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Physiotherapy management of contractures after  
acquired brain injury

Joan Wai-King Leung

Thesis submitted in fulfilment of the requirements for the  
Degree of Doctor of Philosophy

in

Sydney School of Medicine

University of Sydney

September 2014

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## Statement of Authentication

This thesis is submitted to the University of Sydney in fulfilment of the requirement for the Degree of Doctor of Philosophy.

The work presented in this thesis is, to the best of my knowledge and belief, original except as acknowledged in the text. I, Joan Leung, hereby declare that I have not submitted this material, either in full or in part, for a degree at this or any other institution.

Joan Leung



15/05/2014

**Signature:** .....

**Date:** .....

## Candidate's statement

I, Joan Leung, hereby declare that this submission is my own work and that it contains no material previously written by another person except where acknowledged in the text. Nor does it contain material that has been accepted for the award of another degree.

I, Joan Leung, understand that if I am awarded a higher degree for my thesis titled 'Clinical management of contractures after acquired brain injury' being lodged herewith for examination, the thesis will be lodged in the University Library and be available immediately for use. I agree that the University Librarian (or in the case of a department, the Head of Department) may supply a photocopy, electronic copy or microform of the thesis to an individual for research or study or to a library.

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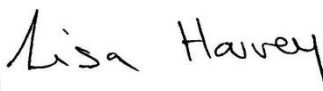
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## Supervisors' statement


As supervisors of Joan Leung's doctoral work, we certify that her thesis 'Physiotherapy management of contractures after acquired brain injury' is sufficiently well prepared to be examined for the Degree of Doctor of Philosophy. To the best of my knowledge, no editorial assistance has been sought in the writing of the thesis.

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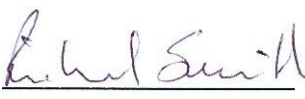
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This thesis was undertaken with the knowledge that contractures are difficult to treat, and studies involving patients with traumatic brain injury (one of the targeted clinical populations for this thesis) are challenging to conduct. Thus, it was a huge relief when the thesis was finally completed. All the work relating to this thesis could not be accomplished without the joint effort and great support of a large number of people.

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## **Publications and presentations**

The information included in this thesis had been published and presented in the following forums:

### **Publications:**

Leung J, Smith R, Harvey L, Moseley A. Impact of a unilateral ankle contracture imposed by an ankle-foot-orthosis on knee biomechanics in stance phase. *Clinical Biomechanics* 2014;29:423-428.

Leung J, Harvey LA, Moseley AM. An intensive program of passive stretch and motor training for the management of severe knee contractures after traumatic brain injury: a case report. *Physiotherapy Canada* 2013;65:223-228.

Leung J, Harvey LA, Moseley AM, Tse C, Bryant J, Wyndham S, Barry S. Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial. *Journal of Physiotherapy* 2012;58:231-240.

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### **Presentations:**

Leung J, Harvey LA, Moseley AM (2014) Physiotherapy for contracture management. *Postgraduate Research Students Society Seminar*, University of Sydney, Sydney, Australia.

Leung J, Harvey LA, Moseley AM (2014) Clinical management of contractures and electrical stimulation. *The Brain Injury Rehabilitation Program Physiotherapy Network Group*, Sydney, Australia.

Leung, J, Harvey, LA, Moseley, AM, Whiteside, B, Simpson, M, Stroud, K (2014) Multimodality approach for contracture management after severe traumatic brain injury: a randomised trial. *37<sup>th</sup> Annual Brain Impairment Conference (Australasian Society of the study of Brain Impairment)*. Fremantle, Australia.

Leung J (2014) An update on contracture management. *Community Physiotherapy Network Group*, Sydney, Australia

Leung J, Harvey LA, Moseley AM, Tse C, Bryant J, Wyndham S, Barry S (2013) Electrical stimulation for contracture management after acquired brain injury a randomized trial. *Australian Physiotherapy Association Conference 2013*. Melbourne, Australia.

Leung J, Harvey LA, Moseley AM, Tse C, Bryant J, Wyndham S, Barry S (2013) Electrical stimulation for contracture management after acquired brain injury a randomized trial. *36<sup>th</sup> Annual Brain Impairment Conference (Australasian Society of the study of Brain Impairment)*. Hobart, Australia.

Leung J, Harvey LA, Moseley AM (2013) An intensive program of passive stretch and motor training for the management of severe knee contractures after traumatic brain injury: a case report. *36<sup>th</sup> Annual Brain Impairment Conference (Australasian Society of the study of Brain Impairment)*. Hobart, Australia.

Leung J, Harvey LA, Moseley AM, Tse C, Bryant J, Wyndham S, Barry S (2012) Wrist contractures and electrical stimulation: a randomized controlled study. *Postgraduate Research Students Society Seminar*, University of Sydney, Sydney, Australia.

Leung J, Harvey LA, Moseley AM (2011) An intensive program of passive stretch and motor training for the management of contractures: a case report. *Australian Physiotherapy Association Conference 2011*. Brisbane, Australia.

Leung J, Smith R, Harvey LA, Moseley AM, Chapparo J (2011) Ankle contractures cause knee hyperextension: a biomechanical study. *Australian Physiotherapy Association Conference 2011*. Brisbane, Australia.

Leung J (2010) Current evidence on passive stretch. *Community physiotherapists Professional Development Day*. Royal Rehabilitation Centre Sydney, Sydney, Australia.

### **Submitted for presentation**

Leung J, Harvey LA, Moseley AM (2014) Physiotherapy for contracture management. *New Horizons: Improving healthcare through research and education*. 31<sup>st</sup> Combined Health Science Conference, Sydney, Australia.

Leung, J, Harvey, LA, Moseley, AM, Whiteside, B, Simpson, M, Stroud, K (2015) Multimodality approach for contracture management after severe traumatic brain injury: a randomised trial. *World Confederation for Physical Therapy Congress 2015*, Singapore.

## Preface

This thesis comprises two main parts: (1) physiotherapy for contractures and (2) impact of contractures on knee biomechanics. The content of the thesis is organised in 7 chapters. Each chapter has its own reference list.

Chapter 1 is an introduction which outlines the general theme of the thesis. Chapter 2 is a literature review which provides an overview of the relevant literature and research on physiotherapy for contractures and the impact of ankle contractures on walking. Chapter 3 is a case report which describes the management of chronic contractures of a young adolescent. The content is presented as published in the *Physiotherapy Canada*. Ethics approvals were obtained from the Human Research Ethics Committees of University of Sydney, Northern Sydney Central Coast Area Health Service and Royal Rehabilitation Centre Sydney. Chapter 4 describes a multi-centre randomised controlled study which investigated the effectiveness of the addition of electrical stimulation to a splinting regimen. Ethics approvals were obtained from the Human Research Ethics Committees of the Northern Sydney Central Coast Area Health Service and all the participating hospitals (namely, Royal Rehabilitation Centre Sydney, Balmain Hospital, Liverpool Hospital). It is presented as published in the *Journal of Physiotherapy*. This paper features in the 'most downloaded' list from Science Direct for the first quarter of 2013. Chapter 5 presents a multi-centre randomised controlled study which investigated the effectiveness of a program combining tilt table standing, electrical stimulation and splinting. It is presented in the format as required by the *Journal of Physiotherapy* where the paper has been accepted for publication. Ethics approvals were obtained from the Human Research Ethics Committees of

the Northern Sydney Central Coast Area Health Service and all the participating hospitals (namely, Royal Rehabilitation Centre Sydney, Liverpool Hospital and Westmead Hospital). Chapter 6 is a published paper which describes the impact of ankle contractures on the knee joint and spatiotemporal parameters. It is presented as published in *Clinical Biomechanics*. Ethics approval was obtained from the Human Research Ethics Committees of the University of Sydney. The conception and design of all the four studies (Chapters 3 to 6) originated from Joan Leung, the PhD Candidate, who was also responsible for data collection, analysis and interpretation of the findings and writing of the manuscripts. She also made major contributions to the revisions of the content of all these manuscripts for publications.

Finally, Chapter 7 provides a summary of the principal findings, clinical implications and limitations of the studies, and suggestions for further research. The procedure manuals for the two randomised controlled trials (Chapters 4 and 5) are included in the Appendices.

## Abstract

Contractures are a common problem after acquired brain injury. They are undesirable because of their potentially serious implications on motor recovery, functional outcomes and care needs. There has been increasing research directed at identifying effective treatment for contractures. Yet no solution has been found. There is also little information on the impact of contractures on the characteristics of gait. Much research is required in these areas to guide clinical practice. This thesis therefore focuses on two main aspects: (1) physiotherapy for the treatment of contractures and (2) impact of ankle contractures on the knee joint on walking.

Passive stretch has been the most widely used method to prevent and correct contractures. However, latest research indicates that regular passive stretch, as typically applied by clinicians, does not produce clinically worthwhile or lasting effects in people with neurological conditions. It is probable that the doses of passive stretch were insufficient in these studies, and passive stretch was not combined with treatments that address the factors that contribute to contractures such as spasticity and muscle weakness. To lend support to this argument, a case report (Chapter 3) is firstly presented. It describes an adolescent whose severe knee contractures did not respond to usual care (12 hours of passive stretch using splinting) but resolved after the dose of passive stretch was increased to 24 hours day using serial casting, and the effect was maintained by an intensive program combining splinting (24 hours of stretch a day) with motor training administered over 10 months. This report is the first documented case involving the use of intensive passive stretch for more than 6 months and a follow-up at 5.5 years post injury. This case report suggests that passive stretch is effective when a high dose is used and when it is combined with treatments that address the contributors to contractures. Most importantly,

interventions have to continue until the underlying causes of contractures are adequately addressed. Admittedly, the case report provides only weak evidence. Spontaneous recovery, however, is unlikely in this case as no progress was made in the initial seven months before serial casting. Nonetheless, further scrutiny within a clinical trial will be needed to confirm the effectiveness of this treatment approach.

Electrical stimulation, through its effects on improving muscle strength and spasticity, is potentially useful for addressing the contributors to contractures. A randomised controlled study (Chapter 4) was therefore conducted to evaluate the effectiveness of electrical stimulation as an adjunct treatment for wrist contractures in people with acquired brain injury. Thirty six adults with stroke or traumatic brain injury participated in a 4-week program. The experimental group received electrical stimulation to wrist and finger extensors for 60 minutes a day, and hand splinting for at least 12 hours a day. The control group only received hand splinting. The adherence to treatment was overall good. The study found a mean (95% CI) between-group difference in wrist extension at week 4 (post-intervention) of 7 degrees (-2 to 15). This effect size exceeded the pre-determined minimally important effect of 5 degrees. However, the estimate was associated with considerable imprecision (as reflected by the wide 95% CI) leading to an uncertainty about the added benefit of electrical stimulation. The effect on wrist extension was lost at follow-up, that is, 2 weeks after cessation of electrical stimulation. The expected treatment effects of electrical stimulation on strength, spasticity and motor control were not observed, and this may be the reason for not finding a large or sustained effect on wrist extension.

Contractures may be best managed by a program combining passive stretch with treatments that address the contributors to contractures. To test this hypothesis, a



randomised controlled study (Chapter 5) was conducted. It assessed the effectiveness of a combined treatment program for ankle contracture management. Thirty six people with first severe traumatic brain injury and ankle contractures participated in the study. Both groups underwent a 6-week program. The experimental group received electrical stimulation, tilt table standing and ankle splinting, while the control group received tilt table standing alone. Adherence to treatment was variable due to reasons including behavioural issues, medical reasons and discharges. The study found that the combined program did not have an effect on passive ankle range of motion (-3, 95% CI -8 to 2), motor function, walking speed and global perceived effect of treatment compared to tilt table standing alone. There was a small reduction of spasticity following the 6-week program (-1, 95% CI -1.8 to -0.1) but this effect dissipated at follow-up 4 weeks after the program. Overall, the study did not find the combined program more effective than tilt table standing alone in the management of contracture for people with severe traumatic brain injury.

Electrical stimulation was used as an adjunct treatment in both randomised controlled studies to address spasticity and muscle weakness; factors that are believed to contribute to contractures. No convincing effect on spasticity, muscle strength or motor function was found. It is possible that an insufficient dose of electrical stimulation was used in these studies. It is also possible that electrical stimulation is ineffective for people with severe motor and cognitive impairments; the target populations of these studies. Future research may have to consider other treatment options as adjunct treatments for contracture management in people with severe brain injury.

The second part of the thesis focused on the impact of ankle contractures on walking. Ankle contractures and knee hyperextension gait are both common problems after stroke and

often co-exist. Although they appear to be closely linked, their relationship is unclear. A within-subject observational study (Chapter 6) was therefore conducted to investigate the impact of simulated ankle contractures on the knee joint during stance. Thirteen healthy subjects participated in the study. An ankle-foot-orthosis was used to simulate ankle contracture of two severities (mild and severe). The study found that ankle contractures are associated with increases in knee extension 5 (95% CI 0 to 10) degrees in the mild contracture condition and 9 (95% CI 2 to 17) degrees in the severe contracture condition. This association implies that ankle contractures may play a role in the knee hyperextension gait; a common gait disorder following acquired brain injury. Ankle contractures are also associated with reductions in extension moment and power generation at the knee. This finding suggests that the movement of knee into hyperextension is largely passive but controlled by increased eccentric knee flexor activity. Different to previous research, this study found more than one gait pattern secondary to ankle contractures. While a majority of our participants adopted a foot-flat pattern and exhibited an increase in knee extension, two participants adopted a toe-walking pattern and showed an increase in knee flexion. This study also confirmed the finding of a previous study that ankle contractures have an adverse effect on spatiotemporal parameters including gait velocity, cadence and step length. In addition, this study showed that all the changes were more profound in the severe contracture condition than mild contracture condition.

In summary, the thesis adds knowledge to the limited evidence on contractures. Effective management of contractures may require passive stretch of high intensity and in combination with treatments that target the underlying causes of contractures. The case report provides an initial support for this proposition. The two randomised controlled trials demonstrate that electrical stimulation administered in conjunction with passive stretch is

not more useful than passive stretch alone for people with severe motor and cognitive impairments. The data of these two studies are potentially useful for future meta-analyses. The suggestions stemming from these studies may provide guidance for further investigations into contracture management. The second part of the thesis investigates the impact of ankle contractures on the knee joint. The observational study establishes the link between ankle contractures and increased knee extension, and identifies the two gait patterns that are associated with simulated ankle contractures. This information contributes to the understanding of gait deviations that are secondary to ankle contractures.

# Chapter 1

## Introduction

### 1.1 Background information

Acquired brain injury, such as stroke and traumatic brain injury, are significant causes of disability in Australia. An estimated 1.8% of the population (376,000 people) suffers a stroke at some time in their lives. There are about 53,000 stroke events,<sup>1</sup> and more than 13,000 cases of hospitalised traumatic brain injury every year.<sup>2</sup> While the cases vary in severity, many survivors have life-long consequences including activity limitations and reduced quality of life.<sup>3</sup> Disability associated with motor impairments is very common after acquired brain injury.<sup>4</sup> Primary impairments (for example, muscle weakness and loss of dexterity) result directly from the neurological lesions, and contribute to functional deficits after stroke or traumatic brain injury. Hence they are the main focuses of rehabilitation. Secondary impairments, such as contractures, arise from adaptations to the primary impairments.<sup>5</sup> They also impose major hindrances to functional recovery, and play a significant role in determining functional outcomes and quality of life. Thus prevention and treatment of secondary impairments are as important as managing primary impairments, and have always been an integral part of the overall management.

Contractures are losses in joint range due to changes in the passive mechanical properties soft tissues spanning joints.<sup>6-10</sup> They are a common secondary impairment following acquired brain injury. Contractures are undesirable because they interfere with

movement,<sup>11,12</sup> restrict function and limit activities of daily living. It is therefore not surprising that contractures are associated with poor rehabilitation outcomes.<sup>13-15</sup> The challenge confronting clinicians is that contractures are difficult to treat, and they often occur and persist despite treatment. This thesis aims to contribute knowledge to two key aspects of contractures: (1) physiotherapy for the treatment of contractures, and (2) impact of contractures on walking. The main focuses are on contractures in adults with stroke and traumatic brain injury. These two clinical populations are selected because their clinical presentations are very similar and contractures are common amongst them.<sup>5,16-23</sup> Throughout the thesis, the term acquired brain injury is used to refer to both stroke and traumatic brain injury. The literature review (Chapter 2) focuses primarily on studies that are relevant to acquired brain injury.

Management of contractures traditionally has been based on theoretical knowledge, anecdotal experience, therapists' preference and trend of practice rather than evidence. Passive stretch has been the main method for prevention and correction of contractures. It is applied by holding joints at or near their end-position with an aim to increase muscle extensibility and therefore joint mobility. Commonly used stretch interventions include positioning, manual stretching, tilt table standing, splinting and serial casting. These interventions are widely used and well accepted because of their seemingly sound biological plausibility and empirical basis.<sup>9,24-29</sup> However, there is mounting evidence that passive stretch, as typically applied by clinicians, is ineffective.<sup>30</sup> This finding has led to increasing doubt about the clinical value of passive stretch and contention regarding its use. Much evidence is needed to guide clinical decisions on contracture management.

In this thesis, a case report (study 3) is presented to describe an adolescent for whom an intensive stretch program was conducted in conjunction with motor training. The aim of this case report is to lend support for using high intensity passives stretch and combining it with treatments to address the underlying causes of contractures. It was written with the clear view that case reports only provide weak evidence. Nonetheless, it is the first documented case that involves passive stretch for more than 6 months, motor training for 1.5 years and a follow-up at 5.5 years post injury. This case report is presented in Chapter 3.

A randomised controlled trial was conducted to investigate the effectiveness of electrical stimulation as an adjunct treatment for contracture management. Electrical stimulation was selected because of its potential effect on addressing muscle weakness and spasticity; factors believed to contribute to contractures. The aim of this study is to investigate the benefit of adding electrical stimulation to a program of passive stretch. The results are presented in Chapter 4.

Furthermore, I hypothesised that contractures were best dealt with by a combined program addressing both muscle shortening and factors that contribute to contractures. The second randomised controlled study was conducted to assess the effectiveness of a program combining splinting and tilt table standing with electrical stimulation. Comparison was made with tilt table standing alone. The results of this study are presented in Chapter 5.

There are certain similarities between these two randomised controlled studies. They both involved people with severe motor and cognitive impairments who were receiving rehabilitation after acquired brain injury; used electrical stimulation in conjunction with passive stretch; relied on measures of muscle extensibility as the primary outcome measure, and spasticity, motor control and activity limitations as the secondary measures. In addition,

they both were pragmatic trials exploring clinical use of intervention programs. Thus, it was important to explore practicality, perceived treatment effect and credibility of program from the view point of therapists and participants. Ankle and wrist joints were chosen because these joints are most vulnerable to contractures.<sup>5,16-18,31</sup>

The second part of the thesis aims to improve the understanding of the impact of contractures on walking. Clinicians need a good understanding of the gait deviations secondary to contractures so that they can identify the causes of gait deviations and apply appropriate treatment strategies to improve mobility, gait and walking efficiency. Ankle contractures are common after brain injury.<sup>23,31</sup> While there are suggestions that ankle contractures are closely related to knee hyperextension gait,<sup>32-34</sup> research findings on this have been conflicting. A within-subject observational study was therefore conducted to investigate the changes of knee biomechanics secondary to ankle contractures. An ankle-foot-orthosis was used to simulate ankle contracture of two severities. Healthy subjects were recruited so as to assess the effect of ankle contractures without confounding by the primary motor impairments after acquired brain injury. The findings of the study are reported in Chapter 6.

Overall, the two main objectives of this thesis are: first, identifying effective treatment for contractures; and second, understanding the effect of ankle contractures on knee biomechanics. We hope that this information will add to the existing evidence that guides the clinical management of contractures. The ultimate aim of this thesis is to improve the health outcome and quality of life of people with acquired brain injury.

## 1.2 References

1. 2009 Survey of Disability, Ageing and Carers. Authoritative information and statistics to promote better health and wellbeing. at [www.aihw.gov.au/cardiovascular-health/prevalence](http://www.aihw.gov.au/cardiovascular-health/prevalence).
2. National Hospital Morbidity Database (NHMD). Authoritative information and statistics to promote better health and wellbeing (AIHW). Australia. 1999-2005.
3. National health survey 2007-2008: Users' Guide (cat. no. 4363.0.55.001). Australian Bureau of Statistics. 2008.
4. Tate RL, Broe GA, Lulham JM. Impairment after severe blunt head injury: the results from a consecutive series of 100 patients. *Acta Neurol Scand* 1989;79:97-107.
5. Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disabil Rehabil* 2006;28:891-7.
6. Botte MJ, Nickel VL, Akeson WH. Spasticity and contracture. Physiologic aspects of formation. *Clin Orthop Relat Res* 1988:7-18.
7. Harburn KL, Potter PJ. Spasticity and contractures. *Phys Med Rehabil State of the Art Reviews* 1993;7:113-32.
8. Tardieu C, Huet de la Tour E, Bret MD, Tardieu G. Muscle hypoextensibility in children with cerebral palsy: I. Clinical and experimental observations. *Arch Phys Med Rehabil* 1982;63:97-102.
9. Williams PE. Effect of intermittent stretch on immobilised muscle. *Ann Rheum Dis* 1988;47:1014-6.
10. Dudek N, Trudel G. Joint contractures. In: Frontera W, Silver J, Rizzo T, eds. *Essentials of physical medicine and rehabilitation musculoskeletal disorders, pain, and rehabilitation*. 2nd ed: Philadelphia: Saunders; 2008:651-5.



11. Cooper JE, Shwedyk E, Quanbury AO, Miller J, Hildebrand D. Elbow joint restriction: effect on functional upper limb motion during performance of three feeding activities. *Arch Phys Med Rehabil* 1993;74:805-9.
12. Grover J, Gellman H, Waters RL. The effect of a flexion contracture of the elbow on the ability to transfer in patients who have quadriplegia at the sixth cervical level. *J Bone Joint Surg Am* 1996;78:1397-400.
13. Denes Z. [Consequence of secondary complications during the rehabilitation of patients with severe brain injury]. *Orv Hetil* 2009;150:165-9.
14. Clavet H, Hebert PC, Fergusson D, Doucette S, Trudel G. Joint contracture following prolonged stay in the intensive care unit. *CMAJ* 2008;178:691-7.
15. Clavet H, Hebert PC, Fergusson DA, Doucette S, Trudel G. Joint contractures in the intensive care unit: association with resource utilization and ambulatory status at discharge. *Disabil Rehabil* 2011;33:105-12.
16. Yarkony GM, Sahgal V. Contractures. A major complication of craniocerebral trauma. *Clin Orthop Relat Res* 1987:93-6.
17. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996;119 ( Pt 5):1737-49.
18. Yip B, Stewart DA, Roberts MA. The prevalence of joint contractures in residents in NHS continuing care. *Health Bull (Edinb)* 1996;54:338-43.
19. Farmer SE, James M. Contractures in orthopaedic and neurological conditions: a review of causes and treatment. *Disabil Rehabil* 2001;23:549-58.
20. Singer BJ, Dunne JW, Singer KP, Jegasothy GM, Allison GT. Non-surgical management of ankle contracture following acquired brain injury. *Disabil Rehabil* 2004;26:335-45.
21. Fergusson D, Hutton B, Drodge A. The epidemiology of major joint contractures: a systematic review of the literature. *Clin Orthop Relat Res* 2007:22-9.

22. Sackley C, Brittle N, Patel S, et al. The prevalence of joint contractures, pressure sores, painful shoulder, other pain, falls, and depression in the year after a severely disabling stroke. *Stroke* 2008;39:3329-34.
23. Kwah LK, Harvey LA, Diong JH, Herbert RD. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J Physiother* 2012;58:41-7.
24. Tabary JC, Tabary C, Tardieu C, Tardieu G, Goldspink G. Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. *J Physiol* 1972;224:231-44.
25. Tabary JC, Tardieu C, Tabary C, Lombard M, Gagnard L, Tardieu G. Neural regulation and adaptation of the number of sarcomeres of the muscle fiber to the length imposed upon it. *J Physiol* 1972;65:Suppl 1:168A.
26. Williams PE, Catanese T, Lucey EG, Goldspink G. The importance of stretch and contractile activity in the prevention of connective tissue accumulation in muscle. *J Anat* 1988;158:109-14.
27. Williams PE, Goldspink G. Changes in sarcomere length and physiological properties in immobilized muscle. *J Anat* 1978;127:459-68.
28. Williams PE, Goldspink G. The effect of immobilization on the longitudinal growth of striated muscle fibres. *J Anat* 1973;116:45-55.
29. Williams PE. Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. *Ann Rheum Dis* 1990;49:316-7.
30. Katalinic OM, Harvey LA, Herbert RD, Moseley AM, Lannin NA, Schurr K. Stretch for the treatment and prevention of contractures. *Cochrane Database Syst Rev* 2010;9:CD007455.
31. Singer BJ, Jegasothy GM, Singer KP, Allison GT, Dunne JW. Incidence of ankle contracture after moderate to severe acquired brain injury. *Arch Phys Med Rehabil* 2004;85:1465-9.
32. Winters TF, Jr., Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. *J Bone Joint Surg Am* 1987;69:437-41.

33. Higginson JS, Zajac FE, Neptune RR, Kautz SA, Burgar CG, Delp SL. Effect of equinus foot placement and intrinsic muscle response on knee extension during stance. *Gait Posture* 2006;23:32-6.
34. Moseley A, Wales A, Herbert R, Schurr K, Moore S. Observation and analysis of hemiplegic gait: stance phase. *Aust J Physiother* 1993;39:259-67.

## **Chapter 2**

### **Literature Review**

#### **2.1 Acquired brain injury**

##### **2.1.1 Overview**

Acquired brain injury is damage to the brain that occurs after birth. It encompasses a variety of conditions such as stroke, traumatic brain injury, hypoxia, substance abuse, toxic exposure, dementia, diffuse infectious disorders and brain tumours. Acquired brain injury is unrelated to congenital disorders, developmental disabilities, or processes that progressively damage the brain.<sup>1</sup> Stroke and traumatic brain injury are the most common forms of acquired brain injury.<sup>2</sup> Stroke results from an abnormality of a cerebrovascular origin in the brain, whereas traumatic brain injury is caused by a trauma from an external force to the brain such as motor vehicle accident, fall and assault. Both conditions bear similar clinical characteristics including sudden disturbances of cerebral functions and signs of upper motor neuron lesions. However, traumatic brain injury, owing to the more diffuse nature of injury, tends to have greater bilateral cortical involvement and often results in more diverse loss of motor control than stroke.<sup>3</sup>

Acquired brain injury is a major cause of disability worldwide.<sup>4</sup> In Australia, 2.2% of the population (that is, 432,700 people) had an acquired brain injury with limitation of activities (disability) and reduced participation (handicap) in 2003.<sup>5</sup> The economic consequences are enormous as people with acquired brain injury are more likely to need assistance with mobility, self-care, daily activities and societal participation than people with disability generally.<sup>5</sup> Stroke alone contributes to approximately 25% of chronic disability costing over AUD 1.3 billion annually.<sup>6</sup> The total estimated cost of traumatic brain injury is over AUD 8.6 billion while the estimated costs of long-term care for people with moderate and severe traumatic brain injury is over AUS 300 million and 900 million, respectively.<sup>7</sup> Improving the health outcomes of people with acquired brain injury would help optimise individuals' independence and also ease the economic burden on society.

### **2.1.2 Impairments after acquired brain injury**

Neurological impairments following an acquired brain injury can be classified as primary and secondary. Primary impairments are deficits that result directly from the brain lesion. There are many types of primary impairments. Motor impairments are amongst the most common and widely recognised primary impairments. Examples of motor impairments are muscle weakness, spasticity and reduced dexterity. They affect an individual's physical capacity to perform everyday activities and thus are a main source of disability. Therefore, much of the focus of rehabilitation is on the management of motor impairments and recovery of the associated functions. However, cognitive impairments, like confusion, loss of memory, reduced executive function and poor concentration, are also common after acquired brain injury.<sup>8-14</sup> These

cognitive and behavioural sequelae can impact on how a person with acquired brain injury can participate in rehabilitation.

Secondary impairments are complications caused by primary impairments, and thus potentially can be minimised or prevented. Like primary impairments, secondary impairments play a major role in determining a person's ultimate level of independence and quality of life. Hence, prevention and management of secondary impairments are as important as that of primary impairments. Contractures are amongst the commonest secondary motor impairments in people with severe acquired brain injury.<sup>15</sup> There are many varied definitions of contractures. In this thesis, contractures are defined as a loss in joint range of motion due to changes in the passive mechanical properties of soft tissues spanning joints.<sup>16-20</sup>

## **2.2 Physiology and measurement of contractures**

Movement is essential for maintaining the passive mechanical properties of soft tissues. Animal models show that soft tissues undergo a remodelling process when they are subjected to prolonged immobilisation in a shortened position.<sup>21-29</sup> Within a muscle-tendon unit, there is a loss of the number of sarcomeres in series,<sup>19,23,25,30,31</sup> and a reduction in tendon length<sup>32</sup> resulting in an overall shortening of the unit.<sup>26</sup> There are also increases in the proportion of intramuscular connective tissue,<sup>19,21,33</sup> the number of perpendicularly oriented collagen fibers<sup>34,35</sup> and cross-bridges between collagen fibres.<sup>36</sup> Both the perimysium and endomysium thicken.<sup>33,35,37</sup> In the periarticular connective tissues, the amount of collagen, water and other

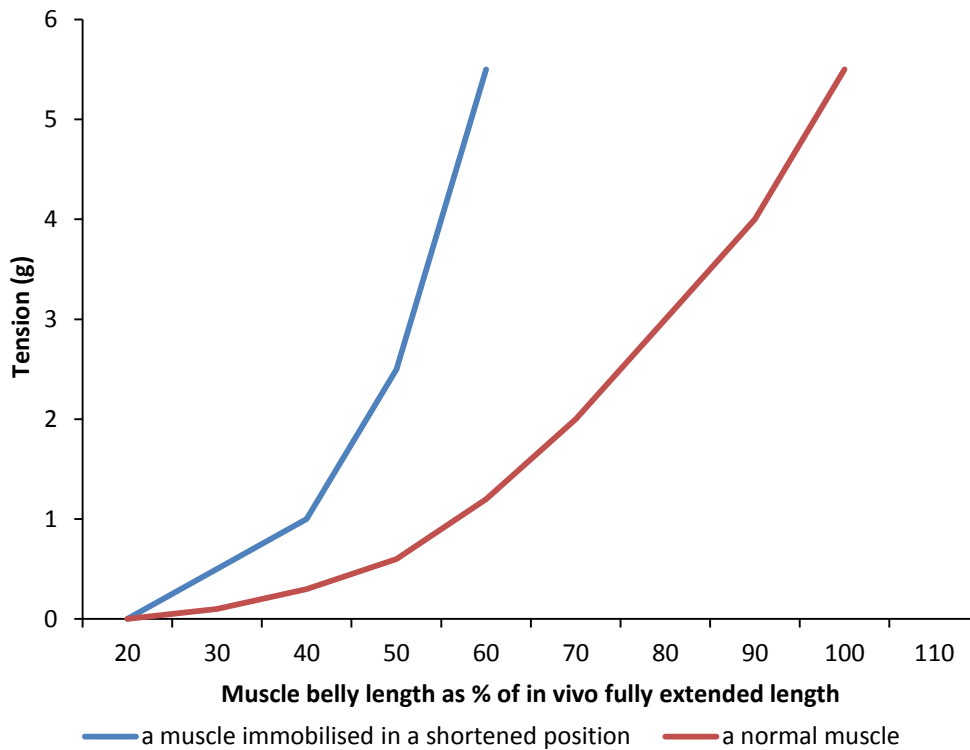
extracellular components reduces while cross-links between fibres increases.<sup>28,29,36</sup> In the capsule and synovium, there are disordered deposition of fibrils, adhesions and reduced gliding between fibres.<sup>16,28</sup> These morphological alterations change the passive mechanical properties of soft tissues. Notably, there are changes in the length and passive stiffness of soft tissues resulting in a reduced length at a given tension. The term extensibility is commonly used to capture these passive mechanical changes in soft tissues. Reduced extensibility in soft tissue structures spanning joints restricts joint range of motion and results in contractures.

The morphological changes associated with contractures have primarily been investigated in animals because these types of investigations require invasive procedures which are not possible in humans. Of late, advancements in medical technologies have permitted investigations in human muscles using non-invasive methods. A study using ultrasound imaging on people with stroke showed that contractures are due to reductions in the length of muscles.<sup>38</sup> This finding is consistent with that observed in animal models.

Immobilisation in a shortened position leads to an increase in passive stiffness of muscles.<sup>18,25,39,40</sup> Passive stiffness is a measure of the passive length-tension properties of a muscle, that is, the tension encountered in a muscle at different lengths without any muscle activity.<sup>25,26</sup> It is described as the ratio of change in tension to change in length. Stiffness is the slope of a length-tension curve. Increases in passive stiffness are demonstrated by increases in the steepness of the length-tension curves. Figure 1 shows a length/tension curve of a mouse muscle immobilised in a shortened position and comparison is made with that of a normal muscle.<sup>25</sup> A muscle immobilised in a shortened position shows a steeper slope. The curve also

shifts to the left of that of a normal muscle. These changes are due to a loss of sarcomeres, and the remaining sarcomeres are stretched.

Figure 1. Length/tension curves for a muscle immobilised in a shortened position and a normal muscle (control).<sup>25</sup>

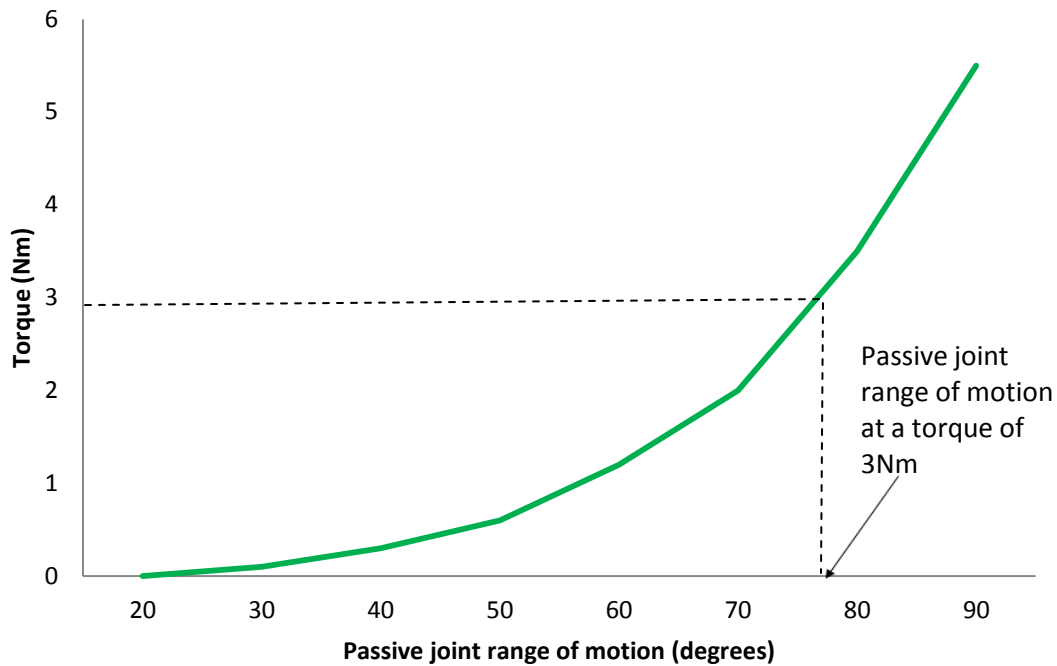


Measurements of passive stiffness are a good way to demonstrate changes in the passive mechanical properties associated with contractures. Passive stiffness is the slope of the torque-displacement curve of a joint (Figure 2). However, the measurement procedure is complex and time-consuming as it involves repeated measurements of passive joint range of motion at various torques or it involves continuous data sampling of both displacement and torque. For



practical reasons, clinicians often choose to measure one point on the torque/displacement curve instead, that is, measure passive joint range with a single known torque as illustrated in Figure 2. Standardisation of torque is an important consideration when using passive joint range of motion to reflect soft tissue extensibility. It minimises measurement errors associated with uncontrolled torques and altered stretch tolerance of an individual.<sup>41-43</sup> Passive range of motion measures the extensibility of all soft tissues spanning the joint, not just muscles. However, muscles that span more than one joint offer a unique opportunity for passive joint range of motion to quantify muscle extensibility by manipulating the position of the joints spanned by the muscles. For instance, extensibility of a bi-articular muscle like gastrocnemius can be reflected by the change in the passive ankle joint range of motion measured with the knee joint flexed and then with the knee extended.<sup>44</sup> This change in passive joint range of motion in one joint resulting from another joint being manipulated into varied positions also highlights the importance of consistent positioning of joints while measuring the target joint when assessing extensibility of muscles than span over more than one joint.

Figure 2. Torque/displacement curve for the ankle joint with the knee extended.<sup>45</sup>



### 2.3 Epidemiology of contracture after acquired brain injury

Contractures are common following acquired brain injury.<sup>46</sup> Up to 84% of patients with craniocerebral trauma<sup>47</sup> and about 50% of patients with stroke develop contractures.<sup>48-51</sup> Reports of incidence however vary substantially.<sup>50</sup> For instance, one year after stroke the incidence ranged from 23%<sup>52</sup> to 60%.<sup>51</sup> Variability also exists in the reported incidence of contracture for specific joints. For example, the reported incidence of ankle contractures after traumatic brain injury varies from 16%<sup>53</sup> to 76%.<sup>47</sup> At present, there is a lack of large multicentre or multi-country cohort studies. Thus most reported incidences have been based

on data collected from individual health institutions which have different standards of care and admission criteria. In addition, while some studies use representative samples by including all consecutive admissions, others use samples of convenience which might not be representative. Other factors which may contribute to the variability in results include differences in the way contractures are defined and measured, and differences in the characteristics of the cohorts (such as, severity of brain injury and stage of recovery). It is, therefore, hardly surprising to see a wide range in the reported incidence of contractures. Notwithstanding the variability, these figures serve to illustrate that contractures are a common secondary impairment of acquired brain injury.

## **2.4 Contributors to the development of contracture after acquired brain injury**

### **2.4.1 Muscle weakness and associated loss of motor function**

Muscle weakness is a typical primary motor impairment of acquired brain injury. Muscle weakness following acquired brain injury is due to the altered neural input secondary to damage to the brain which compromises neuronal recruitment and volitional muscle activation. Up to 81% of individuals with stroke experience muscle weakness on the side contralateral to the cerebral lesion.<sup>54</sup> Following an acquired brain injury, muscles can further be weakened through disuse and immobility. Muscle weakness reduces movements, and in severe cases immobilises a limb and deprives soft tissues of stretch. This predisposes muscles placed in shortened positions to contractures. Similarly, muscle imbalance places joints in fixed positions

and increases the risk of contractures. A number of studies have demonstrated the close relationship between contractures and muscle weakness or related motor deficits such as reduced dexterity functional ability and mobility.<sup>49-51,55-60</sup> Not surprisingly, people without early signs of motor recovery are prone to contractures.<sup>57,58,61</sup>

## **2.4.2 Spasticity**

Spasticity is another common primary impairment of acquired brain injury, and is thought to be a result of neural reorganisation after brain lesions.<sup>62-64</sup> The most widely accepted definition of spasticity is “a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome”.<sup>65</sup> Following a stroke, 19 to 30% of people develop spasticity within 3 months, and the incidence of spasticity increases to as high as 39% after 12 months.<sup>48,66-72</sup> The incidence of spasticity is even higher in people with severe traumatic brain injury, with up to 88% of them developing spasticity within 14 days of injury.<sup>73</sup> Spasticity, like muscle weakness, can induce contractures by immobilising a limb in a fixed position<sup>16</sup> but the remodelling process is likely accelerated by the chronic tonic contractile activity associated with spasticity.<sup>24,74</sup> Spasticity may further contribute to contractures through an alteration of muscle fibre composition<sup>75-79</sup> and increased cross-bridge attachments.<sup>80</sup>

The association of spasticity with contractures has been well documented.<sup>17,48,76,81-88</sup> Both spasticity and contractures contribute to hypertonia;<sup>89</sup> a term that describes the increased resistance of muscles encountered during passive lengthening in people with neurological impairments.<sup>90</sup> Contractures contribute to hypertonia through the increased passive stiffness

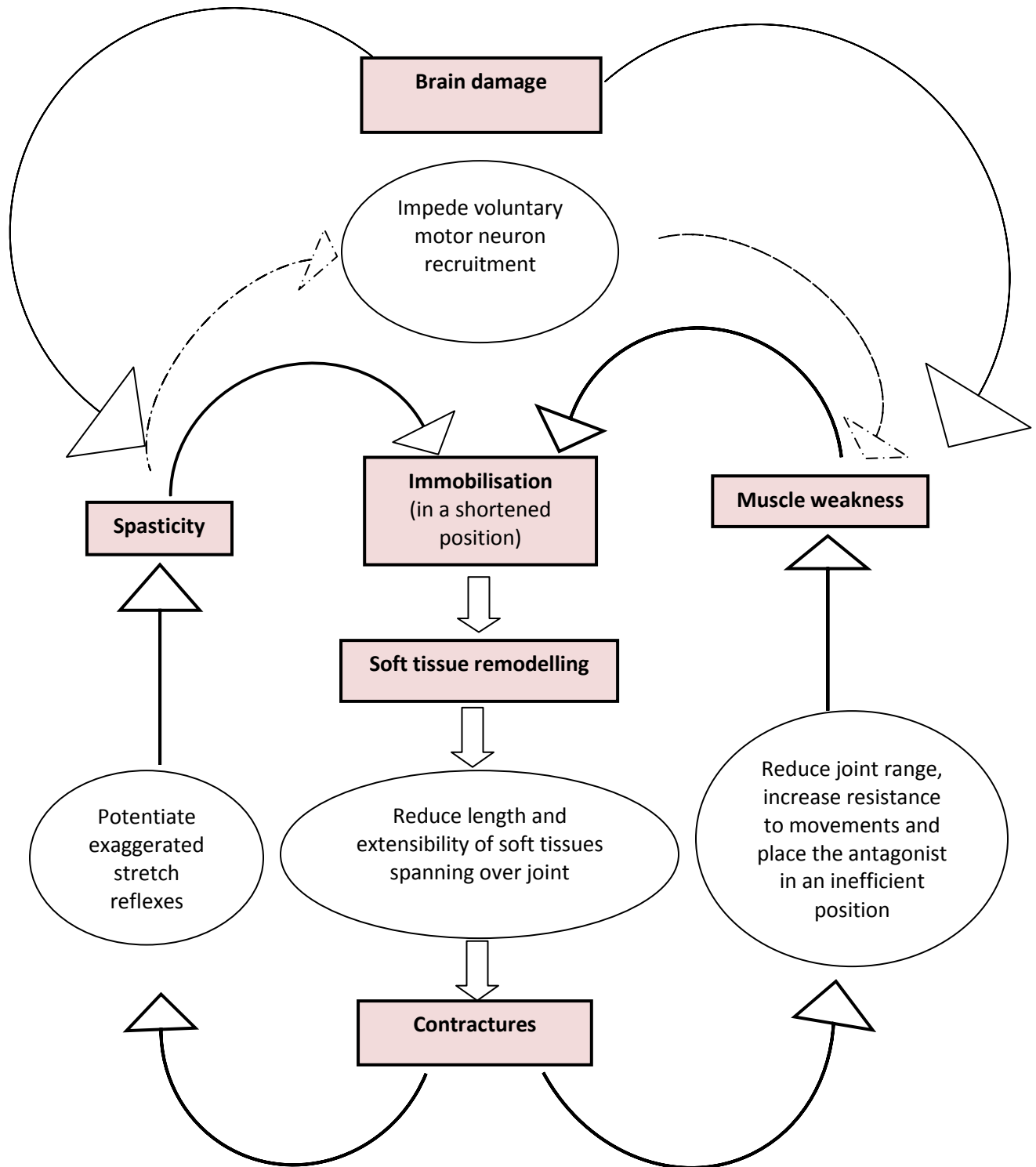
secondary to immobilisation in shortened positions. Whereas, spasticity contributes to hypertonia through hyperexcitable stretch reflex such as hypersensitive muscle spindles and increased tonic muscle activation.<sup>89</sup> Importantly, laboratory examinations of the passive properties of spastic muscles have found significant structural alterations similar to that associated with contractures including shorter resting sarcomere length, greater passive modulus, shorter fascicular length, increased extracellular matrix compliance and endomysial connective tissue proliferation than normal muscles.<sup>91-94</sup> Soft tissues of hemiplegic patients with spasticity have been found to be nearly three times stiffer than that of the age matched controls.<sup>76</sup> All these observations suggest that spasticity plays a significant role in contracture development after acquired brain injury.

### **2.4.3 Combined factors**

Spasticity and muscle weakness are both common primary motor impairments and often co-exist after an acquired brain injury. They individually or concomitantly predispose soft tissues to shortening. Their contribution to contractures has been confirmed by two longitudinal studies. One study identifies spasticity as the main contributor in the first 4 months after stroke and thereafter muscle weakness.<sup>49</sup> The other study reports muscle weakness as the most significant predictor for contractures after stroke, and up to 20% of variation in contracture development can be explained by strength, spasticity, age, pre-morbid function and pain.<sup>50</sup> Importantly, it is the interactions amongst spasticity, weakness and contractures that may add complexity to this problem. The theoretical chains of events are illustrated in Figure 3. Contractures make movements difficult by decreasing the available range of motion in the joint and increasing

resistance to movement.<sup>48,82</sup> Contractures also place the antagonists in a lengthened position making the muscles less efficient in generating tension to produce movements.<sup>25,95</sup> Thus, contractures are likely to promote further immobilisation, cause increased muscle weakness and therefore exacerbate contractures. Thereby a vicious cycle is set up. Similarly, contractures enhance spindle stimulation and cause excessive responsiveness to stretch which in turn can potentiate spasticity.<sup>48,96-101</sup> This excessive responsiveness to stretch may also impede voluntary motor neuron recruitment resulting in further loss of muscle strength and movements, and thus contractures.<sup>102</sup> In summary, contractures are a complex issue involving multiple factors which may reciprocally potentiate and exacerbate each other.<sup>89</sup> These perhaps are the reasons why contractures are difficult to treat. Solutions to contractures may require effective strategies to address not only soft tissue shortening but their contributors so that these chains of events can be interrupted.

Figure 3. A theoretical model of events contributing to the development of contractures after acquired brain injury.



## **2.5 Passive stretch as treatment for contractures**

### **2.5.1 Rationale for clinical use of passive stretch**

Passive stretch is the application of sustained tension to soft tissues in lengthened positions. Animal models indicate that sustained passive stretch increases sarcomere number,<sup>19,21-23,25,103-105</sup> induces muscle growth,<sup>106-109</sup> retards immobilisation-induced atrophy,<sup>26</sup> promotes synthesis of collagen and other extracellular components, orientates collagen to the lines of stress, increases tensile strength and water content within the periarticular tissues, and prevents accumulation of connective tissues.<sup>103,110,111</sup> All these findings suggest that soft tissues undergo structural remodelling and elongate when stretched. This underpins the rationale for using passive stretch for contracture management.

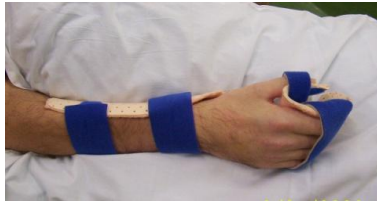
### **2.5.2 Clinical application of passive stretch**

Passive stretch has been the mainstream treatment for management of contractures after acquired brain injury. Muscles have an elastic property. The response of muscles to passive stretch can vary depending on how passive stretch is applied. Brief duration of passive stretch can lead to an immediate increase in joint mobility through viscoelastic deformation of muscles.<sup>112,113</sup> However, the effect of such response is only transient and has limited clinical value. Sustained passive stretch, as observed in animal models,<sup>21,23,25,26,103,104</sup> may induce structural remodelling of muscles. Hence, clinical stretch interventions typically subject muscles to prolonged controlled stretch, with an aim to achieve a lasting increase in muscle length and joint range of motion.



Commonly used stretch interventions include positioning, tilt table standing, splinting and serial casting (Figure 4). Positioning of muscles in a lengthened position is a commonly used stretch method. Examples of this are positioning of shoulders in external rotation in lying and in forward flexion in sitting. Light weights are often applied over the limb to maintain the position. Tilt table standing makes use of an individual's body weight to stretch the ankle plantarflexors. A wedge can be added under the feet to increase the amount of stretch on the ankle plantarflexors. Duration of positioning and tilt table standing is arbitrary and usually ranges from 30 minutes to 60 minutes within a session. Splints generally are used to apply low-load stretch for a number of hours a day. Duration of splinting is usually dictated by an individual's tolerance and skin condition. They are often applied overnight so as not to interfere with therapy. Serial casting is a technique which involves application of successive casts to gradually stretch muscles. During the casting process, passive stretch is applied 24 hours a day over days or weeks. It is not uncommon for people with acquired brain injury to receive multiple stretch interventions to manage their contractures (Figure 4).

Figure. 4. Commonly used stretch interventions.



Hand splinting



Ankle splinting



Knee splinting



Serial casting



Stretching of ankle plantarflexors making use of body weight

Multiple stretch interventions: tilt table standing, positioning of shoulder on a table, serial casting for elbow and splinting for hand.

There are many challenges that confront clinicians in the administration of passive stretch for people with acquired brain injury. Firstly, the dosage of passive stretch is difficult to quantify as it is influenced by many variables including application methods, intensity of force, session duration, treatment frequency and length of intervention period. Secondly, there is a lack of guidance on the dose of passive stretch. While not proven, the dose is likely to vary depending on a number of factors including muscle type and size, age, diagnosis, amount of muscle weakness, loss of dexterity and spasticity, size and location of brain lesion, and severity and chronicity of contractures. At present, there is limited knowledge that guides clinical applications of passive stretch on people with acquired brain injury. There are suggestions that frequency and duration of passive stretch are more important than intensity,<sup>114</sup> and prolonged low-load passive stretch is more effective than high-load brief passive stretch.<sup>115,116</sup> However, this information is based on geriatric populations. A randomised controlled trial investigated the effect of different doses of passive stretch on people with traumatic brain injury. In the trial, 24 hours of passive stretch using serial casting was compared with an hour of stretching using positioning or splinting. It found a treatment effect of 22 degrees, (95% CI 13 to 31) in favour of serial casting, one day post-intervention. When the dose of passive stretch was reduced after 2 weeks, this effect dissipated. This finding suggests that higher dose is more effective than lower dose but this requires more investigation.<sup>117</sup> In addition, the dosage of stretch that is required to achieve a lasting treatment effect remains undetermined. There is also uncertainty regarding stretch methods. Very few studies compared one stretch intervention with another. One trial compared 30 minutes of tilt table standing with night splinting, and found no convincing difference in their effectiveness in preventing ankle

contractures after stroke (1 degree, 95% CI -5 to 7).<sup>118</sup> The authors concluded that both interventions were equally effective however without a control group it is not known whether either intervention was effective.

The lack of clear evidence has led to many different methods and dosages of stretch being used in clinical practice. Therapists' decisions are generally based on anecdotal evidence, clinical experience, personal preference, training, trends of practice or treatment endorsed by particular institutions.

### **2.5.3 Effectiveness of stretch interventions**

A systematic review summarised the findings of 25 randomised controlled trials that investigated the effectiveness of clinical stretch interventions on neurological populations, namely acquired brain injury (stroke, traumatic brain injury), spinal cord injury, Duchenne muscular dystrophy, cerebral palsy and Charcot Marie-Tooth disease.<sup>119</sup> The meta-analyses revealed an immediate treatment effect (defined as <24 hours) of 3 degrees (95% CI 0 to 5); a short-term effect (defined as >24 hours and < 1 week) of 1 degree (95% CI 0 to 3); and a medium-term effect (defined as >1 week) of 0 degree (95% CI -2 to 2). This indicates that passive stretch interventions, as typically administered in clinical settings, do not produce clinically important effects in people with neurological conditions.

The findings of the systematic review contradict the general belief that passive stretch is effective for the prevention and correction of contractures. However, the trials included in the review are of varying quality (PEDro scale<sup>120</sup> ratings between 4/10 and 8/10) so perhaps were vulnerable to bias. The critical question remains: is passive stretch really ineffective? One

possible reason for the findings of the systematic review is that the dosage of passive stretch used was insufficient. There were considerable variability amongst the clinical trials included in the systematic review in terms of stretch methods, session duration, treatment frequency and length of intervention period. Thus, the dosage of stretch used in these trials was difficult to quantify. The systematic review used total stretch time to quantify dose and reported that the median dose of passive stretch was 6.5 hours a day over 30 days. In addition, most of the included trials involved the application of stretch interventions for less than 3 months, and none exceeded 7 months.<sup>119</sup> This dosage may be insufficient. The argument for large doses of passive stretch is supported by the two trials which investigated passive stretch administered for 24 hours a day using serial casting over 1 to 2 weeks.<sup>117,121</sup> Both trials reported a better short-term effect than no stretch or an hour a day of stretch (15 degrees, 95% CI 6 to 23 degrees; 22 degrees, 95% CI 13 to 31). These trials suggest that an intensive program of passive stretch can be effective. One of these trials<sup>117</sup> was excluded from the systematic review because it compared one passive stretch program with another. While the systematic review shows that passive stretch at the doses typically applied in clinical settings is ineffective, no trial has investigated the effectiveness of high intensity passive stretch administered over extended periods of time.

Another plausible explanation for the findings of the systematic review is that contractures are a complex problem involving multiple factors. Spasticity and muscle weakness are amongst the likely contributors. Passive stretch focuses predominantly on reversing soft tissue shortening but does not address the underlying causes. The continual presence of the contributors to contracture may be the reason why passive stretch alone cannot produce a large or sustained

treatment effect on joint range of motion. Hence, an approach combining passive stretch with treatments that address the contributors to contracture may be more effective than passive stretch alone. Evidence for this combined treatment strategy, however, is lacking and warrants investigation. Research is also required to identify treatments that can be used in combination with passive stretch. Electrical stimulation appears to be a suitable choice.

## **2.6 Electrical stimulation as a treatment modality for contracture management**

### **2.6.1 Electrical stimulation and muscle strength**

Electrical stimulation may improve muscle strength of people with neurological disorders because it can elicit strong muscle contractions even when individuals have little or no ability to voluntarily activate muscles.<sup>122</sup> A systematic review analysed the findings of 11 randomised controlled trials, and concluded that electrical stimulation might have a modest beneficial effect in improving muscle strength after stroke.<sup>123</sup> A more recent randomised controlled trial, not included in the systematic review, also indicates electrical stimulation may improve muscle strength in patients with severe stroke although the lower end of the 95% CI suggests some uncertainty (0.5N, 95% CI 0.0 to 1.0).<sup>124</sup> People with contractures often have limited ability to participate in active treatment including conventional strengthening exercises. Electrical stimulation is seemingly an appropriate adjunct modality to address muscle weakness, a plausible contributor to contractures.

## **2.6.2 Electrical stimulation and spasticity**

Electrical stimulation appears to reduce spasticity in people with stroke. Five randomised controlled trials assessed electrical stimulation applied 9 to 60 minutes a day over 3 to 12 weeks. All reported small reductions in spasticity following electrical stimulation when compared to no electrical stimulation or placebo, as reflected by the Modified Ashworth Scale or composite spasticity scale scores.<sup>125-129</sup> The quality of these trials is variable with the PEDro scale<sup>120</sup> scores ranging between 4/10 and 8/10. Only one trial investigated the long-term effects of electrical stimulation on spasticity and reported that the effect dissipated within 3 months after cessation of treatment.<sup>129</sup> Nevertheless, these studies all suggest that electrical stimulation can reduce spasticity at least over the short-term.

## **2.6.3 Electrical stimulation and contracture management**

Electrical stimulation may be useful for contracture management. The first randomised controlled trial investigated the effects of 30 minutes, 5 days a week for 4 weeks, of electrical stimulation on adolescents with cerebral palsy and chronic knee contractures.<sup>130</sup> The finding (4 degrees, 95% CI 0 to 7) was small and inconclusive as indicated by the lower end of 95% confidence interval. The chronicity of contractures, however, may have an impact on outcome. The second randomised controlled trial investigated the effect of electrical stimulation on the passive ankle dorsiflexion range of motion after stroke. It reported a small treatment effect (5 degrees, 95% CI 4 to 7).<sup>126</sup> In this trial, 20 sessions of 9 minutes of supramaximal dose (that is, 20% over that which was required to produce maximum muscle contraction) of electrical stimulation was applied. Supramaximal dose is rarely used clinically because it is

uncomfortable. It is unclear how the researchers overcame this problem. It would be of a clinical interest to determine if a longer but more readily tolerable dose of electrical stimulation is therapeutic. It would also be useful to determine the effect of combining electrical stimulation with passive stretch as no trial has investigated this.

## **2.7 Impact of ankle contractures on gait**

Improving mobility is a common goal in rehabilitation. In this section, a specific focus is placed on the impact of ankle contractures on walking. Ankle contractures are chosen because they are common after acquired brain injury.<sup>47,50</sup> Adequate ankle dorsiflexion motion is required to perform various daily mobility tasks efficiently. Kinematic data suggest that approximately 30 degrees of ankle dorsiflexion is required to stand up from sitting,<sup>131</sup> 5 to 15 degrees to walk,<sup>132</sup> and 27 degrees to descend stairs in a normal fashion.<sup>133</sup> Hence, ankle contractures can be a major source of disability after an acquired brain injury.<sup>134-136</sup> Walking is a function that involves the interaction of multiple joints. A restriction in movement of one joint inevitably influences the position and movement of other joints leading to a change in the total gait pattern. Previous observational analyses have suggested that ankle contractures are a common cause of gait deviations following stroke.<sup>137-139</sup> However, the impact of ankle contractures on the kinematics at other joints and its contribution to secondary gait deviations is not fully understood.



There is a general view that ankle contractures are closely related to knee hyperextension gait,<sup>140,141</sup> a common gait disorder that is found in 40 to 60% of people with stroke.<sup>142</sup> A study observed 13 patients with stroke and ankle contractures and found that they all walked with increased knee extension (or hyperextension) in stance phase.<sup>140</sup> This observation supports a link between ankle contractures and knee hyperextension. However, the same is not observed in people with cerebral palsy and ankle contractures: while some of them walk with knee hyperextension, others walk with increased knee flexion.<sup>136,143-145</sup> The limitation of these clinical observations is that people with neurological disorders almost always have other impairments, like motor weakness, reduced dexterity and spasticity, which also influence their gait. Thus it is difficult to isolate the effect of ankle contractures in clinical populations.

There are non-clinical studies that have investigated the relationship between ankle contractures and knee biomechanics. Using a mathematical model, one study concluded that ankle contractures led to knee hyperextension.<sup>141</sup> However, this finding was refuted by another study which simulated ankle contractures on 12 healthy people and found a significant increase of knee flexion in stance phase instead.<sup>146</sup> Taping was used in this study to restrict ankle dorsiflexion. One drawback of taping is that it can cause discomfort or even pain as it pulls on the skin upon weight bearing. Therefore, participants might have inadvertently held their ankles in plantarflexion to lessen the discomfort. Thus the observed gait pattern might not be a result of ankle contractures only. At present, there is little and conflicting information regarding the relationship between ankle contracture and knee biomechanics. Further investigations are needed to help clarify the impact of ankle contractures on the knee joint. An ankle-foot-orthosis is probably a better choice to simulate ankle contracture than taping because it does

not cause discomfort. An ankle-foot-orthosis also has the advantage that the degree of imposed ankle restriction can be quantified and systematically adjusted. A biomechanical analysis using an ankle-foot-orthosis to simulate ankle contractures will improve the understanding of the changes in knee biomechanics and spatiotemporal characteristics secondary to ankle contractures. It will also be interesting to investigate how the severity of ankle contractures influences the extent of changes in the knee biomechanics as this investigation has not been done previously.

## **2.8 Summary**

This literature review provided an overview of the existing knowledge and research findings regarding contractures. The first part of this thesis focuses on physiotherapy management of contractures. Passive stretch has a sound physiologic rationale and has been the mainstream treatment for contracture management. However, a systematic review did not find passive stretch, as typically applied in the clinic, to be effective. To date, no studies have investigated the administration of high intensity passive stretch over an extended period of time. It is possible that the lack of effect is due to insufficient dosage. Further investigations on passive stretch of large dosages are warranted before a definitive conclusion can be drawn. Moreover, contractures are a complex problem involving multiple factors. Passive stretch focuses predominantly on reversing soft tissue shortening but does not address any of the underlying causes of contracture. This may be the reason why passive stretch alone fails to produce a large

or sustained effect. An approach that combines passive stretch with treatments that target the contributors to contracture development may be more effective than passive stretch alone. Electrical stimulation has potential therapeutic effects on strength and spasticity; the factors believed to contribute to contractures. It is seemingly an appropriate adjunct modality to consider. Investigations of the role of electrical stimulation as an adjunct treatment and the effectiveness of combining passive stretch with electrical stimulation on contracture management may be useful.

The second part of the thesis focuses on the impact of contractures on walking. Ankle contractures are a common problem after acquired brain injury and yet their effect on knee biomechanics is unclear. Improving mobility is a common goal in rehabilitation. Investigation of how ankle contractures affect the kinematics and kinetics of the knee joint will improve the understanding of the mechanisms underlying some of the common gait deviations seen in clinical populations.

## 2.9 Aims of the thesis

There are two parts of the thesis. The first part of the thesis focuses on identifying effective treatment strategies for contractures. The second part of the thesis aims to improve the understanding of the impact of ankle contractures on the knee joint during gait. The following is the outline of these studies.

Chapter 3: A case report describes an adolescent who underwent an intensive treatment program for his severe chronic knee contractures. The aim of the case report is to illustrate the importance of using an adequate dose of passive stretch and concomitant treatment to address the contributors to contracture in the management of contractures.

Chapter 4: A randomised controlled study aims to investigate the benefit of electrical stimulation as an adjunct treatment for wrist contractures. Electrical stimulation was used in addition to a program of splinting. This was compared with a program of splinting alone.

Chapter 5: A randomised controlled study aims to determine the effectiveness of a combined program for ankle contracture management. The program consisted of electrical stimulation, splinting and tilt table standing. This was compared with tilt table standing alone.

Chapter 6: A biomechanical study aims to evaluate the impact of ankle contractures on kinematics and kinetics of the knee joint in the sagittal plane during stance phase. Healthy subjects were recruited so that the effect of ankle contractures could be examined without confounding factors. An ankle-foot-orthosis was used to simulate and quantify ankle

contractures (of two severities). Comparisons were made between walking with and without restrictions of ankle dorsiflexion.

The overall objective of this thesis is to improve the health outcome of people with acquired brain injury through a better understanding of contractures and their management.

## 2.10 References

1. Definition of acquired brain injury. Toronto Acquired Brain Injury Network. 2005.
2. Turner-Stokes L. Rehabilitation following acquired brain injury: National Clinical Guidelines. London (UK): Royal College of Medicine and the British Society of Rehabilitation Medicine. 2003.
3. Boake C, Francisco GE, Ianhoe CB, Korhari S. Brain injury rehabilitation. In: Braddorn RL, ed. Physical medicine and rehabilitation Toronto: Saunders Company; 2000:1073-116.
4. International Brain Injury Association. at <http://www.internationalbrain.org/?q=Brain-Injury-Facts>.)
5. The Australian Bureau of Statistics' 2003 Survey of Disability, Aging and Carers. Australian Bureau of Statistics. 2003.
6. Aboriginal Stroke Project Steering Committee. National stroke unit program: Aboriginal stroke project. National Stroke Foundation of Australia. 2004.
7. Access Economics. The economic cost of spinal cord injury and traumatic brain injury in Australia. Reported by Access Economics for the Victorian Neurotrauma Initiative. Canberra: Access Economics 2009:31.

8. Mueller JA, Dollaghan C. A systematic review of assessments for identifying executive function impairment in adults with acquired brain injury. *J Speech Lang Hear Res* 2013;56:1051-64.
9. Chung C, Pollock A, Campbell T, Durward B, Hagen S. Cognitive rehabilitation for executive dysfunction in adults with stroke or other adult nonprogressive acquired brain damage. *Stroke* 2013;44:e77-8.
10. Mioni G, Rendell PG, Henry JD, Cantagallo A, Stablum F. An investigation of prospective memory functions in people with traumatic brain injury using Virtual Week. *J Clin Exp Neuropsychol* 2013;35:617-30.
11. McDonald S. Impairments in social cognition following severe traumatic brain injury. *J Int Neuropsychol Soc* 2013;19:231-46.
12. Deb S, Lyons I, Koutzoukis C. Neurobehavioural symptoms one year after a head injury. *Br J Psychiatry* 1999;174:360-5.
13. Spitz G, Schonberger M, Ponsford J. The relations among cognitive impairment, coping style, and emotional adjustment following traumatic brain injury. *J Head Trauma Rehabil* 2013;28:116-25.
14. Ruttan L, Martin K, Liu A, Colella B, Green RE. Long-term cognitive outcome in moderate to severe traumatic brain injury: a meta-analysis examining timed and untimed tests at 1 and 4.5 or more years after injury. *Arch Phys Med Rehabil* 2008;89:S69-76.
15. Denes Z. [Consequence of secondary complications during the rehabilitation of patients with severe brain injury]. *Orv Hetil* 2009;150:165-9.
16. Botte MJ, Nickel VL, Akesson WH. Spasticity and contracture. Physiologic aspects of formation. *Clin Orthop Relat Res* 1988:7-18.
17. Harburn KL, Potter PJ. Spasticity and contractures. *Phys Med Rehabil State of the Art Reviews* 1993;7:113-32.

18. Tardieu G, Tardieu C, Colbeau-Justin P, Lespargot A. Muscle hypoextensibility in children with cerebral palsy: II. Therapeutic implications. *Arch Phys Med Rehabil* 1982;63:103-7.
19. Williams PE. Effect of intermittent stretch on immobilised muscle. *Ann Rheum Dis* 1988;47:1014-6.
20. Dudek N, Trudel G. Joint contractures. In: Frontera W, Silver J, Rizzo T, eds. *Essentials of physical medicine and rehabilitation musculoskeletal disorders, pain, and rehabilitation*. 2nd ed: Philadelphia: Saunders; 2008:651-5.
21. Tabary JC, Tabary C, Tardieu C, Tardieu G, Goldspink G. Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. *J Physiol* 1972;224:231-44.
22. Goldspink G, Tabary C, Tabary JC, Tardieu C, Tardieu G. Effect of denervation on the adaptation of sarcomere number and muscle extensibility to the functional length of the muscle. *J Physiol* 1974;236:733-42.
23. Tabary JC, Tardieu C, Tabary C, Lombard M, Gagnard L, Tardieu G. Neural regulation and adaptation of the number of sarcomeres of the muscle fiber to the length imposed upon it. *J Physiol* 1972;65:Suppl 1:168A.
24. Tabary JC, Tardieu C, Tardieu G, Tabary C. Experimental rapid sarcomere loss with concomitant hypoextensibility. *Muscle Nerve* 1981;4:198-203.
25. Williams PE, Goldspink G. Changes in sarcomere length and physiological properties in immobilized muscle. *J Anat* 1978;127:459-68.
26. Herbert RD, Balnave RJ. The effect of position of immobilisation on resting length, resting stiffness, and weight of the soleus muscle of the rabbit. *J Orthop Res* 1993;11:358-66.

27. Akeson WH, Woo SL, Amiel D, Matthews JV. Biomechanical and biochemical changes in the periarticular connective tissue during contracture development in the immobilized rabbit knee. *Connect Tissue Res* 1974;2:315-23.
28. Akeson WH, Amiel D, Abel MF, Garfin SR, Woo SL. Effects of immobilization on joints. *Clin Orthop Relat Res* 1987:28-37.
29. Akeson WH, Woo SL, Amiel D, Coutts RD, Daniel D. The connective tissue response to immobility: biochemical changes in periarticular connective tissue of the immobilized rabbit knee. *Clin Orthop Relat Res* 1973:356-62.
30. Witzmann FA, Kim DH, Fitts RH. Hindlimb immobilization: length-tension and contractile properties of skeletal muscle. *J Appl Physiol* 1982;53:335-45.
31. Heslinga JW, Huijing PA. Effects of short length immobilization of medial gastrocnemius muscle of growing young adult rats. *Eur J Morphol* 1992;30:257-73.
32. Herbert RD, Crosbie J. Rest length and compliance of non-immobilised and immobilised rabbit soleus muscle and tendon. *Eur J Appl Physiol Occup Physiol* 1997;76:472-9.
33. Goldspink G, Williams PE, eds. *Muscle fibre and connective tissue changes associated with use and disuse*: Oxford: Butterworth-Heinemann; 1990.
34. Williams PE, Goldspink G. Connective tissue changes in immobilised muscle. *J Anat* 1984;138 ( Pt 2):343-50.
35. Jarvinen TA, Jozsa L, Kannus P, Jarvinen TL, Jarvinen M. Organization and distribution of intramuscular connective tissue in normal and immobilized skeletal muscles. An immunohistochemical, polarization and scanning electron microscopic study. *J Muscle Res Cell Motil* 2002;23:245-54.
36. Woo SL, Matthews JV, Akeson WH, Amiel D, Convery FR. Connective tissue response to immobility. Correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees. *Arthritis Rheum* 1975;18:257-64.



37. Goldspink G, Williams PE. The nature of the increased passive resistance in muscle following immobilization of the mouse soleus muscle [proceedings]. *J Physiol* 1979;289:55P.
38. Kwah LK, Herbert RD, Harvey LA, et al. Passive Mechanical Properties of Gastrocnemius Muscles of People With Ankle Contracture After Stroke. *Arch Phys Med Rehabil* 2012;93:1185-90.
39. Halar EM, Stolov WC, Venkatesh B, Brozovich FV, Harley JD. Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons. *Arch Phys Med Rehabil* 1978;59:476-84.
40. O'Dwyer N, Neilson P, Nash J. Reduction of spasticity in cerebral palsy using feedback of the tonic stretch reflex: a controlled study. *Dev Med Child Neurol* 1994;36:770-86.
41. Halbertsma JP, Goeken LN, Hof AL, Groothoff JW, Eisma WH. Extensibility and stiffness of the hamstrings in patients with nonspecific low back pain. *Arch Phys Med Rehabil* 2001;82:232-8.
42. Magnusson SP, Simonsen EB, Aagaard P, Sorensen H, Kjaer M. A mechanism for altered flexibility in human skeletal muscle. *J Physiol* 1996;497 ( Pt 1):291-8.
43. Folpp H, Deall S, Harvey LA, Gwinn T. Can apparent increases in muscle extensibility with regular stretch be explained by changes in tolerance to stretch? *Aust J Physiother* 2006;52:45-50.
44. Ben M. Efficacy of stretch interventions on muscle extensibility: University of Sydney, Australia; 2010.
45. Chesworth BM, Vandervoort AA. Comparison of passive stiffness variables and range of motion in uninvolved and involved ankle joints of patients following ankle fractures. *Phys Ther* 1995;75:253-61.
46. Fergusson D, Hutton B, Drodge A. The epidemiology of major joint contractures: a systematic review of the literature. *Clin Orthop Relat Res* 2007:22-9.
47. Yarkony GM, Sahgal V. Contractures. A major complication of craniocerebral trauma. *Clin Orthop Relat Res* 1987:93-6.
48. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996;119 ( Pt 5):1737-49.

49. Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disabil Rehabil* 2006;28:891-7.
50. Kwah LK, Harvey LA, Diong JH, Herbert RD. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J Physiother* 2012;58:41-7.
51. Sackley C, Brittle N, Patel S, et al. The prevalence of joint contractures, pressure sores, painful shoulder, other pain, falls, and depression in the year after a severely disabling stroke. *Stroke* 2008;39:3329-34.
52. Pinedo S, de la Villa FM. [Complications in the hemiplegic patient in the first year after the stroke]. *Rev Neurol* 2001;32:206-9.
53. Singer BJ, Dunne JW, Singer KP, Jegasothy GM, Allison GT. Non-surgical management of ankle contracture following acquired brain injury. *Disabil Rehabil* 2004;26:335-45.
54. Barker WH, Mullooly JP. Stroke in a defined elderly population, 1967-1985. A less lethal and disabling but no less common disease. *Stroke* 1997;28:284-90.
55. Faghri PD, Rodgers MM, Glaser RM, Bors JG, Ho C, Akuthota P. The effects of functional electrical stimulation on shoulder subluxation, arm function recovery, and shoulder pain in hemiplegic stroke patients. *Arch Phys Med Rehabil* 1994;75:73-9.
56. Linn SL, Granat MH, Lees KR. Prevention of shoulder subluxation after stroke with electrical stimulation. *Stroke* 1999;30:963-8.
57. Pandyan AD, Cameron M, Powell J, Stott DJ, Granat MH. Contractures in the post-stroke wrist: a pilot study of its time course of development and its association with upper limb recovery. *Clin Rehabil* 2003;17:88-95.
58. Turton AJ, Britton E. A pilot randomized controlled trial of a daily muscle stretch regime to prevent contractures in the arm after stroke. *Clin Rehabil* 2005;19:600-12.

59. Bohannon RW. Relationship between shoulder pain and selected variables in patients with hemiplegia. *Clin Rehabil* 1988;2:111-17.
60. Nguyen J. Ankle dorsiflexion contracture and walking in early stroke. Proceedings of Australian Physiotherapy Association Conference, 1-5 October 2009.
61. Malhotra S, Pandyan AD, Rosewilliam S, Roffe C, Hermens H. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. *Clin Rehabil* 2011;25:184-91.
62. Sheean G. The pathophysiology of spasticity. *Eur J Neurol* 2002;9 Suppl 1:3-9; discussion 53-61.
63. Brown P. Pathophysiology of spasticity. *J Neurol Neurosurg Psychiatry* 1994;57:773-7.
64. Ivanhoe CB, Reistetter TA. Spasticity: the misunderstood part of the upper motor neuron syndrome. *Am J Phys Med Rehabil* 2004;83:S3-9.
65. Lance JW. Symposium synopsis. . In: Feldman RG, Young RR, Koella WP, eds. *Spasticity: disorder motor control*: Miami: Symposia Specialists; 1980: 585-94:584-94.
66. Welmer AK, von Arbin M, Widen Holmqvist L, Sommerfeld DK. Spasticity and its association with functioning and health-related quality of life 18 months after stroke. *Cerebrovasc Dis* 2006;21:247-53.
67. Watkins CL, Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK. Prevalence of spasticity post stroke. *Clin Rehabil* 2002;16:515-22.
68. Sommerfeld DK, Eek EU, Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. *Stroke* 2004;35:134-9.
69. Thilmann AF, Fellows SJ, Garms E. The mechanism of spastic muscle hypertonus. Variation in reflex gain over the time course of spasticity. *Brain* 1991;114 ( Pt 1A):233-44.
70. Fellows SJ, Ross HF, Thilmann AF. The limitations of the tendon jerk as a marker of pathological stretch reflex activity in human spasticity. *J Neurol Neurosurg Psychiatry* 1993;56:531-7.

71. Mayer NH, Esquenazi A. Muscle overactivity and movement dysfunction in the upper motoneuron syndrome. *Phys Med Rehabil Clin N Am* 2003;14:855-83, vii-viii.
72. Wissel J, Schelosky LD, Scott J, Christie W, Faiss JH, Mueller J. Early development of spasticity following stroke: a prospective, observational trial. *J Neurol* 2010;257:1067-72.
73. Verplancke D, Snape S, Salisbury CF, Jones PW, Ward AB. A randomized controlled trial of botulinum toxin on lower limb spasticity following acute acquired severe brain injury. *Clin Rehabil* 2005;19:117-25.
74. Huet de la Tour E, Tardieu C, Tabary JC, Tabary C. Decrease of muscle extensibility and reduction of sarcomere number in soleus muscle following a local injection of tetanus toxin. *J Neurol Sci* 1979;40:123-31.
75. Edstrom L. Relation between spasticity and muscle atrophy pattern in upper motor neurone lesions. *Scand J Rehabil Med* 1973;5:170-1.
76. Sinkjaer T, Magnussen I. Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain* 1994;117 ( Pt 2):355-63.
77. Edstrom L. Selective changes in the sizes of red and white muscle fibres in upper motor lesions and Parkinsonism. *J Neurol Sci* 1970;11:537-50.
78. Dattola R, Girlanda P, Vita G, et al. Muscle rearrangement in patients with hemiparesis after stroke: an electrophysiological and morphological study. *Eur Neurol* 1993;33:109-14.
79. Dietz V, Ketelsen UP, Berger W, Quintern J. Motor unit involvement in spastic paresis. Relationship between leg muscle activation and histochemistry. *J Neurol Sci* 1986;75:89-103.
80. Carey JR, Burghardt TP. Movement dysfunction following central nervous system lesions: a problem of neurologic or muscular impairment? *Phys Ther* 1993;73:538-47.
81. Bobath B, ed. *Adult hemiplegia: evaluation and treatment*. London: Heinemann Medical Books; 1990.

82. Vattanasilp W, Ada L, Crosbie J. Contribution of thixotropy, spasticity, and contracture to ankle stiffness after stroke. *J Neurol Neurosurg Psychiatry* 2000;69:34-9.
83. Singer B, Dunne J, Singer KP, Allison G. Evaluation of triceps surae muscle length and resistance to passive lengthening in patients with acquired brain injury. *Clin Biomech (Bristol, Avon)* 2002;17:152-61.
84. Chung SG, Van Rey E, Bai Z, Roth EJ, Zhang LQ. Biomechanic changes in passive properties of hemiplegic ankles with spastic hypertonia. *Arch Phys Med Rehabil* 2004;85:1638-46.
85. Given JD, Dewald JP, Rymer WZ. Joint dependent passive stiffness in paretic and contralateral limbs of spastic patients with hemiparetic stroke. *J Neurol Neurosurg Psychiatry* 1995;59:271-9.
86. Lee WA, Boughton A, Rymer WZ. Absence of stretch reflex gain enhancement in voluntarily activated spastic muscle. *Exp Neurol* 1987;98:317-35.
87. Dietz V, Trippel M, Berger W. Reflex activity and muscle tone during elbow movements in patients with spastic paresis. *Ann Neurol* 1991;30:767-79.
88. Perry J. Rehabilitation of spasticity. In: Feldman RG, Young RR, Koella WP, eds. *Spasticity: disordered motor control*. Miami: Symposia Specialists;1980:87-100.
89. Singer B, Dunne J, Allison G. Reflex and non-reflex elements of hypertonia in triceps surae muscles following acquired brain injury: implications for rehabilitation. *Disabil Rehabil* 2001;23:749-57.
90. Stolov WC. The concept of normal muscle tone, hypotonia and hypertonia. *Arch Phys Med Rehabil* 1966;47:156-68.
91. Friden J, Lieber RL. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve* 2003;27:157-64.
92. Lieber RL, Steinman S, Barash IA, Chambers H. Structural and functional changes in spastic skeletal muscle. *Muscle Nerve* 2004;29:615-27.

93. Lieber RL. Biological and biomechanical adaptation to contracture. Proceedings of the Australian Physiotherapy Conference, 1-5 October 2009.
94. Li L, Tong KY, Hu X. The effect of poststroke impairments on brachialis muscle architecture as measured by ultrasound. *Arch Phys Med Rehabil* 2007;88:243-50.
95. Ada L, Canning C, eds. Anticipating and avoiding muscle shortening. In: *Key issues in neurological physiotherapy*: Oxford Butterworth-Heinemann; 1990.
96. Gracies JM. Pathophysiology of spastic paresis. II: Emergence of muscle overactivity. *Muscle Nerve* 2005;31:552-71.
97. Dietz V, Quintern J, Berger W. Electrophysiological studies of gait in spasticity and rigidity. Evidence that altered mechanical properties of muscle contribute to hypertonia. *Brain* 1981;104:431-49.
98. Gowland C, deBruin H, Basmajian JV, Plews N, Burcea I. Agonist and antagonist activity during voluntary upper-limb movement in patients with stroke. *Phys Ther* 1992;72:624-33.
99. Maier A, Eldred E, Edgerton VR. The effects on spindles of muscle atrophy and hypertrophy. *Exp Neurol* 1972;37:100-23.
100. Gioux M, Petit J. Effects of immobilizing the cat peroneus longus muscle on the activity of its own spindles. *J Appl Physiol* 1993;75:2629-35.
101. Gracies JM. Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. *Muscle Nerve* 2005;31:535-51.
102. Gracies JM. Pathophysiology of spastic paresis. II: Emergence of muscle overactivity. *Muscle and Nerve* 2005;31:552-71.
103. Williams PE, Catanese T, Lucey EG, Goldspink G. The importance of stretch and contractile activity in the prevention of connective tissue accumulation in muscle. *J Anat* 1988;158:109-14.
104. Williams PE, Goldspink G. The effect of immobilization on the longitudinal growth of striated muscle fibres. *J Anat* 1973;116:45-55.

105. Williams PE. Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. *Ann Rheum Dis* 1990;49:316-7.
106. Summers TB, Hines HM. Effect of immobilization in various positions upon the weight and strength of skeletal muscle. *Arch Phys Med Rehabil* 1951;32:142-5.
107. Booth FW. Time course of muscular atrophy during immobilization of hindlimbs in rats. *J Appl Physiol* 1977;43:656-61.
108. Fournier M, Roy RR, Perham H, Simard CP, Edgerton VR. Is limb immobilization a model of muscle disuse? *Exp Neurol* 1983;80:147-56.
109. Spector SA, Simard CP, Fournier M, Sternlicht E, Edgerton VR. Architectural alterations of rat hind-limb skeletal muscles immobilized at different lengths. *Exp Neurol* 1982;76:94-110.
110. Akeson WH, Woo SL, Amiel D, Doty DH. Rapid recovery from contracture in rabbit hindlimb. A correlative biomechanical and biochemical study. *Clin Orthop Relat Res* 1977:359-65.
111. Frost HM. Skeletal structural adaptations to mechanical usage (SATMU): 4. Mechanical influences on intact fibrous tissues. *Anat Rec* 1990;226:433-9.
112. Magnusson SP, Simonsen EB, Aagaard P, Kjaer M. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. *Am J Sports Med* 1996;24:622-8.
113. Duong B, Low M, Moseley AM, Lee RY, Herbert RD. Time course of stress relaxation and recovery in human ankles. *Clin Biomech (Bristol, Avon)* 2001;16:601-7.
114. McClure PW, Blackburn LG, Dusold C. The use of splints in the treatment of joint stiffness: biologic rationale and an algorithm for making clinical decisions. *Phys Ther* 1994;74:1101-7.
115. Light KE, Nuzik S, Personius W, Barstrom A. Low-load prolonged stretch vs. high-load brief stretch in treating knee contractures. *Phys Ther* 1984;64:330-3.
116. Steffen TM, Mollinger LA. Low-load, prolonged stretch in the treatment of knee flexion contractures in nursing home residents. *Phys Ther* 1995;75:886-95; discussion 95-7.

117. Moseley AM, Hassett LM, Leung J, Clare JS, Herbert RD, Harvey LA. Serial casting versus positioning for the treatment of elbow contractures in adults with traumatic brain injury: a randomized controlled trial. *Clin Rehabil* 2008;22:406-17.
118. Robinson W, Smith R, Aung O, Ada L. No difference between wearing a night splint and standing on a tilt table in preventing ankle contracture early after stroke: a randomised trial. *Aust J Physiother* 2008;54:33-8.
119. Katalinic OM, Harvey LA, Herbert RD. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: a systematic review. *Phys Ther* 2011;91:1-14.
120. Maher CG, Sherrington C, Herbert RD, Moseley AM, Elkins M. Reliability of the PEDro scale for rating quality of randomized controlled trials. *Phys Ther* 2003;83:713-21.
121. Moseley AM. The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. *Phys Ther* 1997;77:240-7; discussion 8-59.
122. Landau WM, Sahrman SA. Preservation of directly stimulated muscle strength in hemiplegia due to stroke. *Arch Neurol* 2002;59:1453-7.
123. Glinksky J, Harvey L, Van Es P. Efficacy of electrical stimulation to increase muscle strength in people with neurological conditions: a systematic review. *Physiother Res Int* 2007;12:175-94.
124. Rosewilliam S, Malhotra S, Roffe C, Jones P, Pandyan AD. Can surface neuromuscular electrical stimulation of the wrist and hand combined with routine therapy facilitate recovery of arm function in patients with stroke? *Arch Phys Med Rehabil* 2012;93:1715-21 e1.
125. Yan T, Hui-Chan CW, Li LS. Functional electrical stimulation improves motor recovery of the lower extremity and walking ability of subjects with first acute stroke: a randomized placebo-controlled trial. *Stroke* 2005;36:80-5.
126. Bakhtiary AH, Fatemy E. Does electrical stimulation reduce spasticity after stroke? A randomized controlled study. *Clin Rehabil* 2008;22:418-25.



127. Sabut SK, Sikdar C, Kumar R, Mahadevappa M. Functional electrical stimulation of dorsiflexor muscle: effects on dorsiflexor strength, plantarflexor spasticity, and motor recovery in stroke patients. *NeuroRehabilitation* 2011;29:393-400.
128. Sahin N, Ugurlu H, Albayrak I. The efficacy of electrical stimulation in reducing the post-stroke spasticity: a randomized controlled study. *Disabil Rehabil* 2012;34:151-6.
129. Lin Z, Yan T. Long-term effectiveness of neuromuscular electrical stimulation for promoting motor recovery of the upper extremity after stroke. *J Rehabil Med* 2011;43:506-10.
130. Khalili MA, Hajihassanie A. Electrical simulation in addition to passive stretch has a small effect on spasticity and contracture in children with cerebral palsy: a randomised within-participant controlled trial. *Aust J Physiother* 2008;54:185-9.
131. Rodosky MW, Andriacchi TP, Andersson GB. The influence of chair height on lower limb mechanics during rising. *J Orthop Res* 1989;7:266-71.
132. Sutherland DH, Olshen R, Cooper L, Woo SL. The development of mature gait. *J Bone Joint Surg Am* 1980;62:336-53.
133. McFadyen BJ, Winter DA. An integrated biomechanical analysis of normal stair ascent and descent. *J Biomech* 1988;21:733-44.
134. Lamontagne A, Malouin F, Richards CL. Contribution of passive stiffness to ankle plantarflexor moment during gait after stroke. *Arch Phys Med Rehabil* 2000;81:351-8.
135. Siegler S, Moskowitz GD, Freedman W. Passive and active components of the internal moment developed about the ankle joint during human ambulation. *J Biomech* 1984;17:647-52.
136. Tardieu C, Lespargot A, Tabary C, Bret MD. Toe-walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. *Phys Ther* 1989;69:656-62.

137. Moseley A, Wales A, Herbert R, Schurr K, Moore S. Observation and analysis of hemiplegic gait: stance phase. *Aust J Physiother* 1993;39:259-67.
138. Moore S, Schurr K, Wales A, Moseley A, Herbert A. Observation and analysis of hemiplegic gait: swing phase. *Aust J Physiother* 1993;39:271-8.
139. Perry J. *Gait analysis: normal and pathological function*: Thorofare, NJ: Slack Inc; 1992.
140. Winters TF, Jr., Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. *J Bone Joint Surg Am* 1987;69:437-41.
141. Higginson JS, Zajac FE, Neptune RR, Kautz SA, Burgar CG, Delp SL. Effect of equinus foot placement and intrinsic muscle response on knee extension during stance. *Gait Posture* 2006;23:32-6.
142. Cooper A, Alghamdi GA, Alghamdi MA, Altowaijri A, Richardson S. The relationship of lower limb muscle strength and knee joint hyperextension during the stance phase of gait in hemiparetic stroke patients. *Physiother Res Int* 2012;17:150-6.
143. Gage JR. *Gait analysis in cerebral palsy*. London: Mac Keith Press; 1991.
144. Svehlik M, Zwick EB, Steinwender G, Saraph V, Linhart WE. Genu recurvatum in cerebral palsy-- part A: influence of dynamic and fixed equinus deformity on the timing of knee recurvatum in children with cerebral palsy. *J Pediatr Orthop B* 2010;19:366-72.
145. Matjacic Z, Olensek A, Bajd T. Biomechanical characterization and clinical implications of artificially induced toe-walking: differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. *J Biomech* 2006;39:255-66.
146. Goodman MJ, Menown JL, West JM, Jr., Barr KM, Vander Linden DW, McMulkin ML. Secondary gait compensations in individuals without neuromuscular involvement following a unilateral imposed equinus constraint. *Gait Posture* 2004;20:238-44.

## Chapter 3

An intensive program of passive stretch and motor training  
for the management of severe knee contractures after traumatic brain  
injury: a case report

**Statement from co-authors confirming authorship contribution of the  
PhD candidate**

As co-authors of the paper “An intensive program of passive stretch and motor training for the management of severe knee contractures after traumatic brain injury: a case report”, we confirm that Joan Leung has made contributions to the following:

- Conception and design of the paper
- Data collection
- Writing of the manuscript and revisions of the content

*Lisa Harvey*

Lisa Harvey \_\_\_\_\_ Date 27/08/2014\_\_\_\_\_

*Anne Moseley*

Anne Moseley \_\_\_\_\_ Date 27 Aug 2014\_\_\_\_\_

## CASE STUDY

# An Intensive Programme of Passive Stretch and Motor Training to Manage Severe Knee Contractures after Traumatic Brain Injury: A Case Report

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### ABSTRACT

**Purpose:** While contemporary management of contractures (a common secondary problem of acquired brain injury that can be difficult to treat) includes passive stretch, recent evidence indicates that this intervention may not be effective. This may be because clinical trials have not provided a sufficient dose or have not combined passive stretch with other treatments. The purpose of this case report is to describe a programme of intensive passive stretch combined with motor training administered over a 1.5-year period to treat severe knee contractures. **Method:** Five months after traumatic brain injury, an adolescent client with severe contractures in multiple joints underwent an intensive stretch programme for his knee contractures, including serial casting and splinting, which was administered for 10 months in conjunction with a motor training programme administered for 1.5 years. **Results:** The client regained full extension range in his knees and progressed from being totally dependent to walking short distances with assistance; these effects were maintained at follow-up 5.5 years after injury. **Conclusion:** The use of a high dose of passive stretch in conjunction with motor training may be an option to consider for correcting severe contractures following acquired brain injury.

**Key Words:** brain injuries; contracture; exercise therapy; muscle stretching exercises.

### RE'SUME'

**Objectif :** Bien que le contrôle des contractures (un problème secondaire fréquent propre aux lésions cérébrales acquises et pouvant être difficile à traiter) comprenne des étirements passifs, des preuves récentes indiquent que cette intervention pourrait être inefficace. Cela pourrait s'expliquer par le fait que les essais cliniques n'ont pas utilisé des dosages suffisants ou qu'ils n'ont pas jumelé les étirements passifs à d'autres traitements. L'objectif de ce rapport de cas consiste à décrire un programme d'étirements passifs intensifs allié à un entraînement moteur administré durant une période d'un an et demi pour le traitement de contractures sévères au genou. **Méthodologie :** Cinq mois après un traumatisme cérébral, un client adolescent aux prises avec des contractures sévères à de multiples articulations a été soumis à un programme intensif d'étirements pour ses contractures au genou, y compris l'application successive de plâtres et d'attelles. Le programme a été administré pendant 10 mois, en même temps qu'un programme d'entraînement moteur qui a duré un an et demi. **Résultats :** Le client a retrouvé la pleine extension de son genou et ses progrès se sont étendus d'une dépendance totale à la capacité de marcher sur de courtes distances sans aide; ces effets se sont prolongés pendant un suivi de cinq ans et demi après le traumatisme. **Conclusion :** L'utilisation des étirements passifs à dosage élevé en même temps qu'un entraînement moteur pourrait être une avenue à considérer pour corriger les contractures sévères résultant d'un traumatisme cérébral.

Contractures—a reduction in passive joint range due to shortening and stiffening of soft tissues spanning joints—are commonly observed after acquired brain injury (e.g., stroke, traumatic brain injury); incidence ranges from 23% to 85%.<sup>1,2</sup> Contractures are a problem because they limit function, reduce mobility, and are linked to pain.<sup>1</sup> Not surprisingly, contractures contribute to poor motor recovery, prolonged length of hospital stay, and poor rehabilitation outcomes.<sup>3,4</sup>

Contractures develop in the early weeks after acquired brain injury,<sup>5–12</sup> worsen over time,<sup>6,7,10–12</sup> and can persist for protracted periods after brain injury,<sup>5,9</sup> often despite interventions to treat and prevent them. To date, passive stretch and motor training have been the most widely used physical therapy interventions for this purpose. Several randomized controlled studies have reported that passive stretch alone does not produce clinically worthwhile or sustained effects in people with brain

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Figure 1 Resting position in bed on first admission (5 months post injury).

injury.<sup>6,8,12-21</sup> These findings were confirmed by a recent Cochrane review that provided high-quality evidence that passive stretch produces negligible short-term (mean between-group difference 1, 95% CI, 0-3) or long-term (mean between-group difference 0, 95% CI, 2 to 2) changes in joint range of motion (ROM) when administered to people with a variety of neurological conditions. The typical dose of passive stretch applied in the reported trials was 30 minutes to 12 hours per day over 4-12 weeks (median duration 30 days);<sup>22</sup> this dosage of passive stretch may be insufficient. In addition, contracture is a complex problem; passive stretch focuses predominantly on reversing soft-tissue shortening and does not address the underlying causes of contracture, such as poor motor control and spasticity.<sup>9</sup> Combining passive stretch with interventions that address the contributors to contracture is likely to offer a better prospect to effectively manage contracture than passive stretch alone. Importantly, passive stretch may need to continue until the underlying causes are addressed if treatment is to achieve a sustained effect.

This report describes the case of an adolescent with severe traumatic brain injury (TBI) and severe contractures. His knee contractures were managed with programmes involving months of prolonged passive stretch, including serial casting and splinting, combined with 1.5 years of motor training. Ethical approval was granted by the Human Research Ethics Committee of the Royal Rehabilitation Centre Sydney.

## METHODS

### Case description

A 16-year-old boy was involved in a motorcycle accident and sustained a TBI. His first documented Glasgow Coma Scale score (2 weeks after the injury) was 5/15. He had post-traumatic amnesia for more than 6 months,

which indicates the extremely severe nature of his injury. He did not receive rehabilitation and was discharged to a nursing home directly from the acute-care facility 3.5 months after injury. At 5 months post injury, he was admitted to a brain-injury unit for a trial of rehabilitation and to review his nursing-home programme.

On admission, the client was minimally responsive and unable to verbalize. He was confined to a bed with his arms and legs fully flexed and his lower body rotated to the left (see Figure 1). Physical examination revealed multiple contractures. Maximum passive knee extension was 50° on the right and 45° on the left, measured with a goniometer, a common and reliable method for measuring physiologic knee ROM (ICC 0.88).<sup>23</sup> (All measurements were rounded to the nearest 5.) He also had severe contractures in his hips, ankles, shoulders, elbows, wrists, fingers, and trunk. He was able to follow verbal instructions to blink his eyes, open his mouth, turn his head, and, inconsistently, abduct his right shoulder to 30°. Manual muscle testing showed no voluntary control or strength in his lower limbs.<sup>24</sup> He had strong flexor spasticity in all limbs (4/4 on the Modified Ashworth Scale).<sup>25</sup> Functionally, he was fully dependent and required a high level of care; he scored 18/126 on the Functional Independence Measure (FIM),<sup>26</sup> the lowest possible score on this scale (see Table 1).

### Intervention

This case report focuses on the management of the client's knee contractures from admission to the brain-injury unit through to 5 years later. Initially, passive stretch was applied to his knees for approximately 12 hours/day, using ready-made knee orthoses (see Figure 2). Two months after his admission (7 months post-injury), the left knee showed a 10° improvement in extension ROM, but the right knee showed a 5° deterioration (see Table 1). The slow progress and the deterioration in right-knee ROM in the first 2 months after admission prompted the move to a more aggressive stretch programme. Serial casting<sup>27-30</sup> for the right knee therefore began. A series of five casts was applied over 5 weeks. At the same time, the duration of splinting for the left knee was increased to 24 hours/day. The client gained nearly full passive extension in both knees after 5 weeks. Adjustable custom-made knee orthoses were then applied to both knees 24 hours/day to maintain the gain in range. The orthoses were removed only for hygiene and therapy; the client's skin condition was closely monitored.

Motor training and lower-limb strength training were initiated following the correction of the client's knee contractures. He attended the gymnasium for 1 hour/day. Physical therapy followed the principles of motor relearning,<sup>31</sup> with an emphasis on muscle strengthening in functional positions and repetitive training augmented with appropriate encouragement and feedback. Initial training involved exercises to elicit muscle activity and

Table 1 Timeline of Progress in Knee Extension Range of Motion, Knee Extensor Muscle Strength, Spasticity, and Dependence (FIM)

	Time since injury				
	5 mo	12 mo	14 mo	25 mo	5.5 y
Stage of rehabilitation	1st admission	1st discharge	2nd admission	2nd discharge	Follow-up
Stretch interventions	Knee splinting 12 h/d	Serial casting followed by 24 h/d knee splinting	Gradual reduction of knee splinting time	Cessation of knee splinting	
Knee extension ROM					
Right	50 °	Full	Full	Full	Full
Left	45 °	Full	Full	Full	Full
Knee extensor strength, /5					
Right	0	3	3	4	4
Left	0	3	3	4	4
MAS score, /4	4	2	2	1	1
FIM score, /126	18	28	28	32	54

ROM ¼ range of motion; MAS ¼ Modified Ashworth Scale; FIM ¼ Functional Independence Measure.



Figure 2 Knee orthosis used to apply 12 hours of passive stretch per day at the beginning of the client's first admission (5 months post injury).

strengthen the leg muscles, first with the orthoses applied and then without. Over time, the client progressed to assisted cycling, concentric and eccentric exercises, and repetitive sit-to-stand practice. The duration of practice and complexity of tasks were increased gradually; treatment targets were set and progressed as he improved. Progress was slow, as the client exhibited behavioural problems that affected his ability to participate in training. A reward system designed by the team psychologist was used to increase adherence to therapy.

Eight months after admission (13 months post injury), the client had full passive bilateral knee extension and showed improved strength in both lower limbs. The hip flexion contractures also resolved as knee ROM improved. He was able to stand with moderate support from two assistants and bilateral knee orthoses. Although it seemed an appropriate time to start weaning him off the orthoses,

this step was postponed because he was discharged prematurely to secure a nursing-home placement. One month after discharge (14 months post injury), he was readmitted to the brain-injury unit for further rehabilitation as planned. He participated in in-patient rehabilitation for another 11 months, during which he began gait training, first with and later without the support of the knee orthoses. He stopped wearing the knee orthoses 2 months into this second admission (16 months post injury). At the beginning of the gait training, a forearm support frame (Pacer Gait Trainer, Wonderland Rehab & Child Care, Lidcombe, NSW) was used for walking, which provided support to the client's pelvis and had straps to stop his left leg from crossing the midline (see Figure 3). He subsequently progressed to walking with an ordinary forearm support frame. He never developed skin or other problems from the orthoses, and the orthoses had to be modified only once to accommodate changes in weight and muscle bulk. Anti-spasticity medication (dantrolene) was used throughout his in-patient rehabilitation. He also received botulinum toxin injections for his wrists, fingers, elbows, and ankles, but not for his knees. The effect of the injections was mixed: while both elbows and the right wrist and fingers responded well to the injections, the ankles, left wrist, and left fingers did not. The ankle contractures were subsequently corrected with serial casting, and ROM was maintained using customized ankle-foot orthoses (the same approach used for his knees).

## RESULTS

The client was discharged back to a nursing home at 25 months post injury, with a plan for him to eventually move to a group home designed to reintegrate young people with disabilities into the community. At this time, he had anti-gravity strength (grade 3/5) in the knee extensor



Figure 3 Walking with a walking frame (Pacer Gait Trainer) and the assistance of one person (22 months post injury). Note: At the time this photo was taken, the client was wearing an ankle-foot orthosis for his right ankle and receiving serial casting for his left ankle. He later progressed to using an ordinary forearm support frame.

muscles and full passive extension in the knees. He was able to stand with supervision and the support of a rail (see Figure 4) and could walk 140 m with one assistant and a forearm support frame. His FIM score was 32/126 (see Table 1).

The client was reviewed 5.5 years post injury. He was still living in a nursing home and receiving a maintenance programme prescribed by the nursing-home physiotherapist. He maintained full passive knee extension bilaterally and had grade 4/5 strength in the knee extensor muscles (see Table 1). He could perform standing transfers with one person. He used an electrical wheelchair as his primary form of mobility but was learning to walk using a wall-mounted handrail with assistance of one person. His FIM score was 54/126 (see Table 1). He received surgical interventions to correct the deformities of his left wrist and fingers, and was subsequently transferred to a community group home.

## DISCUSSION

Contractures are a common problem after acquired brain injury and can be difficult to manage. High-quality evidence indicates that passive stretch, as typically administered by physical therapists to people with neu-



Figure 4 Standing without knee orthoses but with bilateral ankle-foot orthoses, the support of parallel bars, and supervision from one person (24 months post injury). Note: a ball was used to keep the feet separated and he was wearing customized ankle-foot orthoses.

rological conditions, is ineffective.<sup>22</sup> This case report describes an adolescent with TBI whose severe knee contractures did not respond to usual care but resolved following a high dose of passive stretch combined with intensive motor training and anti-spasticity medication. Long-term follow-up indicated that these effects were sustained.

Severe contractures have significant deleterious effects on mobility and function. Poor mobility and function may also precipitate contracture, thereby setting up a vicious cycle. The client presented in this case report had an extremely severe TBI, multiple joint contractures, severe and generalized spasticity, and very poor motor control. These severe impairments, together with protracted post-traumatic amnesia, suggested a poor prognosis. Yet by the time he was discharged 25 months post injury, the knee contractures had resolved and he could perform standing transfers and walk short distances with assistance. This outcome was better than anticipated.

While it is impossible to rule out the possibility that the observed improvements were due to spontaneous recovery, bias, or other factors, we propose that the resolution of knee contractures enabled the client to participate in motor training and that this, in turn, led to



improvements in strength and control that helped maintain knee ROM. The lack of progress in the first 7 months before the interventions began lends support to our argument.

The approach described here has three strong features. First, a high dose of passive stretch was used. The knee contractures resolved only after an increase in the dose of passive stretch (from 12 to 24 hours/day); this highlights the importance of applying an adequate dose of passive stretch to achieve a therapeutic effect. Second, concomitant intervention (in this case, the individualized motor training programme) was implemented to treat the underlying contributors to contracture; the motor training, a key component of the programme, could not begin until after the correction of the knee contractures. Third, passive stretch was continued until the client had gained adequate strength to maintain the knee ROM. Perhaps these are the keys to successful management of contractures. The use of anti-spasticity medication may also have contributed to the successful outcome.

There is currently controversy about the effectiveness of passive stretch.<sup>22</sup> However, no trial has examined the use of intensive passive stretch administered over extended periods (e.g., 24 hours/day over a few months) and in conjunction with other interventions (in this case, motor training). This could well be the key to successful contracture management and an option to consider for correcting contractures following acquired brain injury. It may be premature to abandon stretch altogether on the basis of the recent Cochrane review.<sup>22</sup> This case study suggests the need for further research. It may be appropriate to consider a clinical trial, but conducting a study involving an intensive programme such as the one reported here will pose challenges for future researchers.

## CONCLUSION

This case study illustrates the resolution of severe and chronic contractures following an intensive programme of passive stretch provided in conjunction with a motor training programme. This approach may provide the answer to contracture management, but it requires further scrutiny within a clinical trial. Until such trials have been conducted, however, this approach may be an option to consider for the correction of severe contractures following acquired brain injury.

## REFERENCES

1. Fergusson D, Hutton B, Drodge A. The epidemiology of major joint contractures: a systematic review of the literature. *Clin Orthop Relat Res.* 2007;456(456):22–9. <http://dx.doi.org/10.1097/BLO.0b013e3180308456>. Medline:17179779
2. Kwah LK, Harvey LA, Diong JH, et al. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J Physiother.* 2012;58(1):41–7. [http://dx.doi.org/10.1016/S1836-9553\(12\)70071-1](http://dx.doi.org/10.1016/S1836-9553(12)70071-1). Medline:22341381
3. Turner-Stokes L, ed. *Rehabilitation following acquired brain injury: national clinical guidelines.* London: RCP, BSRM; 2003.

4. De'nes Z. A ma'sodlagos ka'rosoda'sok jelentose'ge a su'lyos agyka'rosoda'st szvenvedett betegek rehabilita'cio'ja sora'n [Consequence of secondary complications during the rehabilitation of patients with severe brain injury]. *Orv Hetil.* 2009;150(4):165–9. Medline:19144601
5. Singer BJ, Jegasothy GM, Singer KP, et al. Incidence of ankle contracture after moderate to severe acquired brain injury. *Arch Phys Med Rehabil.* 2004;85(9):1465–9. <http://dx.doi.org/10.1016/j.apmr.2003.08.103>. Medline:15375817
6. Turton AJ, Britton E. A pilot randomized controlled trial of a daily muscle stretch regime to prevent contractures in the arm after stroke. *Clin Rehabil.* 2005;19(6):600–12. <http://dx.doi.org/10.1191/0269215505cr891oa>. Medline:16180595
7. Pandyan AD, Cameron M, Powell J, et al. Contractures in the post-stroke wrist: a pilot study of its time course of development and its association with upper limb recovery. *Clin Rehabil.* 2003;17(1):88–95. <http://dx.doi.org/10.1191/0269215503cr587oa>. Medline:12617383
8. Lannin NA, Cusick A, McCluskey A, et al. Effects of splinting on wrist contracture after stroke: a randomized controlled trial. *Stroke.* 2007;38(1):111–6. <http://dx.doi.org/10.1161/01.STR.0000251722.77088.12>. Medline:17122432
9. Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disabil Rehabil.* 2006;28(13–14):891–7. <http://dx.doi.org/10.1080/09638280500535165>. Medline:16777777
10. Ada L, Goddard E, McCully J, et al. Thirty minutes of positioning reduces the development of shoulder external rotation contracture after stroke: a randomized controlled trial. *Arch Phys Med Rehabil.* 2005;86(2):230–4. <http://dx.doi.org/10.1016/j.apmr.2004.02.031>. Medline:15706548
11. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain.* 1996;119(5):1737–49. <http://dx.doi.org/10.1093/brain/119.5.1737>. Medline:8931594
12. Lannin NA, Horsley SA, Herbert R, et al. Splinting the hand in the functional position after brain impairment: a randomized, controlled trial. *Arch Phys Med Rehabil.* 2003;84(2):297–302. <http://dx.doi.org/10.1053/apmr.2003.50031>. Medline:12601664
13. Harvey LA, Batty J, Crosbie J, et al. A randomized trial assessing the effects of 4 weeks of daily stretching on ankle mobility in patients with spinal cord injuries. *Arch Phys Med Rehabil.* 2000;81(10):1340–7. <http://dx.doi.org/10.1053/apmr.2000.9168>. Medline:11030499
14. Harvey L, de Jong I, Goehl G, et al. Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. *Aust J Physiother.* 2006;52(4):251–8. [http://dx.doi.org/10.1016/S0004-9514\(06\)70004-6](http://dx.doi.org/10.1016/S0004-9514(06)70004-6). Medline:17132119
15. Moseley AM, Hassett LM, Leung J, et al. Serial casting versus positioning for the treatment of elbow contractures in adults with traumatic brain injury: a randomized controlled trial. *Clin Rehabil.* 2008;22(5):406–17. <http://dx.doi.org/10.1177/0269215507083795>. Medline:18441037
16. Horsley SA, Herbert RD, Ada L. Four weeks of daily stretch has little or no effect on wrist contracture after stroke: a randomised controlled trial. *Aust J Physiother.* 2007;53(4):239–45. [http://dx.doi.org/10.1016/S0004-9514\(07\)70004-1](http://dx.doi.org/10.1016/S0004-9514(07)70004-1). Medline:18047458
17. Dean CM, Mackey FH, Katrak P. Examination of shoulder positioning after stroke: a randomised controlled pilot trial. *Aust J Physiother.* 2000;46(1):35–40. Medline:11676788
18. Gustafsson L, McKenna K. A programme of static positional stretches does not reduce hemiplegic shoulder pain or maintain shoulder range of motion—a randomized controlled trial. *Clin Rehabil.* 2006;20(4):277–86. <http://dx.doi.org/10.1191/0269215506cr944oa>. Medline:16719026
19. Ben M, Harvey L, Denis S, et al. Does 12 weeks of regular standing prevent loss of ankle mobility and bone mineral density in people with recent spinal cord injuries? *Aust J Physiother.* 2005;51(4):251–6. [http://dx.doi.org/10.1016/S0004-9514\(05\)70006-4](http://dx.doi.org/10.1016/S0004-9514(05)70006-4). Medline:16321132

20. Harvey LA, Byak AJ, Ostrovskaya M, et al. Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries. *Aust J Physiother.* 2003;49(3):176–81. Medline:12952517
21. Harvey LA, Herbert RD, Glinsky J, et al. Effects of 6 months of regular passive movements on ankle joint mobility in people with spinal cord injury: a randomized controlled trial. *Spinal Cord.* 2009;47(1):62–6. <http://dx.doi.org/10.1038/sc.2008.71>. Medline:18574489
22. Katalinic OM, Harvey LA, Herbert RD, et al. Stretch for the treatment and prevention of contractures. *Cochrane Database Syst Rev.* 2010;9(9):CD007455. Medline:20824861
23. Cibere J, Bellamy N, Thorne A, et al. Reliability of the knee examination in osteoarthritis: effect of standardization. *Arthritis Rheum.* 2004;50(2):458–68. <http://dx.doi.org/10.1002/art.20025>. Medline:14872488
24. Hislop HJ, Montgomery J. Daniels and Worthingham's muscle testing: techniques of manual examination. 8th ed. New York: W.B. Saunders; 2007.
25. Waninge A, Rook RA, Dijkhuizen A, et al. Feasibility, test-retest reliability, and interrater reliability of the Modified Ashworth Scale and Modified Tardieu Scale in persons with profound intellectual and multiple disabilities. *Res Dev Disabil.* 2011;32(2):613–20. <http://dx.doi.org/10.1016/j.ridd.2010.12.013> Medline:21232915
26. Hamilton BB, Laughlin JA, Fiedler RC, et al. Interrater reliability of the 7-level Functional Independence Measure (FIM). *Scand J Rehabil Med.* 1994;26(3):115–9. Medline:7801060
27. Verplancke D, Snape S, Salisbury CF, et al. A randomized controlled trial of botulinum toxin on lower limb spasticity following acute acquired severe brain injury. *Clin Rehabil.* 2005;19(2):117–25. <http://dx.doi.org/10.1191/0269215505cr827oa>. Medline:15759526
28. Mortenson PA, Eng JJ. The use of casts in the management of joint mobility and hypertonia following brain injury in adults: a systematic review. *Phys Ther.* 2003;83(7):648–58. Medline:12837126
29. Zablony C, Andric MF, Gowland C. Serial casting: clinical applications for the adult head injured patient. *J Head Trauma Rehabil.* 1987;2(2):46–52. <http://dx.doi.org/10.1097/00001199-198706000-00007>.
30. Booth BJ, Doyle M, Montgomery J. Serial casting for the management of spasticity in the head-injured adult. *Phys Ther.* 1983;63(12):1960–6. Medline:6647551
31. Carr JH, Shepherd RB, editors. *Neurological rehabilitation.* 2nd ed. New York: Churchill Livingstone; 2010.

## Chapter 4

Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial

**Statement from co-authors confirming authorship contribution of the  
PhD candidate**

As co-authors of the paper "Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial", we confirm that Joan Leung has made contributions to the following:

- Development of the research question
- Conception and design of the research
- Collection, analysis and interpretation of data
- Writing of the manuscript and critical revision of the content for publication

Lisa Harvey		Date	27/08/2014
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# Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial

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**Question:** Is electrical stimulation and splinting more effective than splinting alone for the management of wrist contracture following acquired brain injury? **Design:** A multi-centre randomised trial with concealed allocation, assessor blinding, and intention-to-treat analysis. **Participants:** Thirty-six adults with first stroke or traumatic brain injury and mild to moderate wrist flexion contractures. **Intervention:** The experimental group received electrical stimulation to the wrist and finger extensor muscles for 1 hour a day over 4 weeks while the control group did not. Both groups wore a splint for 12 hours a day during this 4-week period. **Outcome measures:** The primary outcome was passive wrist extension measured with a 3 Nm torque and with the fingers in extension. Secondary outcomes included passive wrist extension, wrist and finger extensor strength, wrist flexor spasticity, motor control of the hand, and Global Perceived Effect of Treatment, and perception of treatment credibility. Outcome measures were taken at baseline, at the end of the intervention period (4 weeks), and after a 2-week follow-up period (6 weeks). **Results:** At 4 and 6 weeks, the mean between-group difference (95% CI) for passive wrist extension was 7 degrees (–2 to 15) and –3 degrees (–13 to 7), respectively. Secondary outcomes were statistically non-significant or were of borderline statistical significance. **Conclusion:** It is not clear whether electrical stimulation and splinting is more effective than splinting alone for the management of wrist contracture after acquired brain injury. Therapists' confidence in the efficacy of electrical stimulation for contracture management is not yet justified. **Trial registration:** ACTRN12609000495224. [Leung J, Harvey LA, Moseley AM, Tse C, Bryant J, Wyndham S, Barry S (2012) Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial. *Journal of Physiotherapy* 58: 231–240]

**Key words:** Randomised controlled trial, Acquired brain injury, Contracture, Splinting, Electrical stimulation

## Introduction

Contracture is characterised by a loss of range of motion secondary to adaptive shortening of soft tissues spanning joints (Botte et al 1988, Harburn and Potter 1993). It is a common problem for people with acquired brain injury (Fergusson et al 2007, Kwah et al 2012). Contracture is undesirable because of its potentially serious implications for motor recovery, function, care, hygiene, and posture (Fergusson et al 2007). Thus treating and preventing contracture are often important aspects of rehabilitation.

While passive stretch has been the mainstay of physiotherapy management for contracture, a recent Cochrane systematic review of passive stretch concluded that regular stretch provided for less than 6 months is not effective in people with neurological conditions (Katalinic et al 2010). In the studies included in that review, the stretch was administered in different ways including splints (Harvey et al 2006, Lannin et al 2007), sustained passive stretch and positioning (Ada et al 2005, de Jong et al 2006, Dean et al 2000, Gustafsson and McKenna 2006, Harvey et al 2003, Horsley et al 2007), standing on a tilt table (Ben et al 2005, Harvey et al 2000), and serial casting (Moseley et al 1997). Stretch alone may be ineffective for the treatment and prevention of contracture because it does not address possible underlying causes of contracture, namely muscle weakness and spasticity (Ada et al 2006). Weakness and spasticity are common impairments

after acquired brain injury. They immobilise joints in stereotypical postures predisposing them to contracture (Ada et al 2006, Fergusson et al 2007). Stretch provided in conjunction with interventions addressing weakness and spasticity may be more effective than stretch alone.

Electrical stimulation is increasingly used to increase strength and reduce spasticity in people with acquired brain injury. A systematic review concluded that electrical stimulation has a modest beneficial effect on muscle strength after stroke (Glinsky et al 2007). Two of the

**What is already known on this topic:** Stretch alone may not affect contracture, perhaps because it does not address underlying muscle weakness and spasticity. Electrical stimulation can increase strength and reduce spasticity in some patients at risk of contracture.

**What this study adds:** The effect of electrical stimulation for contracture management was not clear. While further research is needed to clarify the effectiveness of electrical stimulation, it may be reasonable to use electrical stimulation in conjunction with splinting because it is inexpensive and not associated with discomfort or pain. It may be appropriate to use stronger doses of electrical stimulation than that used in the study.

studies included in that review specifically investigated the effect of electrical stimulation on the strength of wrist and finger extensor muscles after stroke and both reported large effect sizes (Bowman et al 1979, Powell et al 1999). There is also initial evidence that electrical stimulation may reduce spasticity. Three randomised trials in people with stroke (Bakhtiary and Fatemy 2008, Cheng et al 2010, Yan et al 2005) showed a reduction of ankle spasticity following electrical stimulation. While these studies were performed in the lower limbs, there is no reason to believe that the response of upper limb muscles to electrical stimulation differs from the response of lower limb muscles. Weakness and spasticity are the two known contributors to contracture. Electrical stimulation, when provided in conjunction with stretch, may help reduce contracture by increasing strength and reducing spasticity.

The possible therapeutic effect of electrical stimulation for contracture management is supported by a trial in people with stroke (Bakhtiary and Fatemy 2008), which reported a small treatment effect of electrical stimulation on passive ankle dorsiflexion range of motion (mean between-group difference 5 degrees, 95% CI 2 to 7). While this trial suggests that electrical stimulation is therapeutic, supramaximal levels of electrical stimulation for 9 minutes a day were applied (ie, the intensity was set at 25% over the intensity needed to produce a maximum contraction). Supramaximal doses are not commonly used clinically because of the associated discomfort. It is not clear how Bakhtiary and Fatemy overcame this problem. We were interested in whether we could replicate these results using a similar protocol of electrical stimulation but with a lower and more readily tolerated intensity of electrical stimulation applied for 1 hour a day rather than 9 minutes a day. We were also interested in combining electrical stimulation with stretch as this has not been investigated previously. Splinting was selected as a way of readily providing sustained stretch. The research question therefore was:

Is a program of electrical stimulation and splinting more effective than splinting alone for the treatment and prevention of wrist contracture following acquired brain injury?

## Method

### Design

An assessor-blinded, randomised controlled trial was undertaken. All participants were randomly allocated to one of two groups: experimental group (electrical stimulation and hand splinting) or control group (hand splinting only). The allocation sequence was computer-generated by a person not involved in participant recruitment. Group allocation was concealed using consecutively numbered, sealed, opaque envelopes which were kept off-site. The envelopes were opened after the baseline assessment, at which time participants were considered to have entered the trial. Follow-up assessments were conducted at the end of the 4-week program (post-intervention) and 2 weeks after that (follow-up). All assessors were blinded to group allocation. The success of blinding was monitored.

### Participants

Patients admitted with a stroke or traumatic brain injury to one of five rehabilitation units in Sydney, Australia, were screened for inclusion between June 2008 and November

2011. The eligibility criteria were: first documented stroke or traumatic brain injury; weakness of wrist and finger extensor muscles (inability to extend wrist and fingers fully in a gravity-eliminated position); and dystonia/flexor spasticity in the wrist and fingers equating to a Tardieu scale score  $\geq 1$  (Tardieu et al 1954), or any loss of extensibility in the extrinsic wrist and finger flexor muscles compared to the unaffected side. People were excluded if they were unable to tolerate the experimental interventions, unlikely to stay in the hospital for four weeks, had severe contracture preventing measurement with our device (ie, inability to passively extend the fingers with the wrist in a neutral position), and had recent wrist or finger fractures, fixed flexion deformities in the individual finger joints, or previous wrist problems limiting range of motion. People with cognitive impairments were not excluded.

### Interventions

Participants in both groups received a 4-week program. The experimental group received 1 hour of daily electrical stimulation, 5 days per week, administered via a digital muscular stimulation unit<sup>a</sup>. Electrical stimulation was applied to the wrist and finger extensor muscles while wearing a hand splint that kept the wrist and fingers in full extension (as tolerated). After the hand splint was applied with the arm supported on a surface, the distal straps were loosened to allow room for the fingers and wrist to extend beyond the splint during stimulation. This was done to optimise the stretch and to strengthen muscles at their shortest length where they are often weakest after stroke (Ada et al 2003). The electrical stimulation was applied through a pair of square electrodes (5 cm  $\times$  5 cm). The following parameters were used: pulse width of 300  $\mu$ s, frequency of 50 Hz, on time of 15 s, off time of 15 s, and a ramping-up period of 1.5 s. The pulse width and frequency of stimulation were selected to optimise the strengthening benefits of the electrical stimulation (Bowman and Baker 1985). The amplitude of electrical stimulation was set at a level to produce maximum tolerable muscle contractions. If participants were unable to indicate tolerable levels of stimulation, the minimum amplitude of stimulation required to generate a palpable muscle contraction was used. At the beginning of each session, participants were instructed to contract the wrist and finger extensor muscles in time with the electrical stimulation. Participants were reminded regularly during each training session but not verbally encouraged with each contraction.

Both the experimental and control groups wore hand splints for 12 hours a day, 5–7 days per week. Custom-made hand splints were used to maintain the maximum tolerated wrist and finger extension. The splints were checked each time they were applied and modified as required to maintain comfort, fit, and stretch. During the 2-week follow-up period, participants in both groups continued to wear the hand splint for 12 hours a day, 5–7 days per week. Electrical stimulation was not applied to the wrists of participants in either group during these 2 weeks. A diary was used to record the duration and frequency of electrical stimulation and splinting.

The electrical stimulation and usual care were administered by physiotherapists working in the participating units over the course of the trial. These physiotherapists were not randomised to participants and consequently they managed

an arbitrary mix of control and experimental participants. The splints were applied by physiotherapists, nursing staff, or physiotherapy assistants (under the supervision of the treating physiotherapists).

Throughout the study, no other stretch-based interventions were administered to the wrist. All participants received usual multidisciplinary rehabilitation provided by the participating units, which included training of hand function as appropriate. No botulinum toxin was administered to the wrist prior to or during the study period. Use of other anti-spasticity medication was not mandated by the trial protocol and was recorded.

### Outcome measures

There were one primary and six secondary outcomes. The primary outcome was passive wrist extension measured with a torque of 3 Nm and with fingers in extension. This was used to reflect the extensibility of the extrinsic wrist and finger flexor muscles. The secondary outcomes were: passive wrist extension with a torque of 2 Nm, strength of the wrist and finger extensor muscles, spasticity of the wrist flexor muscles, motor control of the hand, physiotherapists' and participants' Global Perceived Effect of Treatment, and perception of treatment credibility. Outcome measures were taken at the beginning of the trial (Week 0), after the intervention period (Week 4), and after 2 weeks of follow-up (Week 6) (Figure 2). The outcome measures were taken by one of four blinded and trained assessors who assessed participants of both groups. The post-intervention and follow-up assessments were done more than 24 hours but within 3 days after the splint (and electrical stimulator) had been removed.

Passive wrist extension was measured with the application of two stretch torques (2 and 3 Nm) using a standardised procedure (Harvey et al 1994). Measurements with a torque of 1 Nm were considered initially but abandoned because of problems attaining meaningful results. This procedure has high test-retest reliability (Intra Class Correlation 0.85). The arm and hand were positioned on the measuring device with the participant lying in supine and the shoulder in 30–45 degrees of abduction and the elbow fully extended (see Figure 1). Two participants had the measurements taken in supine with the elbows slightly flexed and three participants were tested in sitting with elbow in 90 degrees flexion because of shoulder or elbow pain. Once the position was determined at the baseline assessment, the same position was used for all subsequent assessments for each participant (post-intervention and follow-up). A pre-stretch was applied to the wrist and finger flexor muscles for 30 seconds. Stretch torques of 1 Nm, 2 Nm, and then 3 Nm were then applied using a spring balance which was kept perpendicular to the hand. Wrist extension (in degrees) at torques of 2 Nm and 3 Nm was measured using a protractor attached to the measuring device.

Strength of the wrist and finger extensor muscles was determined with a dynamometer. This method has a high inter-rater reliability with an Intra Class Correlation Coefficient range of 0.84 to 0.94 (Bohannon 1987). The dynamometer was secured on a purpose-built platform. Participants sat with the arm secured on the platform and were instructed to push their hands against the dynamometer as hard as possible for 3 seconds. They were given 5 attempts with at least 10 seconds rest between each

attempt. The best of 5 measurements was used for analysis. The readings of the dynamometer (in kg) were converted to Newtons and then to torque values (in Nm) by multiplying the reading in Newtons by the distance between the wrist and the point of application of the dynamometer (ie, distal end of the second metacarpal).

Spasticity of wrist flexor muscles was assessed using the Tardieu Scale (Tardieu et al 1954). The Tardieu Scale has a high percentage close agreement with laboratory measures of spasticity (Patrick and Ada 2006). Participants were instructed to relax during the test. The assessor moved the participant's wrist as fast as possible. Reaction to passive stretch was rated on a 5-point scale.

Motor control of the hand was assessed using the hand movement item of the Motor Assessment Scale (Carr et al 1985). The Motor Assessment Scale has a high test-retest reliability with a mean Intra Class Correlation Coefficient of 0.95 (Carr et al 1985). All items were performed without assistance. Participants were scored on the best of three performances.

The Global Perceived Effect of Treatment was rated separately through questionnaires at Week 4 and Week 6 by the treating physiotherapists and participants (or their carers if the participants did not have the capacity to answer the questions). Assistance was provided to participants (or their carers) as needed by staff not otherwise involved in the study. The treating physiotherapists and participants (or their carers) were initially asked if they thought their wrists were better, the same or worse. Those who stated that their wrists were better were asked to rate the improvement between 1 (a little better) and 6 (a very great deal better). Those who stated that their wrists were worse were asked to rate the deterioration between 1 (a little worse) and 6 (a very great deal worse). These data were analysed by combining responses into a single 13-point scale with -6 reflecting a very great deal worse, 0 reflecting no change and +6 reflecting a very great deal better. The minimally important difference was set at 1 point (Schneider and Olin 1996).

Perception of treatment credibility was evaluated by the treating physiotherapists and participants (or their carers) at Week 4 using questionnaires which captured their tolerance to the treatment (scored on a 5-point scale), their perceptions of the worth of the treatment (scored on a 5-point scale), their perceptions of the effectiveness of the treatment (scored on



**Figure 1.** The device to measure passive wrist extension.



a 5-point scale), and their willingness to continue with the same treatment if it were to be provided (scored yes or no). Assistance was provided to participants (or their carers) as needed by staff not otherwise involved in the study. Treating physiotherapists were also asked to indicate if they would administer the treatment to the participants if further management for wrist contracture was needed (scored yes or no). In addition, participants and physiotherapists were asked open-ended questions directed at identifying any issues or concerns about the intervention(s).

**Data analysis**

The sample size was calculated *a priori*. Best estimates indicated that a sample size of 36 participants was required to provide an 80% probability of detecting a between-group difference of 5 degrees for the primary outcome, assuming a standard deviation of 5 degrees (Bakhtiary and Fatemy 2008) and a 10% drop-out rate. The minimally important difference for the primary outcome was set at 5 degrees in line with a number of previous studies on joint contracture (Harvey et al 2000, Harvey et al 2003, Horsley et al 2007, Lannin et al 2007, Lannin et al 2003).

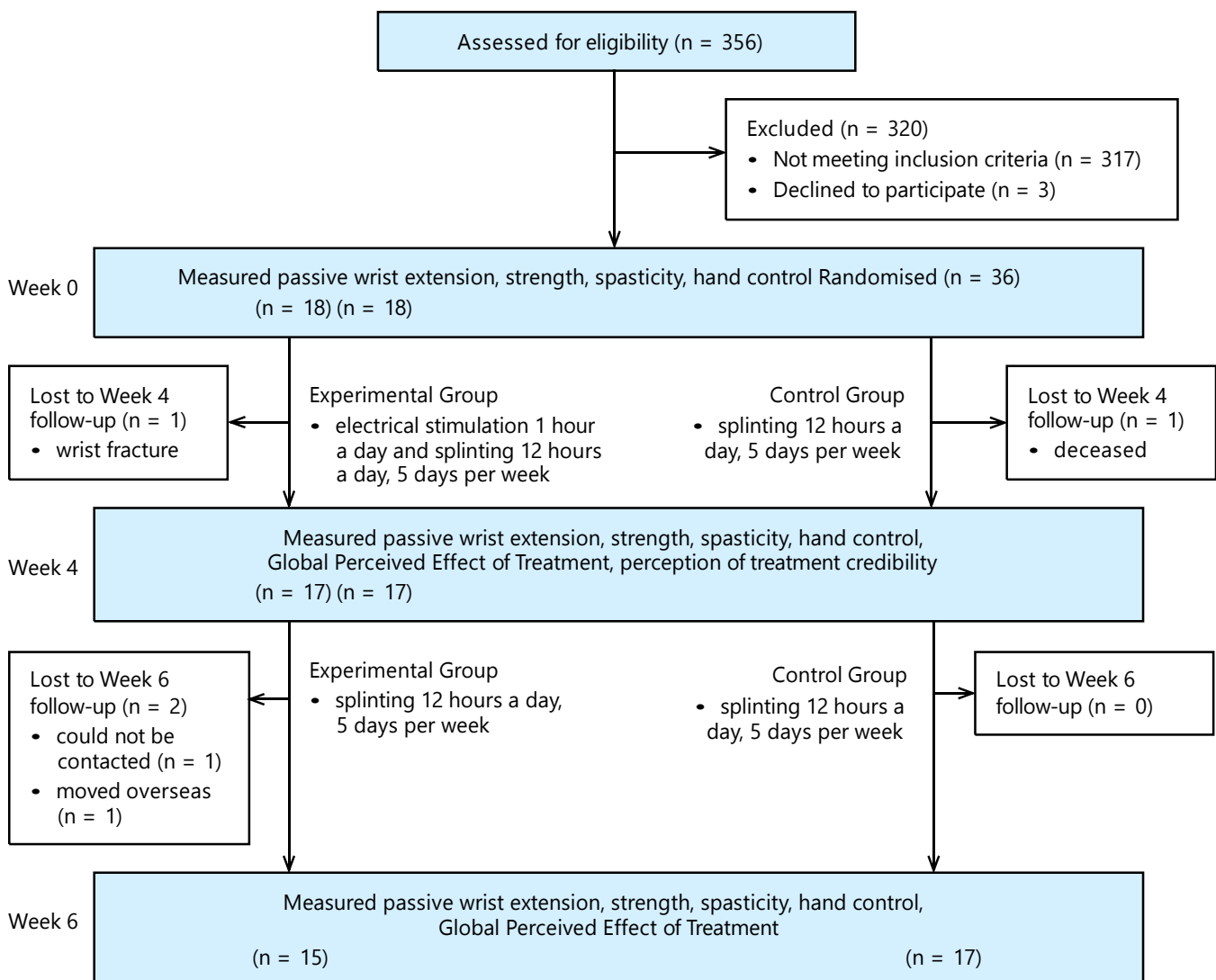
Linear regression analyses were performed to assess the effect of the intervention on passive wrist extension and

strength. The centile routine of Stata<sup>b</sup> was used to derive the median between-group differences and the 95% CIs for the ordinal measures (motor control of hand, spasticity, and Global Perceived Effect of Treatment). The responses from the questionnaires were analysed using chi squared tests. The ratings for treatment effectiveness, treatment worth, and tolerance were dichotomised into < 3 and ≥ 3 for between-group comparisons. The significance level was set at < 0.05. Analyses were conducted separately for the post-intervention and follow-up assessments. Missing data were not imputed. All analyses were performed according to ‘intention-to-treat’.

**Results**

**Flow of participants and therapists through the trial**

A total of 356 patients were screened; 39 met the eligibility criteria but three declined to participate. Hence 36 were recruited and randomised: 31 (86%) had a stroke and 5 (14%) had a traumatic brain injury. Table 1 outlines the demographic and neurological characteristics of the two groups. The flow of the participants through the trial is illustrated in Figure 2. Approximately 15 physiotherapists working in the participating units administered the electrical stimulation and usual care over the course of the trial.



**Figure 2.** Recruitment and flow of participants through the trial.



### Adherence to the trial protocol

Adherence to the electrical stimulation was excellent and adherence to splinting was fair (Table 2). One participant in the experimental group participated in the program for only two days and then declined further electrical stimulation and splinting. He completed all the assessments. Five other participants (two in the experimental group and three in the control group) had poor adherence to the splinting regimen (< 50% adherence). Twelve (33%) participants were unexpectedly discharged home before completion of the program, with seven before the post-intervention assessment and another five after the post-intervention assessment but before the follow-up assessment (six in the experimental group and six in the control group). In all but three cases, their families and carers were relied upon to continue the interventions. In the three cases that this was not possible, an experienced and trained research assistant visited the participants and provided the interventions according to the study protocol.

### Effect of electrical stimulation

All primary and secondary outcome measures are shown in Tables 3 and 4 (individual participant data are presented in Table 5 on the eAddenda). Both groups showed a mean loss in passive wrist extension over the 4-week intervention

period (2 degrees in the experimental group and 9 degrees in the control group). The mean between-group difference at 4 weeks was 7 degrees (95% CI -2 to 15) in favour of the experimental group, which exceeded the pre-determined minimally important level of 5 degrees. However, the 95% CI reflected imprecision around this estimate. At follow-up 2 weeks later, the mean between-group difference was 3 degrees (95% CI -7 to 13) in favour of the control group. There were no convincing treatment effects at 4 or 6 weeks for any of the secondary outcomes although the mean (95% CI) between-group differences of the Global Perceived Effect of Treatment rated by the treating physiotherapists were 1 point (0 to 2) at Week 4 and 3 points (0 to 5) at Week 6.

Tables 6 and 7 show participants' and physiotherapists' perceptions of treatment credibility, respectively. Five participants (3 in the control group and 2 in the experimental group) experienced some discomfort from the hand splints. There were no reports of any adverse events. Overall, the participants of both groups demonstrated no significant between-group differences in their ratings for treatment benefit, worth of treatment, tolerance to treatment, or willingness to continue with treatment. In contrast, the physiotherapists administering the electrical stimulation and splinting protocol reported significantly higher levels

**Table 1.** Baseline characteristics of participants.

Participant characteristics	Randomised (n = 36)	
	Exp (n = 18)	Con (n = 18)
Age at injury (yr), median (IQR)	66 (57 to 75)	48 (34 to 62)
Gender, n male (%)	14 (78)	11 (61)
Cause of injury, n		
Stroke (haemorrhage: infarct: aneurysm)	5: 12: 0	5: 7: 2
TBI (motor vehicle accident: fall: firearm)	1: 0: 0	2: 1: 1
Days from injury to baseline assessment, median (IQR)	42 (32 to 52)	69 (44 to 94)
Baseline GCS <sup>a</sup> , median (IQR)	6 (n = 1)	7 (2 to 12) (n = 4)
Anti-spasticity medication, n (%)	2 (11)	4 (22)

Exp = experimental group, Con = control group, GCS = Glasgow Coma Scale score, TBI = traumatic brain injury, <sup>a</sup>TBI participants only

**Table 2.** Adherence to the trial protocol.

	Exp (n = 18)		Con (n = 18)	
	Expected	Actual median (IQR)	Expected	Actual median (IQR)
Intervention period				
electrical stimulation (hr)	20	22 (20 to 24)	n/a	n/a
splinting (hr)	ε 240	271 (218 to 324)	ε 240	289 (236 to 342)
Follow-up period				
splinting (hr)	ε 120	142 (95 to 189)	ε 120	120 (92 to 148)

Exp = experimental group, Con = control group, n/a = not applicable

**Table 3.** Mean (SD) of groups, mean (SD) difference within groups, and mean (95% CI) difference between groups for passive wrist extension and strength at Weeks 4 and 6.

Outcome	Groups						Difference within groups				Difference between groups	
	Week 0		Week 4		Week 6		Week 4 minus Week 0		Week 6 minus Week 0		Week 4 minus Week 0	Week 6 minus Week 0
	Exp (n=18)	Con (n=18)	Exp (n=17)	Con (n=17)	Exp (n=15)	Con (n=17)	Exp	Con	Exp	Con	Exp minus Con	Exp minus Con
Passive wrist extension at 3 Nm (deg)	56 (11)	58 (10)	54 (11)	49 (12)	47 (13)	52 (15) (n=16)	-2 (8)	-9 (15)	-9 (12)	-6 (15)	-2 to 15 7	-3 (-13 to 7)
Passive wrist extension at 2 Nm (deg)	39 (14)	38 (12)	37 (13)	35 (14)	34 (11)	35 (16)	-2 (10)	-3 (20)	-4 (11)	-2 (20)	-10 to 13	-2 (-14 to 10)
Wrist and finger extensor muscle strength (Nm)	0.7 (1.0)	0.3 (0.6)	2.0 (2.1)	0.6 (1.4)	2.4 (2.7)	0.8 (1.7)	1.2 (1.9)	0.4 (1.2)	1.6 (2.2)	0.6 (1.6)	0.8 (-0.3 to 1.9)	1.0 (-0.4 to 2.4)

Exp = experimental group, Con = control group, Shaded row = primary outcome.

**Table 4.** Median (IQR) of groups and median (95% CI) between-group difference in change for spasticity, hand control and Global Perceived Effect of Treatment at Weeks 4 and 6.

Outcome	Groups						Difference between groups	
	Week 0		Week 4		Week 6		Week 4 minus Week 0	Week 6 minus Week 0
	Exp (n=18)	Con (n=18)	Exp (n=17)	Con (n=17)	Exp (n=15)	Con (n=17)	Exp minus Con	Exp minus Con
Tardieu Scale (0 to 5)								
	(1 to 2)	(1 to 2)	(1 to 2)	(1 to 3)	(1 to 2)	(1 to 2)	(-1 to 1)	(0 to 1)
Motor Assessment Scale score for hand item (0 to 6)	2	2	2	2	2	2	n	n
	(0 to 1)	(0 to 0)	(0 to 3)	(0 to 0)	(0 to 5)	(0 to 0)	(0 to 0)	(0 to 1)
Participants' Global Perceived Effect of Treatment <sub>a</sub> (-6 to 6)	n/a	n/a	n	n	n	n	n	n
			(0 to 3)	(0 to 3)	(0 to 4)	(0 to 2)	(0 to 3)	(0 to 3)
			1	n	(n=14)	(n=15)	1	1
Physiotherapists' Global Perceived Effect of Treatment (-6 to 6)	n/a	n/a				-1		
			(0 to 2)	(-1 to 0)	(0 to 4)	(-2 to 0)	(0 to 2)	(0 to 5)
			(n=15)	(n=14)	(n=10)	(n=10)	1	2

Exp = experimental group, Con = control group, n/a = not applicable. <sup>a</sup>21% (7/34) and 14% (4/29) of the questionnaires were answered by carer on behalf of the participants at the post-intervention assessment and at the follow-up assessment, respectively.

of treatment effectiveness and worth than physiotherapists administering the splinting protocol alone. About half of the physiotherapists who administered the experimental intervention indicated that they would recommend

an electrical stimulation and splinting protocol to the participants if further treatment for wrist contracture was indicated. Similarly, about half of the physiotherapists who

**Table 6.** Participants' perception of treatment credibility at 4 weeks. The ratings for treatment worth and tolerance were dichotomised into <3 and ≥3 for between-group comparisons. 21% (7/34) of the questionnaires were answered by carers on behalf of the participants.

Outcome	Groups		Between-group comparison <i>p</i>
	Exp (n = 17)	Con (n = 17)	
Considered the treatment beneficial, n (%)			0.387
yes	10 (59)	7 (41)	
no	7 (41)	8 (47)	
did not answer	0 (0)	1 (6)	
unsure	0 (0)	1 (6)	
Rating for treatment worth, n (%)			0.866
1 highly worthwhile	3 (18)	6 (35)	
2 reasonably worthwhile	7 (41)	2 (11)	
3 not sure	3 (18)	7 (41)	
4 not too worthwhile	1 (6)	1 (6)	
5 definitely not worthwhile at all	3 (18)	1 (6)	
Rating for tolerance, n (%)			0.492
1 comfortable	5 (29)	4 (24)	
2 slightly uncomfortable	4 (24)	4 (24)	
3 moderately uncomfortable	5 (29)	6 (35)	
4 very uncomfortable but still tolerable	2 (12)	2 (12)	
5 intolerable	1 (6)	0 (0)	
did not answer	0 (0)	1 (6)	
Willing to continue the intervention, n (%)			0.353
yes	11 (65)	9 (53)	
no	5 (29)	8 (47)	
did not answer	1 (6)	0 (0)	

Exp = experimental group, Con = control group

**Table 7.** Physiotherapists' perception of treatment credibility at 4 weeks. The ratings for treatment worth and tolerance were dichotomised into <3 and ≥3 for between-group comparisons. n = number of responses from physiotherapists, not the number of physiotherapists.

Outcome	Groups		Between-group comparison <i>p</i>
	Exp (n = 15)	Con (n = 14)	
Rating for treatment effectiveness, n (%)			0.017
1 very effective	1 (7)	0 (0)	
2 effective	6 (40)	1 (7)	
3 unsure	5 (33)	10 (71)	
4 ineffective	1 (7)	3 (21)	
5 very ineffective	2 (13)	0 (0)	
Rating for treatment worth, n (%)			0.035
1 highly worthwhile	1 (7)	1 (7)	
2 reasonably worthwhile	8 (53)	2 (14)	
3 not sure	1 (7)	4 (29)	
4 not too worthwhile	3 (20)	7 (50)	
5 definitely not worthwhile at all	2 (13)	0 (0)	
Recommended the treatment, n (%)			0.837
yes	8 (53)	8 (57)	
no	7 (47)	6 (43)	

Exp = experimental group, Con = control group

administered the control intervention indicated that they would recommend a splinting protocol alone.

### Blinding of assessors

Blinding of the assessors was reasonably successful. The assessors reported being unblinded in three of the post-intervention assessments and two of the follow-up assessments. On two of these five occasions, a third person not involved in the trial and unaware of the participants' group allocation was asked to read the wrist angle from the protractor while the unblinded assessor did the setup and applied the torque.

### Anti-spasticity medications

Two experimental participants received anti-spasticity medication at baseline. One had the dose increased and the other stopped the medication during the intervention period. In the control group, four participants received anti-spasticity medications at baseline with the dose decreased for two of them during the intervention period. Another participant started anti-spasticity medication during the intervention period and one other participant started it in the follow-up period.

## Discussion

This trial was conducted in an attempt to find a solution to contracture because a Cochrane systematic review indicates that traditional treatment strategies involving passive stretch alone are ineffective. We hypothesised that stretch provided in conjunction with electrical stimulation may be more effective than stretch alone through the possible therapeutic effects of electrical stimulation on strength and spasticity. While the mean between-group difference of 7 degrees in wrist extension was in favour of the experimental group (electrical stimulation and stretch) at Week 4 and exceeded the pre-determined minimally important effect, this estimate of treatment effectiveness was associated with considerable imprecision leading to uncertainty about the added benefit of electrical stimulation (as reflected by the wide 95% CI spanning from -2 to 15). We were also unable to demonstrate a treatment effect of the electrical stimulation on strength and spasticity. This perhaps explains our failure to demonstrate a convincing treatment effect on wrist extension.

The imprecision of our estimate (ie, 95% CI -2 to 15) was greater than expected and greater than a comparable study upon which we based our power calculations (95% CI 4 to 7, Bakhtiary and Fatemy 2008). There are differences between our trial and that of Bakhtiary and Fatemy which may explain these differences. Our trial recruited people with obvious weakness, and either spasticity or reduced extensibility of the long finger flexor muscles after an acquired brain injury regardless of anti-spasticity medication, whereas Bakhtiary and Fatemy recruited patients with spasticity after stroke who were not receiving anti-spasticity medication. It is possible that the two groups of patients respond differently to electrical stimulation. The electrical stimulation protocols were also different. In our trial, electrical stimulation was applied at the maximal tolerable intensity for 1 hour a day whereas Bakhtiary and Fatemy applied supramaximal levels of electrical stimulation (ie, the intensity was set at 25% over the intensity needed to produce a maximum contraction) for 9 minutes a day.

It is not clear how participants tolerated such high doses of electrical stimulation. Another difference is that in our trial electrical stimulation was applied with the wrist held in an extended position in order to optimise any beneficial stretching and strengthening effects. In contrast, Bakhtiary and Fatemy applied electrical stimulation with the ankle unsupported (and presumably in a plantarflexed position). We are not sure if any of these differences between the two trials are important.

There are other factors that may explain the imprecision of our estimate of treatment effectiveness. First, there was considerable variability in the participants' age, length of time post-injury, and degree of spasticity, weakness, motor control, and hand contracture. These factors may vary the way participants responded to the intervention. Second, some participants in our study had difficulty relaxing during measures of passive wrist extension because of pain. Although any inadvertent muscle activity was unlikely to bias the results systematically, it may have added noise to the data leading to an imprecise estimate (ie, wide 95% CI).

Perhaps there are sub-groups of participants who respond more favourably to electrical stimulation than others. For instance, initial strength may be an important determinant of the effectiveness of electrical stimulation. There is growing evidence to suggest that electrical stimulation may be more effective for increasing strength when combined with voluntary movements or functional activity (Alon et al 2008, Bolton et al 2004, Chan et al 2009, de Kroon et al 2002, Ng and Hui-Chan 2007). It is possible that people with some strength in their wrist or finger extensor muscles benefit more from electrical stimulation than those without any strength. However, our sample size was too small to explore this issue with post hoc analyses. While our participants were encouraged to contract the wrist and finger extensor muscles in time with the electrical stimulation, most (72%) participants did not have active wrist and finger movement at baseline and the majority did not have sufficient cognition or concentration to co-operate. Future studies could consider limiting the study cohort to people with some active motor control or using electromyography-triggered electrical stimulation to encourage participants to actively contract their wrist and finger extensor muscles during treatment.

We may have found a clear treatment effect if we had used a stronger dose of electrical stimulation (eg, higher intensity, greater frequency of application, and longer application duration) than the regimen we tested. We applied the electrical stimulation for 1 hour per day, 5 days per week, over 4 weeks. This is in line with the dosage of electrical stimulation provided in a trial reporting a moderate effect of electrical stimulation on wrist and finger extensor muscle strength post-stroke (Bowman et al 1979) but it is less than another trial in which 90 min per day of electrical stimulation was used for 8 weeks (Powell et al 1999). Future studies could investigate the effectiveness of electrical stimulation applied for longer each day and/or over a longer time period. The latter may pose considerable challenges to researchers and clinicians as it is increasingly common for patients to be discharged from hospitals within a few weeks of stroke and it may be difficult to administer the intervention once patients are discharged home.

The feedback from the treating physiotherapists and participants suggest that electrical stimulation is well

tolerated. Adherence to the electrical stimulation protocol was excellent and there were no adverse events. Interestingly, while we did not find a convincing treatment effect on our primary outcome, there was a tendency for the physiotherapists who implemented the electrical stimulation and splint protocol to give a higher score for effectiveness and worth than physiotherapists who implemented the splinting protocol alone (although the lower end of the 95% CI associated with the mean between-group differences indicated no difference). In the absence of any demonstrated treatment effect, this finding may reflect physiotherapists' preconceived beliefs and expectations about electrical stimulation. There was no difference in the number of physiotherapists who indicated that they would recommend an electrical stimulation and splinting protocol versus the number who would recommend a splinting protocol alone.

The results of this trial do not provide conclusive evidence about the effectiveness of electrical stimulation for contracture management. Nor do the results indicate that electrical stimulation is ineffective. Certain sub-groups of patients may respond better to this intervention than others but larger studies will be required to identify them. Until further work has been done in this area, it may be reasonable to apply electrical stimulation for the treatment and prevention of contracture, especially as it is inexpensive, well tolerated, and not associated with harm. v

**Footnotes:** <sup>a</sup>Versports stimulator, Ausmedic Mobility and Rehab, Australia. <sup>b</sup>Stata Data Analysis and Statistical Software, www.stata.com.

**eAddenda:** Table 5 available at [jop.physiotherapy.asn.au](http://jop.physiotherapy.asn.au)

**Ethics:** The study was approved by the ethics committees of the Northern Sydney Central Coast Area Health Service and the participating hospitals. Written consent was obtained from all the participants or their legal guardians before data collection began.

**Competing interests:** Nil

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## References

- Ada L, Canning CG, Low SL (2003) Stroke patients have selective muscle weakness in shortened range. *Brain* 126: 724–731.
- Ada L, Goddard E, McCully J, Stavrinou T, Bampton J (2005) Thirty minutes of positioning reduces the development of shoulder external rotation contracture after stroke: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation* 86: 230–234.
- Ada L, O'Dwyer N, O'Neill E (2006) Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disability and Rehabilitation* 28: 891–897.
- Alon G, Levitt AF, McCarthy PA (2008) Functional electrical stimulation (FES) may modify the poor prognosis of stroke survivors with severe motor loss of the upper extremity: a preliminary study. *American Journal of Physical Medicine and Rehabilitation* 87: 627–636.
- Bakhtari AH, Fatemy E (2008) Does electrical stimulation reduce spasticity after stroke? A randomized controlled study. *Clinical Rehabilitation* 22: 418–425.
- Ben M, Harvey L, Denis S, Glinesky J, Goehl G, Chee S, et al (2005) Does 12 weeks of regular standing prevent loss of ankle mobility and bone mineral density in people with recent spinal cord injuries? *Australian Journal of Physiotherapy* 51: 251–256.
- Bohannon RW (1987) Interrater reliability of hand-held dynamometry. *Physical Therapy* 67: 931–933.
- Bolton DA, Cauraugh JH, Hausenblas HA (2004) Electromyogram-triggered neuromuscular stimulation and stroke motor recovery of arm/hand functions: a meta-analysis. *Journal of the Neurological Sciences* 223: 121–127.
- Botte MJ, Nickel VL, Akeson WH (1988) Spasticity and contracture. Physiologic aspects of formation. *Clinical Orthopaedics and Related Research* 233: 7–18.
- Bowman BR, Baker LL (1985) Effects of waveform parameters on comfort during transcutaneous neuromuscular electrical stimulation. *Annals of Biomedical Engineering* 13: 59–74.
- Bowman BR, Baker LL, Waters RL (1979) Positional feedback and electrical stimulation: an automated treatment for the hemiplegic wrist. *Archives of Physical Medicine and Rehabilitation* 60: 497–502.
- Carr JH, Shepherd RB, Nordholm L, Lynne D (1985) Investigation of a new motor assessment scale for stroke patients. *Physical Therapy* 65: 175–180.
- Chan MK, Tong RK, Chung KY (2009) Bilateral upper limb training with functional electric stimulation in patients with chronic stroke. *Neurorehabilitation and Neural Repair* 23: 357–365.
- Cheng JS, Yang YR, Cheng SJ, Lin PY, Wang RY (2010) Effects of combining electric stimulation with active ankle dorsiflexion while standing on a rocker board: a pilot study for subjects with spastic foot after stroke. *Archives of Physical Medicine and Rehabilitation* 91: 505–512.
- de Jong LD, Nieuwboer A, Aufdemkampe G (2006) Contracture preventive positioning of the hemiplegic arm in subacute stroke patients: a pilot randomized controlled trial. *Clinical Rehabilitation* 20: 656–667.
- de Kroon JR, van der Lee JH, IJzerman MJ, Lankhorst GJ (2002) Therapeutic electrical stimulation to improve motor control and functional abilities of the upper extremity after stroke: a systematic review. *Clinical Rehabilitation* 16: 350–360.
- Dean CM, Mackey FH, Katrak P (2000) Examination of shoulder positioning after stroke: A randomised controlled pilot trial. *Australian Journal of Physiotherapy* 46: 35–40.
- Fergusson D, Hutton B, Drodge A (2007) The epidemiology of major joint contractures: a systematic review of the literature. *Clinical Orthopaedics and Related Research* 456: 22–29.
- Glinesky J, Harvey L, Van Es P (2007) Efficacy of electrical stimulation to increase muscle strength in people with neurological conditions: a systematic review. *Physiotherapy Research International* 12: 175–194.
- Gustafsson L, McKenna K (2006) A programme of static positional stretches does not reduce hemiplegic shoulder pain or maintain shoulder range of motion—a randomized controlled trial. *Clinical Rehabilitation* 20: 277–286.
- Harburn KL, Potter PJ (1993) Spasticity and contractures.

- Physical Medicine and Rehabilitation State of the Art Reviews* 7: 113–132.
- Harvey L, de Jong I, Goehl G, Mardwedel S (2006) Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. *Australian Journal of Physiotherapy* 52: 251–258.
- Harvey L, King M, Herbert R (1994) Test-retest reliability of a procedure for measuring extensibility of the extrinsic finger flexor muscles. *Journal of Hand Therapy* 7: 251–254.
- Harvey LA, Batty J, Crosbie J, Poulter S, Herbert RD (2000) A randomized trial assessing the effects of 4 weeks of daily stretching on ankle mobility in patients with spinal cord injuries. *Archives of Physical Medicine and Rehabilitation* 81: 1340–1347.
- Harvey LA, Byak AJ, Ostrovskaya M, Glinsky J, Katte L, Herbert RD (2003) Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries. *Australian Journal of Physiotherapy* 49: 176–181.
- Horsley SA, Herbert RD, Ada L (2007) Four weeks of daily stretch has little or no effect on wrist contracture after stroke: a randomised controlled trial. *Australian Journal of Physiotherapy* 53: 239–245.
- Katalinic OM, Harvey LA, Herbert RD, Moseley AM, Lannin NA, Schurr K (2010) Stretch for the treatment and prevention of contractures. *Cochrane Database of Systematic Reviews* 9: CD007455.
- Kwah LK, Harvey LA, Diong JH, Herbert RD (2012) Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *Journal of Physiotherapy* 58: 41–47.
- Lannin NA, Cusick A, McCluskey A, Herbert RD (2007) Effects of splinting on wrist contracture after stroke: a randomized controlled trial. *Stroke* 38: 111–116.
- Lannin NA, Horsley SA, Herbert R, McCluskey A, Cusick A (2003) Splinting the hand in the functional position after brain impairment: a randomized, controlled trial. *Archives of Physical Medicine and Rehabilitation* 84: 297–302.
- Moseley AM (1997) The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. *Physical Therapy* 77: 240–247; discussion 248–259.
- Ng SS, Hui-Chan CW (2007) Transcutaneous electrical nerve stimulation combined with task-related training improves lower limb functions in subjects with chronic stroke. *Stroke* 38: 2953–2959.
- Patrick E, Ada L (2006) The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clinical Rehabilitation* 20: 173–182.
- Powell J, Pandyan AD, Granat M, Cameron M, Stott DJ (1999) Electrical stimulation of wrist extensors in poststroke hemiplegia. *Stroke* 30: 1384–1389.
- Schneider LS, Olin JT (1996) Clinical global impressions in Alzheimer's clinical trials. *International Psychogeriatrics* 8: 277–288.
- Tardieu G, Shentoub S, Delaure R (1954) A la recherche d'une technique de mesure de la spasticite. *Revista de Neurologia* 91: 143–144.
- Yan T, Hui-Chan CW, Li LS (2005) Functional electrical stimulation improves motor recovery of the lower extremity and walking ability of subjects with first acute stroke: a randomized placebo-controlled trial. *Stroke* 36: 80–85.


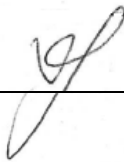
## Chapter 5

Standing, electrical stimulation and splinting are no better than standing alone for management of ankle plantarflexion contractures in people with traumatic brain injury: a randomised controlled trial

**Statement from co-authors confirming authorship contribution of the  
PhD candidate**

As co-authors of the paper "Standing, electrical stimulation and splinting is no better than standing alone for ankle contractures in people with brain injury: a randomised controlled trial", we confirm that Joan Leung has made contributions to the following:

- Development of the research question
- Conception and design of the research
- Collection, analysis and interpretation of data
- Writing of the manuscript and critical revision of the content for publication

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**Standing, electrical stimulation and splinting are no better than standing alone for management of ankle plantarflexion contractures in people with traumatic brain injury: a randomised controlled trial**

**ABSTRACT**

**Question:** Is using a combination of standing, electrical stimulation and splinting more effective than standing alone for the management of ankle contractures after severe brain injury? **Design:** A multi-centre randomised trial with concealed allocation, assessor blinding and intention-to-treat analysis. **Participants:** Thirty-six adults with severe traumatic brain injury and ankle plantarflexion contractures. **Intervention:** All participants underwent a 6-week program. The intervention group received tilt table standing, electrical stimulation and ankle splinting. The control group received tilt table standing alone. **Outcome measures:** The primary outcome was passive ankle dorsiflexion with a 12Nm torque. Secondary outcomes included passive dorsiflexion with lower torques (3, 5, 7 and 9Nm), spasticity, Functional Independence Measure score for the walking item, walking speed, Global Perceived Effect of Treatment and perceived treatment credibility. Outcome measures were taken at baseline (Week 0), end of intervention (Week 6), and follow-up (Week 10). **Results:** The mean between-group differences (95% CI) for passive ankle dorsiflexion at Week 6 and Week 10 were -3 degrees (-8 to 2) and -1 degree (-6 to 4), respectively, in favour of the control group. There was a small mean reduction of 1 point in spasticity at Week 6 (95% CI -1.8 to -0.1) in favour of the intervention group but this effect disappeared at Week 10. There were no differences for other secondary outcome measures except that physiotherapists' perceived treatment credibility. **Conclusion:** Tilt table standing, electrical stimulation and splinting is not better than tilt table standing alone for the management of ankle contractures after severe brain injury.

**Trial Registration:** ACTRN12608000637347

**Key words:** Traumatic Brain Injury, Contracture, Splinting, Electrical Stimulation, Stretch.

## INTRODUCTION

Contractures are a common secondary problem after acquired brain injury.<sup>1,2</sup> Traditional treatment for contractures has primarily involved passive stretch. However, a systematic review found that commonly-used passive stretch interventions do not produce clinically worthwhile effect.<sup>3</sup> Two reasons may explain this finding. Firstly, the dose of passive stretch used in the included trials may be insufficient (median dose: 6 hours a day over 30 days). It has been demonstrated in a randomised controlled trial that 24 hours a day of passive stretch produced a greater effect on joint range than an hour a day of passive stretch (between-group difference of 22 degrees, 95% CI 13 to 31), and when the dose of passive stretch was reduced its effect diminished.<sup>4</sup> Secondly, passive stretch focuses primarily on increasing the length of soft tissues but does not address the factors which are believed to contribute to contractures such as muscle weakness and spasticity. The continual presence of these factors, such as spasticity and muscle weakness,<sup>1,5</sup> may explain why passive stretch fails to produce a large or sustained effect.

Effective management of contractures may therefore require combining a high dose of passive stretch with treatments that address the underlying causes of contracture. A case report has described an intensive program of a high dose of passive stretch combined with motor training for the correction of chronic knee contractures.<sup>6</sup> However, case reports only provide weak evidence. High quality evidence is needed to verify the effectiveness of this approach.

The purpose of this study is to compare a multimodal treatment program (combining tilt table standing, splinting and electrical stimulation) with a single modality treatment program (tilt table standing alone). People with severe traumatic brain injury were targeted because contractures are common in this clinical population. Tilt table standing and splinting were investigated because both are commonly used and together they increase total stretch dose. Electrical stimulation is used because of its potential therapeutic effects on muscle weakness and spasticity; the two known contributors to contractures. A systematic review<sup>7</sup> and a randomised controlled trial<sup>8</sup> suggest that electrical stimulation increases strength after acquired brain injury. Five randomised controlled trials also report a decrease in spasticity with electrical stimulation.<sup>9-13</sup> In addition, people with contractures often have severe motor impairments and therefore very limited ability to participate in active treatment. Electrical stimulation can elicit muscle contractions in people with little or no ability to voluntarily

contract muscles.<sup>14</sup> Hence, it is seemingly an appropriate adjunct treatment for contractures in the target population. The research question therefore was: Is combining tilt table standing, electrical stimulation and ankle splinting more effective than tilt table standing alone in the treatment of ankle contractures following severe traumatic brain injury?

## **METHOD**

### **Design**

A multi-centre, assessor-blinded, randomised controlled study was undertaken. All participants were randomly allocated to one of two groups using a blocked randomisation schedule: intervention group (tilt table standing, electrical stimulation and ankle splinting) or control group (tilt table standing only). The random allocation sequence was computer-generated by a person not involved in participant recruitment. Group allocation was concealed using consecutively numbered, sealed, opaque envelopes which were kept off-site. After baseline assessment, the investigator contacted a person who was not involved in the study to reveal the group allocation. End of intervention and follow-up assessments were conducted at Week 6 and Week 10, respectively.

### **Participants, therapists and centres**

All patients admitted with a traumatic brain injury to one of three Metropolitan Brain Injury Rehabilitation Units in Sydney (namely, Royal Rehabilitation Centre Sydney, Liverpool Hospital and Westmead Hospital) were screened between January 2009 and December 2014. They were invited by their physiotherapists to participate in the study if they fulfilled the following criteria:

- 1) first documented traumatic brain injury
- 2) a score of 4 or less on the walking item of Functional Independence Measure (i.e., an inability to walk 17 metres without physical assistance or 50 metres with supervision)
- 3) presence of an ankle contracture (defined as passive dorsiflexion ankle range of motion less than 5 degrees at a torque of 12Nm; measured using the device specified in the study)
- 4) ability to participate in the assessment and intervention program
- 5) no unstable medical conditions or recent ankle fractures
- 6) no other neurological conditions such as spinal cord injury or cerebrovascular disease
- 7) anticipated length of stay in hospital of at least 6 weeks
- 8) no Botulinum Toxin injection to ankle joint within 3 months

The study was approved by the ethics committees of the Northern Sydney Central Coast Area Health Service (Australia) and the participating hospitals. Written consent was obtained from all the participants or their legal guardians.

### **Intervention**

Participants in both groups received a 6-week program. The intervention group received 30 minutes of tilt table standing, electrical stimulation to the ankle dorsiflexor muscles, 5 days per week and ankle splinting 12 hours a day, at least 5 days a week. Participants were stood on the tilt table as vertical as they would tolerate. A wedge was placed under the foot to maximise the stretch to the plantarflexor muscles. Electrical stimulation was applied to the dorsiflexor muscles while participants stood on the tilt table. The electrical stimulation was used like this in an attempt to increase the strength of the dorsiflexor muscles in their shortest length where they are often weakest.<sup>15</sup> Electrical stimulation was applied using a digital neuromuscular stimulation unit<sup>a</sup> through a pair of square electrodes (5cm x 5cm). Parameters were: pulse width of 300  $\mu$ s, frequency of 50 Hz, on time of 15 s, off time of 15 s, and a ramping-up period of 1.5 s. These parameters were selected to optimise any strengthening benefits.<sup>16</sup> The amplitude of electrical stimulation was set to produce maximum tolerable muscle contractions. For participants who were unable to indicate tolerable levels of stimulation, the amplitude of stimulation was set to generate a palpable muscle contraction. Participants were encouraged to voluntarily contract their muscles with the electrical stimulation but most were in post-traumatic amnesia and had severe cognitive and motor deficits which limited their abilities to actively participate in therapy. Both tilt table standing and electrical stimulation were administered by physiotherapists. The intervention group also wore an ankle splint<sup>b</sup> for at least 12 hours a day, 5 days per week. The splints positioned the ankles in maximum tolerable dorsiflexion. They were applied by physiotherapists, nursing staff or physiotherapy assistants as directed by the treating physiotherapists.

Participants in the control group only received tilt table standing for 30 minutes, 3 times a week. They did not stand with a wedge under the foot. In short, the intervention programs of the two groups differed in three ways. Firstly, the intervention group received 30 sessions of tilt table standing while the control group only received 18 sessions. Secondly, the intervention group received maximum stretch (by using a wedge where applicable) while standing on the tilt table while the control group did not receive stretch beyond a plantigrade position. Thirdly, the intervention group received electrical stimulation and ankle splinting

while the control group did not. During the 4-week follow-up period, participants in both groups stood on a tilt table 30 minutes, 3 times a week, without a wedge. No electrical stimulation or splinting was administered to the ankle during this time. Over the course of the trial, all participants received usual multidisciplinary rehabilitation provided by the participating units as appropriate. This consisted of physiotherapy, occupational therapy, speech therapy, recreational therapy and psychological therapy. Physiotherapy included an individualised motor training program which, where appropriate, included practice of sit to stand, walking and standing. The usual care for both groups involved positioning of participants' feet in dorsiflexion while seated and lying. No other passive stretch-based interventions were administered to the ankle during the trial. Physiotherapists were assigned to participants on admission (i.e., prior to recruitment). Thus, the physiotherapists managed an arbitrary mix of control and intervention participants. Diaries were used to record all interventions. No other passive stretch-based interventions were administered to the ankle. In addition, no Botulinum Toxin injection was administered to the ankle during the study period. Use of anti-spasticity medication was not mandated by the study protocol but was recorded. Assessors and medical staff were blinded to group allocation but treating physiotherapists and participants were not. Success of assessor blinding was monitored.

### **Outcome measures**

There were one primary and nine secondary outcomes. The primary outcome was passive ankle dorsiflexion measured with a torque of 12Nm with the knee in extension. This was used to reflect the extensibility of the bi-articular ankle plantarflexor muscles. The secondary outcomes were passive dorsiflexion range at 3Nm, 5Nm, 7Nm and 9Nm, spasticity, Functional Independence Measure score for the walking item, walking speed, physiotherapists' and participants' Global Perceived Effect of Treatment and perceived treatment credibility. All outcome measures were taken at the beginning of the study (Week 0), end of the intervention (Week 6), and follow-up (Week 10). The outcome measures were taken by one of the five blinded and trained assessors who assessed participants of both groups. The end of intervention and follow-up assessments were conducted at least 24 hours and within 3 days after the last session of intervention.

Passive ankle dorsiflexion was measured using a specially made device and standardised procedure.<sup>17</sup> This torque-controlled procedure has a high test-retest reliability (Intra Class Correlation 0.95). With the participant lying supine and the ankle firmly positioned on the

footplate, a standardised torque was applied to the ankle by hanging weights from the rim of the wheel (Figure 1). A pre-stretch was administered by applying a constant ankle dorsiflexion torque of 12Nm for 3 minutes. Passive ankle dorsiflexion range was then measured with progressively larger torques: 3Nm, 5Nm, 7Nm, 9Nm and then 12 Nm. Various torques were used for two reasons. Firstly, joint angle could change in response to a treatment for a low torque but not a high torque or vice versa. Secondly, multiple torque-displacement values could provide information about the torque-angle relationship which cannot be gauged from just one single measure. The angle of the footplate and the inclination of tibia were measured using a digital inclinometer. The procedure was modified for two participants (both in the control group) who were too restless to comply with the standard procedure. Modifications included exclusion of pre-stretch and reversing the order of measurements by starting with the largest torque (12Nm). This was to ensure that the primary outcome measure (joint angle with 12Nm) was obtained. The same procedure was used for all the assessments for these two participants. This modified procedure was also used for a third participant (in the control group) who became too agitated in the follow-up assessment to adhere to the standard procedure. No other changes were made to the outcome measures or protocol since the commencement of the study.

Spasticity of ankle plantarflexor muscles was rated based on the reaction to passive stretch at high speed (not angle of catch) using the 5-point scale of the Tardieu Scale.<sup>18</sup> The Tardieu Scale has a high percentage agreement with laboratory measures of spasticity.<sup>19</sup> Participants were instructed to relax during the test in supine with the lower leg supported on a roll. The assessor moved the participant's ankle as fast as possible.

Activity limitation was assessed using the walking item of the Functional Independence Measure and the 10-metre walk test (ICC 0.998).<sup>20</sup> Functional Independent Measure has a high inter-rater reliability for all the motor items including walking (ICC 0.84 to 0.97).<sup>21</sup> The 10-meter walk test was only conducted on participants who could walk without physical assistance. The same walking aide was used in all assessments by those who required walking aides on the initial assessment. Participants were asked to walk over a 14-metre walkway as fast as possible. Time taken to walk the middle 10 metres was used to calculate walking speed. Walking speed was recorded as zero in those who could not walk without physical assistance.

The Global Perceived Effect of Treatment was rated by the treating physiotherapists and participants (or their carers if the participants did not have the capacity to answer the questions). Using separate questionnaires, the treating physiotherapists and participants (or their carers) were initially asked if they thought the ankle was better, the same or worse. They were then asked to rate the improvement or deterioration between 1 (a little better/a little worse) and 6 (a very great deal better/a very great deal worse). These responses were then combined into a single 13-point scale with -6 reflecting a very great deal worse, zero reflecting no change and +6 reflecting a very great deal better.

Perceived treatment credibility was evaluated by both the treating physiotherapists and participants (or their carers) at Week 6 using separate questionnaires. Participants were asked to provide ratings for tolerance to treatment, perceived treatment worth and perceived treatment benefit using 5-point scales (Figure 6). They were also asked if they were willing to continue with the same treatment if it was to be provided (scored as “yes” or “no”). Treating physiotherapists were asked to rate their perceived treatment worth and treatment effectiveness using 5-point scales (Figure 7), and indicate if they would recommend the same protocol to the participants if further treatment was needed for the ankle (scored as “yes” or “no”). Physiotherapists and participants were also asked to report any issues or concerns about the intervention(s) and how they were managed using open-ended questions.

### **Data analysis**

The sample size was calculated *a priori* based on best estimates. A sample of 36 participants was recruited to provide an 80% probability of detecting a between-group difference of 5 degrees for the primary outcome, assuming a standard deviation of 5 degrees<sup>22</sup> and a 10% drop-out rate. The minimally worthwhile treatment effect for the primary outcome was set at 5 degrees in line with a number of previous studies on contractures.<sup>23-28</sup>

Linear regression analyses were performed to assess passive dorsiflexion, walking speed and Global Perceived Effect of Treatment. One-factor ANOVA was used to analyse categorical data namely Functional Independent Measure score for the walking item and spasticity. Chi squared tests were used to analyse perceived treatment credibility. The significance level was set at < 0.05. Analyses were conducted separately for the end of intervention and follow-up assessments. Missing data were not imputed. All analyses were performed according to ‘intention-to-treat’.

## **RESULTS**

### **Flow of participants and therapists through the study**

A total of 681 patients with traumatic brain injury were screened over 5 years from January 2009 to December 2013. Ultimately, 36 patients were randomised. The flow of the participants through the study is illustrated in Figure 2. Table 1 outlines the demographic and injury characteristics of the intervention and control groups. The characteristics of the two groups were similar. The median (IQR) length of post-traumatic amnesia was 180 (115 to 180) and 125 (90 to 180) for the intervention group and control group, respectively. This reflects the severe nature of participants' brain injury. Most participants were in post-traumatic amnesia at the time of recruitment as indicated by the median (IQR) time between injury and baseline assessment. In addition, majority of the participants could not walk or needed a lot of assistance on walking. Only 6 participants (those who scored 4 for the walking item of the Functional Independence Measure) could participate in the 10-metre walk test at baseline. The number of participants who could participate in the walk test increased to 17 and 18 at end of intervention and follow-up assessments respectively. Those who could not participate in the walk test (that is, unable to walk 14 metres without physical assistance) had their walking speed recorded as zero in accordance with the study protocol. The data of all participants were entered into the analysis for walking speed irrespective of whether they participated in the walk test or not. Approximately 14 physiotherapists working in the participating units administered the interventions as per group allocation and provided usual care over the course of the study. All participants (except one) were assessed in hospital. Data collection was completed in April 2014.

### **Adherence to the study protocol**

Adherence to the various aspects of the intervention was summarised in Table 2. The overall adherence was fairly good but there was considerable variability due to a number of reasons. For instance, adherence of tilt table standing was reduced for in the intervention period due to fainting, storming, fatigue or behavioural issues (10 participants) and tilt table standing was discontinued in the follow-up period due to medical or psychological reasons, or early discharge (3 participants). The adherence to electrical stimulation was reduced primarily due to the reduced standing time and not related to any intolerance of electrical stimulation. The adherence to splinting was reduced because of behavioural issues (3 participants), poor tolerance (1 participant) and skin problems (1 participant).



One participant violated the protocol and received Botulinum Toxin injection for his ankle 4 days into the follow-up period. The use of anti-spasticity during the course of the study was summarised in Table 3. Importantly, the doctors prescribing the medications were blinded to participants' group allocation. There were also minor deviations from the protocol related to the timing of assessments (Table 2). The deviations were due to early discharges, public holidays, medical problems and acute illnesses. The blinding of the assessors was reasonably successful. Assessors were unblinded in two of the end of intervention assessments and one of the follow-up assessments. In two of these assessments, a third person, who was otherwise not involved in the study, was asked to take the readings from the dynamometer for the passive ankle range.

### **Effect of multimodal treatment**

The mean between-group differences (95% CI) for passive ankle dorsiflexion with 12Nm at Week 6 and Week 10 were -3 degrees (-8 to 2) and -1 degree (-6 to 4), respectively (Figure 3). Both were in favour of the control group (that is, the control group had 3 degrees and 1 degree more passive dorsiflexion, on average, compared to the intervention group at Week 6 and Week 10 respectively) and less than the pre-specified minimally worthwhile treatment effect of 5 degrees. There was a mean reduction in spasticity of 1 point (95% CI -1.8 to -0.1) at Week 6, favouring the experimental group, but this effect disappeared at Week 10. No between-group differences were found in walking speed, Functional Independence Measure score for the walking item, and participants' and physiotherapists' Global Perceived Effect of Treatment. All the primary and secondary outcome measures are shown in Tables 4 and 5 (individual participant data are presented in Table 6 on the eAddenda).

Overall, there were no differences between groups for participants' tolerance to treatment, perceived treatment benefit, perceived treatment worth, and willingness to continue with treatment. In contrast, the physiotherapists administering the intervention for the intervention group rated perceived treatment effectiveness and perceived treatment worth higher than the physiotherapists administering the control intervention. They were also twice as likely as the physiotherapists administering the control intervention to recommend the intervention protocol to the participants if further treatment for ankle contracture was indicated (81% vs

39%). Table 7 and 8 show participants' and physiotherapists' perceived treatment credibility, respectively.

## **DISCUSSION**

This study compared a multimodal treatment program with a single modality treatment program for contracture management. It was conducted because a systematic review indicates that passive stretch alone is ineffective.<sup>3</sup> We hypothesised that a program of tilt table standing combined with electrical stimulation and splinting may be more effective than tilt table standing alone for the treatment of contracture. We added electrical stimulation because it may improve strength and reduce spasticity and thus address important contributors to contracture. We provided splinting and additional sessions of tilt table standing sessions to the intervention group in order to increase the dose of passive stretch. Contrary to our expectations, the study showed that 6 weeks of regular standing on a tilt table combined with electrical stimulation and ankle splinting did not provide added benefits when compared to a less intensive program of tilt table standing alone in people with severe traumatic brain injury and ankle contractures. The upper end of the 95% confidence interval associated with the mean between-group difference of ankle range did not reach the pre-specified minimally worthwhile treatment effect of 5 degrees. This indicates that the failure to detect a treatment effect was not due to an inadequate sample size. Despite the findings, the physiotherapists who implemented the multimodal program scored treatment effectiveness and worth higher than physiotherapists who implemented the tilt table standing alone. They were also twice as willing to recommend the treatment they provided compared to those implemented tilt table standing alone. This is possibly a reflection of the physiotherapists' preconceived beliefs and expectations about the multimodal program.

A number of reasons may explain our failure to demonstrate a treatment effect. Firstly, the control group received some passive stretch (tilt table standing) although in a considerably lower dose than the intervention group. This was done because tilt table standing is often used in people with brain injury for purposes other than stretching. For example, it is used to get patients upright and to provide initial training for standing. We therefore could not justify depriving participants in the control group of this intervention. However, the inclusion of tilt table standing for the control group inevitably reduced the treatment contrast between the intervention and control groups, possibly diluting any possible treatment effects of the multimodal program. Secondly, our study recruited participants with severe traumatic brain

injury and ankle contractures. These participants often had severe cognitive and behavioural impairments and complex medical issues. These characteristics imposed considerable challenges for the implementation of treatment program. This reduced adherence might have influenced the outcome.

Electrical stimulation was used in this study to address the contributors to contracture, namely muscle weakness and spasticity. The feedback from both participants and physiotherapists indicated that the use of electrical stimulation was feasible. However our study did not find an improvement in joint range. Electrical stimulation was applied for 30 minutes a day, 5 days a week over 6 weeks. This dose may be insufficient. A trial which used a supramaximal dose of electrical stimulation (9 minutes a day over 4 weeks) found a small effect on joint range (5 degrees, 95% CI 3 to 8) and spasticity when compared with a group without electrical stimulation.<sup>22</sup> Our participants with severe traumatic brain injury, however, may not be able to tolerate supramaximal doses or longer durations of electrical stimulation. In addition, electrical stimulation was applied to the ankle dorsiflexor muscles with the ankle in maximal dorsiflexion. This was done to maximise stretch and to strengthen the dorsiflexor muscles in their inner range where they are often weakest.<sup>15</sup> The induced muscle contractions were isometric. It is not clear whether we would have obtained different results if we applied electrical stimulation in a different way or applied electrical stimulation to the gastrocnemius muscles instead.

Another possible reason for not finding an effect is that most of our participants (64%) had severe weakness or no muscle activity (grade 2 or less) in their ankle dorsiflexor muscles at baseline, and many also did not have the cognitive ability to contract their ankle muscles in synchronisation with the electrical stimulation. There is increasing evidence supporting the combination of electrical stimulation with volitional muscle contractions for motor training.<sup>29-37</sup> The potential value of electrical stimulation may be undermined if participants are unable to work voluntarily with the electrical stimulation. Three other trials have investigated electrical stimulation in people with acquired brain injury and severe motor impairments but the findings of all three are inconclusive.<sup>23,38,39</sup> Our findings suggest that electrical stimulation is not effective for contracture management in people with severe traumatic brain injury. However, these findings may not generalisable to other clinical conditions or patients with less severe brain injury.

Our results indicate that there was no difference between a single modality treatment program of tilt table standing and a multimodal treatment program combining tilt table standing, electrical stimulation and ankle splinting. While it is always tempting to look at within-group changes in trials like this and use the data to conclude that both programs were equally effective (or ineffective), this is not a valid interpretation without a control group that had no intervention. We did not attempt to assess the effectiveness of individual modalities. Our finding, however, did suggest that the addition of splinting was not therapeutic. This finding is consistent with previous clinical trials on splinting which also failed to demonstrate treatment effects.<sup>27,28,40</sup>

In summary this study, along with the many others that have preceded it, does not provide a solution to contractures. We selected tilt table standing, electrical stimulation and ankle splinting because they are commonly used in people with severe brain injury and their effectiveness when used in combination have never been investigated. In addition, they are amongst the few modalities that can be used in people with severe brain injury who have a limited ability to actively participate in treatment. Despite our failure to demonstrate a treatment effect, the findings of this study should not deter further research on this topic. Future research could investigate combining high dosages of passive stretch with medical interventions such as anti-spasticity medications, Botulinum Toxin injections and, where possible, repetitious practice of functional activities.

## REFERENCE

1. Kwah LK, Harvey LA, Diong JH, Herbert RD. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J Physiother* 2012;58:41-7.
2. Fergusson D, Hutton B, Drodge A. The epidemiology of major joint contractures: a systematic review of the literature. *Clin Orthop Relat Res* 2007:22-9.
3. Katalinic OM, Harvey LA, Herbert RD. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: a systematic review. *Phys Ther* 2011;91:1-14.
4. Moseley AM, Hassett LM, Leung J, Clare JS, Herbert RD, Harvey LA. Serial casting versus positioning for the treatment of elbow contractures in adults with traumatic brain injury: a randomized controlled trial. *Clin Rehabil* 2008;22:406-17.
5. Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disabil Rehabil* 2006;28:891-7.
6. Leung J, Harvey LA, Moseley AM. An intensive programme of passive stretch and motor training to manage severe knee contractures after traumatic brain injury: a case report. *Physiother Can* 2013;65:223-8.
7. Glinsky J, Harvey L, Van Es P. Efficacy of electrical stimulation to increase muscle strength in people with neurological conditions: a systematic review. *Physiother Res Int* 2007;12:175-94.
8. Rosewilliam S, Malhotra S, Roffe C, Jones P, Pandyan AD. Can surface neuromuscular electrical stimulation of the wrist and hand combined with routine therapy facilitate recovery of arm function in patients with stroke? *Arch Phys Med Rehabil* 2012;93:1715-21 e1.
9. Malhotra S, Pandyan AD, Rosewilliam S, Roffe C, Hermens H. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. *Clin Rehabil* 2011;25:184-91.
10. Yan T, Hui-Chan CW, Li LS. Functional electrical stimulation improves motor recovery of the lower extremity and walking ability of subjects with first acute stroke: a randomized placebo-controlled trial. *Stroke* 2005;36:80-5.
11. Sabut SK, Sikdar C, Kumar R, Mahadevappa M. Functional electrical stimulation of dorsiflexor muscle: effects on dorsiflexor strength, plantarflexor spasticity, and motor recovery in stroke patients. *NeuroRehabilitation* 2011;29:393-400.
12. Sahin N, Ugurlu H, Albayrak I. The efficacy of electrical stimulation in reducing the post-stroke spasticity: a randomized controlled study. *Disabil Rehabil* 2012;34:151-6.
13. Lin Z, Yan T. Long-term effectiveness of neuromuscular electrical stimulation for promoting motor recovery of the upper extremity after stroke. *J Rehabil Med* 2011;43:506-10.
14. Landau WM, Sahrman SA. Preservation of directly stimulated muscle strength in hemiplegia due to stroke. *Arch Neurol* 2002;59:1453-7.
15. Ada L, Canning CG, Low SL. Stroke patients have selective muscle weakness in shortened range. *Brain* 2003;126:724-31.
16. Bowman BR, Baker LL. Effects of waveform parameters on comfort during transcutaneous neuromuscular electrical stimulation. *Ann Biomed Eng* 1985;13:59-74.
17. Harvey L, Byak A, Ostrovskaya M, Glinsky J. Reliability of a device designed to measure ankle mobility. *Spinal Cord* 2003;41:559-62.

18. Tardieu G, Shentoub S, Delaure R. A la recherche d'une technique de mesure de la spasticite. *Rev Neurol* 1954;91:143-4.
19. Patrick E, Ada L. The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clin Rehabil* 2006;20:173-82.
20. van Loo MA, Moseley AM, Bosman JM, de Bie RA, Hassett L. Inter-rater reliability and concurrent validity of walking speed measurement after traumatic brain injury. *Clin Rehabil* 2003;17:775-9.
21. Donaghy S, Wass PJ. Interrater reliability of the Functional Assessment Measure in a brain injury rehabilitation program. *Arch Phys Med Rehabil* 1998;79:1231-6.
22. Bakhtiary AH, Fatemy E. Does electrical stimulation reduce spasticity after stroke? A randomized controlled study. *Clin Rehabil* 2008;22:418-25.
23. Leung J, Harvey LA, Moseley AM, et al. Electrical stimulation and splinting were not clearly more effective than splinting alone for contracture management after acquired brain injury: a randomised trial. *J Physiother* 2012;58:231-40.
24. Harvey LA, Batty J, Crosbie J, Poulter S, Herbert RD. A randomized trial assessing the effects of 4 weeks of daily stretching on ankle mobility in patients with spinal cord injuries. *Arch Phys Med Rehabil* 2000;81:1340-7.
25. Harvey LA, Byak AJ, Ostrovskaya M, Glinsky J, Katte L, Herbert RD. Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries. *Aust J Physiother* 2003;49:176-81.
26. Horsley SA, Herbert RD, Ada L. Four weeks of daily stretch has little or no effect on wrist contracture after stroke: a randomised controlled trial. *Aust J Physiother* 2007;53:239-45.
27. Lannin NA, Cusick A, McCluskey A, Herbert RD. Effects of splinting on wrist contracture after stroke: a randomized controlled trial. *Stroke* 2007;38:111-6.
28. Lannin NA, Horsley SA, Herbert R, McCluskey A, Cusick A. Splinting the hand in the functional position after brain impairment: a randomized, controlled trial. *Arch Phys Med Rehabil* 2003;84:297-302.
29. Taylor L, Lewis GN, Taylor D. Short-term effects of electrical stimulation and voluntary activity on corticomotor excitability in healthy individuals and people with stroke. *J Clin Neurophysiol* 2012;29:237-43.
30. Alon G, Levitt AF, McCarthy PA. Functional electrical stimulation (FES) may modify the poor prognosis of stroke survivors with severe motor loss of the upper extremity: a preliminary study. *Am J Phys Med Rehabil* 2008;87:627-36.
31. Bolton DA, Cauraugh JH, Hausenblas HA. Electromyogram-triggered neuromuscular stimulation and stroke motor recovery of arm/hand functions: a meta-analysis. *J Neurol Sci* 2004;223:121-7.
32. Chan MK, Tong RK, Chung KY. Bilateral upper limb training with functional electric stimulation in patients with chronic stroke. *Neurorehabil Neural Repair* 2009;23:357-65.
33. de Kroon JR, van der Lee JH, IJzerman MJ, Lankhorst GJ. Therapeutic electrical stimulation to improve motor control and functional abilities of the upper extremity after stroke: a systematic review. *Clin Rehabil* 2002;16:350-60.
34. de Kroon JR, IJzerman MJ, Chae J, Lankhorst GJ, Zilvold G. Relation between stimulation characteristics and clinical outcome in studies using electrical stimulation to improve motor control of the upper extremity in stroke. *J Rehabil Med* 2005;37:65-74.

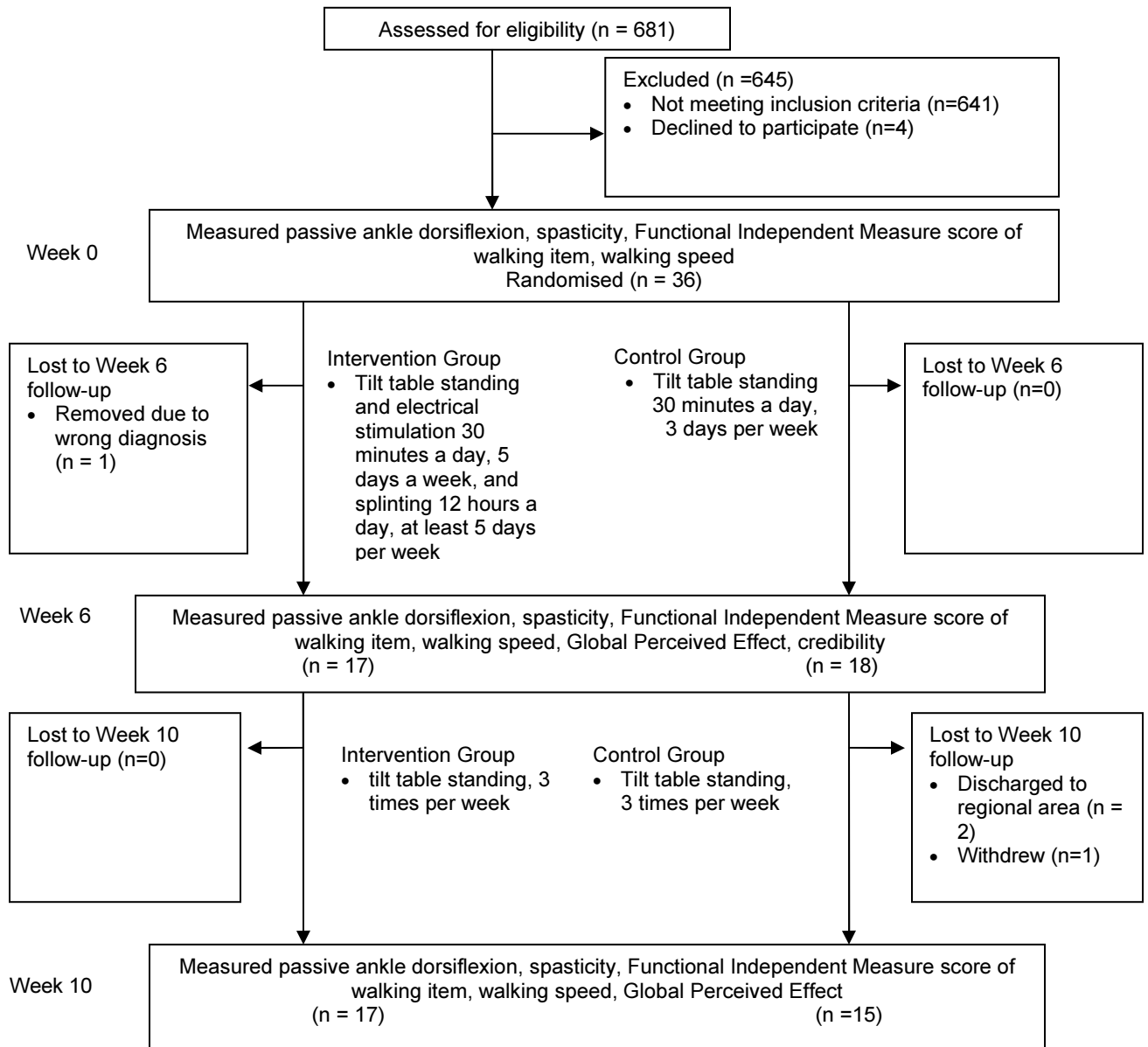
35. Ng SS, Hui-Chan CW. Transcutaneous electrical nerve stimulation combined with task-related training improves lower limb functions in subjects with chronic stroke. *Stroke* 2007;38:2953-9.
36. Shindo K, Fujiwara T, Hara J, et al. Effectiveness of hybrid assistive neuromuscular dynamic stimulation therapy in patients with subacute stroke: a randomized controlled pilot trial. *Neurorehabil Neural Repair* 2011;25:830-7.
37. Stein RB, Everaert DG, Roy FD, Chong S, Soleimani M. Facilitation of corticospinal connections in able-bodied people and people with central nervous system disorders using eight interventions. *J Clin Neurophysiol* 2013;30:66-78.
38. de Jong LD, Dijkstra PU, Gerritsen J, Geurts AC, Postema K. Combined arm stretch positioning and neuromuscular electrical stimulation during rehabilitation does not improve range of motion, shoulder pain or function in patients after stroke: a randomised trial. *J Physiother* 2013;59:245-54.
39. Malhotra S, Rosewilliam S, Hermens H, Roffe C, Jones P, Pandyan AD. A randomized controlled trial of surface neuromuscular electrical stimulation applied early after acute stroke: effects on wrist pain, spasticity and contractures. *Clin Rehabil* 2012.
40. Harvey L, de Jong I, Goehl G, Mardwedel S. Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. *Aust J Physiother* 2006;52:251-8.

**Figure 1.** The device to measure passive ankle dorsiflexion.

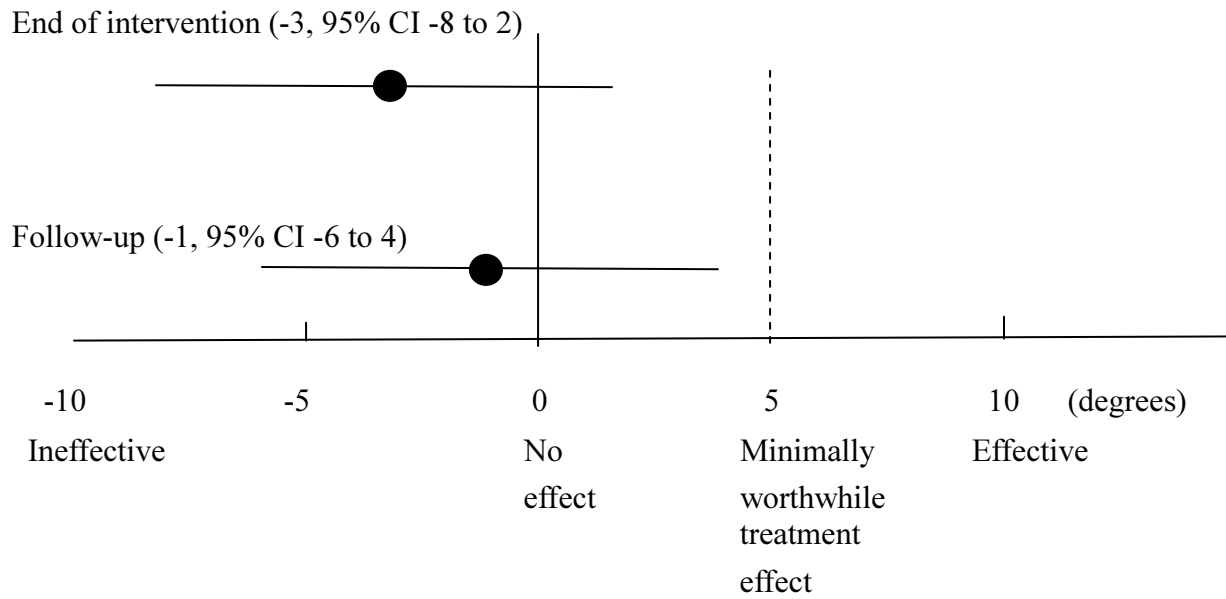




**Figure 2.** Recruitment and flow of participants through the study.



**Figure 3.** The mean between-group difference (and 95% CI) for passive ankle dorsiflexion at 12 Nm at end of intervention and follow-up.



The dots represent the mean between-group differences and the lines represent the 95% CI.

**Table 1.** Baseline characteristics of participants.

Participant characteristics	Int (n = 17)*	Con (n = 18)
Age at injury ( <i>yr</i> ), mean (SD)	38 (14)	38 (15)
Gender, n male (%)	14 (82)	15 (83)
Cause of injury, n (motor vehicle accident : fall : assault: other)	10 : 4 : 2 : 1	14 : 1 : 2 : 1
Days from injury to baseline assessment, median (IQR)	140 (96 to 226)	83 (66 to 161)
Baseline Glasgow Coma Scale score, mean (SD)	5 (3)	5 (3)
Post-traumatic amnesia duration: median (IRQ) <sup>a</sup>	180 (115 to 180)	125 (90 to 180)
Anti-spasticity medication, n (%)	8 (47)	7 (39)
FIM scale score for walking, median (IRQ)	1 (1 to 1)	1 (1 to 1)
Ankle dorsiflexor strength, n (grade 0 : 1 : 2 : 3 : 4 : 5)	7 : 0 : 6 : 0 : 4 : 0	6 : 3 : 1 : 0 : 8 : 0

Int = intervention group, Con = control group, FIM = Functional Independence Measure

<sup>a</sup> Post-traumatic amnesia duration was transcribed as 180 days for the participants with protracted (>6 months) but undetermined length of post-traumatic amnesia

\*One participate was withdrawn from the intervention group immediately following recruitment

**Table 2.** Adherence to the study protocol.

	Int (n=17)		Con (n =18)	
	protocol	actual median (IQR)	protocol	actual median (IQR)
Intervention period				
tilt table standing ( <i>min</i> )	900	890 (780 to 900)	540	540 (517 to 568)
electrical stimulation ( <i>min</i> )	900	870 (800 to 900)	n/a	n/a
splinting ( <i>hr</i> )	≥ 360	359 (197 to 436) (n =16)*	n/a	n/a
Follow-up period				
tilt table standing ( <i>min</i> )	360	330 (270 to 380)	360	360 (328 to 360) (n =15)
Timing of assessments				
6-week assessment (wks)	6	7 (6 to 8)	6	6 (6 to 7)
10-week assessment (wks)	10	10 (10 to 10)	10	10 (10 to 10)

Int = intervention group, Con = control group, n/a = not applicable

\*The data on splinting of 1 participant were missing.

**Table 3.** The use of anti-spasticity medication during the course of the study

	Baseline		Intervention period		Follow-up period	
	n (%)		n (%)		n (%)	
	Int (n=18)	Con (n=18)	Int (n=17)	Con (n=18)	Int (n=17)	Con (n=15)
On anti-spasticity medication	8 (44)	7 (39)	8 (47)	8 (44)	8 (47)	8 (53)
Increased dose	n/a	n/a	3 (18)	2 (11)	0 (0)	0 (0)
Stopped medication	n/a	n/a	1 (6)	0 (0)	0 (0)	0 (0)
Started medication	n/a	n/a	1 (6)	1 (6)	0 (0)	0 (0)
Decreased dose	n/a	n/a	0 (0)	0 (0)	0 (0)	1 (7)
Changed medication	n/a	n/a	0 (0)	1 (6)	0 (0)	1 (7)

Int = intervention group, Con = control group, n/a = not applicable

**Table 4.** Mean (SD) of groups, mean (SD) difference within groups, and mean (95% CI) difference between groups for passive ankle dorsiflexion at Week 0, 6 and 10.

Outcome	Groups						Difference within groups				Difference between groups	
	Week 0		Week 6		Week 10		Week 6 minus Week 0		Week 10 minus Week 0		Week 6 minus Week 0	Week 10 minus Week 0
	Int	Con	Int	Con	Int	Con	Int	Con	Int	Con	Int minus Con	Int minus Con
Passive ankle dorsiflexion at 12 Nm ( <i>deg</i> )	-5 (6) (n = 17)	-6 (6) (n = 18)	-5 (6) (n = 16)	-3 (9) (n = 17)	-2 (5) (n = 16)	-3 (7) (n = 15)	-1 (6)	2 (8)	2 (7)	4 (7)	-3 (-8 to 2)	-1 (-6 to 4)
Passive ankle dorsiflexion at 9 Nm ( <i>deg</i> )	-8 (6) (n = 17)	-8 (6) (n = 16)	-9 (6) (n = 16)	-8 (7) (n = 15)	-7 (5) (n = 16)	-6 (8) (n = 13)	-1 (5)	0 (5)	2 (6)	2 (7)	-1 (-5 to 3)	-1 (-6 to 4)
Passive ankle dorsiflexion at 7 Nm ( <i>deg</i> )	-11 (6) (n = 17)	-11 (6) (n = 16)	-11 (6) (n = 16)	-10 (8) (n = 15)	-10 (5) (n = 16)	-10 (7) (n = 12)	1 (5)	0 (5)	2 (5)	2 (7)	1 (-3 to 5)	0 (-5 to 5)
Passive ankle dorsiflexion at 5 Nm ( <i>deg</i> )	-15 (6) (n = 17)	-13 (5) (n = 16)	-15 (6) (n = 16)	-15 (6) (n = 14)	-13 (5) (n = 16)	-14 (8) (n = 12)	1 (6)	-1 (5)	2 (5)	1 (7)	2 (-2 to 6)	1 (-3 to 6)
Passive ankle dorsiflexion at 3 Nm ( <i>deg</i> )	-17 (7) (n = 17)	-16 (6) (n = 16)	-17 (6) (n = 16)	-17 (7) (n = 14)	-16 (6) (n = 16)	-15 (9) (n = 12)	1 (7)	-1 (6)	1 (6)	1 (9)	2 (-3 to 7)	0 (-6 to 5)

Int = intervention group, Con = control group, Shaded row = primary outcome.

Note: Passive ankle dorsiflexion data for two participants at the end of intervention (one in exp group and one in control group) were not included in the analyses because of a technical problem with data collection. The decision to exclude these data was made before analysing the results.

Note: Angle data in the first 6 columns is expressed relative to a neutral position where a negative angle denotes degrees of plantarflexion from neutral.

**Table 5.** Mean (SD) of groups and mean (95% CI) between-group difference in change for spasticity, walking speed, Functional Independence Measure scale score for walking item and Global Perceived Effect of Treatment at Week 0, 6 and 10.

Outcome	Groups						Difference between groups	
	Week 0		Week 6		Week 10		Week 6 minus Week 0	Week 10 minus Week 0
	Int (n = 17)	Con (n = 18)	Int (n = 17)	Con (n = 18)	Int (n = 17)	Con (n = 15)	Int minus Con	Int minus Con
Tardieu Scale (0 to 5)	2 (1)	1 (1)	2 (1)	3 (1)	3 (1)	2 (1)	-1 (-1.8 to -0.1)	0 (-1.2 to 0.6)
Walking speed (m/s)	0.1 (0.2)	0.1 (0.4)	0.3 (0.5)	0.4 (0.5)	0.4 (0.6)	0.4 (0.5)	-0.1 (-0.4 to 0.2)	0 (-0.4 to 0.4)
Functional Independence Measure scale score for walking item (1 to 7)	2 (1)	2 (1)	3 (2)	3 (2)	3 (3)	3 (3)	0 (-1.4 to 0.9)	0 (-1.9 to 0.9)
Participants' Global Perceived Effect of Treatment <sup>a</sup> (-6 to 6)	n/a	n/a	1 (2) (n = 15)	2 (2) (n = 16)	2 (2) (n=15)	3 (3) (n=13)	-1 (-3 to 0)	-1 (-3 to 0)
Physiotherapists' Global Perceived Effect of Treatment (-6 to 6)	n/a	n/a	1 (1)	1 (2)	1 (1) (n = 14)	1 (1)	0 (-1 to 2)	0 (-1 to 1)

Int = intervention group, Con = control group, n/a = not applicable

<sup>a</sup> 55% (11/31) and 54% (15/28) of the responses were provided by carers on behalf of the participants at the end of intervention assessment and at the follow-up assessment, respectively.

Note: A negative between-group difference reflects a treatment effect in favour of the intervention group for the Tardieu Scale.

**Table 7.** Feedback from participants on perceived treatment effectiveness and treatment credibility at Week 6. 55% (11/31) of the questionnaires were answered by carers on behalf of the participants.

Outcome	Groups		Between-group comparison <i>p</i>
	Int (n = 15)	Con (n = 16)	
Considered the treatment beneficial, n (%)			0.886
yes	9 (60)	10 (63)	
no	6 (40)	6 (37)	
did not answer	0 (0)	0 (0)	
unsure	0 (0)	0 (0)	
Rating for treatment worth, n (%)			0.563
1 highly worthwhile	4 (27)	8 (50)	
2 reasonably worthwhile	6 (40)	6 (37)	
3 not sure	2 (13)	2 (13)	
4 not too worthwhile	1 (7)	0 (0)	
5 definitely not worthwhile at all	0 (0)	0 (0)	
Did not answer	2 (13)	0(0)	
Rating for tolerance, n (%)			0.157
1 comfortable	1 (7)	6 (37)	
2 slightly uncomfortable	6 (40)	7 (44)	
3 moderately uncomfortable	3 (20)	1 (6)	
4 very uncomfortable but still tolerable	4 (26)	2 (13)	
5 intolerable	0 (0)	0 (0)	
did not answer	1 (7)	0 (0)	
Willing to continue the intervention, n (%)			0.583
yes	14 (93)	14 (87)	
no	1 (7)	2 (13)	
did not answer	0 (0)	0 (0)	

Int = intervention group, Con = control group



**Table 8.** Feedback from physiotherapists on perceived treatment effectiveness and treatment credibility at Week 6.

Outcome	Groups		Between-group comparison <i>p</i>
	Int (n = 17)	Con (n = 18)	
Rating for treatment effectiveness, n (%)			0.046
1 very effective	0 (0)	0 (0)	
2 effective	9 (53)	3 (17)	
3 unsure	8 (47)	13 (72)	
4 ineffective	0 (0)	2 (11)	
5 very ineffective	0 (0)	0 (0)	
Rating for treatment worth, n (%)			0.025
1 highly worthwhile	0 (0)	1 (6)	
2 reasonably worthwhile	12 (70)	4 (22)	
3 not sure	4 (24)	7 (39)	
4 not too worthwhile	1 (6)	6 (33)	
5 definitely not worthwhile at all	0 (0)	0 (0)	
Recommended the treatment, n (%)			0.009
yes	14 (82)	7 (39)	
no	3 (28)	11 (61)	

Int = intervention group, Con = control group

Note: n = number of responses from physiotherapists, not the number of physiotherapists.

## Chapter 6

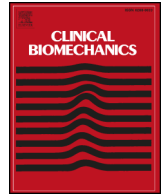
The impact of simulated ankle plantarflexion contracture on the knee joint during stance phase of gait: a within-subject study

**Statement from co-authors confirming authorship contribution of the  
PhD candidate**

As co-authors of the paper "The impact of simulated ankle plantarflexion contracture on the knee joint during stance phase of gait: a within-subject study", we confirm that Joan Leung has made contributions to the following:

- Conception and design of the research
- Collection, analysis and interpretation of data
- Writing of the manuscript and critical revision of the content

Richard Smith	<u>Richard Smith</u>	Date <u>5<sup>th</sup> May 2014</u>
Lisa Harvey	<u>Lisa Harvey</u>	Date <u>27/08/2014</u>
Anne Moseley	<u>Anne Moseley</u>	Date <u>27 Aug 2014</u>
Joe Chapparo	<u>Joe Chapparo</u>	Date <u>20-5-14</u>



# The impact of simulated ankle plantarflexion contracture on the knee joint during stance phase of gait: A within-subject study



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## abstract

**Background:** Ankle plantarflexion contractures are common in adults with neurological disorders and known to cause secondary gait deviations. However, their impact on the knee joint is not fully understood. The aims of this study are to describe the effect of simulated plantarflexion contractures on knee biomechanics during the stance phase and on the spatiotemporal characteristics of gait.

**Methods:** Mild (10-degree plantarflexion) and severe (20-degree plantarflexion) ankle contractures were simulated in thirteen able-bodied adults using an ankle-foot-orthosis. A no contracture condition was compared with two simulated contracture conditions.

**Findings:** There was an increase in knee extension, sometimes resulting in hyperextension, throughout stance for the two contracture conditions compared to the no contracture condition (mean increase in knee extension ranged from 5° to 9°; 95% CI 0° to 17°). At the same time, there were reductions in extension moment and power generation at the knee. Simulated plantarflexion contractures also reduced gait velocity, bilateral step length and cadence. All these changes were more pronounced in the severe contracture condition than mild contracture condition. While the majority of participants adopted a foot-flat pattern on landing and exhibited an increase in knee extension during stance, two participants used a toe-walking pattern and exhibited an increase in knee flexion.

**Interpretation:** Ankle plantarflexion contractures are associated with an increase in knee extension during stance phase. However, some people with simulated ankle contractures may walk with an increase in knee flexion instead. Ankle plantarflexion contractures also adversely affect gait velocity, step length and cadence.

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## 1. Introduction

Ankle plantarflexion contractures are common in individuals with neurological disabilities and known to cause major problems during gait (Kwah et al., 2012; Yarkony and Sahgal, 1987). Observational studies suggest that ankle plantarflexion contractures cause inadequate foot clearance, hitching of the pelvis and abduction of the hip during swing phase (Perry, 1974, 1992; Rose and Gamble, 1994). They also result in reduced hip extension during stance phase along with premature contralateral initial foot contact and reduced step length (Perry, 1974, 1992; Rose and Gamble, 1994). However, the impact of ankle plantarflexion contractures on knee biomechanics during stance is not clear. Clinicians need to better understand the influence of ankle

plantarflexion contractures on knee biomechanics for clinical practice. For example, without a good understanding of the possible effects of ankle plantarflexion contractures, clinicians may try to correct a gait deviation at the knee by providing knee control exercises when the underlying impairment is a plantarflexion contracture.

It is widely believed that ankle plantarflexion contractures in people with neurological conditions cause knee hyperextension during gait (Moseley et al., 1993; Perry, 1974, 1992). An observational study of 13 patients with stroke and restricted ankle dorsiflexion reported that all of them walked with increased knee extension in stance phase (Winters et al., 1987). This observation supports the link between ankle plantarflexion contractures and increased knee extension. However, the same is not observed in people with cerebral palsy and ankle plantarflexion contractures. While some of these individuals walk with knee hyperextension, others walk with excessive knee flexion (Gage, 1991; Matjacic et al., 2006; Svehlík et al., 2010; Tardieu et al., 1989). The limitation with these observational studies is that people with stroke and cerebral palsy have a range of other impairments

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such as spasticity, and loss of strength and dexterity, which make it difficult to isolate the effect of ankle plantarflexion contractures. The observed knee position may not be solely due to ankle plantarflexion contractures.

A mathematical model of bipedal motion lends support to the hypothesis that ankle plantarflexion contractures cause knee hyperextension (Higginson et al., 2006). Yet the findings of this model have been contradicted by a study of able-bodied participants which indicated an increase in knee flexion at initial and mid-stance when ankle plantarflexion contractures were simulated with tape (Goodman et al., 2004). However, taping may not be a good model for exploring the effects of contractures because taping can cause pain as the tape pulls on the skin with weight bearing. Thus it is possible that the participants in this study might have actively held their ankles in plantarflexion and walked on their toes to ease the pain. Hence, the observed changes in knee biomechanics might not be due to the simulated ankle plantarflexion contractures but an attempt by the participants to reduce the pain associated with the tape. Further work is required to verify the finding of the mathematical model and determine the effects of ankle plantarflexion contractures on knee kinematics. This information will be useful for therapists to identify the underlying cause of abnormal knee biomechanics when walking.

An ankle-foot-orthosis may be a superior method to simulate ankle contractures because, unlike taping, it is not associated with pain and can be adjusted to restrict ankle movement in a quantifiable way. Therefore, the primary objective of this study was to determine the effect of plantarflexion contractures simulated with an ankle-foot-orthosis on knee biomechanics in able-bodied people. Comparisons were made between a no contracture condition and two contracture conditions, namely a mild contracture condition (10-degree plantarflexion) and a severe contracture condition (20-degree plantarflexion). The relationships between the severity of simulated plantarflexion contracture and the extent of changes in the knee biomechanics were also examined.

The secondary objective of this study was to assess the impact of ankle plantarflexion contractures on spatiotemporal parameters. A previous study reported that simulated ankle contractures reduce gait velocity, stride length, cadence, step lengths and single support time on the affected side (Goodman et al., 2004). We wanted to take this opportunity to confirm these findings.

## 2. Methods

### 2.1. Participants

Thirteen healthy adults (five males and eight females) under the age of 50 years and without any pathology limiting strength or movement of their hips, knees and ankles, participated in the study. People older than 50 years were excluded because older people walk slower and have a more variable gait velocity between strides compared to younger people (Hollman et al., 2007). The mean (SD) age was 30 years (7), and height and weight were 165 cm (6) and 64 kg (10), respectively. Written informed consent was obtained from all the participants. The study was approved by the ethics committee of the University of Sydney.

### 2.2. Simulation of contractures using an ankle-foot-orthosis

A purpose-built ankle-foot-orthosis was used to simulate ankle plantarflexion contractures (Fig. 1). The orthosis was designed to restrict ankle dorsiflexion at two pre-determined positions, mild (10-degree plantarflexion) and severe (20-degree plantarflexion) ankle contractures, while allowing full plantarflexion. The orthosis could also be unlocked to allow full ankle joint mobility. The orthosis was worn on the right leg only.



Fig. 1. The purpose-built ankle-foot-orthosis that restricted ankle dorsiflexion while allowing full plantarflexion (dorsiflexion stops of two different sizes were inserted into the joint of the orthosis on either side to simulate the “mild” and “severe” contracture conditions).

### 2.3. Testing procedure

Four experimental conditions were conducted in the same order for all participants. The ankle-foot-orthosis was worn for all conditions except the normal walking condition. The order of testing was:

1. No contracture condition – ankle-foot-orthosis with no restriction in ankle movement.
2. Mild contracture condition – ankle-foot-orthosis with ankle dorsiflexion restricted to 10-degree plantarflexion (i.e.,  $-10^\circ$ ).
3. Severe contracture condition – ankle-foot-orthosis with ankle dorsiflexion restricted to 20-degree plantarflexion (i.e.,  $-20^\circ$ ).
4. Normal walking condition – no ankle-foot-orthosis. This condition was included to evaluate the effect of wearing the ankle-foot-orthosis.

Participants were not given instructions on how to walk. Instead they were free to walk in whatever way they felt comfortable. The same shoes were worn for all conditions. Comfort and fit of the ankle-foot-orthosis were established before each assessment. Participants were asked to practise walking down the 10-metre walkway prior to data collection until they were acclimated to the test environment. Trials were considered successful if the right foot hits the designated force plate although participants were unaware that a force plate was being used and they were blinded to its location. Five successful trials were obtained for each condition. Thus there were 20 trials from each participant giving a total of 260 walk trials. 4/260 trials were excluded. Two trials were excluded because the participants' foot missed the force plate. Two other trials were excluded because of unacceptably high ground reaction forces. These trials were replaced by one of the four remaining trials in the condition selected at random.

## 2.4. Gait analysis

Data were collected using a fourteen-camera motion analysis system (Eagle camera/Cortex 2, Motion Analysis Corporation, Santa Rosa, USA) which tracked the position of body segments over a 10-metre walkway via reflective markers. This system has high reliability for measuring three-dimensional lower limb kinematics in the sagittal plane (mean coefficient correlation: 0.96 to 0.99) (Mackey et al., 2005). Participants wore minimal clothing and had 33 reflective surface markers taped to specific anatomical landmarks (namely, forehead, cervical spine, sternum, mid thoracic, sacrum, both tragi, anterior superior iliac spines, greater trochanters, mid-thighs, medial femoral condyles, lateral femoral condyles, upper tibias, lower tibias, lateral tibias, medial malleoli, lateral malleoli, calcanei, fifth metatarsal heads and first metatarsal heads). The Skeleton Builder model was used. A main feature of this model is that it automatically scales the bone lengths to the subject's actual bonelengths. The camera system recorded the motion of each marker while the force plate recorded ground reaction forces as the participant walked. The positions of the right thigh, shank and foot during one stance phase were exported into KinTools software (Motion Analysis Corporation, Santa Rosa, USA) for kinematic analysis of knee angular displacement and angular velocity in the sagittal plane. The three-dimensional internal moments and intersegment joint forces were calculated using inverse dynamics and by modelling the leg as a collection of rigid links or segments to calculate the moment about the knee in the sagittal plane. Gender, height and weight were entered into the Mass Model Editor of the KinTools software to ensure that the mass and inertial properties of each segment were taken into account when calculating the kinetic data (de Leva, 1996). The joint power calculation was confined to the product of the components of the joint moments and joint velocity vectors perpendicular to the sagittal plane. The data were time-normalised to a percentage of the total stance time. The mean and the 95% confidence intervals were calculated across trials and across subjects. In addition, each participant's gait pattern was observed as they walked and described. The descriptions were confirmed by observing the joint movements and the location of ground reaction forces frame by frame on a computer.

## 2.5. Spatiotemporal parameters

Gait velocity, ipsilateral and contralateral step length, cadence, and percent single-support duration of the ipsilateral leg were derived from the foot-contact and toe-off events of the gait cycle using the KinTools software.

## 2.6. Statistics

Maximum ankle dorsiflexion during stance derived from the ankle kinematic data was used to confirm the success of the ankle-foot-orthosis in simulating plantarflexion contracture. In addition, the no contracture condition was compared with the normal walking condition to evaluate the effect of wearing the ankle-foot-orthosis on knee kinematics.

Comparisons were made between the no contracture condition and the simulated contracture conditions to evaluate the effect of plantarflexion contractures on knee kinematics during stance phase and spatiotemporal variables. Statistical analyses were performed for (1) peak knee flexion on loading in early stance phase, (2) peak knee extension during mid-late stance, (3) peak value of knee extension moment throughout stance phase, (4) peak values of knee flexion moment throughout stance phase, (5) energy expenditure at the knee throughout stance phase, (6) gait velocity, (7) ipsilateral and (8) contralateral step length, (9) cadence, and (10) percent ipsilateral single-support duration.

SPSS software (version 18) was used to perform all analyses. General linear models (nested repeated measures) were used for statistical comparisons. For the spatiotemporal, kinematic and joint moment

variables, data from each trial for each participant were entered into the models. Energy expenditure at the knee during the stance phase of one gait cycle was calculated as the product of mean absolute value of knee power throughout stance phase and stance duration. The mean values of the five trials were entered into the models. General linear models were also used to determine the relationships between the severity of simulated ankle contractures and extent of changes in the knee kinematics and spatiotemporal parameters. The significance level was set at  $p < 0.05$ . All data are presented as means and 95% confidence intervals unless otherwise stated.

## 3. Results

### 3.1. Simulation of contractures using an ankle-foot-orthosis

The use of ankle-foot-orthosis to simulate plantarflexion contractures was successful. The mean (SD) maximum ankle dorsiflexion during stance phase in the no contracture condition was 5 (3) degrees compared to  $-8$  (3) degrees in the mild contracture condition and  $-17$  (3) degrees in the severe contracture condition. No participant reported any pain from wearing the orthosis.

The knee kinematics of the no contracture condition (i.e., wearing the orthosis but unlocked and without restriction) and the normal walking condition (i.e., no orthosis) were very similar and there were no statistically significant between-group differences in either mean knee flexion on loading ( $-0.3^\circ$ ,  $-3.2$  to  $2.6$ ), or the peak knee extension in mid-stance ( $-1.1^\circ$ ,  $-5.4$  to  $3.1$ ). The kinematics of these two conditions are presented in Fig. 2.

### 3.2. Effect of simulated contractures on knee kinematics

There was an increase in knee extension throughout stance phase in the simulated contracture conditions compared to the no contracture condition (see Table 1 and Fig. 3). Peak knee flexion on loading was  $5^\circ$  (0 to 10) less in the mild contracture condition and  $9^\circ$  (2 to 17) less in the severe contracture condition compared to the no contracture condition. Peak knee extension during mid-late stance was  $8^\circ$  (4 to 12) more in the mild contracture condition and  $8^\circ$  (0 to 15) more in the severe contracture condition compared to the no contracture condition. These increases in knee extension in the two contracture conditions were statistically significant when compared to the no contracture condition. A linear positive relationship was found between severity of contracture and both peak knee flexion on loading and peak knee extension during mid-late stance ( $P = 0.016$  and  $0.005$ , respectively), that is,

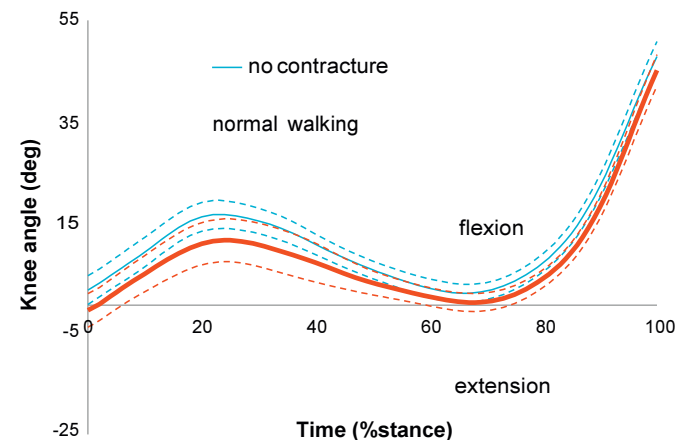


Fig. 2. Knee angular displacement curves for the no contracture condition (i.e., wearing ankle-foot-orthosis with no restriction in ankle movement) and normal walking condition (i.e., no ankle-foot-orthosis). Curves normalised to percent stance time. Thick solid lines represent mean values, dotted lines represent 95% confidence interval of mean values.



**Table 1**  
Effect of simulated ankle plantarflexion contracture on knee kinematics, knee kinetics and spatiotemporal parameters.

	Mean (SD)			Mean difference (95% CI)	
	No contracture	Mild contracture	Severe contracture	No versus mild contracture	No versus severe contracture
Peak knee flexion on loading (degrees)	12 (4)	6 (7)	2 (10)	-5 (-10 to 0) <sup>a</sup>	-9 (-17 to -2) <sup>a</sup>
Peak knee extension during mid-late stance (degrees)	-2 (3)	6 (7)	6 (9)	8 (4 to 12) <sup>a</sup>	8 (0 to 15) <sup>a</sup>
Peak knee extension moment throughout stance (Nm/kg)	0.50 (0.18)	0.28 (0.14)	0.16 (0.11)	-0.24 (-0.34 to -0.14) <sup>a</sup>	-0.36 (-0.49 to -0.23) <sup>a</sup>
Peak knee flexion moment throughout stance (Nm/kg)	0.29 (0.15)	0.58 (0.18)	0.69 (0.27)	0.26 (0.11 to 0.40) <sup>a</sup>	0.39 (0.22 to 0.57) <sup>a</sup>
Energy expenditure at the knee joint (joules)	0.17 (0.06)	0.15 (0.06)	0.16 (0.08)	0.01 (-0.06 to 0.03)	0.01 (-0.08 to 0.05)
Velocity (m/s)	1.2 (0.2)	1.0(0.2)	0.8(0.4)	-0.2 (-0.4 to -0.1) <sup>a</sup>	-0.4 (-0.7 to -0.2) <sup>a</sup>
Contralateral step length (m)	0.62 (0.13)	0.54 (0.13)	0.42 (0.24)	-0.08 (-0.15 to -0.004) <sup>a</sup>	-0.20 (-0.35 to -0.05) <sup>a</sup>
Ipsilateral step length (m)	0.65 (0.08)	0.56 (0.15)	0.55 (0.15)	-0.09 (-0.13 to -0.04) <sup>a</sup>	-0.10 (-0.14 to -0.06) <sup>a</sup>
Cadence (step/s)	1.03 (0.28)	0.99 (0.47)	0.85 (0.24)	-0.05 (-0.21 to 0.12)	-0.19 (-0.28 to -0.10) <sup>a</sup>
Percent ipsilateral single support duration (%)	40.6 (10.3)	41.1 (9.9)	39.1 (10.50)	0.5 (-3.2 to 4.1)	-1.5 (-6.5 to 3.4)

<sup>a</sup> Significant difference comparing the no contracture condition with the contracture conditions (P < 0.05).

increased severity of contracture was associated with increases in knee extension during stance.

### 3.3. Effect of simulated contractures on knee kinetics

There was a reduction in the knee extension moment about the knee throughout stance phase when the contracture conditions were compared to the no contracture condition (see Table 1 and Fig. 3). There was also a reduction in knee peak power generation but an increase in knee power absorption in mid-stance, while a reduction in knee power absorption was observed at early and late stance (Fig. 3). Simulated contractures did not change the energy expenditure at the knee (Table 1).

### 3.4. Description of gait patterns

Twelve of the participants in the mild contracture condition and 11 of the participants in the severe contracture condition were observed to place their right foot flat on the ground during early and mid-stance. These observations were confirmed by checking the joint movements and the location of the ground reaction forces at foot contact on a computer. All these participants bore weight on the rear part of their feet and showed increased knee extension.

One participant, however, adopted a different walking pattern to others and walked on his toes in both contracture conditions, and a different participant was observed to walk on his toes in the severe contracture condition. The tracking process confirmed that the ground reaction forces were under their fore-feet at foot contact. This toe-walking pattern was accompanied by a distinctly different knee angle displacement pattern and was characterised by an increase in knee flexion throughout stance. In the mild contracture condition, a 12-degree increase in the peak knee flexion on loading and a 5-degree reduction in the peak knee extension were found compared to the no contracture condition. In the severe contracture condition, a 10-degree increase in the peak knee flexion on loading and a 10-degree reduction in peak knee extension were recorded. The kinematics of these two walking patterns (i.e., foot-flat pattern and toe-walking pattern) is presented in Fig. 4.

### 3.5. Effect of simulated contractures on spatiotemporal parameters

Reductions were observed in gait velocity, cadence, and bilateral step length but not percent single-support duration when the contracture conditions were compared with the no contracture condition (Table 1). Contralateral was shorter than ipsilateral step length. Significant relationships were found between the changes in these spatiotemporal variables and severity of contracture (P-values ranged from 0.000 to 0.008).

## 4. Discussion

Simulated ankle plantarflexion contractures in able-bodied people resulted in an increase in knee extension during stance. During normal walking there are two extension peaks that occur at foot contact and in the early part of mid-stance. The knee does not fully extend in either of these phases. This phenomenon is described as 'stance phase knee flexion' (Whittle, 2007). This normal stance phase flexion of the knee was lost and replaced by full extension or even hyperextension during the two contracture conditions. This increase in knee extension or hyperextension occurs within the limit of the normal laxity of the soft tissue structures around the knee. This finding is consistent with Perry's original suggestion in 1974 that restricted ankle dorsiflexion reduces the forward rotation of the lower leg over the foot during stance phase, with a resultant increase in knee extension and sometimes hyperextension (Perry, 1974). This finding is also consistent with a mathematical model linking ankle plantarflexion contractures with increased knee extension (Higginson et al., 2006). Ankle plantarflexion contractures are common after acquired brain injury (Kwah et al., 2012; Yarkony and Sahgal, 1987), thus it may be one reason for the high incidence of knee hyperextension in people with neurological disabilities (Cooper et al., 2012; Knutsson and Richards, 1979).

Simulated ankle contractures resulted in a marked reduction in the knee extension moment during early-mid stance. This indicates that as the knee goes into hyperextension, the net concentric knee extensor muscle activity is reduced. This suggests that the knee hyperextension is passive and is brought upon largely by forces that are not generated by the knee joint. These external forces can be generated by the increased plantarflexion moment associated with ankle plantarflexion contractures (Lamontagne et al., 2000) and the large extension moment as the centre of body mass moves anterior to the knee joint (Moseley et al., 1993). The simultaneous increases in knee flexion moment and knee power absorption in mid-stance suggest increased activity of the knee flexors as they work eccentrically to slow down the knee as it moves into hyperextension. Thus while the concentric knee extensor activity reduces, the eccentric knee flexor activity increases. It is therefore not surprising that there is no difference in the overall energy expenditure in the knee joint when the simulated contracture conditions were compared to the no contracture condition. Unlike able-bodied people, eccentric control of knee flexors is often impaired in people with neurological disorders and may cause the knee to hyperextend abruptly.

The majority of participants exhibited a foot-flat pattern and showed reduced peak knee flexion on loading and increased knee extension in mid-late stance with simulated contractures. However, two participants adopted a different walking strategy and used a toe-walking pattern with increased knee flexion. Both patterns have their own shortcomings. A foot-flat pattern is associated with knee hyperextension which over time may lead to genu recurvatum. A toe-walking pattern, on the

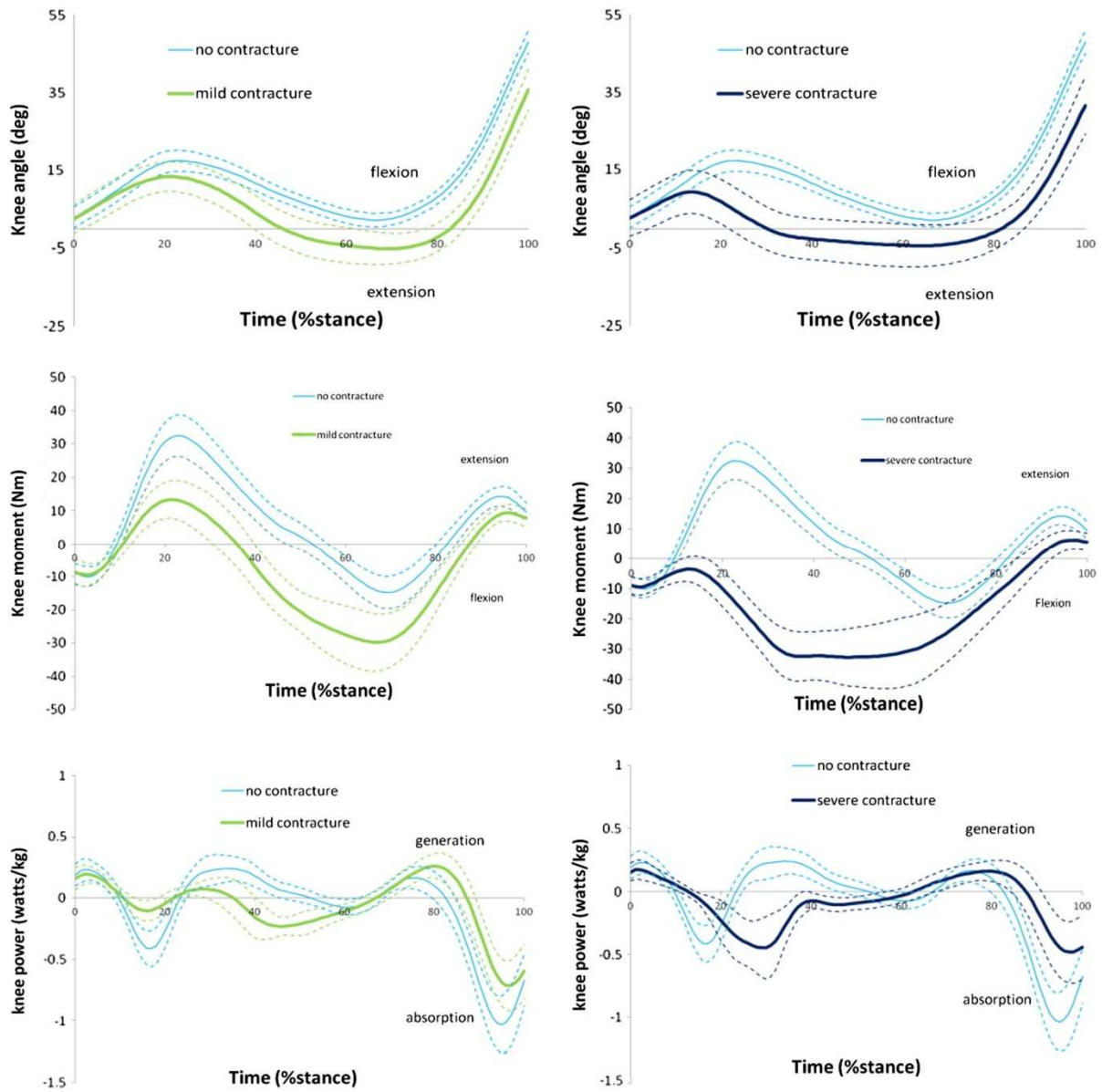


Fig. 3. Knee angular displacement curves (deg), moments (Nm/kg) and powers (watts/kg) for the no contracture and the contracture conditions. Curves normalised to percent stance time. Left column: no contracture versus mild contracture; Right column: no contracture versus severe contracture. Solid lines represent mean values, and dotted lines represent 95% confidence interval of mean values.

other hand, may encourage further loss in ankle dorsiflexion range and further contractures.

In this study, healthy participants were recruited to avoid confounding by other neurological impairments such as spasticity and loss of strength and dexterity. We postulate that the same two gait patterns (i.e., foot-flat and toe-walking patterns) can be expected in people with a neurological condition. However, the determinants that drive a person to use one pattern instead of another are yet to be determined. It is likely that the gait pattern used is contingent upon a person's ability (or inability) to meet the kinetic requirements of the two gait patterns. Comparison of the kinetics of these two patterns will provide useful information to better understand the determinants of the gait patterns observed in people with pathology and an ankle plantarflexion contracture.

Simulated ankle contractures are associated with a decrease in walking speed, bilateral step length and cadence. This finding is similar to those of Goodman et al. (2004). The reductions were more

pronounced in the severe ankle contraction condition than the mild ankle contraction condition. In addition, a greater reduction was found in the contralateral step length than the ipsilateral step length. This suggests that ankle plantarflexion contractures impede the forward movement of the contralateral leg. This may explain the reduced walking speed and cadence. All these observations suggest that ankle plantarflexion contractures compromise walking ability. We, however, cannot rule out the possibility that participants' walking may improve with some more practice.

Our study was conducted on healthy participants. Thus care has to be exercised when extrapolating these results to clinical populations as people with a neurological condition generally have less ability than able-bodied people to adapt to impairments, including contractures. The impact of ankle plantarflexion contractures on gait and spatiotemporal parameters may be more profound in people with neurological conditions than that observed in this study.



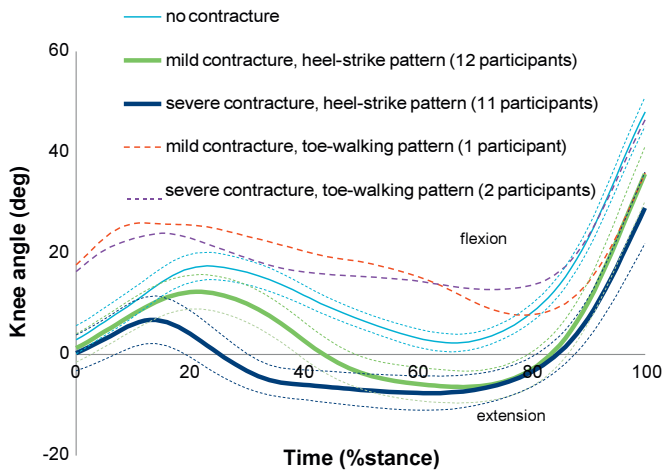


Fig. 4. Knee angular displacement curves of the two distinct gait patterns (i.e., heel-strike pattern and toe-walking pattern). Curves normalised to percent stance time. Thick solid lines represent the heel-strike pattern in the contracture conditions, dotted lines represent the toe-walking pattern in the contracture conditions, and thin solid line represents the no contracture condition.

In our study, the ankle-foot-orthosis effectively restricted ankle dorsiflexion range to approximately  $-10^\circ$  and  $-20^\circ$  for the two contracture conditions while allowing free ankle plantarflexion. Our kinematic data shows that the ankle-foot-orthosis provides a good way to simulate plantarflexion contractures in a quantifiable way. No participants reported any pain and more importantly, wearing the ankle-foot-orthosis without a simulated contracture (no contracture condition) did not produce any change in the knee angular displacement when compared to not wearing it (normal walking condition). However, an ankle-foot-orthosis only restricts range of movement at the ankle. It effectively simulates contractures resulting from a loss of length in the mono-articular structures spanning the ankle joint but not the bi-articular structures spanning both the ankle and knee joints. Although it is possible that shortening of bi-articular structures such as gastrocnemius has a similar impact on knee biomechanics, further investigations are needed. Another limitation of this study is that the findings were derived from kinematic and kinetic data. Further studies involving electromyography will be required to verify our interpretations.

## 5. Conclusion

Simulated plantarflexion contractures are associated with an increase in knee extension (or knee hyperextension) during stance. This kinematic change is accompanied by a reduction in knee extension moment and power generation. Although the majority of participants adopted a foot-flat and knee hyperextension pattern of walking, two participants adopted a toe-first and knee flexion pattern of walking. Simulated plantarflexion contractures also reduce gait velocity, bilateral step length and cadence.

## Ethics approval

The study was approved by the Ethics Committee of the University of Sydney. Written consent was obtained from all the participants before data collection began.

## Competing interests

Nil.

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## References

- Cooper, A., Alghamdi, G.A., Alghamdi, M.A., Altowajri, A., Richardson, S., 2012. The relationship of lower limb muscle strength and knee joint hyperextension during the stance phase of gait in hemiparetic stroke patients. *Physiother. Res.* 17, 150–156.
- de Leva, P., 1996. Adjustments to Zatsiorsky-Seluyanov's segment inertia parameters. *J. Biomech.* 29, 1223–1230.
- Gage, J.R., 1991. *Gait Analysis in Cerebral Palsy*. Mac Keith Press, London.
- Goodman, M.J., Menown, J.L., West Jr., J.M., Barr, K.M., Vander Linden, D.W., McMulkin, M.L., 2004. Secondary gait compensations in individuals without neuromuscular involvement following a unilateral imposed equinus constraint. *Gait Posture* 20, 238–244.
- Higginson, J.S., Zajac, F.E., Neptune, R.R., Kautz, S.A., Burgar, C.G., Delp, S.L., 2006. Effect of equinus foot placement and intrinsic muscle response on knee extension during stance. *Gait Posture* 23, 32–36.
- Hollman, J.H., Kovash, F.M., Kubik, J.J., Linbo, R.A., 2007. Age-related differences in spatio-temporal markers of gait stability during dual task walking. *Gait Posture* 26, 113–119.
- Knutsson, E., Richards, C., 1979. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 102, 405–430.
- Kwah, L.K., Harvey, L.A., Diong, J.H., Herbert, R.D., 2012. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J. Physiother.* 58, 41–47.
- Lamontagne, A., Malouin, F., Richards, C.L., 2000. Contribution of passive stiffness to ankle plantarflexor moment during gait after stroke. *Arch. Phys. Med. Rehabil.* 81, 351–358.
- Mackey, A.H., Walt, S.E., Lobb, G.A., Stott, N.S., 2005. Reliability of upper and lower limb three-dimensional kinematics in children with hemiplegia. *Gait Posture* 22, 1–9.
- Matjacic, Z., Olensek, A., Bajd, T., 2006. Biomechanical characterization and clinical implications of artificially induced toe-walking: differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. *J. Biomech.* 39, 255–266.
- Moseley, A., Wales, A., Herbert, R., Schurr, K., Moore, S., 1993. Observation and analysis of hemiplegic gait: stance phase. *Aust. J. Physiother.* 39, 259–267.
- Perry, J., 1974. Kinesiology of lower extremity bracing. *Clin. Orthop. Relat. Res.* 18–31.
- Perry, J., 1992. *Gait Analysis: Normal and Pathological Function*. Slack Inc., Thorofare, NJ.
- Rose, J., Gamble, J.G., 1994. *Human Walking*. Williams and Wilkins, Baltimore.
- Svehlik, M., Zwick, E.B., Steinwender, G., Saraph, V., Linhart, W.E., 2010. Genu recurvatum in cerebral palsy – part A: influence of dynamic and fixed equinus deformity on the timing of knee recurvatum in children with cerebral palsy. *J. Pediatr. Orthop. B* 19, 366–372.
- Tardieu, C., Lespargot, A., Tabary, C., Bret, M.D., 1989. Toe-walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. *Phys. Ther.* 69, 656–662.
- Whittle, M.W., 2007. *Gait Analysis: An Introduction*. Butterworth-Heinemann: Elsevier.
- Winters Jr., T.F., Gage, J.R., Hicks, R., 1987. Gait patterns in spastic hemiplegia in children and young adults. *J. Bone Joint Surg. Am.* 69, 437–441.
- Yarkony, G.M., Sahgal, V., 1987. Contractures. A major complication of craniocerebral trauma. *Clin. Orthop. Relat. Res.* 93–96.

## **Chapter 7**

### **Discussion**

#### **7.1 Physiotherapy for contractures**

##### **7.1.1 Overview**

One aim of this thesis was to advance knowledge on contracture management after acquired brain injury. Passive stretch has been the most commonly used physical intervention for prevention and correction of contractures. However, there is controversy about its use. A systematic review concludes that passive stretch, as typically applied, does not provide clinically important effects for people with neurological conditions.<sup>1</sup> It is probable that the studies to date had not used a sufficient dose of passive stretch, and passive stretch had not been combined with treatments that address the contributors to contractures. The theoretical framework underlying the combined treatment approach is that contractures are a complex problem involving multiple contributors. Passive stretch aims solely to reverse soft tissue shortening but does not address any of the contributors. The continual presence of these contributors is probably the reason why passive stretch alone fails to produce a worthwhile or lasting effect. I therefore hypothesised that combining passive stretch with treatments that address the contributors of contracture would be more effective than passive stretch alone.

##### **7.1.2 Principal findings**

The case report (Chapter 3) illustrated a long-term resolution of severe contractures following an intensive program of combining passive stretch with motor training. It is the

first documented case that involves administration of passive stretch over an extended period of time (10 months). The treatment program had certain key features that might have contributed to its outcome. Firstly, a high dose of passive stretch was used (24 hours a day). Secondly, passive stretch was administered in conjunction with treatment (in this case, motor training) to address muscle weakness; a factor believed to contribute to contractures. Thirdly, passive stretch was continued until this factor had been adequately addressed. This program is very different to that of previous clinical trials on passive stretch; none of which applied passive stretch more than 6 months or combined passive stretch with treatments to address contributors of contractures. The features of this program may hold the key to contracture management but robust evidence is needed to support such an approach.

The first randomised controlled trial (Chapter 4) investigated the effectiveness of electrical stimulation on wrist range of motion as an adjunct treatment for contracture management in people with acquired brain injury and poor hand control. It found a mean between-group difference of 7 degrees which exceeded the predetermined minimally important effect of 5 degrees. However, the estimate was associated with uncertainty (95% CI: -2 to 15 degrees). No treatment effect was found on muscle strength, spasticity and motor function.

The second randomised controlled trial (Chapter 5) compared a multimodal treatment program with a single modality treatment program for the management of ankle contractures in people with traumatic brain injury who had severe motor and cognitive impairments. The trial found that tilt table standing combined with electrical stimulation and ankle splint was not more effective than tilt table standing alone on joint range of motion (-3 degrees, 95% CI -8 to 2), motor function (0, 95% CI -1.4 to 0.9) and walking speed

(-0.1, 95% CI -0.4 to 0.2). A small reduction in spasticity was observed immediately following the interventions (-1, 95% CI -1.8 to -0.1), but this effect dissipated in 4 weeks.

### **7.1.3 Implications and suggestions for further investigations**

Passive stretch has a sound physiological rationale and has been the mainstream treatment for contractures. The finding of a systematic review<sup>1</sup> contradicts the long-held belief that passive stretch is an effective treatment for contracture management. Systematic reviews provide the highest level of evidence. This finding imposes a major challenge to a common therapy practice. Should clinicians abandon passive stretch based on the findings of the systematic review? The outcome of the case report (Chapter 3) suggests that clinicians should consider using large doses of passive stretch, combining passive stretch with treatments that address the contributors to contractures and continuing with passive stretch until the contributors have been adequately addressed. However, evidence based on case studies is inherently weak. Further research is needed to support this treatment approach.

The two randomised controlled trials both compared a program combining passive stretch and electrical stimulation with a program of passive stretch alone. They both failed to demonstrate that the program of passive stretch and electrical stimulation was superior to passive stretch alone. One probable explanation is that the dose of electrical stimulation may have been insufficient. In these two clinical trials, electrical stimulation was used 30-60 minutes a day over 4 to 6 weeks. Larger doses may be required to achieve a therapeutic effect but these were not considered for the trials because of the concern that participants may not be able to tolerate higher intensity or longer duration of stimulation. Another probable explanation is that electrical stimulation is ineffective in people with severe motor

and cognitive impairments. A recent study showed that people with some active control respond more favourably to electrical stimulation than those without.<sup>2</sup> There is also increasing evidence that supports combining electrical stimulation with voluntary muscle contractions for strength and functional training.<sup>3-11</sup> In the two clinical trials reported in this thesis, the vast majority of the participants did not have the motor or cognitive ability to contract muscles voluntarily in time with electrical stimulation. The potential effects of electrical stimulation could have been undermined if participants could not work voluntarily with it. It seems logical to suggest future studies on electrical stimulation restrict the inclusion criterion to people with some active motor control. Further studies may also consider using electromyography-triggered electrical stimulation to encourage voluntary muscle contractions. However, people with contractures often have poor motor function and limited ability to participate in active treatment.<sup>12-14</sup> Therefore, both these suggestions for further studies may be of limited practical value. In this thesis, both randomised controlled trials compared a combined treatment program with a single modality program. Neither trial included a control arm that received no intervention. Therefore we can only make conclusions about the relative effectiveness of the two treatment programs. The effectiveness of these programs compared to no intervention remains unclear. It may be useful for future studies to compare a multi-modal intervention group with a control group that does not receive any intervention other than usual care.

Our findings coincide with that of two recent comparable studies on people with stroke.<sup>15,16</sup> One investigated the use of electrical stimulation for the wrist joint.<sup>15</sup> Another investigated the effectiveness of a program combining electrical stimulation with passive stretch on shoulder range.<sup>16</sup> Comparison was made with no or sham stimulation. Their findings were both inconclusive (wrist: 4 degrees, 95% CI -7 to 15; shoulder external rotation 13 degrees,

95% CI 1 to 24; abduction 0 degree, 95% CI -17 to 18; flexion 9 degrees, 95% CI -17 to 35). Similar to the clinical trials in this thesis, these studies were conducted on people with poor motor control. The congruency of the results adds credibility to our finding that electrical stimulation may not be effective for people with severe brain injury. However, this finding may not hold for other clinical populations or people with less impairment. In addition to electrical stimulation, my second clinical trial also used splinting 12 hours a day. The failure of splinting to demonstrate a treatment effect in this trial is consistent with other studies on splinting.<sup>17-20</sup> Clinicians may have to re-think the use of electrical stimulation and splinting for people with severe brain injury for contracture management.

In all, much research is still required in the area of contracture management. At present, there is strong evidence that passive stretch alone is ineffective. This thesis proposes combining passive stretch with treatments that address contributors to contractures. While this concept may not be new, the case report and the two randomised controlled trials in this thesis are amongst the very few studies that have examined such an approach. The findings of the two randomised controlled trials, along with two other recently published studies,<sup>16,21</sup> do not provide support for using electrical stimulation for contracture management in people with severe motor or cognitive deficits. Nonetheless, researchers should not be discouraged from conducting further investigations on combining passive stretch with treatments that address the contributors to contractures. This thesis only focused on physical interventions. Clinicians and researchers can consider other treatment options such as combining passive stretch with medical interventions like anti-spasticity medications and Botulinum Toxin injections. Further effort is needed to identify interventions that can effectively tackle the contributors to contractures and are applicable

to people with contractures. Future research into various combinations of treatment may provide a much needed solution for contracture management.

## **7.2 The impact of ankle contractures on gait**

### **7.2.1 Overview**

The second part of my thesis examined the impact of ankle contractures on the biomechanics of the knee during gait. Ankle contractures are known to cause secondary gait deviations. However, their impact on the knee joint during gait is not fully understood. Ankle contractures and knee hyperextension gait are both common problems after stroke.<sup>22-</sup><sup>24</sup> While they often co-exist, the relationship between the two is unclear as research findings on this have been conflicting.<sup>25,26</sup> An observational study was therefore conducted to determine the impact of ankle contractures on knee biomechanics.

### **7.2.2 Principal findings**

The study (Chapter 6) confirms that ankle contractures are associated with an increase in knee extension in stance (5 degrees to 9 degrees, 95% CI 0 to 17 degrees). This finding supports Perry's original statement that restricted ankle dorsiflexion reduces the forward rotation of the lower leg segment over the foot during stance phase leading to increased knee extension (or hyperextension).<sup>27</sup> It also concurs with a mathematical model<sup>25</sup> that links ankle contractures with increased knee extension. The study found reductions in extension moments and power generations secondary to ankle contractures. These findings suggest that the movement of the knee into extension is largely passive but controlled by increased eccentric flexor activity. Despite the changes in knee kinematics and kinetics, simulated

ankle contractures did not change the overall energy expenditure at the knee joint during stance phase. Different to other previous studies,<sup>25,26</sup> the study reveals more than one gait pattern secondary to ankle contractures. While the majority of participants used a foot-flat pattern and exhibited an increase in knee extension, a few of them adopted a toe-walking pattern and showed an increase in knee flexion. This finding is in congruence with the mixed walking patterns observed in people with cerebral palsy and ankle contractures.<sup>28-31</sup> The study also confirms the finding of a previous study<sup>26</sup> that ankle contractures reduce walking speed, cadence and bilateral step length. All the changes in kinematics, kinetics and spatiotemporal parameters were more profound in the severe contracture condition than mild contracture condition.

### **7.2.3 Implications and suggestions for further investigations**

The observational study (Chapter 6) establishes the association between ankle contractures and an increase in knee extension (or knee hyperextension) during stance. Knee hyperextension gait is a common gait disorder with 40-60% of people with stroke walking with such gait pattern.<sup>24</sup> The association implies that ankle contractures can play a role in this gait disorder.

Two distinctive walking patterns secondary to ankle contractures were observed in the study: foot-flat pattern and toe-walking pattern. It is likely that ankle contractures would result in the same two walking patterns in clinical populations. However, the determinants for adopting one pattern and not the other are unclear and warrants further investigation. Both patterns can lead to undesirable long-term outcomes. A foot-flat pattern, with its association with knee hyperextension, can lead to genu recurvatum over time. A toe-walking pattern, on the other hand, may encourage further loss in ankle dorsiflexion range



and thus accentuate contractures. Further studies are needed to identify effective treatment to correct these gait patterns or mitigate their negative impacts.

The study was conducted on healthy participants to avoid confounding by other impairments. Caution has to be exercised when extrapolating the results to clinical populations. Firstly, people with a neurological condition are less able to adapt to impairments, like contractures, than able-bodied people. Thus, the effects of ankle contractures on both gait and spatiotemporal parameters are likely to be more profound in these people than observed in the study. Secondly, the healthy participants of the study showed increased knee flexion moment and knee power absorption in the simulated contracture conditions. The increased eccentric knee flexor activity probably slows the knee down as it moves into hyperextension. However, people with neurological conditions and impaired knee control may not have the ability to increase eccentric knee flexor activity. In such cases, an abrupt knee hyperextension can be expected.

The study shows that an ankle-foot-orthosis provides an effective and quantifiable way to simulate fixed ankle contractures. However, it is not without limitations. While it effectively simulates contractures resulting from shortening of a mono-articular structure spanning over the ankle (such as soleus), it does not mimic contractures resulting from shortening of biarticular structures spanning the knee and ankle (such as gastrocnemius). Its rigid properties also limit its ability to mimic the dynamic nature of ankle restriction imposed by spasticity. While the impact of shortening of gastrocnemius or spasticity of ankle plantarflexors on knee biomechanics may be similar to that observed in the study, further investigations are needed to confirm this. In addition, the findings of the study are based on

kinematic and kinetic data. Verification of the interpretations using electromyography will be useful.

### **7.3 Summary**

The overall aim of this thesis was to advance knowledge on contractures. The first part of the thesis suggests that it is premature to abandon passive stretch. Instead, it proposes combining passive stretch with treatments that address the contributors to contractures. The case report was firstly presented to support this proposition. It described an adolescent whose severe knee contractures did not respond to low dose of passive stretch but resolved after the dose of passive stretch was increased, and the effect was maintained by an intensive program combining stretch with motor training. This case report provides a preliminary justification for an intensive program of passive stretch administered in combination with motor training for the correction of severe contractures. The first randomised controlled study examined the effectiveness of electrical stimulation as an adjunct treatment for contracture management. The finding of this study was inconclusive. The second randomised controlled study compared a program combining tilt table standing, electrical stimulation and ankle splinting with a program of tilt table standing alone. It did not find one program more effective than the other. These findings indicate that electrical stimulation and splinting may not be useful for contracture management in people with severe motor and cognitive impairments. These studies contribute to the evidence base for contracture management. The data of these studies may be useful for future meta-analyses to determine the role electrical stimulation in rehabilitation. The second part of this thesis focused on the impact of ankle contractures on the knee joint. An observational study

established the association between ankle contractures and an increase in knee extension. It also identified the two gait patterns that are associated with ankle contractures. In addition, it confirmed the adverse effects of ankle contractures on spatiotemporal parameters. These findings may improve clinicians' understanding of the gait deviations caused by ankle contractures. I hope that the information generated from this thesis will provide useful guidance for future research and clinical practice in contracture management.

## 7.4 References

1. Katalinic OM, Harvey LA, Herbert RD. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: A systematic review. *Phys Ther* 2011;91:1-14.
2. Doucet BM, Griffin L. High-versus low-frequency stimulation effects on fine motor control in chronic hemiplegia: a pilot study. *Top Stroke Rehabil* 2013;20:299-307.
3. Alon G, Levitt AF, McCarthy PA. Functional electrical stimulation (FES) may modify the poor prognosis of stroke survivors with severe motor loss of the upper extremity: a preliminary study. *Am J Phys Med Rehabil* 2008;87:627-36.
4. Taylor L, Lewis GN, Taylor D. Short-term effects of electrical stimulation and voluntary activity on corticomotor excitability in healthy individuals and people with stroke. *J Clin Neurophysiol* 2012;29:237-43.
5. Bolton DA, Cauraugh JH, Hausenblas HA. Electromyogram-triggered neuromuscular stimulation and stroke motor recovery of arm/hand functions: a meta-analysis. *J Neurol Sci* 2004;223:121-7.

6. Chan MK, Tong RK, Chung KY. Bilateral upper limb training with functional electric stimulation in patients with chronic stroke. *Neurorehabil Neural Repair* 2009;23:357-65.
7. de Kroon JR, van der Lee JH, IJzerman MJ, Lankhorst GJ. Therapeutic electrical stimulation to improve motor control and functional abilities of the upper extremity after stroke: a systematic review. *Clin Rehabil* 2002;16:350-60.
8. de Kroon JR, IJzerman MJ, Chae J, Lankhorst GJ, Zilvold G. Relation between stimulation characteristics and clinical outcome in studies using electrical stimulation to improve motor control of the upper extremity in stroke. *J Rehabil Med* 2005;37:65-74.
9. Ng SS, Hui-Chan CW. Transcutaneous electrical nerve stimulation combined with task-related training improves lower limb functions in subjects with chronic stroke. *Stroke* 2007;38:2953-9.
10. Shindo K, Fujiwara T, Hara J, et al. Effectiveness of hybrid assistive neuromuscular dynamic stimulation therapy in patients with subacute stroke: a randomized controlled pilot trial. *Neurorehabil Neural Repair* 2011;25:830-7.
11. Stein RB, Everaert DG, Roy FD, Chong S, Soleimani M. Facilitation of corticospinal connections in able-bodied people and people with central nervous system disorders using eight interventions. *J Clin Neurophysiol* 2013;30:66-78.
12. Pandyan AD, Granat MH, Stott DJ. Effects of electrical stimulation on flexion contractures in the hemiplegic wrist. *Clin Rehabil* 1997;11:123-30.
13. Turton AJ, Britton E. A pilot randomized controlled trial of a daily muscle stretch regime to prevent contractures in the arm after stroke. *Clin Rehabil* 2005;19:600-12.
14. Malhotra S, Pandyan AD, Rosewilliam S, Roffe C, Hermens H. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. *Clin Rehabil* 2011;25:184-91.

15. Malhotra S, Rosewilliam S, Hermens H, Roffe C, Jones P, Pandyan AD. A randomized controlled trial of surface neuromuscular electrical stimulation applied early after acute stroke: effects on wrist pain, spasticity and contractures. *Clin Rehabil* 2013;27:579-90.
16. de Jong LD, Dijkstra PU, Gerritsen J, Geurts AC, Postema K. Combined arm stretch positioning and neuromuscular electrical stimulation during rehabilitation does not improve range of motion, shoulder pain or function in patients after stroke: a randomised trial. *J Physiother* 2013;59:245-54.
17. Harvey L, de Jong I, Goehl G, Mardwedel S. Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. *Aust J Physiother* 2006;52:251-8.
18. Lannin NA, Herbert RD. Is hand splinting effective for adults following stroke? A systematic review and methodologic critique of published research. *Clin Rehabil* 2003;17:807-16.
19. Lannin NA, Cusick A, McCluskey A, Herbert RD. Effects of splinting on wrist contracture after stroke: a randomized controlled trial. *Stroke* 2007;38:111-6.
20. Lannin NA, Horsley SA, Herbert R, McCluskey A, Cusick A. Splinting the hand in the functional position after brain impairment: a randomized, controlled trial. *Arch Phys Med Rehabil* 2003;84:297-302.
21. Malhotra S, Rosewilliam S, Hermens H, Roffe C, Jones P, Pandyan AD. A randomized controlled trial of surface neuromuscular electrical stimulation applied early after acute stroke: effects on wrist pain, spasticity and contractures. *Clin Rehabil* 2012.
22. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979;102:405-30.
23. Winters TF, Jr., Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. *J Bone Joint Surg Am* 1987;69:437-41.

24. Cooper A, Alghamdi GA, Alghamdi MA, Altowaijri A, Richardson S. The Relationship of Lower Limb Muscle Strength and Knee Joint Hyperextension during the Stance Phase of Gait in Hemiparetic Stroke Patients. *Physiother Res Int* 2011.
25. Higginson JS, Zajac FE, Neptune RR, Kautz SA, Burgar CG, Delp SL. Effect of equinus foot placement and intrinsic muscle response on knee extension during stance. *Gait Posture* 2006;23:32-6.
26. Goodman MJ, Menown JL, West JM, Jr., Barr KM, Vander Linden DW, McMulkin ML. Secondary gait compensations in individuals without neuromuscular involvement following a unilateral imposed equinus constraint. *Gait Posture* 2004;20:238-44.
27. Perry J. Kinesiology of lower extremity bracing. *Clin Orthop* 1974;102:18-31.
28. Gage JR. *Gait analysis in cerebral palsy*. London: Mac Keith Press; 1991.
29. Svehlik M, Zwick EB, Steinwender G, Saraph V, Linhart WE. Genu recurvatum in cerebral palsy--part A: influence of dynamic and fixed equinus deformity on the timing of knee recurvatum in children with cerebral palsy. *J Pediatr Orthop B* 2010;19:366-72.
30. Matjacic Z, Olensek A, Bajd T. Biomechanical characterization and clinical implications of artificially induced toe-walking: differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. *J Biomech* 2006;39:255-66.
31. Tardieu C, Lespargot A, Tabary C, Bret MD. Toe-walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. *Phys Ther* 1989;69:656-62.

## Appendices

Procedure manuals for the two randomized controlled trials

(For Chapter 4 and Chapter 5)

# Appendix 1

## **PROCEDURE MANUAL**

(version one, 01 April 2008)

**A bi-modal approach to prevent and treat wrist contracture after  
acquired brain injury:  
a randomized controlled study  
(For Chapter 4)**

**A collaborative research project conducted  
by  
Royal Rehabilitation Centre Sydney  
Liverpool Hospital  
Balmain Hospital**

**Joan Leung  
Brain Injury Unit  
Royal Rehabilitation Centre Sydney**



## **1. BRIEF DESCRIPTION OF THE PROJECT**

The aim of the study is to investigate the effectiveness of a bimodal approach in the management of contracture. A multi-centre randomised controlled study with concealed allocation and assessor blinding will be conducted. In the study, participants will be randomly allocated into receiving either a programme combining hand splinting and electrical stimulation or a programme using hand splinting alone. The effectiveness of the two programmes in maintaining the length of the extrinsic wrist and finger flexors will be compared. The participants are people with stroke or traumatic brain injury admitted to one of the rehabilitation units of Royal Rehabilitation Centre Sydney, the rehabilitation unit in Balmain Hospital, or the Brain Injury Unit at Liverpool Hospital.

## **2. METHODOLOGY**

### **2.1 Experimental design**

A multi-centre randomised controlled study with concealed allocation and assessor blinding will be conducted.

### **2.2 Eligibility criteria**

36 participants who fulfil the following eligibility criteria will be recruited in the study:

- first diagnosis of acquired brain injury
- weakness of wrist extensors (inability to extend wrist and fingers fully in a gravity eliminated position ie., strength < grade 2)
- presence of reduced flexibility, spasticity (ie., Tardieu Scale score  $\geq 1$ ) or hypertonia in the wrist and finger flexor muscles
- inpatient rehabilitation stay anticipated to be at least 4 weeks
- ability to participate in a splinting regimen and an electrical stimulation programme
- passive wrist extension to at least neutral with fingers extended (ie. ability to place the wrist in neutral with fingers extended on a flat surface)
- no medical conditions that contraindicate full participation in the hand splinting or electrical stimulation programme
- no recent wrist or finger fractures
- no fixed flexion deformities in the individual finger joints
- no previous wrist problems that limit passive wrist range of motion, and
- informed consent from the participant or the person responsible for him or her.

### **2.3 Screening procedure**

Investigators/physiotherapists of the five participating units will screen all existing patients and all new admissions. Outcomes of screening will be recorded on the *Screening Log*. When an eligible patient is identified, Investigators will notify the Principal Investigator (Mrs Joan Leung) to arrange an assessment by a blinded assessor. The blinded assessor will confirm participant's eligibility.

The use of the *Screening Log* will ensure all potentially eligible patients are considered for recruitment. Investigators of each site will submit copies of the *Screening Log* to the Principal Investigator (Mrs Joan Leung) each month so recruitment rate can be monitored.

### **2.4 Recruitment**

*Participant Information Sheet/Person Responsible Information Sheets* and *Consent Forms* will be provided to each of the participating units. The *Information Sheet* describes how the study will be conducted, the potential benefits and risks, confidentiality issues and right to withdrawal. The

*Consent Form* will be used to obtain formal consent to participate from the participant or person responsible for him or her.

Potential participants who fit the eligibility criteria or the persons responsible for them will be given the *Participant Information Sheet/Person Responsible Information Sheet*. An investigator will explain the study protocol to them. No pressure to participate will be placed on them under any circumstances. They will be informed that participation in the study is entirely voluntary and they are free to withdraw from the study at any stage without any effect on their current and future treatment. Those who agree to participate will be given the *Consent Form* to sign and participate in the study.

Health care interpreters will be used for potential participants who cannot speak English.

## **2.5 Random allocation**

Participants will be randomly allocated to one of two groups: the *splinting plus electrical stimulation* group and *splinting only* group. The allocation sequence is computer generated by a person not involved in the recruitment. Allocation will be concealed using consecutively numbered, sealed, opaque envelopes. The envelopes will be kept off-site in a centralised registry. After completion of all the initial assessments, the investigator will contact xxxxx at the central registry of the Rehabilitation Studies Unit by phone (02) xxxxxxxx or email xxxxx@med.usyd.edu.au who will confirm the participant's eligibility and number, write the name of the participant and date of allocation on the envelope, then open the envelope to reveal the group allocation for the participant.

## **3 INTERVENTIONS**

Participants will be randomly allocated to one of following two groups:

### *1) Splinting plus electrical stimulation group*

Participants in the *splinting plus electrical stimulation* group will receive a 4-week programme of using a hand splint for 12 hours per day, 5-7 days per week, and electrical stimulation to the wrist and finger extensors for 60 minutes per day, 5 days per week. The wrist will be splinted in maximum tolerable extension. The electrical stimulation will be applied to the wrist extensors with the wrist held in maximum tolerable extension using the splint.

### *2) Splinting only group*

Participants in the *splinting only* group will receive a 4-week programme of using a hand splint for 12 hours per day, 5-7 days per week. The wrist will be splinted in a maximal tolerable extension.

No other stretch or electrical stimulation treatments for the wrist and fingers will be administered during the trial.

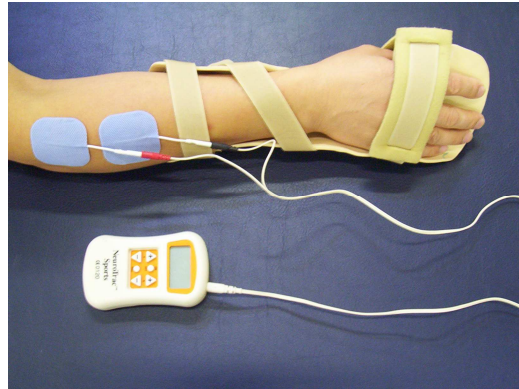
## **3.1 Intervention procedures**

The standard procedure and precautions set up by the Australian Physiotherapy Association (2001) and the participating units will be strictly followed.

### A) Electrical stimulation

- Neuro Trac Sports dual channel stimulation units will be used
- Electrical stimulation will be applied to the wrist and finger extensors with the wrist in maximum tolerable extension in a hand splint.
- Electrical stimulation will be delivered with:
  - a pulse width of 300  $\mu$ s at a frequency of 50hz
  - an on time of 15 seconds and off time of 15 seconds, and
  - a ramping-up period of 1.5 second

-Electrodes will be placed on dorsal aspect of the proximal half of the forearm. Positioning of the electrodes may require minor adjustments to achieve maximum tolerable muscle contraction with least discomfort (see photo below).



-The amplitude of electrical stimulation will be set at a level to produce maximum muscle contractions and finger extension within the tolerance of the participants. Treating physiotherapists will monitor participants' response closely, and increase the intensity when appropriate as the participant may acclimate to the stimulation after an initial period of stimulation during a session and over the intervention period.

-For participants whose tolerance could not be ascertained, lesser amplitude just enough to produce finger extension (submaximal contraction) will be applied.

-1-hour of electrical stimulation can either be applied continuously or be divided up into a few shorter sessions totaling 1-hour

-The participants will be instructed to work with the stimulator by actively extending the wrist and fingers as much as possible when the stimulation is on.

-Each application of electrical stimulation will be recorded in the participant's diary.

#### B) Wrist splinting

-Custom-made hand splints will be used for the splinting regimen in both groups.

-The wrist and fingers will be splinted in maximum tolerable extension for 12 hours a day.

-The splint is generally worn at night but application time can be varied to minimise interference with hygiene and therapy.

-The skin condition of all the participants will be closely monitored when initially wearing the splint.

-The splint will be reviewed every week and modified to maintain maximum stretch as needed.

-The time of application and removal of the splint each day should be recorded on *the weekly splinting chart* for each participant. The wearing time for each day is then calculated and recorded in the participant's *diary*.

#### 4 OUTCOME MEASURES

One primary and seven secondary outcome measures will be used to compare the effectiveness of the two treatment programmes. The primary outcome measure is the extensibility of the extrinsic wrist and finger flexor muscles using a torque of 3Nm. The secondary outcomes are extensibility of the extrinsic wrist and finger flexor muscles with a torque of 1Nm and 2Nm, strength of the wrist extensors, spasticity of the wrist flexors, motor control and global perceived effect of treatment, and credibility of treatment. With the exception of global perceived effect of treatment and credibility of treatment, the outcome measures will be taken at the beginning of the trial

(baseline). All outcomes will be assessed at the end of the 4-week treatment programme (i.e., post-intervention) and 2 weeks later (i.e., follow-up).

The outcome measures will be assessed by blinded and trained assessors. Participants will be told prior to the assessment not to disclose group allocation to the assessor. Therapists will ensure participants in the *Splinting plus electrical stimulation group* do not attend the assessment session with an electrical stimulator. The success of blinding will be recorded.

Post intervention measurements will be conducted no sooner than 24 hours but within 3 days after cessation of the last treatment session. In situations where post-intervention assessment cannot be arranged in time, the experimental interventions will continue till the assessment can be arranged.

None of the post-intervention measurements will be disclosed to the treating physiotherapists or participants until after they have completed the last questionnaires at follow-up.

## **5 ASSESSMENT PROCEDURES**

All the assessment outcomes will be recorded in the Assessment Form.

### **(1) The extensibility of the extrinsic wrist and long finger flexors**

The extensibility of the extrinsic wrist and long finger flexor muscles will be determined by measuring passive wrist extension range with the fingers held in an extended position at standardised torques. Measurements will be taken at 1Nm, 2Nm and then 3Nm. For participants who cannot tolerate 3Nm, the highest tolerable torque at baseline will be used instead, and the same torque will be used for all the subsequent assessments for these participants. A standardised procedure and torque application as described by Harvey et al (1994) will be used. This procedure has a high reliability, with an intra-class correlation coefficient of 0.85 and 80% CI within 5 degrees (Harvey et al 1994).

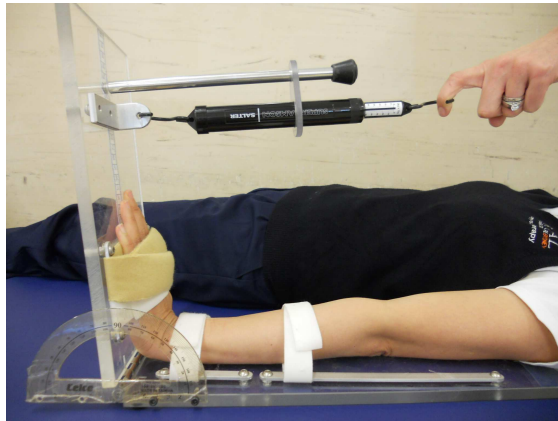
#### **SET-UP**

1. Position the participant in supine with elbow extended and shoulder in 30-45 degrees of abduction.
2. Pre-stretch the wrist with fingers by manually extending the wrist and fingers to the point where tightness is felt but the participant does not experience pain, maintain this position for 30 seconds.
3. Locate and mark the mid-carpal joint.
4. Position the arm on the device and align the mid-carpal joint with the hinge of the device.
5. Lift the movable plate of the device under the hand a few times to ensure the participant's palm is pressing firmly against the plate during passive wrist extension.
6. Secure the fingers, hand and forearm firmly on the device using the straps.
7. Record the position of the tip of the middle finger using the tape attached underneath the movable plate. The same position will be used for every subsequent and follow-up measurement.
8. Ask the participant to relax.

#### **MEASUREMENT**

1. Ensure the palm is firmly placed within the device and the fingers are fully extended during the whole procedure.
2. Apply an extension torque of 1Nm using a calibrated spring.

3. Measure the angle of the wrist using the goniometer attached to the base of the device (see photo below).
4. Repeat the measurements with an extension torque of 2Nm and then 3Nm.



## (2) Strength of wrist extensors

Strength of wrist extensors will be determined with a dynamometer. The inter-rater reliability of testing strength using a dynamometer is high, with correlation coefficients ranging from 0.84 to 0.94 ( $p < .001$ ) (Bohannon et al 1987). To enhance reliability, a specially built device will be used to stabilise the forearm and standardise the position of the dynamometer for every measurement. The device consists of a base with 4 forearm stabilisers and a holder for the dynamometer (see photo in the next page).

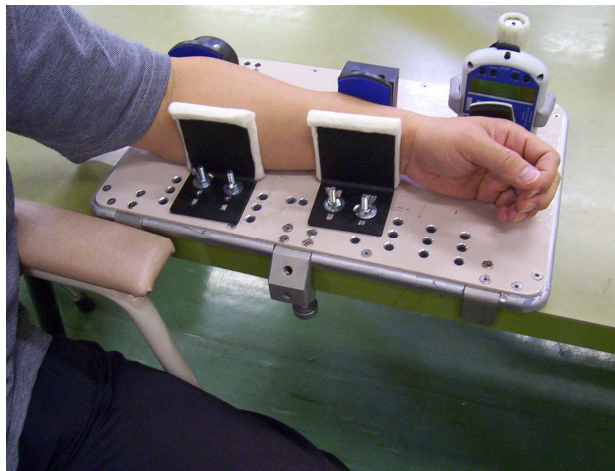
### SET-UP

1. Position the participant sitting next to a non-movable table (place the table against a wall if it has wheels) and rest the arm to be measured on it (see photo below). If the participant has less than neutral shoulder external rotation range, position him or her sitting at a right angle to the table (i.e., facing the table) instead.
2. Note the position in the *Assessment Form*. The same position will be used for all the subsequent assessments.
3. Measure and record the distance between the top part of the seat and the top of the table. The same distance will be used for all subsequent assessments.
4. Position the participant's forearm on the device.
5. Align the distal end of the 2nd metacarpal (index finger) with the distal end of transducer of the dynamometer.
6. Adjust the position of the four forearm stabilisers to secure the forearm firmly in position.
7. Secure the measuring device on the side of the table.
8. Measure and record the distance between the wrist joint and the tip of the 2<sup>nd</sup> metacarpal.

### MEASUREMENT

1. Ask the participant to move at the wrist and push the hand against the transducer of the dynamometer as hard as possible.

2. Record the maximum reading off the dynamometer.
3. Repeat the procedure 5 times with a 10-second rest between each.
4. The best measurement will be used for analysis.



### (3) Spasticity of wrist flexors

Spasticity of wrist flexors will be assessed using the Tardieu Scale (Tardieu et al 1988). There is a high percentage agreement between laboratory measures of spasticity and the high-speed component of the Tardieu Scale, indicating that the Tardieu Scale is a valid clinical tool for the measurement of spasticity (Patrick et al 2006).

#### SET-UP

1. Position the participant in sitting.
2. Stabilise the forearm of the participant with one hand while extending the participant's wrist with the other as fast as possible (faster than the rate of natural drop of the limb segment under gravity). The fingers will be supported in extension during the procedure.
3. Ask the participant to relax.

#### MEASUREMENT

Rate the reaction to passive stretch using this 5-point scale:

- 0: No resistance throughout the course of the passive movement, with no clear catch at a precise angle
- 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 clonuses (<10 seconds when maintaining pressure) occurring at a precise angle.
- 4: Infatigable clonus (>10 seconds when maintaining pressure) occurring at a precise angle.

### (4) Motor control

Motor control will be assessed using the hand movement item of the Motor Assessment Scale (Carr et al 1985). It tests the participant's ability to perform motor tasks on a scale of 0 to 6 where 0 equates to poor function and 6 represents good function. The Motor Assessment Scale has a high reliability with an average inter-rater correlation of 0.95 and an average test-retest correlation of 0.98 (Carr et al 1985).

#### SET-UP

1. Position the participant in sitting

2. Ask the participant to perform all items (listed below) without assistance (unless specified) and repeated three times, if necessary.
3. Provide demonstration or repeat instructions if necessary.
4. Provide general encouragement and verbal cues to gain maximal performance on test items but no specific feedback as to whether the movement pattern.

## MEASUREMENT

Score on the best performance from 3 attempts. Tick the items that are passed and cross those that are failed from the following list:

- Sitting, extension of wrist. (Patient sits at a table with forearm resting on the table. Therapist places cylindrical object in palm of patient's hand. Patient is asked to lift object off the table by extending the wrist. Do not allow elbow flexion).
- Sitting, radial deviation of wrist. (Therapist places forearm in mid pronation-supination, i.e., resting on ulnar side, thumb in line with forearm and wrist in extension, fingers around a cylindrical object. Patient is asked to lift hand off table. Do not allow elbow flexion or pronation).
- Sitting, elbow into side, pronation and supination. (Elbow unsupported and at a right angle. Three-quarter range is acceptable).
- Reach forward, pick up large ball of 14cm (5in) diameter with both hands and put it down. (Ball should be placed on table at a distance that requires elbow extension, wrist neutral or extended. Palms should be kept in contact with the ball)
- Pick up a polystyrene cup from table and put it on table across other side of body. (do not allow alteration in shape of cup)
- Continuous opposition of thumb and each finger more than 14 times in 10 seconds. (Each finger in turn tabs the thumb, starting with index finger. Do not allow thumb to slide from one finger to the other, or to go backwards).

Detailed description of criteria for each task and general rules for administering the MAS are attached in the appendix (Carr et al, 1985).

### (5) Global perceived effect of treatment

Global perceived effect of treatment will be collected from the participant (or the person responsible for him or her) and the treating physiotherapist at the end of the 4-week treatment programme (i.e., post-intervention) and 2 weeks later (i.e., follow-up). To keep the assessors blinded, a staff member who is not involved in the study will conduct the questionnaire for the participant (or the person responsible for him or her).

### (6) General perception of credibility of interventions

General perception of credibility of interventions will be rated by the participant (or the person responsible for him or her) and the treating physiotherapist at the end of the 4-week treatment programme (i.e., post-intervention). To keep the assessor blinded, a staff member who is not involved in the study will conduct the questionnaire for the participant (or the person responsible for him or her).

## 6. ADHERENCE TO INTERVENTION PROGRAM

The treating physiotherapist will use a diary to record adherence with the treatment programme for the participant in the *Splinting plus electrical stimulation group* or the *Splinting only group*. Any protocol violations, and reasons for these violations, will also be recorded in the diary. These diaries will be analysed to determine adherence to the treatment programmes.

## **7. CONCOMITANT THERAPY**

All the participants will receive the usual multi-disciplinary rehabilitation provided by the units including an individualised motor training programme. Efforts will be made to avoid administration of botulinum toxin for the upper limb during the study. The change in anti-spasticity medications and use of botulinum toxin injections will be recorded in the diary.

## **8. MISSING DATA**

If participants decide to withdraw from the study, a request will be made to them to have the outcomes assessed as planned. The data will be used for analysis.

If participants are discharged prior to completion of the 4 weeks of intervention, they will be encouraged to continue with the treatment programme at home. Training will be provided to the participants and/or their family (or carers) to implement the splinting regimen (for participants in either groups) and electrical stimulation (for those in the *Splinting plus electrical stimulation group*), and record in the diary. If necessary, a research assistant will visit 5 days per week to apply electrical stimulation, and family (or carers) will remove it at the end of the required time.

## **9. MINIMISATION OF PERFORMANCE BIAS**

Treating doctors will be blinded to the group allocation to minimise treatment bias. Treating physiotherapists will document in the medical notes that treatment for wrist is carried out as per study protocol without giving further specifications. Other procedures for managing contracture, such as botulinum toxin injections, will be avoided until the study is completed.

## **10. COMMON QUESTIONS**

1) Under what circumstances will patients be considered unable to participate in the treatment programmes?

Answer: Patients with problems contraindicating the application of a wrist splint or electrical stimulation are excluded. The examples are:

- rate controlled cardiac pacemaker
- broken skin
- inability to tolerate even the smallest amount of electrical stimulation
- poor cooperation

Patients who had botulinum toxin injections will be able to participate as long as the injections were done more than 3 months ago.

2) If both wrists are eligible for inclusion, which wrist will be selected for the study?

Answer: The most affected wrist will be selected.

3) What are the ways to enhance adherence to the splinting regimen?

Answer: It is a good idea to continue to use the existing strategies that have worked well in your unit. Following are some suggestions that may enhance adherence further:

- physiotherapists apply splints late in the afternoon and leave them on overnight
- nursing staff record in the splinting chart (left in the patient's room) when removing the splint in the morning
- physiotherapists check the splinting chart in the morning each day to check if the splint has been removed early and, if so, the reason for this
- physiotherapists place a splinting chart over bed-side as a reminder for nursing staff
- physiotherapists use bandage or tape to secure the splints if participants have a known tendency to remove splints
- physiotherapists change/modify splint when there are problems with fitting or discomfort



- if the participants is not consistently completing 12 hours of splinting per day, please contact Joan Leung on (02) xxxxxxxx

4) What are the ways to enhance adherence to the electrical stimulation regimen?

It is a good idea to continue to use the existing strategies that have worked well in your unit.

Following are some suggestions that may enhance adherence further:

- physiotherapists apply electrical stimulation during therapy sessions
- physiotherapists monitor the participant's response to the stimulation
- if the participant cannot tolerate the stimulation, physiotherapist finds out the reason(s) for this and take appropriate actions to address it (e.g. reduce the intensity if the participant cannot tolerate the intensity)
- if the participant is not consistently completing 1-hour stimulation, please contact Joan Leung on (02) xxxxxxxx

5) What happens if the participant is eligible for both the ankle study and wrist study?

Answer: The participant will be recruited for the ankle study first. Two weeks after completion of the 6-week intervention period (i.e., 8 weeks into the ankle study), the participant will be screened for recruitment for the wrist study. He or she can enter the wrist study if still eligible.

6) What to do if a participant request to withdraw from the study?

Answer: If a participant indicates that he or her wants to withdraw from the study, this is the recommended procedure to take:

- have a discussion with the participant/person responsible for him or her to find out the reason(s)
- identify if there is anything that can be done to resolve the issue(s)
- inform Joan Leung on (02) xxxxxxxx who will meet with the participant/person responsible for him or her if it is appropriate to do so
- if the participant still decides to withdraw, encourage him or her to have the outcomes assessed as scheduled, and inform Joan Leung on (02) xxxxxxxx to organise an exit assessment after obtaining his or her agreement
- inform the participant to fill the revocation of consent form

## 11. REFERENCES

Bohannon RW, Andrews AW (1987). Interrater reliability of hand-held dynamometry. *Physical Therapy* 67:931-3.

Carr JH, Shepherd RB, Nordholm L, Lynne D (1985). Investigation of a new Motor Assessment Scale for stroke patients. *Physical Therapy*; 65: 175-80.

EPA practice standards and notes on specific EPAs. Guidelines for the clinical use of electrophysical agents (section 2), Australian Physiotherapy Association 2001.

Harvey L, King M, Herbert R (1994). Test-retest reliability of a procedure for measuring extensibility of the extrinsic finger flexor muscles. *Journal of Hand Therapy*; 7: 51-4

Patrick E and Ada L (2006). The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clinical Rehabilitation*; 20: 73-82.

Tardieu C, Lespargot A, Tabary C, Bret MD (1988). How long must soleus muscle be stretched each day to prevent contracture? *Developmental Medicine and Child Neurology*; 30: 3-10.

# Appendix 2

## **PROCEDURE MANUAL**

(Version two, 12 May 2008)

**A multi-modal approach to prevent and treat ankle contracture after  
traumatic brain injury:  
a randomized controlled study  
(For Chapter 5)**

**A collaborative research project conducted  
by  
Brain Injury Rehabilitation Program Units  
of  
Royal Rehabilitation Centre Sydney  
Liverpool Hospital  
Westmead Hospital**

**Joan Leung  
Brain Injury Unit  
Royal Rehabilitation Centre Sydney**

## **1. BRIEF DESCRIPTION OF THE PROJECT**

The primary objective of the project is to determine the benefit of a multiple modal physical approach for the treatment and prevention of contractures in adults with traumatic brain injury. An assessor blinded, multi-centre, randomised controlled study will be conducted. In this study, the ankle will be selected as a model to assess the effectiveness of the intervention. The multiple-modal physical approach will include tilt table standing combined with ankle splinting and electrical stimulation. This will be compared to usual care, namely tilt table standing alone. The participants will be people with traumatic brain injury admitted to the brain injury rehabilitation units of the Royal Rehabilitation Centre Sydney, Liverpool Hospital and Westmead Hospital.

## **2. METHODOLOGY**

### **2.1 Experimental design**

An assessor-blind, multi-centre, randomised controlled study will be conducted.

36 participants who fulfil the following eligibility criteria will be recruited in the study:

- diagnosed with first traumatic brain injury
- unable to walk 17 metres without physical assistance or 50 metres with supervision ie., a score of <5 on the Functional Independence Measure walking item (Hamilton et al 1994).
- have passive dorsiflexion ankle range of motion <5 and >-15 degrees with the application of 12Nm of torque
- willing and able to participate in a regimen of ankle splinting and tilt table standing. The ability to tolerate electrical stimulation will not be an inclusion criterion.
- do not have unstable medical conditions or recent ankle fractures
- unlikely to be discharged in 6 weeks, and
- provide consent to participate in the study (the participants or the person responsible for them)

### **2.2 Screening procedure**

The Investigators and physiotherapists of the three brain injury units will screen all existing patients and all new admissions. Outcomes of screening will be recorded on the *Screening Log* (see Appendix). Passive dorsiflexion range of motion of patients will be measured with a goniometer and manual stretch. When an eligible participant is identified, the Investigators will notify the Principal Investigator (Mrs Joan Leung by phone 9808 9215 or email Joan.Leung@royalrehab.com.au) to arrange an assessment by a blinded assessor. The blinded assessor will confirm participant's eligibility.

Patients who are potentially eligible (eg. those who fulfil all the other criteria but have > 5 degrees of ankle dorsiflexion range of motion) will be recorded on the *Screening Log* and monitored using a *Monitoring Log*. Investigators will cease monitoring when a change in eligibility is deemed unlikely.

The investigator at each site will submit a copy of the *Screening Log* to Mrs Joan Leung (fax number 9809 9027) each month so that the recruitment rate can be monitored. The use of the *Screening Log* and *Monitoring Log* will ensure all potentially eligible patients are considered for recruitment.

### **2.3 Recruitment**

*Participant Information Sheets /Person Responsible Information Sheets* and *Consent Forms* (see Appendix) will be provided to each of the participating units. The *Participant/ Person Responsible Information Sheets* describe how the study will be conducted, the potential benefits and risks,

confidentiality issues and right to withdrawal. The *Consent form* will be used to obtain formal consent to participate from the participants or the persons responsible for them.

Potential participants or the persons responsible for them will be given the *Participant / Person Responsible Information Sheets*. An investigator will explain the study protocol to them. No pressure to participate will be placed on the potential participant under any circumstances. They will be informed that participation in the study is entirely voluntary and they are free to withdraw from the study at any stage without any effect on their current or future treatment. Those who agree to participate will be given the *Consent Forms* to sign and commence involvement in the study.

Health care interpreters will be used for potential participants who cannot speak English.

#### **2.4 Random allocation**

Participants will be randomly allocated to one of two groups: the multi-modality group (tilt table standing plus splinting and electrical stimulation) and single modality group (tilt table standing only). The allocation sequence will be computer generated by a person not involved in the recruitment. Allocation will be concealed using consecutively numbered, sealed, opaque envelopes. The envelopes will be kept off-site in a centralised registry. After completion of the initial assessment by a blinded assessor, the investigator will contact xxxxx at the central registry of the Rehabilitation Studies Unit of the Royal Rehabilitation Centre Sydney by phone (02) xxxxxxxxx or email xxxxx@med.usyd.edu.au who will then open the envelope to reveal the group allocation for the participant.

### **3. INTERVENTIONS**

Participants will be randomly allocated to one of following two groups:

#### *1) The multi-modal group*

Participants in the multi-modal group will receive a 6-week programme of 12 hours ankle splinting per day plus 30 minutes of passive standing on a tilt table using a wedge under the feet to maximise stretch and electrical stimulation while standing on a tilt table. All interventions will be applied 5 days per week.

#### *2) The Single-modality group*

Participants in the single-modality group will receive a 6-week programme of 30 minutes of passive standing on a tilt table. No wedge will be used under the feet. The intervention will be applied 3 days a week.

During the 4-week post-intervention period, participants in both groups will continue to stand on a tilt table for 30 minutes, 3 times a week, with no wedge. Splints and electrical stimulation will not be used for ankles in either group.

All participants will receive the usual multi-disciplinary rehabilitation provided by the three brain injury units including an individualised motor-training programme. No other passive stretch-based interventions will be administered to the ankle during the trial.

The standard procedure and precautions set up by the Australian Physiotherapy Association and the participating units will be strictly followed. These are described for each intervention, below.

#### *1) Tilt table standing*

- The multi-modal group:

- maximum tolerable stretch will be applied to the ankle plantarflexors

- treating physiotherapists will check to ensure that the heels are slightly off the supporting surface at the start of the stretch time
  - a wedge will be used to increase the intensity of stretch where applicable
- The single-modality group:
    - A piece of foam will be placed under the feet to minimise the amount of stretch
    - no wedge will be used
    - the ankles will not be stretched beyond a plantigrade position
  - Vital signs including blood pressure, oxygen saturation, heart rate, sweating, lip colour, facial expression and level of arousal will be monitored as per standard care to determine tolerance.
  - The inclination of the tilt table will be dictated by the participants' tolerance and increased gradually until an upright position is achieved.
  - The 30 minutes standing can be conducted in one session or with rest periods as necessary.
- 2) Electrical stimulation
- For the multi-modal group only
  - Neuro Trac Sports dual channel stimulation unit will be used
  - Electrical stimulation will be applied to the ankle dorsiflexors (namely tibialis anterior and extensor digitorum longus) while standing on a tilt table
  - Settings are:
    - a pulse width of 300  $\mu$ s at a frequency of 50hz
    - an on time of 10-15 seconds and off time of 20 seconds, and
    - a ramping-up period of 1.5 second and a ramping-down period of 1 seconds
- The electrical stimulation programme will be pre-set and installed under 'PC1'.
- The amplitude of electrical stimulation will be set at a level to produce maximum muscle contractions within participants' tolerance. Treating physiotherapists will monitor participants' response closely.
  - For participants whose tolerance cannot be ascertained, a smaller amplitude will be used which is just large enough to produce muscle contractions.
  - Electrical stimulation will not be used on participants who have contraindications (i.e., rate controlled cardiac pacemaker and broken skin) or are unable to tolerate even the smallest amount of stimulation. Inability to receive electrical stimulation will not be an exclusion criterion.
  - Electrode placement is illustrated in the pictures below



- Instructions:
- Clean the skin with alcohol prior to placing the electrodes.
  - Place the electrodes just lateral to the anterior border of the tibia in the proximal 1/2 of the lower leg.
  - Re-adjust the position the electrodes to achieve maximum tolerable muscle contraction with least discomfort.

### 3) Ankle splinting

- Commercially available splints (Formit) will be used to splint the ankles (see supplier's instruction sheet in the Appendix).
- The appropriate size will be selected for the participants
- The splints will generally be worn at night however this can be varied to minimise interference with hygiene and therapy.
- Ankles will be splinted in maximum tolerable dorsiflexion.
- The skin condition of the participants will be closely monitored.
- Different splints can be used if there are problems with the Formit and care will be taken to ensure that they provide adequate stretch.

## 4. OUTCOME MEASURES

The primary outcome will be passive dorsiflexion range at 12Nm. The secondary outcomes will be passive dorsiflexion range at 3Nm, 5Nm, 7 Nm and 9Nm, spasticity, activity limitation, global perceived effect of treatment and global perception of credibility of the interventions. All the primary and secondary measures will be taken at the beginning of the trial (ie. baseline), at the end of the 6-week programme period (ie. post-intervention) and 4 weeks later (ie. follow-up). No baseline measurements will be required for the global perceived effect of treatment, and the general perception of credibility of the interventions.

Post-intervention measurements will be conducted no sooner than 24 hours but within 3 days after cessation of the last treatment session. In situations where post-intervention assessment cannot be arranged within the timeframe, the experimental interventions will continue till the assessment can be arranged.

The outcome measures will be taken by blinded and trained assessors. In order to ensure blinding, participants will be told prior to the assessment not to disclose group allocation to the assessor. Therapists will ensure participants in the multi-modal group do not attend the assessment session with an ankle splint or electrical stimulator. The success of blinding will be recorded.

## 5. ASSESSMENT PROCEDURES

All the assessment outcomes will be recorded in the Assessment Form.

### 1) *Passive ankle dorsiflexion range*

Passive ankle dorsiflexion range will be measured by a footplate with a standardised torque as described by Harvey et al (2003). This assessment procedure has high intra-rater reliability with intraclass correlation coefficient of 0.95 (95% CI, 0.91-0.98) (Harvey et al 2003).

### SET-UP

1. Secure the ankle measurement device at one end of a plinth.
2. Lay the participant supine with the knee positioned in flexion and the ankle firmly secured in the footplate of the device using two straps. Align the ankle with the axis of rotation of the device by varying
  - i) the number of footpads

- ii) the length of the heel strap  
Then passively push the knee into extension.
- 3. Check the base of the footplate to ensure the participant's heel is pressing firmly against the footplate.
- 4. Ask the participant to relax.
- 5. Test the set-up. Apply a torque of 12Nm by hanging the specified weight from the rim of the wheel. If the foot slides off the plate, re-adjust the foot straps, vary the number of footpads or select a new length for the heel strap. Repeat the procedure until the heel stays firmly against the plate when the torque is applied.
- 6. Record the length of strap and the number of each of the footpads used in the assessment form. The same has to be used for all the measurements for the same participant throughout the study.

## MEASUREMENT

1. Ensure the heel is firmly within the device during the whole procedure.
2. Apply a constant ankle dorsiflexion torque of 12Nm
3. Maintain the stretch torque for 3 minutes before taking any measurements.
4. Reduce the torque to 3Nm.
5. Measure the angle of the footplate by placing the bottom of the inclinometer (marked with yellow tape) on the bottom of footplate (marked with yellow tape). Record the angle and the tilt of the foot-plate.
6. Measure the tibial inclination by placing the inclinometer horizontally on the lower one-half of the lower leg. Record the angle and position of the tibia.
7. Increase the torque to 5Nm. Repeat steps 5 and 6.
8. Repeat the measurements with the application of a torque of 7Nm, then 9Nm and finally 12Nm

(Please refer to the users instruction manual for the ankle measurement device for more detailed information and illustrations).

### 2) *Spasticity of ankle plantarflexors* (Held and Pierrot-Deseilligny 1969, Tardieu et al 1988)

Spasticity of ankle plantarflexors of participants will be rated using the Tardieu Scale (Tardieu et al 1988). In a supine position, the participant's leg will be positioned in slight external rotation and with knee slightly flexed. The blinded assessor will stabilize the shank of the participant with one hand while moving the participant's ankle with the other as fast as possible (faster than the rate of natural drop of the limb segment under gravity). Participants will be instructed to relax during the test.

Reaction to passive stretch will be rated on a 5-point scale:

- 0: No resistance throughout the course of the passive movement, with no clear catch at a precise angle
- 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 maintaining pressure) occurring at a precise angle.

A high percentage of agreement with laboratory measures of spasticity indicates that the Tardieu Scale, when conducted at high speed, is a valid clinical tool for the measurement of spasticity in ankle plantarflexors (Patrick and Ada 2006).

### 3) *Activity Limitations*

a) The score for the walking item of the Functional Independence Measures (FIM) will be used to quantify the activity limitations. The inter-rater reliability of the FIM motor items is from 0.84 to 0.97 when administered on people with traumatic brain injury (Donaghy 1998).

The assessor will rate the walking item using a 7-point scale:

- 7 walks a minimum of 150 feet (50metres) without assistive device safely
- 6 walks a minimum of 150 feet (50metres) but uses a brace or prosthesis on leg, special adaptive shoes, walking aids, or take more than reasonable time or there are safety considerations.
- 5 walks only short distances (a min of 50 feet or 17 metres) independently with or without device. Takes more than reasonable time, or there are safety considerations OR walks with standby supervision, cuing or coaxing to go a min of 150feet (50m)
- 4 performs 75% or more of locomotion effort to go a minimum of 150 feet (50metres)
- 3 performs 50 to 74% of locomotion effort to go a minimum of 150 feet (50metres)
- 2 performs 25% to 49% of locomotion effort to go a minimum of 50 feet (17metres)
- 1 performs less than 25% of locomotion effort to go a minimum of 50 feet (17metres) or requires 2 people to assist, or unable to walk a min of 50 feet

b) Activity limitations will also be determined by the 10-metre walk test. Time taken to walk the central 10 metres of a 14-metre walkway (i.e., there is a 2-metre distance at the beginning and end of the test to allow for acceleration and deceleration) will be measured. The assessor will ask the participants to walk as fast as possible. Walking speed will be calculated and used for analysis. The same walking aide will be provided in all assessments. This test will only be conducted on participants who can walk without physical assistance.

#### 4) *Strength of ankle dorsiflexors*

Strength of ankle dorsiflexors will be determined using manual strength testing (Kendall et al 2005). The data will not be used as an outcome measure but for group comparison at baseline.

- 0 no voluntary dorsiflexor muscle contraction
- 1 trace of voluntary dorsiflexor muscle contraction
- 2 active ankle dorsiflexion to full range in a gravity-eliminated position (side-lying position)
- 3 active ankle dorsiflexion to full range against gravity (supine with knee bent, or in sitting)
- 4 ankle dorsiflexion against moderate resistance (supine with knee bent, or in sitting)

#### 5) *Global perceived effect of treatment and credibility of interventions*

Global perceived effect of treatment and general perception of credibility of interventions will be collected from participants/person responsible for them and treating physiotherapists using questionnaires. To keep the assessors blinded, a staff member who is not involved in the study will conduct the questionnaire for participants/person responsible for them.

**All the post-intervention measurements will not be disclosed to the treating physiotherapists or participants until after they have completed the last follow-up assessment.**

## 6. ADHERENCE TO THE INTERVENTIONS

The treating physiotherapists will use a *diary* to record adherence with the programmes of experimental and control participants. Any protocol violations and reasons for violations will also be recorded in the diary. Changes in the use of anti-spasticity medications and botulinum toxin injections, if any, will also be recorded in the diary. These diaries will be analysed to determine adherence to the treatment and control programmes.

## 7. OTHER INFORMATION

Demographics and injury details will be recorded in the *Demographics Form*.



## 8. MISSING DATA

If participants decide to withdraw from the study or are discharged prior to completion, they will be asked if they are willing to have their final measurement taken. The data will be used for the intention-to-treat analysis.

## 9. MINIMISATION OF PERFORMANCE BIAS AND OTHER CONFOUNDERS

Treating doctors will be blinded to the group allocation to minimise treatment bias. Treating physiotherapists will document in the medical record that treatment for the ankle was carried out as per study protocol without providing further specifications. Efforts will be made to avoid administration of botulinum toxin for the ankles during the study.

## 10. COMMON QUESTIONS

1) Under what circumstances will patients be considered unable to participate in the splinting regime or tilt table standing?

Answer: Patients with contraindications to the application of an ankle splint or tilt table standing are excluded. Examples are:

- severe equinovarus ankle contracture
- severe hip or knee flexion contracture
- unstable medical conditions
- orthopaedic conditions which require non-weight bearing or immobilisation of ankle
- dysautonomia to a degree that compromises compliance with experimental interventions

2) Can ankles be stretched in standing using a standing frame instead of on a tilt table?

Answer: Ankles can be stretched using a standing frame provided that the correct duration (i.e., 30 minutes) and intensity (strong stretch for the multi-modal group) of stretch is administered during standing as per group allocation.

3) If both ankles are eligible for inclusion, which ankle will be selected for the study?

Answer: The most affected ankle will be selected.

4) What are the ways to enhance compliance with the splinting regimen?

Answer: It is a good idea to continue to use the existing strategies that have worked well in your unit. The following are some suggestions that may enhance compliance further:

- physiotherapist to apply splint late in the afternoon and leave it on overnight
- nursing staff to record in the splinting chart (left in the patient's room) when removing the splint in the morning
- physiotherapist to check the splinting chart in the morning each day to check for any violations and reasons for violations
- physiotherapist to place a splinting regimen chart over bed-side as a reminder for nursing staff
- physiotherapist to use bandage or tape to secure the splints if participants have a known tendency to remove splints
- physiotherapist to change/modify splint when there are problems with fitting or discomfort

5) Can we recruit patients who had previously received Botulinum Toxin injections?

Answer: Yes, we can recruit them provided that the injections were administered > 3 months ago.

## 11. REFERENCES

Donaghy S and Wass PJ (1998). Interrater reliability of the functional assessment measure in a brain injury rehabilitation program. *Archives of Physical Medicine Rehabilitation*; 97: 1231-1236.

Hamilton BB, Laughlin JA, Fiedler RC et al. Interrater reliability of the 7-level Functional Independent Measure (FIM). *Scandinavian Journal of Rehabilitation Medicine*; 26: 115-119.

Harvey L, Byak A, Ostrovskaya M, Glinsky J (2003) Reliability of a device designed to measure ankle mobility. *Spinal Cord*, 41: 559-562.

Held JP and Pierrot-Deseilligny E (1969). *Reeducation motrice des affections neurologiques*. Paris: Bailliere: 31-42.

Kendall FP, Kendal-McCreary E, Provance (2005). *Muscles: testing and function with posture and pain*, fifth edition. Lippincott, Williams and Williams.

Patrick E and Ada L (2006). The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clinical Rehabilitation*; 20: 173-182.

Tardieu C, Lespargot A, Tabary C, Bret MD (1988). How long must soleus muscle be stretched each day to prevent contracture? *Developmental Medicine and Children Neurology*; 30: 3-10.