargrade, ROM 7.5 = Range of motion walker set at 7.5° of dorsiflexion, OCG 1 = Orthokinetic compression garment class 1, OCG 2 = Orthokinetic compression garment class 2, $R_{95\%}$ = 95% range for change. *indicates Hmax:Mmax ratios outside of the $R_{95\%}$.

Subject No.	BL 1	DFO	ROM 90	ROM 7.5	OCG 1	OCG 2	BL 2	Mean of BL 1 and BL 2	$R_{95\%}$
2	0.55	0.61	0.73	0.7	0.7	0.67	–	0.55	0.37–0.73
3	0.2	0.19	0.17	0.26	0.24	0.25	0.19	0.2	0.02–0.38
4	0.71	0.48*	0.6	0.73	0.5*	0.57	0.68	0.7	0.52–0.88
5	0.67	0.7	0.64	0.77	0.65	0.67	0.67	0.67	0.49–0.85
8	0.49	0.54	0.56	0.66	0.68	0.66	0.63	0.56	0.38–0.74
9	0.74	0.75	0.68	0.7	0.77	0.76	0.7	0.72	0.54–0.90
10	0.96	0.96	0.8	0.89	1.02	0.93	0.95	0.96	0.78–1.14
11	1.07	0.74*	0.87	0.94	0.91	0.93	0.9	0.99	0.81–1.17
12	0.65	0.67	0.7	0.61	0.67	0.69	0.64	0.65	0.47–0.83
13	0.66	0.66	0.71	0.69	0.57	0.63	0.62	0.64	0.46–0.82

data on a subject-by-subject basis. The $R_{95\%}$ interval for the Hmax:Mmax ratio was found to be 0.18. Table II includes the lower and upper bounds for the $R_{95\%}$ for each individual subject. The DFO and OCG class 1 both fell below the $R_{95\%}$ for subject number four and the DFO fell below the $R_{95\%}$ for subject number 11 (Table II).

It must be mentioned that Hmax:Mmax ratios greater than one were recorded for subjects number 10 and 11. There are two possible explanations for these greater-than-one ratios. Firstly, it is possible that the true maximum M-response was not recorded as increases in the stimulus intensity was ceased too early. Testing was terminated when there was no apparent increase in the M-response with an increase in stimulus intensity. This was done in

order to reduce subject fatigue and discomfort from further increases in stimulus voltage. Because of variability in responses from trial to trial, it is possible that, in these cases, stimulus voltage was not increased sufficiently to the true maximum. Secondly, the peak M-response amplitude may have been missed because the increments between stimulus intensities were not small enough (Figure 2). However decreasing the increment size would have greatly increased the length of the testing session and the effect of subject fatigue. The errors in estimates of the Hmax:Mmax ratios were small and did not affect the overall result.

One of the faulty Hmax:Mmax ratios was a baseline measure for subject 11 which may raise concerns regarding the calculation of the $R_{95\%}$. However, when the faulty Hmax:Mmax ratio is amended to one, the $R_{95\%}$ decreases to 0.15 and the DFO condition for subject 11 is still well outside of the $R_{95\%}$. As the discrepancies are very small we do not believe that this has affected the validity of the measurements or the results of the statistical analysis.

Discussion

The results of this study indicated that overall the tone-reducing devices were ineffective in altering soleus reflex excitability in subjects with stroke while standing. However, when the results were analyzed on an individual basis, the Hmax:Mmax ratios for subject 4 and subject 11 significantly decreased with the DFO condition and additionally for subject four the OCG class 1 condition.

Naslund et al.[44] came across similar findings when examining the effects of dynamic ankle-foot orthoses (AFOs) on children with spastic diplegia. On a group level, the dynamic AFOs appeared to be ineffective, however, when analyzed individually the results showed that some children could benefit from the orthoses.

Upon closer inspection of subjects 4 and 11, there did not appear to be any characteristics that set them apart from the rest of the subject group to explain why their Hmax:Mmax ratios

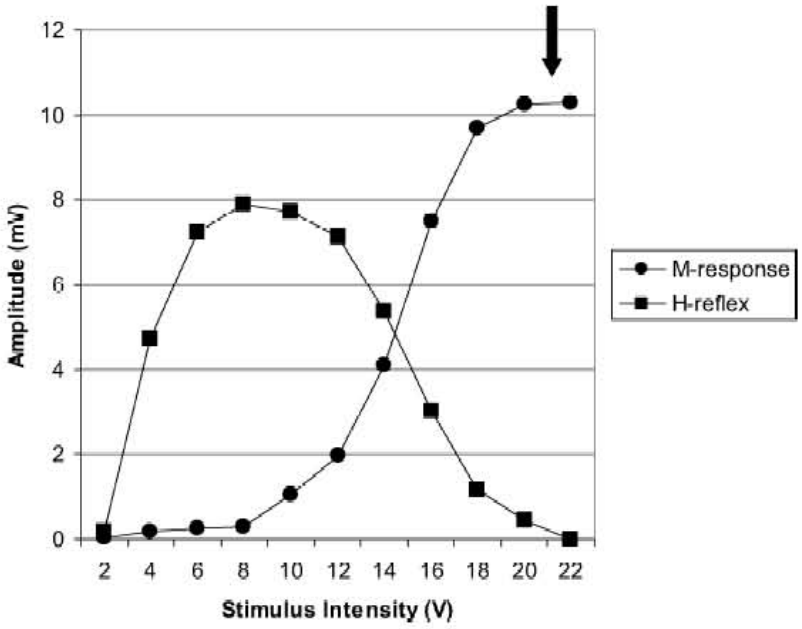

Figure 2. A typical plot of the H-reflex and M-response recruitment curves identifying the maximum M-response amplitude.

decreased (Table I). Lack of solid evidence-based literature on tone-reducing orthotic devices also makes it difficult to determine why the DFO and OCG class 1 reduced their reflex excitability over the other devices. However, this may reinforce the idea that individuals with spasticity vary in their responses to treatment.

The overall ineffectiveness of the devices challenges the claims of previous authors who have supported the use of tone-reducing devices to reduce spasticity.[14] Questions may arise regarding the specific design and choice of tone-reducing devices used in this study. Regarding the DFOs, their design and construction carefully followed detailed instructions in descriptive literature[7,8,12] and therefore poor orthosis design is unlikely to be a contributing factor. It is possible that the DFOs may be more effective during a dynamic activity such as walking and this warrants further investigation.

OCGs are said to have an inhibitory effect on reflex excitability[6] presumably through the stimulation of cutaneous mechanoreceptors. One of the only experimental studies published found that the use of OCGs on the upper limbs of 20 subjects with spastic hemiplegia following stroke resulted in improved neuromuscular function assessed through postural carriage, reaction speed, muscle strength and active ROM.[45] The authors provided no explanation for how the OCGs were able to improve function but suggested that they improve exteroceptive and proprioceptive facilitation.

Experimental studies on the effects of lycra orthoses have found varied results. Corn et al.[46] assessed lycra orthoses on the upper limbs of four children with spasticity. One child experienced a decline in the quality of his upper limb movements, another child showed initial improvements that were not maintained over time and the other two children showed no significant changes. The authors gave little explanation for their non-significant findings except to suggest that the effects of lycra orthoses are variable and individual. On the other hand, studies conducted by Gracies et al.[47,48] found that upper limb lycra orthoses were effective in reducing wrist and finger flexor spasticity as assessed by the Tardieu scale.[48] The authors of these studies attributed the improvements to the tonic stretch applied by the garments rather than cutaneous stimulation. Although these studies have tested the lycra orthoses on upper limbs, their application and effectiveness should be similar for the lower limbs.

One of the main components of OCGs is the compression applied to limbs. It has been demonstrated that circumferential pressure applied to the leg decreases the soleus H-reflex in able-bodied and symptomatic subjects.[37,49] The pressures applied to the leg in these studies ranged from 36.7–40.8 mmHg which is greater than what was applied in the present study giving a possible reason for their overall ineffectiveness. However, there is no information about the optimum level of circumferential pressure for reducing spasticity in the leg. Class 1 and 2 compression socks were used. According to standards for compression socks,[50] compression up to 32 mmHg should have been achieved although direct pressure measurements were not made in this study. The results of this current study suggest that OCGs that apply less than 32 mmHg do not have a significant neurophysiologic effect on muscle tone addressing a need to review current compression therapy for the management of spasticity.

The amount of muscle stretch utilized in this study can be considered quite minimal in comparison to the amount of stretch applied by other investigators. Kanter et al.[18] tested soleus reflex excitability with the soleus stretched to the full range of dorsiflexion motion and Hwang[17] stretched the soleus muscle with the ankle in 20° of dorsiflexion. However, Burke et al.[19] and Robinson et al.[20] demonstrated that inhibition of the H-reflex could be shown with dorsiflexion movement as small as 4–5°. Therefore, the dorsiflexion angle of 7.5° that was used in the present study should have been sufficient to produce an effect. The reason

why greater dorsiflexion angles were not investigated is because they would not be functional or safe in AFOs for ambulation. All of the previous studies tested able-bodied subjects in a prone position and therefore did not need to consider subject comfort, safety or stability in weight-bearing.

One key feature of this study was that the effectiveness of the tone-reducing devices was examined in full weight-bearing. This is important as it has been shown that subject positioning significantly affects the H-reflex which is a task and posture dependent measure.[51,52] Peripheral inputs as well as supraspinal centers exert a larger inhibition of the soleus H-reflex circuit in a standing position compared to lying or sitting.[52]

It is possible that evaluating the tone-reducing devices in a standing position resulted in overall non-significant differences as the spinal reflexes of the subjects were subjected to greater peripheral and/or cortical influences. This may be particularly true as postural perturbations were caused by the artificial stimulus possibly increasing the effect of peripheral and cortical influences required to maintain postural standing balance. Nevertheless, it is important that these devices are tested in real life circumstances to gauge their effect on functional activities.

This study has provided a stepping off point for investigations into the neurophysiologic effects of tone-reducing orthoses on spasticity. Further testing is warranted to examine the effects of such devices when combined with biomechanical AFO management and during functional activities such as walking.

Conclusion

This study has found that overall the DFO, OCG and effect of muscle stretch had no significant effect on soleus reflex excitability in subjects with spasticity while standing.

Acknowledgements

We thank Geoff Cumming, DPhil, of the School of Psychological Science, La Trobe University, for his assistance with our statistical analysis.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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Appendix C

Ibuki, A., Bach, T., Rogers, D. and Bernhardt, J. (2010) 2009 ISPO Australia Best Research Paper Award: An investigation of the neurophysiologic effect of tone-reducing AFOs on reflex excitability in subjects with spasticity following stroke while standing. *Prosthetics and Orthotics International* 34(2):1454-165.

Prosthetics and Orthotics International
June 2010; 34(2): 154–165

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2009 ISPO AUSTRALIA BEST RESEARCH PAPER AWARD

An investigation of the neurophysiologic effect of tone-reducing AFOs on reflex excitability in subjects with spasticity following stroke while standing

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Abstract
Tone-reducing ankle-foot orthoses (TRAFOs) are said to improve the control and functioning of spastic lower limbs by their biomechanic and neurophysiologic effects. Unfortunately, there is limited evidence in literature to support the theory that TRAFOs can effectively decrease spasticity in the foot and ankle neurophysiologically. The primary purpose of this investigation was to determine the neurophysiologic effect of TRAFOs on soleus muscle reflex excitability in subjects with spasticity following stroke while standing. A repeated-measures intervention study was conducted on 15 adult subjects with stroke who were recruited from the community. Custom-made articulated ankle-foot orthoses (AFOs) and TRAFOs with orthokinetic compression garments (OCGs) were fabricated for each subject. Five conditions were tested: (1) Shoes only, (2) AFO, (3) TRAFO, (4) TRAFO with OCG, (5) shoes only, to determine if the TRAFOs were most effective in decreasing spasticity as assessed by the ratio of maximum Hoffmann reflex amplitude to maximum muscle response amplitude (Hmax:Mmax ratio) of the soleus. The results found that there were no significant treatment effects for the interventions ($F = 0.992$, $df = 2.167$, $p = 0.388$), however, when analysed subject-by-subject, four subjects displayed significant increases in their Hmax:Mmax ratios to at least one treatment condition. Overall, the results demonstrated that the tone-reducing devices had no significant neurophysiologic effect on soleus reflex excitability in subjects with spasticity, however individual responses showed that the TRAFOs increased spasticity in some individuals.

Keywords: *Lower limb orthotics, muscle spasticity, stroke, tone-reducing, H-reflex*

Introduction

Walking is a complex motor function that can be severely compromised when an individual suffers a stroke resulting in weakness and spasticity in the lower limbs.[1] Spasticity is the result of disordered sensori-motor control that presents as intermittent or sustained involuntary muscle activation.[2] The primary brain lesion and the resultant changes in the

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ISSN 0309-3646 print/ISSN 1746-1553 online © 2010 ISPO
DOI: 10.3109/03093641003649405

lower limbs can cause an energy inefficient gait that is physically challenging requiring a high level of concentration.[3] Safety can also be compromised with an increased risk of stumbling and falling.[3] Such gait can often be improved by an ankle-foot orthosis (AFO).[4]

Patients with spasticity generally have a combination of biomechanic and neurophysiologic (tone control) problems in the lower limbs.[5] A special group of AFOs termed tone-reducing AFOs (TRAFOs) aim to address these problems by reducing spasticity through the incorporation of tone-reducing mechanisms that inhibit the motoneurons that innervate spastic muscles.[6,7] Any orthosis can be modified to include tone-reducing features which supposedly enhance orthotic control when used appropriately.[8]

Although tone-reducing is not the same as spasticity-reducing (which is what the orthoses are purported to do), the term 'tone-reducing' is commonly used throughout literature and is known among clinicians when referring to these distinct orthotic devices. To minimise confusion with terminology, the term 'tone-reducing' has only been used in this manuscript when referring to the orthotic devices, not when referring to spastic muscles.

Commonly used tone-reducing interventions include inhibitory footplates,[5,9–11] pressure on tendons,[12,13] and orthokinetic compression garments (OCGs)[13,14] to name a few. Researchers examining tone-reducing orthoses have found improvements in gait[10,12,15] and function[14,16–19] but have failed to demonstrate inhibitory effects on spasticity. These studies have been conducted on patients with spasticity secondary to a number of different causes such as stroke and cerebral palsy and have assessed tone-reducing interventions on both the lower limbs and the upper limbs.

Literature regarding TRAFOs is scarce and literature on the effect of TRAFOs for subjects with stroke is even scarcer. The majority of TRAFO literature consists of subjective studies that infer a reduction in spasticity based on observed changes in biomechanic and functional variables.[7,12,20–24] Other criticisms include the lack of objective documentation of change in measured variables, lack of control groups and the potential bias of unblinded assessors.[25] The overall aim of TRAFO research is to evaluate effectiveness in reducing spasticity; however, no study to date has used a measurement tool sufficient to quantify this. It is possible that the improvements seen following TRAFO use observed in previous studies were simply due to the biomechanic effect of the orthoses rather than any neurophysiologic effect. Despite the lack of evidence to demonstrate any additional neurophysiologic benefits, tone-reducing orthosis concepts are still promoted by clinicians today.

In order to study the effects of TRAFOs sufficiently and determine their effect on spasticity, the neurophysiologic effects must be measured independently of the biomechanic effects. This is not simple and may explain the limitations of most previous studies. In our laboratory, we have recently used the Hoffmann reflex (H-reflex)[26–28] to measure the neurophysiologic effect of specific tone-reducing modifications either separately or in combination through the direct measurement of the excitability of motoneurons that innervate spastic muscles.[29] To our knowledge, there has only been one previous investigation that has used the H-reflex to assess the effect of prefabricated AFOs on able-bodied subjects.[30] The AFOs tested in this study did not include any tone-reducing features.

The soleus H-reflex is elicited by electrically stimulating the tibial nerve and recording the subsequent muscle responses.[26] Stimulation of the alpha motoneurons causes a direct muscle response (M-response) with a latency of about 10 ms. The stimulation also depolarises the muscle spindle afferents which results in a monosynaptic excitation of the same motoneurons causing a second muscle response (H-reflex) with a latency of about 35 ms. Typically, responses are measured over a range of stimulus strengths from low stimuli which barely elicit a response to large stimuli which elicit a supramaximal response.

These responses form a recruitment curve (Figure 1),[26] and the ratio of the maximum H-reflex amplitude to the maximum muscle response amplitude (Hmax:Mmax ratio) can be used to eliminate the influence of variability with recording or subject physiology.[31,32]

The soleus H-reflex is a valid and reliable tool[33] that can be used to examine the effects of TRAFOs on the reflex excitability of motoneurons that innervate spastic muscles. A change in the Hmax:Mmax ratio following the application of a TRAFO would indicate a neurophysiologic change in the excitability of motoneurons innervating the soleus muscle indicating a change in the level of spasticity.

The purposes of this investigation were to evaluate the neurophysiologic effects of articulated AFOs and TRAFOs on the reflex excitability of subjects with spasticity and to compare the neurophysiologic effects of both types of orthoses to determine if the TRAFOs offer any neurophysiologic benefits over standard AFO designs. The TRAFOs were identical to the AFOs except that they included a tone-reducing footplate design. The TRAFOs were also tested with the addition of an OCG in an attempt to further enhance the speculated tone-reducing effects. We hypothesised that the Hmax:Mmax ratio, as a measure of reflex excitability indicative of the level of spasticity, would be reduced with the TRAFO conditions.

Methods

Subjects

Subjects who had previously suffered stroke were recruited from the community for inclusion in this project through advertisements in print media, the Stroke Association of Victoria, community stroke support groups and the National Stroke Research Institute (Victoria). Subjects were included in the study based on the following criteria:

- Hemiparetic secondary to a stroke suffered at least 12 months prior to the study;
- Independent community ambulators (gait aids acceptable);
- A good current level of health;
- Spasticity in the soleus muscle of the affected side as determined by a grade equal to or greater than 2 on the Tardieu test adapted for stroke;

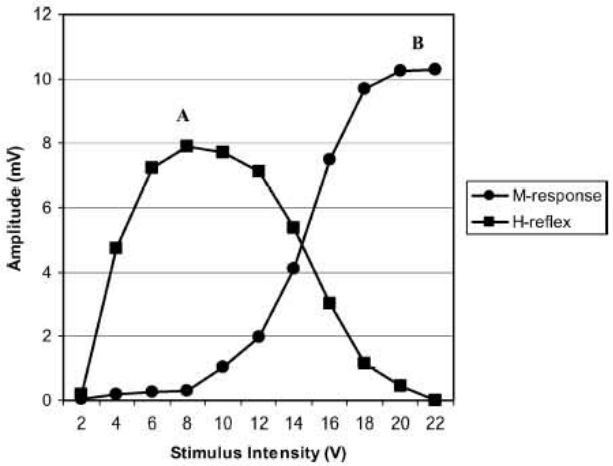

Figure 1. A schematic diagram of the H-reflex and M-response recruitment curves. A = maximum H-reflex amplitude, B = maximum M-response amplitude.

- Able to follow instructions and give informed consent; and
- Able to passively dorsiflex the affected ankle joint to at least neutral (90°) with the knee extended.

Apparatus

All of the equipment used for this experiment were located in the Biomechanics laboratory at La Trobe University, Victoria, Australia. H-reflexes were initiated percutaneously using a Grass SD9B Stimulator (Grass Instruments Co. Quincy, MA, USA) and a custom-built stimulus amplifier (Tain Electronics, Victoria, Australia). Electromyographic (EMG) signals were amplified (1000 gain) bandpass filtered between DC and 2 KHz, monitored on a four-channel oscilloscope (Medelec Ltd. Surrey, UK) and measured with an EMG system (La Trobe University, Victoria, Australia). PowerLab/410 (ADInstruments, Bella Vista, NSW, Australia) was used to digitize EMG and stimulator signals. Power spectral analysis of the EMG signals indicated that almost all of the signal power was below 200 Hz. Data were sampled at 20 kHz for 0.128 sec and processed using PowerLab software (Scope version 3.3). A Cardiometrix Artifact Eliminator (Cardiometrix, Bothell, WA, USA) was used to ensure that the impedance between electrodes was below 5 kohms.

Two 10–1 voltage reduction converters (La Trobe University, Victoria, Australia) were required to reduce EMG and stimulus signals within the range of the A/D converter on the PowerLab. A Tektronix pulse generator (Tektronix, Shanghai, China) was also used to generate a trigger pulse to start the data acquisition as the trigger pulse from the Grass stimulator was too short to initiate data acquisition directly.

Procedures

The project was approved by the Faculty Human Ethics Committee of the Faculty of Health Sciences at La Trobe University (FHEC07/187). All subjects provided informed consent before being included in the study. Subjects attended two sessions, the first for assessment and casting and the second for testing.

Detailed assessments were completed on each subject by a qualified orthotist to determine their level of impairment with particular attention to the presence and degree of spasticity. A plaster-of-Paris wrap cast was taken of their affected leg with the ankle joint held in a neutral position (90°) and the subtalar joint in neither inversion or eversion. Articulated AFOs and articulated TRAFOs were fabricated using the plaster casts (Figure 2).

Orthosis fabrication

Custom-made AFOs and TRAFOs were fabricated from the same positive cast for each patient. This ensured that the devices were identical except for the tone-reducing features of the TRAFOs. Both the AFOs and TRAFOs were moulded with 5 mm polypropylene and fabricated with Tamarack flexure ankle joints (Free motion model 740, Becker Orthopedic, Troy, MI, USA) and plantarflexion stops at 90°.

The AFOs were fabricated first following standard cast modifications for AFOs with full length footplates.[34] No pitch was modified into the casts as the AFOs were to be worn with flat firm-soled post-operative shoes (OTS, Victoria, Australia). Once the AFOs had been fabricated, the plaster casts were repaired and modified with the tone-reducing footplate features to fabricate articulated TRAFOs. These tone-reducing features are well

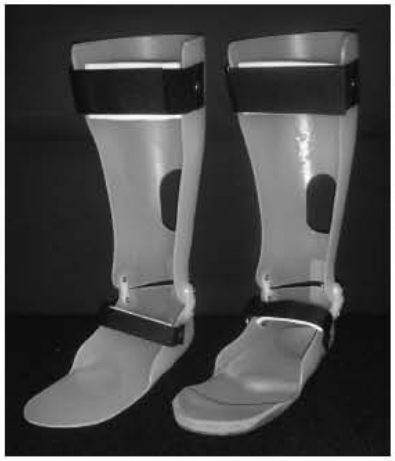

Figure 2. Custom-made articulated AFO and TRAFO with 90° plantarflexion stops.

documented in literature.[5,10,11,35] The tone-reducing footplates were fabricated using sheets of high density foam (EVA 250 kg.m^3) that were vacuum moulded onto the sole of the casts and then shaped. This achieved a good contour of the tone-reducing footplate features and provided a stable plantar base for the TRAFOs. Plastic was then moulded over the casts and tone-reducing footplates to fabricate the TRAFOs.

Rectangular cut-outs (3 × 5 cm) were made in the posterior shells of the AFOs and TRAFOs to allow for EMG electrode placement over the soleus muscle. Calf and ankle straps were attached and where needed threaded screws were drilled into the plantarflexion stops to adjust the stops to 90°.

Class one (18–21 mmHg) Venosan (Salzmann AG, St. Gallen, Switzerland) below knee compression garments were used to create the OCGs which are an added tone-reducing feature.[14,36,37] Pieces of smooth inelastic vinyl were specifically cut from measurements of the subject's leg to create inactive fields over the posterior half of the leg. The pieces of vinyl were fitted directly against the subject's leg inside the compression sleeve which was then worn like a sock under the TRAFO.

Testing procedures

Subjects were asked to refrain from ingesting stimulating substances and from performing strenuous exercise for 12 h prior to the testing session. In preparation for electrode placement, a small area of skin on the affected leg over the soleus muscle was shaved, abraded, and cleansed with alcohol. Surface electrodes (Ag/AgCl 10 mm disk monitoring electrodes) were placed using Hugon's method.[38] The center-to-center electrode distance was 30 mm. The ground electrode was positioned over the fibular head which was chosen as an electrically silent area that did not interfere with the AFOs. The anode was positioned on the anterior thigh just proximal to the patella and the cathode was positioned in the popliteal fossa over the tibial nerve.

For each subject the Hmax:Mmax ratio was tested under five conditions: (i) Shoes only (baseline); (ii) AFO; (iii) TRAFO; (iv) TRAFO with OCG; and (v) Shoes only (baseline return). Subjects were given an accommodating period to become accustomed to the sensation of the stimulating electrode in the standing position before testing began. A walking frame was positioned in front of the subjects and a chair was placed behind them.

Subjects were instructed to bear equal weight through both limbs throughout the testing; however, the actual amount of weight-bearing through each leg was not measured. Subjects were also instructed to use the frame only as a balance aid, to remain relaxed, and to fix their gaze on a target placed on a wall 3 m in front of them. For each of the five testing conditions, H-reflex and M-response recruitment curves were constructed.

For the baseline conditions, postoperative shoes were worn by all subjects to standardize footwear. The order of the three orthosis conditions (AFO, TRAFO and TRAFO with OCG) was randomized by pulling pieces of paper with the conditions out of a box to prevent a series effects. A brief accommodating period was given for each condition to allow the subjects to assume a comfortable standing position.

In all conditions the duration and frequency of the electrical stimulations were the same. The stimulus duration was 1msec and the frequency was 0.2 Hz.[32] Recruitment curves were constructed by gradually increasing stimulus voltage from below threshold for the H-reflex to supramaximal for the M-response (Figure 1). Four stimulations were recorded at each level of stimulus voltage. The mean trace at each level of stimulus intensity was obtained and used to graph the recruitment curves for the H-reflex and the M-response. In this way a variable number of 15–25 samples were obtained from each subject to construct the curves. The ratio of maximum H-reflex amplitude to maximum M-response amplitude was recorded as the Hmax:Mmax ratio.

Analysis

We estimated that 18 subjects would be required to find a difference of $p = 0.05$ between the conditions with 80% confidence based on previous unpublished investigations in our laboratory. A one-way repeated measures Analysis of Variance (ANOVA) was used to calculate differences between the conditions. *Post hoc* tests of within condition contrasts were used to determine which pair-wise comparisons were significantly different. Mauchly's test of sphericity was used to determine whether distributions were normal, and if they were not, a Greenhouse-Geiser adjustment was used.[39] Statistical significance was set at $\alpha = 0.05$. All statistical analyses were performed using the SPSS Statistics 17 (SPSS Inc, Chicago, USA).

Because individuals with neurological disorders are unique,[40] data were examined to determine whether any treatments resulted in significant changes within an individual. The 95% range for change for the Hmax:Mmax ratio was used as the criterion.[41] According to Chinn[42] the 95% range of change ($R_{95\%}$) can be calculated as:

$$R_{95\%} = 1.96SD(y_1 - y_2)$$

where y_1 and y_2 are the mean Hmax:Mmax ratios for the two baseline conditions and SD is the standard deviation of the means. However, using 1.96 as the critical value for z assumes a large sample and is highly optimistic. For a more conservative estimate, the critical value for t with 14° of freedom ($t = 2.1448$) was used as the multiplier. Any measures that fell outside of the range were considered to be indicative of a significant response to the intervention.

Results

Fifteen subjects were recruited over a period of 12 months. The subject characteristics can be found in Table I. The ANOVA revealed that there were no significant differences in the

Table I. Subject characteristics. X = quality of muscle reaction, Y = angle of muscle reaction in plantarflexion.

Subject	Gender	Age (years)	Time since most recent stroke (years)	Stroke type	Side of lesion	Tardieu test (X, Y)	Currently wearing AFO	Type of AFO	Gait aids required	Sensory deficit
1	M	52	26	Haemorrhage	L	2, 20°	Y	Leaf-spring AFO	Y	Y
2	M	58	10	Ischemia	L	2, 42°	Y	Solid AFO	Y	N
3	M	65	3	Ischemia	L	2, 12°	N		N	Y
4	M	63	6	Haemorrhage	R	3, 20°	N		Y	N
5	M	60	5	Haemorrhage	L	3, 7°	Y	Hinged AFO with plantarflexion stop	Y	N
6	F	57	2	Ischemia	L	2, 24°	Y	Hinged AFO	Y	N
7	F	50	15	Haemorrhage	L	3, 8°	N		Y	Y
8	F	60	9	Haemorrhage	R	3, 22°	Y	Solid AFO	Y	Y
9	F	41	2	Haemorrhage	L	2, 14°	N		N	N
10	F	66	4	Ischemia	L	2, 18°	N		Y	Y
11	M	59	17	Haemorrhage	L	4, 36°	N		Y	Y
12	F	60	5	Haemorrhage	R	2, 30°	N		N	N
13	M	71	6	Ischemia	R	3, 22°	N		N	N
14	M	71	35	Haemorrhage	L	3, 22°	N		Y	N
15	F	38	4	Haemorrhage	R	3, 26°	Y	Soft prefabricated ankle brace	Y	N
	8 Male, 7 Female	58.07	9.93	10 Haemorrhage, 5 Ischemia	5 Right, 10 Left	2.6, 21.53°	6 Yes, 9 No		11 Yes, 4 No	6 Yes, 9 No

Hmax:Mmax ratios between any of the conditions ($F = 0.992$, $df = 2.167$, $p = 0.388$) (Figure 3). The $R_{95\%}$ was used to further examine the data to determine whether any substantial changes within individual subjects had been masked by the averaging process. The individuality of the impairments of stroke survivors makes it reasonable and acceptable to analyse the data on a subject-by-subject basis to determine individual changes. The $R_{95\%}$ interval for the Hmax:Mmax ratio was found to be 0.12. Four subjects had significant responses to one or more of the interventions when analysed using the $R_{95\%}$ (Table II). All of

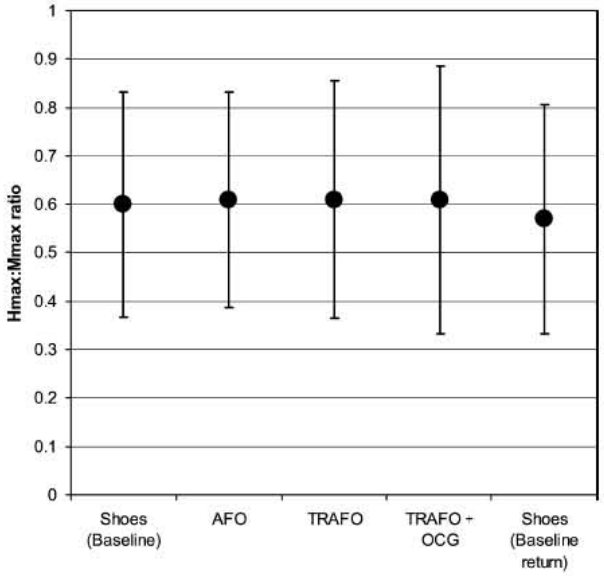

Figure 3. Mean Hmax:Mmax ratios across all subjects.

Table II. Raw data of all subjects across all conditions showing which conditions fell outside of the $R_{95\%}$ as indicated by *. BL1 = Baseline (Shoes only), BL2 = Baseline return (Shoes only).

Subject	BL1	AFO	TRAFO	TRAFO + OCG	BL2	Mean BL1 and BL2	$R_{95\%}$
1	0.57	0.6	0.59	0.6	0.54	0.56	0.44–0.68
2	0.6	0.47	0.44	0.46	0.42	0.51	0.39–0.63
3	0.2	0.22	0.24	0.25	0.2	0.2	0.08–0.32
4	0.59	0.58	0.61	0.62	0.55	0.57	0.45–0.69
5	0.69	0.75	0.85*	1.03*	0.69	0.69	0.57–0.81
6	0.61	0.62	0.58	0.59	0.58	0.6	0.48–0.72
7	0.64	0.62	0.62	0.59	0.67	0.66	0.54–0.78
8	0.91	0.92	0.98	1.04*	0.89	0.9	0.78–1.02
9	0.29	0.46*	0.29	0.21	0.19	0.24	0.12–0.36
10	0.39	0.42	0.41	0.35	0.38	0.39	0.27–0.51
11	0.68	0.77	0.8*	0.79	0.66	0.67	0.55–0.79
12	1	0.97	0.99	0.92	0.99	1	0.88–1.12
13	0.3	0.24	0.29	0.24	0.35	0.33	0.21–0.45
14	0.58	0.6	0.58	0.58	0.62	0.6	0.48–0.72
15	0.9	0.84	0.84	0.86	0.85	0.88	0.76–1

the significant responses were increases in the Hmax:Mmax ratio demonstrating increases in the reflex excitability. One subject responded to the AFO condition, two subjects responded to the TRAFO condition and two subjects responded to the TRAFO with the addition of the OCG.

Discussion

The results of this study indicated that overall, the AFOs and TRAFOs had no significant neurophysiologic effect on reflex excitability demonstrating that the tone-reducing features of the TRAFOs were ineffective compared to otherwise identical AFOs. The TRAFOs included tone-reducing footplates advocated by a number of investigators[5,9,11,17] and were tested both with and without the addition of OCGs also thought to have tone-reducing effects.[7,9,11,13,14,36]

This study has demonstrated a useful method for measuring directly the effect of orthoses on the reflex excitability of patients with spasticity. Previous studies have inferred changes in reflex excitability in subjects wearing TRAFOs from changes in balance and gait[7,10,12,15,22,43] but these studies have failed to separate the biomechanic effects of the orthoses from their neurophysiologic effects. The H-reflex technique enables a direct measurement of the reflex excitability and therefore allows assessment of the neurophysiologic effects separate from the biomechanic effects of the orthoses.

Previous investigators have found improvements in gait,[7,9,12,22,44] EMG activity,[7] posture,[12] and joint position[45] with the use of TRAFOs. However, most of these studies have compared an orthosis condition with a no-orthosis condition or have compared different orthosis designs making it impossible to separate the neurophysiologic and biomechanic effects. No previous study has directly examined the effect of these tone-reducing features on reflex excitability when incorporated into AFO management.

In individual subjects who demonstrated reflex excitability changes with the orthoses, the excitability was increased in all cases. In no cases did the TRAFO features demonstrate a significant decrease in motoneuron excitability. These results are the opposite of those found in a previous investigation in our laboratory[29] where subjects who responded to the tone-reducing devices all demonstrated decreases in their MN excitability. Previous investigators have explained that due to the complex nature of spasticity, the way in which it responds to treatment is unpredictable and it is not uncommon for an intervention to cause an effect in one person and not another.[46,47] It remains possible that some individuals may exhibit reductions in motoneuron excitability as a result of TRAFO modifications but none were observed in the present study.

Only one other study has examined the effects of orthoses on lower limb reflex excitability using H-reflex measurements. Nishikawa and Grabiner[30] found excitatory effects of orthoses (approximately 10%) when examining semirigid prefabricated ankle braces compared to a no-brace condition in 11 able-bodied subjects. They studied peroneal reflex excitability measured by H-reflex amplitudes and attributed the immediate increase in reflex excitability following brace application to stimulation of mechanoreceptors particularly cutaneous receptors. The results of Nishikawa and Grabiner[30] and the results from the analysis of individual hemiparetic subjects in the present study suggest that orthoses may have a predominantly excitatory effect on lower limb motoneurons.

In this study, subjects were assessed during static standing. However, it is during walking when changes in plantar pressures, joint angles and muscle length are believed to have the greatest effect on spasticity.[3,48] Despite this, previous authors have stated that significant changes can be observed when a patient with spasticity simply stands on a tone-reducing

footplate without walking.[11] This is due to the inhibition of reflexogenous areas on the sole of the foot by the diminution of plantar pressures.[11] No such changes were observable in the present study. There is a clear need for further testing of the effects of TRAFO features during ambulation while controlling the biomechanic effects of the orthoses.

Despite the fact that subjects were instructed to bear equal weight through both limbs during the testing period, this was not measured. It is known that stroke patients generally favour their unaffected side when weight-bearing[49] and it is possible that subjects were not bearing enough weight through their affected limbs to achieve the presumed tone-reducing effects of the footplates. Future investigations should measure the amount of weight-bearing to ensure the footplates are being adequately loaded and for consistency between the conditions.

It must be noted that in two subjects Hmax:Mmax ratios greater than one were recorded (Table II). This suggests that a slightly greater number of motoneurons was excited by reflex activation than was excited by direct stimulation of the motor nerve, however our current knowledge of the H-reflex says that obtaining Hmax:Mmax ratios greater than one should not be possible.[26,27] The most likely explanation for this is a recording error where the peak M-response amplitude was missed because increments between stimulus intensities were not sufficiently small (Figure 1). However decreasing the increment size would have required a greater number of increments and would have increased the length of the testing session increasing the probability of subject fatigue. The discrepancies are very small and we do not believe that this has affected the validity of the measurements or the results of the statistical analysis.

A further limitation of this investigation is the small sample size. Difficulty in recruiting subjects who met all of the inclusion criteria resulted in a smaller sample size; however, based on the results of the current investigation as well as previous investigations conducted in our laboratory,[29] we are confident that the inclusion of three more subjects would not have substantially altered the results.

This study highlights the need for care in prescribing orthoses for patients with spasticity. Despite the requirement for biomechanical control, it is possible that orthoses with or without tone-reducing features may increase spasticity. It has been suggested previously that the use of orthoses for patients with spasticity may be detrimental due to the activation of reflexes which result in undesirable muscle contractions.[11,50] Careful thought needs to be put into the design of orthoses for patients with spasticity to minimize these detrimental effects by taking into account the materials used for fabrication, trimlines, areas where the orthosis contacts the limb and joint ranges of motion allowed by the orthoses.

Conclusion

Overall, the orthosis conditions had no significant effect on soleus reflex excitability in patients with spasticity while standing. In four patients, one or more of the orthosis conditions significantly increased their reflex excitability challenging the presumption that tone-reducing orthoses decrease spasticity.

We presented a method for testing the effect of TRAFOs on spasticity that separates biomechanic from neurophysiologic effects. There is no doubt that the biomechanic effects of AFOs are useful for patients with spasticity;[51–56] however, there is no further evidence to suggest that the addition of tone-reducing modifications enhance AFO function while standing. With this being the case, the time and costs associated with fabricating and fitting TRAFOs over standard AFO designs may be unjustified especially if they have the opposite

effect of increasing spasticity. Further investigations of the neurophysiologic effects of TRAFOs while walking are required to substantiate this.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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